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A Philological, Epidemiological, and Clinical Analysis of the Plague of Athens

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**A PHILOLOGICAL, EPIDEMIOLOGICAL, AND CLINICAL ANALYSIS OF THE
PLAGUE OF ATHENS**

by

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Senior Honors Project

Spring, 2013

1. Abstract

In the summer of 430 B.C. during the Peloponnesian War, a plague hit Athens a few days after the Spartans besieged the city. The plague raged continuously for two years and broke out again in 427 B.C. Most of the population was infected, and approximately 25% of the population died. Thucydides wrote *History of the Peloponnesian War*, which is the main literary source for the plague and other events in the Peloponnesian War. Although Thucydides took great pains to carefully describe the clinical features of the disease, physicians and classicists disagree on the identification of the disease. In the past hundred years, scholars have argued for over thirty-nine diseases, but no conclusive argument has been made for a particular disease. In order to narrow down the possible diseases, I used a descriptive epidemiological analysis of Thucydides' description to determine modes of transmission. A respiratory disease with a means of persistence or a vector-borne reservoir disease (insect or animal) are the two modes of transmission most consistent with the epidemiological information. Finally, using Thucydides' description of the clinical features, I concluded that *Rickettsia prowazekii* was the disease of the Athenian Plague.

2. The Plague's Historical Context

Understanding the historical context of the plague is essential to define the epidemiologic factors that enabled the plague to flourish. Between 431-404 B.C., the Peloponnesian War raged between the Delian League (Athens and various Greek poleis) and the Peloponnesian League (Sparta and their allies). The Spartans were the masters of land battle, while the Athenians controlled the sea with their exceptional navy. The Athenian leader, Pericles, took into account the superiority of the Spartan land forces and ordered a drastic population move. Thucydides

describes Pericles' radical strategy in 2.13, which ensured that the population was protected from the invading Spartans. The people of Attica relocated within the Long Walls, which stretched from Athens to the ports of Piraeus and Phalerum. The two ports provided access to food and other goods through the use of Athenian allies on the Mediterranean Sea. Thanks to the funds provide by the Delian League, the Athenians possessed plenty of capital to continue their supply line and remove the threat of starvation. With the majority of the population of Attica crowded within these walls, the Spartans freely ravaged the countryside with little resistance. Eventually, Pericles' strategic relocation would exacerbate the effects of the plague.

Even though the population of Attica was supposed to be within the Long Walls, some Athenians remained outside the walls during non-siege times. Gomme (1933) argued, although, that after the first massive Spartan siege, a large number of individuals remained in the city year-round. Unfortunately for the Spartans, they were unable to leave their homeland for long periods of time. The fear of a helot revolt kept the army close, which resulted in seasonal sieges mostly in the summertime. Pericles did not leave to countryside completely to the wills of the Spartans. Cavalry raids occurred sporadically against the Spartan forces, as commanded by Pericles, to bolster Athenian morale.¹ These events and circumstances of the first year of the war created a situation that would eventually lead to a devastating plague that killed around 25 percent of the Athenian population. To this day, the cause of the plague is undetermined, but certainly not for the lack of trying by scholars over the past hundred years.

3. The Arguments

¹ "The Peloponnesian War," n.d.

The secondary literature used for this paper began in 1839 with Littré and continued to modern day arguments about the identity of the disease. Since the topic has been discussed or written about for nearly 500 years, reading every journal or article making mention of the plague is an inefficient use of time. Only a brief review of the evolution of arguments and possible diseases will be useful here. In general, two for each suggested disease or theory; this process resulted in over thirty-six articles with over thirty-nine diseases being mentioned as the sole disease or in combination with one another. The list of possible diseases includes but is not limited to: influenza², smallpox³, measles⁴, typhus⁵, scarlet fever⁶, bubonic plague⁷, pneumonic plague⁸, ergotism⁹, Marburg-Ebola virus¹⁰, Rift Valley fever¹¹, leptospirosis¹², tularaemia¹³, typhoid fever¹⁴, toxic shock syndrome¹⁵, malaria, cholera¹⁶, dengue¹⁷, shigellosis, poliovirus, scurvy, anthrax¹⁸, diphtheria, erysipelas, Guillain-Barre syndrome¹⁹, syphilis²⁰, meningitis, yellow fever, glanders, rabies, hantaviruses, arenavirus, rickettsialpox, and alimentary toxic aleukia²¹. Any disease without a citation was broadly introduced by Durack (2000) or Morens and Littman (1992) without citations to the original source.

² Langmuir, 1985; Holladay, 1986

³ Morens & Littman, 1992; Littman & Littman, 1969; Retief, 1998; Durack, 2000

⁴ Shrewsbury, 1950; Cunha, 2004; Page, 1953

⁵ Morens & Littman, 1992; Weiss, 1992; Durack, 2000; MacArthur, 1954; Gomme, 1933

⁶ Rolleston, 1937; Shrewsbury, 1950; Page, 1953

⁷ Cantlie, 1900; Hooker, 1958; Perry, 1997; Ganem, 1968

⁸ Perry, 1997; Ganem, 1968; Hooker, 1958

⁹ Kobert, 1899; Salway & Dell, 1955

¹⁰ Scarrow, 1988; Olson et al., 1996; Holden, 1996; Dixon, 1996

¹¹ Morens & Chu, 1986

¹² Wylie & Stubbs, 1983

¹³ Wylie & Stubbs, 1983

¹⁴ Papagrigorakis et al., 2006; Grote, 1888

¹⁵ Langmuir, 1985; Holladay, 1986

¹⁶ Durack, 2000

¹⁷ Keil, 1951

¹⁸ McSherry, 1992

¹⁹ Langmuir, 1985; Holladay, 1986

²⁰ Keil, 1951

²¹ Bellemore J et al., 1994

As this lengthy list indicates, the problems of identifying the Athenian plague continue to multiply until the “answers” have become a quantitatively overwhelming. Philologists argue about how to translate Thucydides, while scientists and physicians argue about which disease best fits Thucydides’ clinical description. No articles offer a concrete argument, or if they do, another article can be found to refute the original claim. By the late 1970s and early 1980s, a general consensus gradually emerged in the literature that conclusively determining the disease was beyond our abilities due to the scant amount of information available to us and the restrictions of the sources we do have.²² The search to identify the disease by relating clinical features presented by Thucydides to modern-day disease reached a standstill. Fortunately, a new method developed in the 1990s jump-started discussions on the topic. Morens and Littman (1992) wrote an article called “Epidemiology of the Plague of Athens,” where they used an epidemiological approach to narrow down the possible diseases of the Athenian plague. Their methods restrict the possible modes of transmission to a zoonotic or vector-borne disease or a respiratory disease with unusual means of persistence. Although they made more progress than most scholars, Morens and Littman (1992) were still limited by the sources available to conclusively determine the disease.

Even after the gains made by Morens and Littman (1992), multiple scholars have suggested determining the disease is simply beyond our reach, due to philological, epidemiological, or evolutionary factors.²³ The presented methodological problems need to be addressed before further analysis of the possible diseases. If any of these hindrances cannot be removed, there will be a critical obstacle in the way of determining the disease. It is true that when only the clinical symptoms and general features are taken into account, the described

²² McNeill & William, 1976; Longrigg, 1980; Poole & Holladay, 1979

²³ Longrigg, 1980; Poole & Holladay 1979

disease does not add up to any modern-day known disease. The reasons for this may be the following: the plague was caused by multiple infectious agents, the disease of the plague is now extinct or evolved beyond recognition, or the Greek technical terms Thucydides used cannot be fully understood by contemporary medical science. I believe the third argument is the most logical on epidemiological and molecular grounds.

The possibility that the plague resulted from co-infection with multiple agents is epidemiologically highly unlikely. Since the disease was confined to the Athenians, Thucydides and others believed in a singular disease, because each eruption of the disease over five years was classified with the same features. It is highly unlikely two diseases “worked” together for five years to display the same clinical features. Furthermore, Thucydides states that individuals had naturally acquired immunity after an infection with the plague. If there was a simultaneous outbreak of multiple diseases, Thucydides would not have concluded a specific acquired immunity. Even if immunity was acquired for one disease, there would have been the possibility of a second infection of the other disease.

The extinction of the disease is doubtful based on evolutionary and genetic factors. Microorganisms evolve quickly to evade humans’ immune defense system; therefore, they usually avoid possible extinction as long as the host persists.²⁴ They need to balance their virulence effects with their ability to spread to another host, in order to increase the possibility of persistence.²⁵ In general, organisms with DNA mutate more slowly than RNA viruses.²⁶ DNA viruses such as smallpox have a much slower mutation rate than RNA viruses such as

²⁴ “Understanding Evolution,” n.d.

²⁵ Galvani, 2003

²⁶ Sanjuan R et al., 2003

arboviruses.²⁷ DNA polymerase, the enzyme that replicates DNA, has a greater ability to fix mutations, which decreases the overall mutation rate and possibilities for bacterial evolution.²⁸ These evolutions have occurred at the molecular level throughout the years, but the clinical symptoms have remained relatively the same for bacteria and DNA viruses. The epidemiological features of bacteria and viruses have stayed even more constant. On the other hand, RNA viruses mutate so rapidly that they would have undergone substantial evolutionary change. The consequences of RNA viruses' fast mutation rate will be discussed later in the paper in relation to arboviral diseases.

Many of the highly debated diseases for the plague (measles, bubonic plague, and smallpox) have been around for thousands of years with minimal evolution of their clinical manifestations. Smallpox dates back more than 3,000 years, and, even though it has undergone evolution of its clinical manifestation, the changes enhanced the survival, rather than quickening the extinction, of the disease. There is no evidence smallpox has ever undergone a large epidemiological change.²⁹ Additionally, measles and bubonic plague, both of which have been around for more than 1,000 years, appear to hardly change at all. Littman states, "There is little empirical evidence or theoretical reason to suspect extinction of any human disease except by purposeful eradication, as with smallpox."³⁰ The first argument that the plague was caused by multiple infectious agents is highly improbable epidemiologically and will not be considered further. The second argument is only pertinent to RNA viruses with exceptionally high mutation rates that may result in the clinical and/or epidemiological features of the disease being

²⁷ Babkin & Shchelkunov, 2006

²⁸ Domingo et al., 1996

²⁹ "The Epidemiology of Smallpox," n.d.

³⁰ Morens & Littman, 1992

unrecognizable. This work aims to support the third argument that Thucydides cannot be fully understood by contemporary medical science.

Multiple papers have been published discussing the proper translation and understanding of Thucydides' text. Longrigg mentions a possible argument that "Thucydides' description of the plague is a purely literary invention for historiographical purposes."³¹ Another argument by Harrison explains that Thucydides' vagueness and over-condensation of style is due to his uncertainty using medical language.³² One can find a plethora of such arguments commenting on Thucydides' text and style. Page and Parry represent the two most common positions taken on the proper way to translate Thucydides, which will be discussed in a subsequent section. Determining the proper translation of Thucydides is undeniably the most important step in determining the disease.

4. Interpreting Thucydides' Account

In Book Two chapters forty-seven to fifty-five, of his *History of the Peloponnesian War*, Thucydides records a *nosos* (disease) that ravaged Athens between 430 and 429 B.C. Thucydides' literary account is the main source for the plague of Athens and other events in the Peloponnesian War. The account began in the summer of 430 B.C. The first days of summer brought with them another invasion of the Spartans into Attica and, days later, the arrival of the plague into the city of Athens. Thucydides attempts to explain the geographical and temporal movements of the plague. The specific details of this account will be discussed later in the epidemiological analysis. Description of the clinical and distinctive features of the plague begins in chapter 49 verse 2 and continues until chapter 51 verse 5. Thucydides' writing on the plague

³¹ Longrigg, 1980

³² Harrison, 1906

of Athens presents many issues that hinder scholars from determining the disease if one does not understand his style of writing or the medical paradigm of the time. In order to circumvent these problems, scholars need to understand the methods, medical knowledge, and motives of the author.

Thucydides was an upper class Athenian citizen who lived through the war, participated in the war as a general, and contracted the plague. Each of these aspects of Thucydides' life brings into question whether any of these factors influenced his writings. Thucydides' description needs to be compared with modern records and an attempt must be made to determine the proper meaning of the text. This can only be accomplished, as Page succinctly says, "by determining how far the Greek is expressed in the technical terms of contemporary medical science."³³ Thucydides is often believed to be a relatively accurate writer and objective historian. He focused on collecting evidence and analytically determining the cause and effect of events. Additionally, Thucydides wrote for the purpose of instruction and future reference, rather than entertainment. The methods and purpose of Thucydides' work are addressed directly in *History of the Peloponnesian War*. Thucydides states,

"As to the deeds done in the war, I have not thought myself at liberty to record them on hearsay from the first informant or on arbitrary conjecture. My account rests either on personal knowledge or on the closest possible scrutiny of each statement made by others. The process of research was laborious, because conflicting accounts were given by those who had witnessed the several events, as partiality swayed or memory served them."³⁴

³³ Page, 1953, 97

³⁴ Thuc., 1.21

As he tells us himself in 2.48.3, Thucydides contracted the plague and witnessed first-hand the effects of the plague of Athens, which would have greatly informed him of the progression and clinical features of the disease. Thucydides had the personal experience and wrote with the purpose to not only understand the past, but also the things that may happen in the future. Each of these factors contributed to the validity and accuracy of his writings.

Additionally, Thucydides was most likely familiar with the Hippocratic writings of the contemporary school, since most of the literature available at the time was medically based. Cochrane makes one of the strongest cases that Thucydides' understanding of the historical method and values mirror the doctrines of the Hippocratic School.³⁵ Page also observes similarities between Thucydides' statement of purpose in recording the plague and multiple passages in the Hippocratic treatises. The parallelism of the two passages, when compared side-by-side, is particularly distinct. The beginning of the *Prognosticon* in the Hippocratic treatise states,

“The first duty of the physician is to practice forecasting. If he foreknows and foretells at the sick-bed the present, the past, and the future, and describes in detail what the sick man has omitted from his own account. He will create confidence that he understands what is the matter with his patients, who will then take heart and entrust themselves to his care.

*Moreover, the value of his treatment depends on his ability to foretell the future from the present symptoms.”*³⁶

Thucydides' statement of purpose mirrors the previous statement in structure and methods.

³⁵ Cochrane, 1929

³⁶ Hippocrates. 1.1

“Each individual, whether doctor or layman, is free to relate his personal opinion about the origin of the plague, and the causes of this unprecedented disturbance, if he can find any powerful enough to account for it. *For my part, I shall describe it just as it was, and provide evidence in the light of which the student may have some knowledge in advance, and so have the best chance of recognizing it if it should ever recur.*”³⁷

Thus both Thucydides’ *History* and the Hippocratic treatises stress the importance of providing evidence for prognosis, rather than stressing the diagnosis. Furthermore, Thucydides’ description of the plague following this passage closely resembles the outline of the *Epidemics*, as Page and Cochrane also mention. Thucydides begins with the *katastasis* (general conditions at the time of outbreak), states fact-based observations, names the crisis day for victims, and concludes with complications for those who survived crisis day.³⁸

Thucydides’ reputation as an ardent observer, precise writer, and rational thinker, combined with the significant parallels to Hippocratic writings of the time, builds a strong argument for the credibility of Thucydides. He could have put the causes of the disease above the prognosis, since the cause is of much more importance in historical writing. This was not the case; Thucydides merely supplemented his historical writing with medical terminology and ideology. The presence of approximately forty technical words within chapters forty-nine and fifty that do not appear anywhere else in the *History* also signifies some deviation from his typical historical writing in the need for special vocabulary. Thucydides clearly had access to medical theory and Hippocratic writings, which he stylistically mirrored in his account of the plague.

³⁷ Thuc., 2.48.3

³⁸ Page, 1953, 98

The extent of understanding he had of medical terminology is another question. The two main points of view on this are represented by two classicists, Page and Parry. Page, by examining how deeply the plague description is articulated in the customary terms of contemporary medicine, argues that Thucydides was familiar with and understood doctors' terminology. Page conducted an extensive philological analysis of Thucydidean terminology for parts of the human body, adjectives, and verbs. The conclusions of Page's investigations are the following: (1) Most of the nouns, verbs, and adjectives are standard terms that generally carry the same meanings of medical writing during Thucydides' generation. (2) All but six terms used by Thucydides are used by Hippocratic physicians and those six remaining terms are related to the standard terminology. (3) Thucydides' terms are rarely found anywhere else but medical and scientific treatises. (4) None of Thucydides' medical terminology conflicts with the medical understanding.³⁹ Parry responded to Page sixteen years later, arguing that the "vocabulary of the description of the plague is not entirely, is not even largely, technical" and the majority of vocabulary was used in everyday life.⁴⁰ Generally, Parry believed Thucydides' understanding of medical terms or the methods of physicians was overstated by Page. However, the textual evidence linking Thucydides with the Hippocratic methods, clearly supports Page's argument.

In summary, Thucydides clearly had an understanding of the medical literature of the time and used it as a resource for the specific terminology demanded in order to describe the disease. Since he wrote his *History* for a non-medical audience, the account is more literary. Attempts to tailor his description were for the purpose of making the information more accessible to the general population and did not interfere with the medical facts. Some of the vocabulary may have been purposely altered for the audience, but it is much more likely the

³⁹ Page, 1953, 110

⁴⁰ Parry, 1969, 113

ambiguity about the disease lies in the inherent difference between contemporary and ancient theories of disease. Ancient medical persons believed diseases to be a humoral imbalance within one person, while our contemporary society builds our medical knowledge on an understanding of pathophysiology. The two differing doctrinal approaches require the translator and reader to be suspicious of complete adherence to a translation. The terminology should not be restricted to one term, but must be allowed to “breathe.” This will present ambiguity in the clinical translation but prevent incorrect diagnosis.

Although the clinical symptoms present many problems when trying to determine the disease of the plague, the epidemiological information is less subject to error than description of the disease. For example, it is harder to observe the seasons or year incorrectly, than determining whether a skin lesion is a blister or rash. Additionally, the modern and ancient paradigm of understanding time, geography, and population demographics are similar. On the other hand, the modern and ancient paradigm of medical terminology has altered greatly. In relation to molecular epidemiology as mentioned above, starting with epidemiology leaves less room for error in determining the characteristics of the plague. Taking this into account, a descriptive epidemiological approach will be used to analyze Thucydides’ description of the disease to determine the disease in relation to person, place, and time.

5. Epidemiologic Information - Modes of Transmission

Epidemiology is the study of the distribution and determinants of health-related states or events in specified populations. In an epidemiological approach, the disease is characterized by its behavior in a population, which is contrasted with the clinical approach that characterizes the disease on the individual level. The clinical approach will be discussed in a subsequent section.

Possible modes of transmission will be eliminated based on their deviation from the plague's epidemiologic information. These modes of transmission are either direct or indirect. Direct transmission is the transfer of an infective agent from a host to a new host without an intermediate such as food, water, air, or animals.⁴¹ Direct modes of transmission include person-to-person and transplacental transmission. Indirect transmission is the transfer to a new host via an intermediate such as water, food, air, materials in the environment, or animals. Indirect modes of transmission include airborne, vehicle-borne, and vector-borne. Vehicle-borne diseases are transmitted by inanimate objects, while vector-borne diseases are transmitted by a live carrier. The modes of transmission discussed and the diseases suggested by past scholars, whether independently suggested or in conjunction with other diseases, are summarized in Table 1.

5A. Person

Thucydides clearly states that the plague consumed various population subgroups with equal voracity. Women, children, and men were affected. Slaves, metics, and citizens were affected. Army and civilians were affected.⁴² The only high-risk subgroups Thucydides mentions are physicians and those in crowded conditions, although he does not distinguish between a case fatality or attack rate.⁴³ If an individual survived the disease they acquired immunity to future fatal infections, although Thucydides mentions a second nonfatal attack could occur.⁴⁴ In terms of the Athenian diet, little was altered in wartime that would account for an epidemic. The Long Walls to Piraeus allowed food to reach Athens. With plenty of capital to spare and the means to get there, Athens traded with their allies all throughout the Mediterranean. Vitamin C deficiency could have occurred in the population, since the Spartans employed a scorched earth policy to

⁴¹ "Modes of Transmission," n.d.

⁴² Thuc., 2.49.1; Thuc., 2.53.4

⁴³ Thuc., 2.47

⁴⁴ Thuc., 2.51

destroy Attica's crops. The scorched earth policy involves the destruction of valuable resources that can be used by the enemy. Vitamin A deficiency, on the other hand, was unlikely because of the access to fish via Piraeus.

5B. Place

Thucydides spends little time and provides little analysis on the origins of the plague, although he mentions that the plague may have started in Ethiopia, spread to Egypt and Libya, and spread to the Persian Empire, before finally arriving to Athens.⁴⁵ Thucydides mentioned the directionality of the spread from Piraeus up into Athens. The Long Walls, housing crowded refugees from the countryside, offered the pathway for the spread. Thucydides remarks that Athens was hit the hardest of all.

Even though the demographic origins of the plague are unknown, the epidemic was probably ship-borne. The plague arrived at Piraeus first, which was one of the major Mediterranean ports at that time. The nature of Piraeus coupled with the fact that most pre-modern pandemics and epidemics, such as dengue, bubonic plague, cholera, and smallpox, were ship-borne lends to the likelihood of the plague arriving this route. One example of the plague transported by ship was mentioned by Thucydides in Bk. 2.58. Hagnon's naval expedition occurred between July and August of 430 B.C. and carried 4,000 troops in route to besiege the Potidaeans. The ship sailed during the height of the plague for five days, and when the navy reached their destination, the epidemic struck. Of particular note and interest is the observation that the starved and besieged Potidaeans were not affected by the plague. Another account of

⁴⁵ Thuc., 2.48

failed transmission is between the Athenians and Spartans, which will be discussed later at length.

The water supply of either of these cities, Athens or Piraeus, was unlikely contaminated. Athens had river-fed aqueducts from the Illisus River, wells, and several springs, while Piraeus' water came from cisterns, as noted by Morens and Littman.⁴⁶ Additionally, Athens is uphill from Piraeus and contains porous limestone under the soil. These conditions would not create an environment for the disease to travel from Piraeus to Athens; therefore, the probability that all of these sources were contaminated at the same time is incredibly low. Thucydides briefly attributes the spread of the disease to "poor ventilation" in the Long Walls, but this remark echoes the belief in the miasmatic theory of disease at the time and should not be highly considered. There is no doubt the living conditions within the Long Walls were brutal. Thucydides states,

"For having no houses but dwelling at that time of the year in stifling booths, the mortality was now without all form; and dying men lay tumbling one upon another in the streets, and men half dead about every conduit through desire of water. The temples also where they dwelt in tents were all full of the dead that died within them".⁴⁷

Aristophanes also speaks to this overcrowding and poor hygiene in the comedy *Knights* stating the refugees were squatting in birds' nests.⁴⁸ Gomme (1933) estimated the base population of Athens to be around 155,000 composed of 25,000 metics, 60,000 citizens, and 70,000 slaves. Major believed the population rose to over 400,000 during times of siege, Froland suggested over 500,000, and Rostovtzeff estimated around 315,000. During this time of the outbreak of the plague, Gomme (1933) and Hansen (1988) estimate the Athenian population was

⁴⁶ Morens & Littman, 1992, 275

⁴⁷ Thus., 2.51

⁴⁸ Aristophanes, 792

around 300,000 to 400,000. Xenophon believed Athens contained 10,000 houses, which housed an average of ten persons per household, but with the advent of the siege, this number shot up to approximately 40.⁴⁹ Many of the refugees entering the Long Walls were placed into crowded refugee camps. No matter which of these estimated populations figures is correct, Athens did not have the capacity to maintain a sustainable living environment for so great a population.

Thucydides mentions the lack of burial resources in 2.52.4, which resulted in corpses remaining in the streets for extended periods of time.

5C. Time

Thucydides observed that the plague hit Athens in the summer of 430 B.C. - probably in early May- a few days after the Spartans besieged Athens and ravaged the countryside. A second and third wave came in the summer of 428 B.C. and the winter of 427-426 B.C. Thucydides mentions a rekindling of the plague for an additional year in the winter of 427-426 B.C. that lasted another year. These figures suggest the plague lasted for at least two years continuously, but possibly four to five years. The disease was not season-bound. Thucydides remarks that the plague had exposing outbreaks, but it was uninterrupted.⁵⁰

5D. Multi-factored Epidemiological Information

The Hagnon expedition mentioned above provides more epidemiological information than simply evidence of ship-borne transmission. After reaching Potidaea, the army suffered and lost approximately twenty-five percent of their forces, 1,050 men, in a period of forty days. The 3,000 besieging Athenians already in Potidaea were also infected, although no statistics of the mortality rate were recorded by Thucydides. The incubation period, case fatality rate, and attack

⁴⁹ Morens & Littman, 1992, 288

⁵⁰ Thuc., 2.53

rate cannot be confidently determined by the information Thucydides provided, but both the case-fatality rate and attack rate can be roughly estimated to 25-100 percent. In 2.50.1, Thucydides observes that illnesses within the population eventually developed into the plague. The plague was apparently widespread throughout the population, which suggests a high attack rate. In the time of war, sheep and cattle within the city were transported to Euboea. Only birds, mice, rats, and dogs remained in the cities.⁵¹ Thucydides' unfamiliarity with the disease and the rapid spread suggest a virgin soil epidemic. Thucydides states, "so great a plague and mortality of men was never remembered to have happened in any place before."⁵² He also mentions that distinctive symptoms, signs, and the rapidity with which it extended through the population had not previously occurred.

6. Modes of Transmission Elimination

General modes of transmission and specific diseases within certain modes of transmission can be eliminated solely from the epidemiological information collected from Thucydides' description. The discussed modes of transmission include vehicle-borne, common-source acquisition, person-to-person, and reservoir vector-borne transmission. Each of these transmissions is considered separately.

6A. Vehicle-borne Transmission and Common-Source Acquisition

Common-source and vehicle-borne epidemics in open populations are generally waterborne or foodborne. Scurvy is a disease brought on by a deficiency of Vitamin C in the diet. As noted previously, the Athenians most likely had a Vitamin C deficiency in their diet, but the symptoms of scurvy would take weeks, possibly months to progress. The disease Thucydides

⁵¹ Morens & Littman, 1992

⁵² Thuc., 2.47

described happened within the matter of days. The Athenian population may have experienced scurvy, but the likelihood of this being the cause of the epidemic is minimal.

As discussed earlier, all the water sources from Piraeus to Athens would not have been simultaneously contaminated. Athens is not only uphill from Piraeus and contains porous limestone under the soil, but also the sources from which they obtained their water were too varied and decentralized. The potable water from Piraeus was in unconnected cisterns, away from other water sources and the sewage systems. Epidemics caused by enteric diseases are generally seen in areas with one-source water systems such as the one occurring in London in the middle of the nineteenth century. Athens' water systems could hardly be called sophisticated.

Ergotism, poisoning resulting from grain infected with fungi, has been suggested as a disease, specifically as an explanation for the peripheral gangrene seen in victims. Athens had massive storage systems for grain within the city, but these sources of grain would have been depleted within two years. For the remainder of the war, grain was imported from various locations, since Athenians still had access to the Aegean due to their extensive fleet. If some harvest survived the scorching methods of the Spartans, it would unlikely be contaminated for multiple growing seasons. Even if the crops were contaminated for years, the grain it produced would not have been enough to feed the Athenian population. Another problem with this transmission is that the Spartans would have taken the crops before they burned the earth, yet Thucydides specifically states that they did not contract the disease.

Also, the Athenian expedition to Potidaea and the following outbreak of the Plague among the besieging population and army is highly improbable via vehicle-borne transmission. The likelihood of massive contamination of all the grain or water sources entering into Athens

for approximately five years is not reasonable enough to entertain. Although ergotism, typhoid fever, alimentary toxic aleukia, shigellosis, scurvy, and cholera have been offered up as possible diseases, common-source acquisition and vehicle-borne transmission are not satisfactory candidates for the causative agent of the Athenian plague.

6B. Person-to-Person Transmission

Person-to-person transmission has three subcategories: fecal-oral, sexually transmitted, and respiratory. Most enteric pathogens can be acquired through vehicle-borne and fecal-oral transmission; even when both routes are considered, an enteric disease would not be able to reach throughout the city. Neither route would reach tens of thousands of Athenians causing an epidemic; it was already mentioned there was no water source that reached throughout the city. By excluding these modes of transmission, cholera, shigellosis, poliomyelitis, and typhoid fever can be removed as possible candidates. Typhoid fever will be briefly revisited later in light of recent paleopathological evidence.

Despite the varying sexual orientation of Spartan and Athenian men, sexually transmitted diseases (STDs) are clearly unlikely candidates. Thucydides specifically mentions all population subgroups –excluding physicians- were equally affected. STDs would be seen predominantly in sexually active subgroups and not in children. It is highly unlikely the STDs could be spread throughout a population of at least tens of thousands individuals in a matter of weeks, even when you consider men returning from naval expeditions. Syphilis can be excluded as a possible disease. Syphilis takes years, even decades, to develop systemic and neurological symptoms. It does not kill a patient quickly.

Morens and Littman (1992) mention three aspects of respiratory transmission that correspond to the plague: population subgroups were equally affected, upper respiratory symptoms occurred in initial stages of disease, and there was correlation to crowding. Thucydides' description of the temporal length of the epidemic and overcrowding of the city rules out most respiratory diseases. Respiratory diseases do not over-winter and would have died out quickly in a crowded population of 300,000-400,000 individuals.⁵³ There is a small likelihood of a respiratory disease entering the population explosively, remaining in a closed and crowded population between two to five years, and not "burning" through the susceptible individuals in the population. Respiratory epidemics peak quickly in their number of infected individuals, diminish rapidly, and rarely become endemic within a population. Also, of particular note, the Spartan army was not infected with the disease, even though they were known to have various contacts with the Athenian army and citizens in the countryside. Diseases transmitted solely via the respiratory route with no means of persistence can be excluded, including measles, meningitis, influenza and staphylococcal diseases. Different forms of these diseases such as TSS, influenza with TSS, and Guillain-Barre can be excluded. Also, measles and influenza can be excluded on the grounds that survivors had naturally acquired immunity, even though immunity acquired from influenza is only for a few years after the infection.⁵⁴

In order for a person-to-person transmitted respiratory disease to be considered, the long-term incidence of the disease in the population and the absence of transmission to populations that the Athenians certainly had contact with must be explained. One explanation for the reemergence of the plague is a respiratory disease with the means of persistence.

⁵³ Morens & Littman, 1992

⁵⁴ Gill & Murphy, 1977

6C. Reservoir Vector-borne (Zoonotic) Transmission

The zoonotic diseases suggested by scholars are not epidemic. If a human is infected with glanders, leptospirosis, rabies, anthrax or tularemia, they are considered an accidental or dead-end host. A dead-end host is an intermediate host that does not allow transmission to another host.⁵⁵ The infectious zoonotic sources for these diseases (rabbits, horses, etc.) would not have been widespread or in enough abundance to cause an outbreak of epidemic proportions. Especially in the case of anthrax, cattle and sheep in the city of Athens were relocated to Euboea. Animal hides with anthrax spores would not be a sufficient enough source for an epidemic, thus limiting the contact between the reservoir and human.

6D. Reservoir Vector-borne (Insect) Transmission

The bubonic plague, arboviral diseases, and typhus are likely candidates within this category of transmission, while malaria is an unlikely candidate for the plague of Athens. Children under fifteen are particularly susceptible to malaria. Over sixty-five percent of the cases are within this population, which is not congruent with Thucydides' observations. Malaria is more common in rural areas than in cities.⁵⁶ Also, malaria has particular seasons where transmission of the disease occurs and its seasonality is not compatible with that of the plague of Athens. The clinical symptoms are discussed later, but it is relevant to note here that the clinical features of malaria are inconsistent with the plague. Finally, Hippocrates and other physicians were familiar with malaria, therefore Thucydides would not have stated "So great a plague and mortality of men was never remembered to have happened in any place before."⁵⁷

⁵⁵ "dead end host," n.d.

⁵⁶ Cui, 2012

⁵⁷ Thuc., 2.47

7. Possible Diseases

One possibility is a respiratory disease with a means of persistence. This persistence can include a scattered rural population reintroducing the causative agent back into the Athenian population or fomite persistence.⁵⁸ Smallpox fits the epidemiologic criteria needed of a respiratory disease to be maintained within the Athenian population for at least two years. The second possibility is insect-borne disease; such as the bubonic plague, arboviral diseases, and typhus. These remaining diseases will be analyzed based on the clinical features presented by Thucydides.

8. Philological Analysis of Thucydides' Description

As noted previously, an accurate translation of Thucydides' description of the plague is of the utmost importance. Five critical clarifications of vocabulary will be used to better translate Thucydides and compose the most accurate list of clinical symptoms. The first of these clarifications appears in the sentence, “κατέσκηπτε γὰρ ἐς αἰδοῖα καὶ ἐς ἄκρας χεῖρας καὶ πόδας, καὶ πολλοὶ στερισκόμενοι τούτων διέφευγον, εἰσὶ δ' οἱ καὶ τῶν ὀφθαλμῶν.”⁵⁹ In the R. Wagner edition of *History*, the phrase is translated, “It affected the genitals, the fingers, and the toes, and many of those who recovered lost the use of these members; some, too, went blind.”

στερισκόμενοι, is a third person, masculine, plural, passive, or middle participle of sterew. The antecedents of τούτων are aidoia, ceiras, and podas and the participle agrees with polloi. The most appropriate translation, due to the causal nature of the verb, is ‘many survived because they lost these parts.’⁶⁰ The participle can be applied to two subsequent events mentioned in the text: (1)

⁵⁸ Morens & Littman, 1992

⁵⁹ Thuc., 2.48.8

⁶⁰ Thuc., 186, R. Warner translation

being deprived of fingers, toes, and genitalia, and (2) inflammation of the eye with successive blindness. Amputation of the extremities does not need to be inferred here, although scholars such as Lucretius incorrectly translated the term in the active meaning ‘cutting off.’⁶¹ Page, Liddell and Scott, and Morgan generally agree with this translation and the passive nuance of the participle, which solidifies gangrene as a clinical symptom.

Some scholars have argued for the bubonic plague as the plague of Athens. Their argument centers around the assumption that in another sentence Thucydides meant to say *boubwn* instead of *elkesin*, which means wound. The phrase in questions is, “ἀλλ’ ὑπέρυθρον, πελιτνόν, φλυκταίναις μικραῖς καὶ ἔλκεσιν ἐξηνηθηκός.”⁶² In the R. Wagner edition of *History*, the phrase is translated, “‘the skin’ was rather reddish and livid, breaking out into small pustules and ulcers.” *Elkesin* most commonly refers to a lesion on the body and is a term of general reference, but context should decide whether ‘sore,’ ‘ulcer,’ ‘wound,’ or other terms are appropriate. *Boubwn* was commonly used in the 400’s as “swelling” rather than “wound” and was used to describe buboes. Other definitions for *boubwn* are groin, glands, and swollen glands. So, was Thucydides mistaken in his terminology? In one of Menander’s dramas written in the 300’s, a farmer complains of a *Boubwn* that has arisen on his groin from an *elkos* on his foot from a spade. These were words of a layman with little to no knowledge of medical terminology, so if this man did not mix up the terms, it is not likely Thucydides would do such a thing. Thucydides observed lesions or wounds and not buboes. In terms of whether the phrase should be translated as ulcer, pustles, rash, or skin lesion, the purpose of this analysis, finding the exact meaning of the phrase is not important. Instead, the presence or absence of symptoms like a rash rather than whether it was a blister, pustule, sore, or ulcer will be considered.

⁶¹ Bailey, 1947

⁶² Thuc., 2.48.5

Kardian, based on etymology, seems to offer a simple translation: ‘heart.’ Although in the phrase, “καὶ ὁπότε ἐς τὴν καρδίαν στηρίζειεν, ἀνέστρεφέ τε αὐτὴν καὶ ἀποκαθάρσεις χολῆς πᾶσαι ὅσαι ὑπὸ ἰατρῶν ὀνομασμένα εἰσὶν ἐπῆσαν”⁶³, Kardian is defined as ‘cardiac orifice of the stomach’ or ‘stomach.’⁶⁴ In the R. Wagner edition of *History*, the phrase is translated, “Next the stomach was affected with stomach-aches and with vomiting of every kind of bile that was given a name by the medical profession.” This is an important clarification to determine if vomiting of bile was a symptom. This alternative meaning is attested in medical writings of Thucydides’ time, which proves vomiting was indeed a symptom.

9. Clinical Features and General Features

In 49.1-51.2 of Thucydides’ *History of the Peloponnesian War*, the symptoms of the plague are described from “head to trunk (kardia, swma 49.3-5), the bowels (koilia 49.6), and extremities (akrwthria 49.7)” as the disease progressed through the body.⁶⁵ Table 2 is a summary of Thucydides’ account of Athenian plague symptoms and general features compiled from original translations of specific vocabulary, the Penguin translation and Page’s translation.

10. Final Elimination of Diseases

As discussed earlier, the clinical and general features alone do not add up to a specific disease. When these features are applied to the diseases narrowed down by epidemiologic grounds, a final causative is concluded.

10A. Elimination of Smallpox

⁶³ Thuc., 2.48.3

⁶⁴ Liddell & Scott, 877

⁶⁵ Thuc., 182, R. Warner Translation

Smallpox is a respiratory disease with fomite persistence, which can explain the explosive and re-emergent characteristics of the plague of Athens. The disease of smallpox, *Varicella major* or *Varicella minor*, has a lengthy incubation period of twelve days and has less transmissibility than measles or influenza, which decreases the ‘burn-through’ rate in the population. Assuming a minimum base population of 100,000 people per four sq. miles, a twelve-day serial generation time and two adequate contacts, the plague if caused by smallpox would last as long as eleven months.⁶⁶ Even after the epidemic burns through the population, it can be reintroduced through dried secretions. Epidemiologically, smallpox seems like a relative fit, but the clinical and general features of the epidemic observed by Thucydides eliminate smallpox as a possibility. Thucydides observed gangrene, which is not a symptom of smallpox. Also, Thucydides did not observe pock-marks, even though they are distinctive features of the disease. If Thucydides contracted the disease, he would have had the pockmarks on his body and face. Such a distinct side effect would have been mentioned by Thucydides, especially if he was disfigured and mentioning it would have provided a better identification of the disease. Thucydides mentions amnesia and delirium as side effects in some cases, but neither of these are characteristic side effects of smallpox. Physical prostration is a characteristic of smallpox, but in 2.49.5 Thucydides specifically mentions that patients are able to move. The deviation from Thucydides’ description of the plague’s clinical features eliminates smallpox as a possible disease on clinical grounds. Smallpox does not fit into the general features either. In 2.51 Thucydides says a second nonfatal attack could occur, but a smallpox victim has naturally acquired immunity.⁶⁷ This type of immunity is the result of memory cells retained from B and T-cell populations, which were created in response to the smallpox pathogen. This immunity is life-

⁶⁶ Morens & Littman, 1992

⁶⁷ “Epidemiology of Smallpox,” n.d.

long. Other problems with smallpox are the mortality rate among specific population subgroups and that it discriminately attacks the younger populations. Poole and Holladay (1979) stated that eighty to ninety percent of smallpox mortalities occur in children. Thucydides specifically states the disease did not discriminate against age groups. Another inconsistency in the argument for smallpox is that the person-to-person respiratory transmission would most likely result in the Spartan population contracting the disease. Kobert (1899) argued it was smallpox with ergotism, but ergotism was already excluded on epidemiologic grounds. All of these factors decidedly remove smallpox as a viable disease of the plague of Athens.

10B. Elimination of the Bubonic Plague

Multiple scholars, including Williams and Hooker, have presented the bubonic and pneumonic plague as diseases for the plague.⁶⁸ Thucydides clearly describes gangrene of the extremities in 2.49.7-8, which occurs in individuals with the bubonic plague. On the other hand, the characteristic buboes of the bubonic plague were not noted by Thucydides. Many scholars argue that Thucydides' exclusion of the symptom was an unintentional omission. As noted earlier, such a mistake was very unlikely in an educated and well-versed historian with knowledge of Hippocratic medical literature. If the bubonic plague was the disease, Thucydides would have observed faster death rates between one to three days. The bubonic plague is simply not consistent with Thucydides' account.

10C. Elimination of Arboviral Diseases

Morens and Chu (1986) suggested an arbovirus as the causative agent of the plague of Athens, focusing on Rift Valley fever. Morens and Chu suggested that epidemiologically, these

⁶⁸ Williams, 1958

reemergent and explosive diseases have the greatest similarity to the Athenian plague. Dengue particularly has multiple documentations of its explosive epidemic-inducing behavior.⁶⁹ The arbovires have an RNA genome and have a particularly high mutation rate. This high mutation rate leads to quick evolutionary change as compared to bacteria or DNA viruses. The arboviral diseases would first show these mutations are at the molecular level with clinical and epidemiologic levels being the last affected.⁷⁰ Although the arboviral diseases fit epidemiologically, they do not fit the clinical or general features of Thucydides' description. Rift Valley fever often only has mild fever or no symptoms. There is no gangrene or diarrhea, and there is a less than 1% mortality rate. Yellow fever has less than 5% mortality rate and natural acquired immunity. Most of these arboviruses have mild symptoms with such a low mortality rate they are an unlikely candidate for the plague of Athens.

11. Response to Paleopathological Evidence

Paleopathology is the study of ancient disease. It can give us a look into the disease's evolution, ancient population conditions, and long-term associations between disease, human biology, and culture.⁷¹ The principal source of paleopathology is skeletal remains, which can be used to determine a person's history of disease and health. In 2001 at a Kerameikos cemetery, a 2500 year old mass grave dating back to the plague of Athens was discovered. Doctor Manolis Papagrigrorakis studied the skeletal remains and was able to extract *S. enterica serovar Typhi*. He concluded that this pathogen was the causative ancient of the plague. DNA recovered from three skeletons' teeth resulted in two strands of DNA that were similar to a *Salmonella* species. The first problem with the study is the extremely small sample taken. The second problem is

⁶⁹ McCoy, 1964

⁷⁰ Holmes, 1998

⁷¹ Augderheide & Rodriguez-Martin, 1998

Papagrigorakis's phylogenetic analysis: "if another, yet unknown pathogen.... was the actual cause of the plague of Athens, it would have to be closely related to *S. enterica* and definitely closer than *S. typhimurium*."⁷² This analysis is based on an 8% and 7% divergence between the obtained DNA sequence and *S. typhimurium* and *S. enterica* serovar Typhi, respectively. The divergence means that of the whole DNA sequence the two strains differed from each other between 8% and 7%. Shapiro *et al* states that the sequence obtained is more similar to *S. enterica*, although the two *Salmonella* strands being compared to the discovered DNA are strikingly similar. There is less than 1% difference to the discovered DNA sequence. The analysis does not confirm typhoid as the disease, but rather suggests it is simply *Salmonella* in origin. In addition to the phylogenetic analysis, we know Typhoid was endemic in ancient Greece from Hippocratic accounts, so the presence of the disease does not conclude it was a disease. If typhoid was not endemic, its presence at the same time the plague occurred would be of particular note, but this is not the case.

12. Why Typhus?

Typhus, argued for by Macarthur (1954), Crawford (1914), Keil (1951), and Ferguson,⁷³ is the best fit for the disease of the plague of Athens. Arguments made previously against typhus by Shrewsbury (1950) and Page (1953) were contingent on *Rickettsia prowazeki* being acquired by contact with rats and the disease spread further by the louse. Typhus was historically eliminated as a possible disease, because some scholars believed rats were rare in ancient Greece.⁷⁴ They believed *mus* was a specific term for mouse and a general term for rodent, but

⁷² Papagrigorakis, 2006

⁷³ Ferguson, 151-3

⁷⁴ McNeill, 1976

did not include the rat.⁷⁵ Now *Rickettsia prowazeki*, the disease of epidemic typhus, and *Rickettsia mooseri*, the disease of Murine typhus, are recognized as two individual species, due to the work of Mooser and Zinser in the 1930s.⁷⁶ *Rickettsia mooseri* is propagated to a human population by the rat flea, while *Rickettsia prowazeki* is propagated by the body louse. The separation of epidemic and murine typhus nullifies the previous arguments made against the disease by Shrewsbury (1950) and others. The argument for the absence of rats in Athens could be useful for the case against the plague, but it was already excluded on clinical grounds and will not be discussed further.

12A. Clinical Argument for Typhus

Here Page describes the movement of a Typhus infection, but I will add parentheticals referring back to Thucydides' text describing the clinical features.

“The onset is rapid, with severe headache (kefalhs qermai iscurai), suffused eyes (twñ ofqalmwn epuqhmata kai flogwsis), and foul breath (pneuma atopon kai duswdes hfiei). Hoarseness is common (bragcos), cough and some kind of bronchial disorder universal (meta bhcos iscurou). Vomiting is not characteristic, but may occur (apokaqarqeis colhs). The body suffers internally a strong sensation of heat (de entos ekaeto), which may not be apparent to the touch (kai to men exwqen aptomenw swma out' agan qermon). The skin-eruption may be livid in colour as well as red (flukatainai mikrais kai elkesin exhnqhkos). Further developments include gangrene (ex akrais ceiras kai podas kai polloi steriskomenoi toutwn diefeugon), with hemorrhage and diarrhea (diarroiias). Loss of memory (lhqh elambane) and mortification of fingers and toes are common complications

⁷⁵ Liddell & Scott, 1155

⁷⁶ Mooser et al., 1931

(akrwthriwn antilhyis); and there are records of impairment of the eyesight (steriskomenoi twn ofqalmwn).”⁷⁷

The clinical symptoms of typhus are strikingly similar to and parallel Thucydides’ description.

12B. Epidemiologic Argument for Typhus

Epidemic typhus, caused by the agent *Rickettsia prowazeki*, is a common vector-borne disease that is known for explosive epidemics seen in crowded populations and in war-time. Epidemiologically, typhus remains in crowded populations for extended periods of time and remains or persists in survivors. The ten to sixty percent mortality rate range for epidemic typhus includes the twenty-five percent or more mortality rate deduced from the Hagnon expedition. The natural acquired immunity with possible nonfatal recurrence observed by Thucydides is characteristic of typhus. After extended periods of time, a previously infected individual may relapse back into the disease to the point that they become infectious, but this second infection is not fatal. Unlike smallpox, typhus has no age predilection. The mutation rates of the DNA containing *Rickettsia prowazeki* is relatively minimal. This suggests any evolutionary change on an epidemiologic and clinical scale is unlikely and lends more credibility to the similarities observed between the ancient strain of typhus and modern strain.⁷⁸

The body louse, *Pediculus humanus humanus*, is the vector for the pathogenic bacteria through its infected feces and resides in clothing.⁷⁹ The louse is particularly found in the areas of clothing that touch the body such as the neck, groin, armpits, and waistline. *Pediculus*

⁷⁷ Page, 1953, 114

⁷⁸ Drake et al. 1998

⁷⁹ Fournier et al., 2002

humanus humanus contracts *Rickettsia prowazeki* upon ingestion of host's infected blood.

Within seven to ten days of the bacterial infection, the louse dies or survives with a lifelong infection. Since the louse is only a human parasite, it can also die within seven to ten days if a host is absent. The most common mode of transmission for *Rickettsia prowazeki* is inoculation by a louse with infected feces that contains the disease. The incubation period of the disease is between six and sixteen days, but the most common is twelve. Epidemic typhus occurs specifically in crowded populations with large populations of lice. The louse resides in the clothing, and when the host moves around or scratches their clothing, the lice will move to the outer clothing. This makes the transfer to another person easier, since the louse only crawls. From the writings of Thucydides and Aristophanes, we know Athens was overcrowded and had a decline in normal standards of sanitation. In order for epidemic typhus to be a plausible disease, there had to be an infestation of lice in the Athenian population. In the *Peace* in line 540, Aristophanes references the abundance of lice in the city. Also, the First Book of the *Epidemics* describes the presence of lice in Corinth.

Epidemic typhus fits the plague of Athens clinically and epidemiologically. Due to the overcrowding and infestation of lice, there were perfect conditions for epidemic to flourish for multiple years in Athens. There is one remaining unanswered question in this mystery: 'how did the Spartans fail to contract the plague, even after multiple points of contact with possibly the population in Attica and the Athenian army?'

13. The Spartan Mystery Solved

The main point of contact between the Spartans and Athenians was on the battle field. Pericles' strategy to move the civilian population of Attica within the Long Walls decreased the points of contact between the Spartan army and the civilian Attica population. Thucydides states,

“The Peloponnesians were no sooner entered Attica but the sickness presently began, and never came into Peloponnesus, to speak of, but reigned principally in Athens and in such other places afterwards as were most populous.”⁸⁰

When viewing the aforementioned question in terms of typhus, the length of contact, clothing customs, and exchange of clothing between armies or between armies and civilians are important factors to address. Clothing in Ancient Greece was particularly expensive and time consuming to make. Civilian Athenians going into the Long Walls would have taken all of their clothing with them; essentially, they would have taken the plague with them. Even if clothing was left behind, the short life-span of the lice without a human host makes it very unlikely the lice would still be alive by the time the Spartan army arrived in Attica. Both of the scenarios make the transmission of lice between the Spartan army and the civilian population highly improbable.

The frequent points of contact between the Athenians and the Spartans were through battle. If there was a point of transmission, the battlefield would be the most probable location. The louse does not jump between individuals, nor does it move quickly. The only highly probable mode of transmission for the louse to reach the Spartan population would be if the Spartans took the louse-infected clothing off the dead Athenian soldiers. The next question to explain this mystery is whether or not the Spartans would have taken Athenian clothing.

⁸⁰ Thuc., 2.54

Athenians and Spartans fought with armor and tunics covering their bodies. The armor they did use was scant, had little direct contact with the body, and was not made out of cloth that would house lice. In the Archaic period, the tunics were made out of wool, but in the Classical period, the material was linen. During the later 5th century B.C., the Lacedaemonians began to wear a new style of tunic called exomis.⁸¹ The material left the right shoulder and arm open for more mobility in combat. The Spartans kept their traditional style of the himation, while the Greeks replaced the himation with the chlamys during the Classical period. In addition to the different style, the Spartans wore crimson himations and tunics. Lycurgus ordered this change because the color least resembled women's clothing and was war-like.⁸² In most cases, the cloak was not used in battle and only worn during leisure time. Since the Spartans wore a different style and color of tunic than the Athenians, the likelihood that the Spartans stripped dead Athenians for their clothing is highly unlikely. This would explain the Spartans not contracting the plague, even after direct person-to-person contact.

14. Conclusion

From the diseases presented by previous scholars, modes of transmission were eliminated based on the epidemiological information presented by Thucydides in *The History of the Peloponnesian War*. One possibility was a respiratory disease with a means of persistence. This persistence can include a scattered rural population that continues to reintroduce the causative agent back into the population or fomite persistence. Smallpox fit the epidemiologic criteria needed of a respiratory disease to be maintained within the Athenian population for at least two years. The second possibility is insect vector-borne disease, such as the bubonic plague, arboviral

⁸¹ Sekunda, 1998

⁸² Plutarch, 430

diseases, and typhus are the most likely candidates. These remaining diseases were analyzed based on the clinical features presented by Thucydides. Smallpox, bubonic plague, and arboviral diseases had relatively similar epidemiological features to the Athenian plague, but did not match the clinical and general features presented by Thucydides. Typhus not only fit the Athenian plague epidemiologically, but also clinically. The mysterious lack of transmission to the Spartans can be explained by the vector of *Rickettsia prowazeki* and the clothing customs of the Spartans.

| Table 1. Possible infectious diseases of the Plague of Athens classified by agent and mode of transmission | | | | | |
|--|------------------|------------------------------|---------------|--------------|-----------|
| | | Modes of Transmission | | | |
| Agent | Person to person | Transplacental | Vehicle-borne | Vector-borne | Airborne |
| Virus | smallpox | | smallpox | | smallpox |
| | measles | | | | measles |
| | influenza | | | | influenza |
| | poliomyelitis | | | | |

| | | | | | |
|----------|-------------------------------|----------|-----------------------------------|-------------------|------------|
| | Marburg-Ebola meningitis | | | | |
| | | | | yellow fever | |
| | | | | dengue fever | |
| | | | | Rift Valley fever | |
| | | | | rabies | |
| | | | | hantavirus | |
| | | | | arenavirus | |
| Bacteria | syphilis | syphilis | | | |
| | scarlet fever | | scarlet fever | | |
| | shigellosis | | shigellosis | | |
| | meningitis | | | | |
| | staphylococcal | | | | |
| | erysipelas | | | | |
| | toxic shock syndrome (toxins) | | | | |
| | | | diphtheria | | diphtheria |
| | | | typhoid fever | | |
| | | | cholera | | |
| | | | | anthrax | anthrax |
| | | | | pneumonic plague | |
| | | | | bubonic plague | |
| | | | | glanders | |
| | | | | typhus | |
| | | | | tularaemia | |
| | | | | leptospirosis | |
| Protozoa | | | | malaria | |
| | | | | rickettsialpox | |
| Fungal | | | ergotism | | |
| | | | alimentary toxic aleukia (toxins) | | |

| Progression through the body | Clinical symptoms associated with progression | Points of death or deviated temporal components of symptoms |
|---|---|---|
| Kardia , swma (the head to the trunk) 49.2-5 | 1. Heat in the head; redness and burning in the eyes; blood-red throat and tongue; abnormal and | |

| | | |
|--|--|--|
| | <p>malodorous breath (49.2)</p> <p>2. Sneezing and hoarseness with violent coughing; <i>vomiting of bile</i> (49.3)</p> <p>3. Empty retching and convulsions/spasms (49.4)</p> <p>4. Normal external body temperature; flushed and livid skin; <i>small blisters and sores</i>; total body hyperaesthesia and restlessness; unquenchable thirst; alleviate body heat via immersion into water (49.5)</p> | <p>The retching may occur after the previous symptoms ceased or much later.</p> <p>Majority died from this internal fever on the seventh or ninth day.</p> |
| koilia (the bowels) 49.6 | 1. Lesions form in bowels and uniformly fluid diarrhea (49.6) | Terminal exhaustion may occur apparently caused by diarrhea. |
| akrwthria (the extremities) 49.7 | 1. <i>Seizure of extremities</i> (genitalia, fingers, and toes), convalescent amnesia (49.8) | Many survived at this stage with the <i>losing the use of extremities or eyes</i> . ⁸³ |
| <p>Other general features of note for the plague include the following: birds and animals most likely died upon consumption of infected dead bodies (50.1-2), weak and strong person equally susceptible (51.3), contagious and communicable infection (51.4), and acquired immunity (51.5) with chance of second nonfatal attack. Nearly all the verbs are iterative optative or imperfect, which speaks to Thucydides observing the symptoms as recurring phenomena.</p> | | |

Works Cited

Anderson RM. "Directly Transmitted Viral and Bacterial Infections of Man," *The Population Dynamics of Infectious Diseases: Theory and Applications* (New York 1982) 1-37.

⁸³ Morgan, 1994

- Augderheide, A. and Rodriguez-Martin, C. eds. *Cambridge Encyclopedia of Human Paleopathology* New York: Cambridge University Press, 1998.
- Aristophanes. Knights. *The Internet Classics Archive*. Retrieved Jan 1 2013 from
<<http://classics.mit.edu//Aristophanes/knights.html>>
- Babkin IV, Shchelkunov SN. Time scale of Poxvirus evolution. *Molecular Biology* 2006; 40: 16-19
- Bailey C. *T. Lucretius C. De rerum natura*. Oxford: 1947; 3.
- Baziotopoulou-Valavani E. A mass burial from the cemetery of Kerameikos. In Stamatopoulou M, Yeroulanou M, eds. *Excavating Classical Culture: Recent Archaeological Discoveries in Greece*. Oxford, England: Archaeopress; 2002; 187-201. *Studies in Classical Archaeology*; vol I.
- Bellemore J, Plant IM, Cunningham LM. Plague of Athens- fungal poison? *J Hist Med Allied Sci*. 1994; 49: 521-543.
- Cantlie J. Plague: Hot to recognize, prevent and treat plague. London, England: *Cassell* 1900:1-68
- Cartledge P. *Sparta and Lakonia: A Regional History 1300-362 BC*. Routledge. Second Edition.
- Cochrane CN. *Thucydides and the Science of History*. 1929.
- Crawford R. *Plague and pestilence in Literature and Art*. Oxford; 1914: 23-41, 212-22
- Cui L, Yan G, Sattabongkot J, Cao Y, Chen B, Chen X, Fan Q, Fang Q, Jongwutiwes S, Parker D, Sirichaisinthop J, Kyaw MP, Su XZ, Yang H, Yang Z, Wang B, Xu J, Zheng B, Zhong D, Zhou G (2012). "Malaria in the Greater Mekong Subregion: Heterogeneity and complexity". *Acta Tropica* 121 (3): 227–39.
- Cunha BA. Osler on typhoid fever: differentiating typhoid from typhus and malaria. *Infect Dis*

Clin North Am 2004; 18: 111-125.

Cunha BA. The cause of the Plague of Athens: plague, typhoid, typhus, smallpox, or measles?

Infect Dis Clin North Am 2004; 18: 29-43.

Dagnino J. What was the plague of Athens? *Rev Chilena Infectol* 2011; 28: 374-380.

dead-end host. (n.d.) *The American Heritage Medical Dictionary*. (2007). Retrieved Jan 1 2013

from <<http://medical-dictionary.thefreedictionary.com/dead-end+host>>

Dixon B. Ebola in Greece? *Br Med J* 1996; 313:430

Domingo E, Escarmís C, Sevilla N, Moya A, Elena SF, Quer J, Novella IS, Holland JJ (June

1996). "Basic concepts in RNA virus evolution". *The FASEB Journal : Official*

Publication of the Federation of American Societies for Experimental Biology 10 (8):

859-64.

Drake J W, Charlesworth B, Charlesworth D, Crow JF, Rates of spontaneous mutation. *Genetics*

1998; 148:1667-1686

Durack DT, Littman RJ, Benitez RM, Mackowiak PA. Hellenic holocaust: a historical clinico-

pathologic conference. *Am J Med* 2000; 109:391-7.

Epidemiology of smallpox. (n.d.) World Health Organization. Retrieved Dec 20 2012 from

<http://whqlibdoc.who.int/smallpox/9241561106_chp4.pdf>

Ferguson T. In a letter to A.W. Gomme. *HCT* ii. 151-3

Froland A. The great plague of Athens 430 BC. *Dan Medicinhist Arbog* 2010; 38: 63-80.

Gill PW, Murphy AM. Naturally acquired immunity to influenza type A: a further prospective

study. *Med J Aust* 1977; 2: 761-5.

Harrison JE. The Ancient City, Its Character and Limits and Sanctuaries in the Citadel. *Primitive*

Athens as Described by Thucydides. Cambridge: 1906; 1:5-65

Hippocrates (W.D. Smith trans.) *Hippocrates: Epidemics Book 2,4-7 (Loeb Classical Library)*

Boston: Harvard University Press; 1994.

Hippocrates (W.H.S. Jones trans.) *Hippocrates: Nature of Man IV (Loeb Classical Library)*.

Boston: Harvard University Press; 1994.

Holden C. Athenian plague probe. *Science* 1996;274:1307

Holladay AJ. The Thucydides syndrome: another view. *N Engl J Med* 1986;15:1170-3.

Holmes E. Molecular Epidemiology and evolution of emerging infectious diseases. *British Medical Bulletin* 1998; 54: 533-543.

Fournier PE, Ndiokubwayo JB, Guidran J, Kelly PJ, Raoult D. Human pathogens in body and head lice. *Emerg Infect Dis* 2002; 8: 1515-8

Froland A. The great plague of Athens 430 BC. *Dan Medicinhist Arbog* 2010; 38:63-80.

Galrani A. Epidemiology meets evolutionary ecology. Berkeley, California: *TRENDS in Ecology and Evolution* 2003; 18: 132-137.

Ganem DE. Plasmids and pestilence- Biological and clinical aspects of bubonic plague- Medical Staff Conference. University of California, San Francisco. *West J Med* 1986; 144:447-451.

Gomme AW. *The Population of Athens in the Fifth and Fourth Centuries BC*. Oxford, England: Basil Blackwell; 1933; 22-44.

Grote G. *A History of Greece*. 1888; 10:78.

Hansen MH. Athenian population losses 431-403 B.C. and the number of Athenian citizens in 431 B.C. In: Hansen MH, ed. *Three Studies in Athenian Demography*. Copenhagen, Denmark: Munkgaard; 1988; Historisk-Filosofiske Meddelelser; vol LVI

Keil H. The Louse in Greek Antiquity, with comments on the Diagnosis of the Athenian Plague

- as recorded by Thucydides. *Bull. Hist. Med.* 1951; 25:305-23.
- Kobert R. Uber die Pest des Thucydides, *Janus* 1899; 4: 240-251.
- Langmuir AD, Worthen TD, Solomon J, Ray CG, Peterson E. The Thucydides syndrome: a new hypothesis for the cause of the plague of Athens. *N Engl J Med* 1985;313:1027-30.
- Littman RJ, Littman ML. The Athenian plague: smallpox. *Trans Am Philol Assoc* 1969; 100: 261-275.
- Littman RJ. The Plague of Athens: Epidemiology and Paleopathology. *Mount Sinai Journal of Medicine* 2009; 46: 456-467.
- Littre E. Deuxieme livre des epidemies. *Oevres completes d' Hippocrate*. Paris: 1839; 5: 43-71
- Longrigg J. The great plague at Athens. *Hist Sci* 1980; 18: 209-225.
- MacArthur WP. The Athenian plague: a medical note. *Classical Q* 1954; 4: 171-174
- Maia C. "Some Mathematical Developments on the epidemic Theory Formulated by Reed and Frost," *Human Biol.* 24 (1952) 167-200
- Major RH. *Fatal Partners, War and Disease*. New York 1941; I 9-13.
- Massell TB. What Caused the Plague of Athens? *The Western Journal of Medicine* 1986; 145: 104-105.
- Mausner JS, Kramer S. "Descriptive Epidemiology: Person, Place, and Time," *Epidemiology- An Introductory Text* (Philadelphia 1985) 118-153.
- McArthur WP. The Athenian plague: a medical note. *Classical Quarterly* 1954; 48:171-174.
- McCoy OR. "Dengue. Epidemiologic Considerations," in J.B. Coates, E.C. Hoff, P.M. Hoff, Medical Department, United States Army (eds), *Preventative Medicine in World War II, Volume VII: Communicable Disease. Arthropodborne Diseases Other Than Malaria*, (Washington, D.C. 1964) IV 29-40.

McNeill, William H. *Plagues and People*, New York: Anchor Books, 1976.

McSherry J, Kilpatrick R. The plague of Athens. *J R Soc Med* 1992;85:713

Modes of Transmission. *The Open University*. (2013). Retrieved Jan 1 2013 from
<<http://labspace.open.ac.uk/mod/oucontent/view.php?id=439261§ion=20.4.4>>

Morens DM, Chu MC. The plague of Athens. *N Engl J Med* 1986; 314:855.

Morens DM, Littman RJ. Epidemiology of the Plague at Athens. *Trans Am Philol Assoc* 1992;
122: 271-304.

Morens DM, Littman RJ. Thucydides' syndrome reconsidered: new thoughts on the 'Plague of
Athens.' *Am J Epidemiol* 1994; 140:621-28

Morgan TE. Plague or poetry? Thucydides on the epidemic at Athens. *Trans Am Philol Assoc*
1994; 124: 197-209.

Mooser H, Castaneda MR, Zinsser H. The Transmission of the Virus of Mexican Typhus from
Rat to Rat by *Polyplax Spinulosus*. *J Exp Med* 1931; 54: 567-575

Nutton V. *Ancient Medicine*. London, England: Routledge; 2004.

Ochman H, Elwyn S, Moran NA. Calibrating bacterial evolution. *Proc Natl Acad Sci USA* 1999;
96:12638-12643.

Olson PE, Hames CS, Benenson AS, Genovese EN. The Thucydides syndrome: Ebola deja vu?
(or Ebola reemergent?) *Emerg Infect Dis* 1996; 2:155-6.

Olson PE, Benenson AS, Genovese EN. Ebola/ Athens revisited. *Emerging Infectious Disease*
Journal 1998; 4: 1-3.

Page DL. Thucydides' description of the great plague. *Classical Quart* 1953; 3:97-119.

Papagrigorakis MJ, Yapijakis C, Synodinos PN, Baziotopoulou-Valavani E. DNA examination of
ancient dental pulp indicates typhoid fever as a probable cause of the Plague of Athens.

- Int J Infect Dis* 2006; 10: 206-214.
- Parry A. The Language of Thucydides' Description of the Plague of Athens. *BICS* 1969; 16:106-118
- Perry RD, Fetherston JD. *Yersinia pestis*: the agent of the plague. *Clin Microbiol Rev* 1997; 10:35-66.
- Plutarch, The Life of Lycurgus, trans. John Dryden. *The Internet Classics Archive*; 1683.
- Poole JCF, Holladay AJ. Thucydides and the Plague of Athens. *Classical Q* 1979;29:282-300.
- Retief FP, Cilliers L. The epidemic of Athens, 430-426 BC. *SAMI* 1998; 88: 50-53.
- Rolleston JD, *The History of the Acute Exanthemata*. London; 1937: 49.
- Rostovtzeff M. *The Social and Economic History of the Hellenistic World*. Oxford 1941; I 95.
- Sallares R. *Malaria and Greek History*. Manchester, England: Manchester University Press; 2002.
- Sallares R. *The Ecology of the Ancient Greek World*. Ithaca, NY: Cornell University Press; 1991.
- Salway P, Dell W. Plague at Athens. *G & R* 1955; 24:62-70
- Sanjuan R, Nebot M, Chirico N, Mansky LM, Belshaw R. viral Mutation Rates. *J. Virol* 2010; 84: 9733-9748.
- Scarborough J. Thucydides, Greek medicine and the plague at Athens: a summary of possibilities. *Episteme* 1970;4: 77-90
- Scarrow GD. The Athenian plague: a possible diagnosis. *Ancient Hist Bull* 1988; 2:4-8.
- Secunda N. *The Spartan Army*. Great Britain: Osprey Publishing Limited, 1998: 20-29.
- Shapiro B, Rambaut A, Gilbert TM. No proof typhoid caused the Plague of Athens (a reply to Papagrigorakis et al.). *International Journal of Infectious Disease* 2006; 10: 334-335.
- Shrewsbury JDF. The plague of Athens. *Bull Hist Med* 1950;24:1-24.

The Peloponnesian War. 2004. Encyclopaedia Britannica Online. 29Apr2013

<<http://www.thelatinlibrary.com/historians/narrative/peloponnesianwar1.html>>

Theodorides J. The plague of Athens. *Journal of the Royal Society of Medicine* 1995; 88: 363.

Thucydides. *The History of the Peloponnesian War*. R Warner, trans. London, England: Penguin Books Ltd; 1954 and 1972

Thucydides. *The Landmark Thucydides: A Comprehensive Guide to the Peloponnesian War*. R Crawley, trans. New York, NY: Simon & Schuster; 1996; *Touchstone Books*

Thucydides. *Thucydides: the History of the Peloponnesian War*. Vols I and II. CF Smith, trans. Boston, MA: Harvard University Press; 1919 and 1921; *Loeb Classical Library*

Understanding evolution. 2013. *University of California Museum of Paleontology*. 12Feb2013
<<http://evolution.berkeley.edu/>>.

Weiss E. In: Lederberg J, editor. *Encyclopedia of microbiology*, Vol. 3. San Diego, USA: Academic Press; 1992. p. 585-610.

Williams EW. The sickness at Athens. *Greece and Rome*, xxvi 1957: 98-103.

Wylie JH, Stubbs HW. The Plague at Athens 430-428 BC: Epidemic and Epizootic." *CQ* 33:6-11.

Xenophon. *Xenophon in Seven Volumes*, 7. E. C. Marchant, G. W. Bowersock, tr. *Constitution of the Athenians*. Harvard University Press, Cambridge, MA; William Heinemann, Ltd., London. 1925.

Zinsser H. *Rate, Lice and History*. Boston, MA: Little, Brown and Company; 1934.