

May 2020

Applying Modern Immunology to the Plague of Ancient Athens

Juhi C. Patel

University of Tennessee, Knoxville, jpatel36@vols.utk.edu

Follow this and additional works at: <https://trace.tennessee.edu/pursuit>



Part of the [Ancient History, Greek and Roman through Late Antiquity Commons](#), [Disease Modeling Commons](#), and the [Epidemiology Commons](#)

Recommended Citation

Patel, Juhi C. (2020) "Applying Modern Immunology to the Plague of Ancient Athens," *Pursuit - The Journal of Undergraduate Research at The University of Tennessee*: Vol. 10 : Iss. 1 , Article 7.

<https://doi.org/10.7290/pur10mkys>

Available at: <https://trace.tennessee.edu/pursuit/vol10/iss1/7>

This article is brought to you freely and openly by Volunteer, Open-access, Library-hosted Journals (VOL Journals), published in partnership with The University of Tennessee (UT) University Libraries. This article has been accepted for inclusion in Pursuit - The Journal of Undergraduate Research at The University of Tennessee by an authorized editor. For more information, please visit <https://trace.tennessee.edu/pursuit>.

Applying Modern Immunology to the Plague of Ancient Athens

Cover Page Footnote

The author would like to thank Dr. Aleydis Van de Moortel at the University of Tennessee for research supervision and advice.

1.1 Introduction.

After the Persian wars in the early fifth century BC, Athens and Sparta had become two of the most powerful city-states in Greece. At first, they were allies against the common threat of the Persians. However, in the aftermath of the Persian wars, political disagreements between the two leading powers led to the formation of two opposing groups: the Delian League and the Peloponnesian League. The Delian League led by Athens was a sea-based naval power, and the Peloponnesian League led by Sparta was a land-based power with a formidable army (Rhodes 1988, 23). Our main written source for the war between those two confederations is the late-5th-century-BCE Athenian historian and eye-witness Thucydides, who wrote the *History of the Peloponnesian War*. Thucydides tells us that in 431 BC, Pericles, then the leader of Athens, devised a strategy for dealing with the superior Spartan land army by bringing a large part, if not all, of the rural population of Attica into the city walls of Athens and its harbor Piraeus. With this action, Pericles surrendered the Attic countryside to Spartan raiding, and Athens became de facto a besieged fortification. However, he maintained control of the harbor Piraeus to safely import food and other necessities by ship to supply the Athenian population within the city walls. Athens and Piraeus were connected by the Long Walls, which protected the road from the harbor to the main city and ensured the Athenians safe access to the port of Piraeus.

When the refugees from the Attic countryside came into the city, they had to find shelter within the walls of the city. The influx of so many refugees caused overcrowding, which contributed to poor sanitary conditions (Th. 2.17.1). Already in the second year of the war, in 430 BC, a devastating epidemic disease broke out in Athens. Despite the presence of a first-hand account from Thucydides with details of the disease, modern historians have been unable to come to a consensus about the identity of the pathogen responsible for the epidemic.

The present study will examine the different factors involved in the spread of the plague in ancient Athens at the beginning of the Peloponnesian war and investigate how the refugee crisis caused by the military strategy of Pericles affected the rapid spread of the plague. Nowadays, countries experiencing high rates of urbanization are also suffering high rates of epidemic diseases. Many of these regions have poor living conditions with overcrowding and insufficient sanitation systems. Now that we know more about the causes of epidemic diseases in overcrowded conditions, we can use this knowledge to understand the conditions in ancient Athens during the plague and devise simple protocols for slowing down or preventing the spread of the disease.

The main written primary source I am using in the present study is *The History of the Peloponnesian War* by Thucydides. Other important primary evidence is archaeological: skeletal remains claimed to be of plague victims,

physical remains of houses, public buildings, water supply systems, sanitation systems, burial sites, etc. In addition I am consulting a variety of secondary sources that discuss population size and living conditions in Athens before and during the war, the factors that may have influenced the spread of the disease, and the potential identification of the disease. Using the most probable identifications of the Athenian plague, I will use a function derived from an SIR model (“S” representing the number of susceptible people, “I” the number of infections, and “R” the number of recovered or immune people) used in modern immunology to compare the conditions of the spread of these modern diseases to the conditions in Athens during the plague. In modern times, standard immunological protocols have been developed for a number of epidemic diseases that have been suggested as candidates for the Athenian plague, and I will apply these hypothetical protocols to data for ancient Athens to see how they could have slowed down or stopped the spread of the disease.

1.2 The Plague of Athens.

Thucydides reports that Athens suffered the worst from the unknown disease, which affected anyone, no matter whether healthy or sick. He reports the following symptoms of the disease: headache, red eyes, red throat, bad breath, sneezing, hoarseness, coughing, vomiting, convulsions, body blisters, fever, thirst, restlessness, sleeplessness, bowel ulcers, diarrhea, gangrene, memory loss, the presence of an animal reservoir, and immunity (Th. 2.49.2-13). Based on the symptoms provided by Thucydides, modern historians had focused primarily on diseases such as smallpox, measles, and typhoid fever as potential identifications of the plague.

In 2006 researchers used a mass grave with 150 bodies discovered in the cemetery of Kerameikos to conduct molecular DNA testing on the dental pulp of three teeth selected randomly from the bodies in the tomb (Papagrigorakis et al. 2006). Using suicide PCR, which stands for Polymerase Chain Reaction, and primers of seven different diseases, they amplified the genes of interest. The tested diseases were plague, typhus, typhoid fever, anthrax, tuberculosis, cowpox, and cat-scratch disease. By using a sample of microbial DNA from the teeth together with an enzyme that synthesizes DNA and a microbe-specific primer that signals the enzyme where to begin synthesizing new DNA, they are able to amplify the genes of interest (NCBI 2017). Once a single gene from the target DNA has been amplified, its genome is sequenced and inserted into the GenBank® sequence database. The database compares the isolated sequence to all known sequences available, and provides a list based on base pair similarity. In this study, the researchers found a 93% similarity in the *narG* gene to modern *Salmonella enterica* serovar *Typhi*, which causes typhoid fever. They knew that it could not be the

modern strain of this disease because there was not a 100% homology between the base pairs. They explain the 93% similarity by suggesting that there may have been a mutation of the strain over time. Indeed, such mutation was indicated by genetic sequencing of the ancient *narG* gene which showed the presence of 28 base pair changes, 25 of which were in the final codon. The changes in the final codon do not change its identity, so there are no biological consequences. However, the three mutations that are not in the final codon likely resulted in more significant changes. In fact, genetic testing into the examination of *S. typhi* genome 5% of the *S. typhi* genome has been inactivated by the presence of pseudogenes, which is indicative of significant biological changes, suggesting that the bacterial genome has mutated to better adapt its pathogenesis. Over time, genetic mutations may have allowed *S. typhi* to reduce its routes of invasion and focus on single human infection. (Wain et al. 2002: 165). This may explain why modern typhoid fever does not affect animal reservoirs, whereas it may have done so in ancient Athens, where Thucydides recorded animal infection by the plague.

Cross-examination of the primary evidence provided by Thucydides and the primary archaeological evidence provided by Papagrigorakis et al. highlights a key discrepancy- the animal reservoir. The simple experiment performed by Papagrigorakis et al. makes it difficult to pinpoint the reason behind this difference. It is clear from their discussion that Papagrigorakis et al. did not compare the obtained DNA with more than seven pathogens. They simply stopped the study once they received a positive result, presumably because this type of study was very expensive at the time. However, the available database of pathogens has since expanded significantly, and it has become much cheaper and more efficient to run PCR.

Thus, although an old strain of typhoid fever was most likely the causative agent that killed the Athenians whose teeth were analyzed, the results of this research are not adequate to definitively identify the disease. The experiment should be repeated with a larger sample size, and the genome(s) should be compared to more than the seven tested pathogens. We should at least consider the other diseases discussed by historians based on the similarity of their symptoms to those described by Thucydides. The ideal way to do this would be to sequence the entire genome of the DNA extracted from the teeth, and not just one gene at a time as they have done. However, this would be a very expensive, multi-million dollar, project.

Since the DNA evidence is somewhat inconclusive, we can use Thucydides' list of symptoms and compare these with the symptoms of known diseases. The following table lists other possible identifications of the plague considered by modern historians (Table 1). Some diseases such as bubonic plague can be eliminated immediately, as we know that the reservoirs for the disease--rats--were most likely not present in ancient Greece during this time period (Vigne 1994). In

the following sections, I will choose three diseases-- typhoid, measles, and smallpox-- which were selected on the basis of their greatest similarity to Thucydides' description of the symptoms to conduct the SIR modeling. One key difference is these three diseases, unlike the Athenian plague, do not affect an animal reservoir. The frequency of person- to-person and person-to-animal contact must have increased enormously within the walls of Athens at the time of the outbreak of the war due to the increase of inhabitants as a result of Pericles' strategy. The following section will discuss the spike in population density and provide estimates for the size and density of Athens' population after the influx of refugees.

Common name	Microbial agent	Mode of Transmission	Symptoms	Incubation Period	Length of Symptoms	Reservoir	Lethal	Researchers	Sources
Plague of Athens	Unknown	Contact with infected person, plus more unidentified	headache, red eyes, red throat, bad breath, sneezing, hoarseness, coughing, vomiting, convulsions, body blisters, fever, thirst, restlessness, sleeplessness, bowel ulcers, diarrhea, gangrene, memory loss	Unknown	most succumbed after 7-9 days (stages 1-2)	dogs, birds	yes	M.J. Papaioannidis et al 2006	Thucydides
Typhoid	Salmonella enterica serovar typhi	contaminated food/water, from infected person	fever, headache, weakness, stomach pain, diarrhea, cough, rash, loss of appetite, delirium	1-3 weeks	3-4 weeks	humans	yes	J.F.D.Shrewsbury	CDC, Mayo Clinic
Measles	Rubeola virus	droplet/airborne	mild fever, cough, runny nose, red/watery eyes, sore throat, red spots, rash, high fever	7-14 days	6-13 days	humans	yes- pre-vaccine	R.J.Littman & M.Littman (1969)	CDC and WHO
Smallpox	Varicella virus	direct and prolonged face-to-face contact	high fever, head and body aches, vomiting, rash sores in mouth, pustules over body	10-14 days	22-24 days	Humans	yes- 3/10 died	C.H.Eby & H.D.Ejlen (1962)	CDC
Glanders	Burkholderia mallei	from animals, contaminated water	fever with chills, aches, chest pain, headache, ulcers, diarrhea	1-5 days	several weeks	animals (horses)	yes- if in bloodstream	P.Salway & W.Dell (1955)	CDC
Ergot Toxin	Claviceps fungi	consumption	vomiting, fever, burning, pain, weakness, gangrene, convulsions, hallucinations	N/A	N/A	none	sometimes	J.A.H. Wylie & H.W.Stubbs	Miedaner 2015
Leptospirosis	Leptospira bacteria	Contact with infected animal urine or contaminated water	High fever, headache, chills, muscle ache, vomiting, jaundice, diarrhea, rash, red eyes	2 days to 4 weeks	few days to 3 weeks	Animals: cattle, pigs, dogs, rodents	rare		CDC
Lassa Fever	Lassa virus	Ingestion or inhalation, person to person	fever, malaise, weakness, headache, sore throat, diarrhea, cough, vomiting, pain, shock, seizures	1-3 weeks	14 days	Multimammate rat	yes- rarely	J.M.H.Hopper (1992)	WHO
Alimentary Toxic Aleukia	Contaminated wheat	ingestion	leukopenia, chest pain, rash, sepsis, bleeding from nose and mouth	15 minutes (mycotoxins)	4.5 hours	animals	sometimes	J.Bellemore, I.M.Plant & M.Cunningham	WHO and Lutsky
Epidemic Typhus	Rickettsia prowazekii	ingestion	fever and chills, body aches and pain, nausea, vomiting, stomach pain, cough, rash, gangrene	within 2 weeks	2 weeks	humans	Yes	W.P.MacArthur (1954)	CDC and WHO
Anthrax	Bacillus anthracis	airborne, contact with contaminated spores	mild fever, sore throat, fatigue, ache, chest pain, cough, nausea, high fever, shock, meningitis	1 day to 2 months	variable	livestock and game animals	yes		CDC and Mayo Clinic
Tuberculosis	Mycobacterium tuberculosis	droplet	coughing, chest pain, fatigue, fever, chills, sweats, weight loss	2-12 weeks	weeks to months	humans	yes		CDC and Mayo Clinic
Bubonic Plague	Yersinia pestis	bite of infected fleas	Sudden onset of fever, headache, chills, weakness, swollen lymph nodes	1-7 days	As short as 24 hours	fleas, rodents (rats, mice)	yes		CDC and WHO
Cat-scratch disease	Bartonella henselae	bite/scratch from infected cat	skin swollen and red with lesions, fever, headache, poor appetite, exhaustion	3-14 days	2-4 months	cats, fleas	no		CDC and NCBI

Table 1. Hypotheses for the identification of the plague of Athens. This table lists diseases discussed by modern historians and scientists. The first row lists the symptoms provided by Thucydides. Symptoms in common with Thucydides' description are in bold print. The stages of the symptoms are differentiated by a color change, i.e. the first stage is represented in blue, the second in green, the third in orange, and the fourth in yellow.

1.3 Population Densities in the Walled Areas of Athens and Piraeus.

1.3.1 Population Estimates of Ancient Athens, ca 431 BCE.

The polis of Athens encompassed the whole of Attica (around 2,527 km²), and was much larger than the walled areas of Athens and Piraeus (Morris 2005:15). Our primary source for the population size of the Athenian state is Thucydides, and modern historians disagree on how to interpret population data. This is why it has been difficult for modern historians to come to a consensus about the exact size of the population of the walled area of Athens and Piraeus after the refugee influx in 431 BCE.

Modern historians have used various methods to determine population size based on passages by Thucydides and other historians. In particular, Thucydides gives data about the numbers of land soldiers and triremes (ancient war ships) available to Athens at the beginning of the war in 431 BCE. In a speech to the Athenian assembly trying to bolster their morale as the war broke out, Pericles mentions that there were 13,000 hoplites of the active army and 16,000 hoplites on home duty to defend Athens. In addition, he mentions that there were 1200 members of the cavalry, 1600 archers on foot, and 300 seaworthy triremes (Th. 2.13.6-8; Table 2 below).

A prominent modern historian, A. W. Gomme, used those military data as the foundation for his estimate. Adding together the number of male citizens, women, and children (172,000), metics (28,500), and slaves (115,000) gives a total of 315,500 people living in the Athenian polis in 431 BCE (Gomme 1933: 21, 26; Table 2). A higher estimate of 353,500 people can be proposed on the basis of estimates by Hansen (1988). The lowest reasonable estimate 292,950 people can be proposed on the basis of calculations by van Wees (2004: 241-243), who references Thucydides and Hansen in his analysis. The calculations for each estimate are shown in Table 2. Having reviewed the various population estimates of the Athenian polis in 431 BCE, I believe that it is reasonable to work with simplified estimates of 300,000, 350,000, and 400,000 inhabitants.

	Thucydides (2.13.6)	Gomme	Hansen	van Wees	Morris
Hoplites (both citizens and metics, old and young)	29,000 (13,000 in active duty and 16,000 in the garrisons and home-guard)	29,000 (subtracted 4000 to account for metics)	60000 male Athenian citizens, including citizens unfit for military service and above military age	23,700	
Cavalry	1200			1000	
Bowmen	1600				
Trireme Crew	44,400 (based on an estimate of 180 seaworthy fighting triremes and 120 transport triremes); moreover, there were other ships in the Athenian navy not mentions by Thucydides				
Thetes		18,000		(18,000)	
Women and children (and fighting men)	76,200 x 3 = 228,600 (also includes metics)	(25000+18000) x 4 = 172,000	60000 x 3.5 = 210000	(24,700+18,000) x 3.5 = 149,450	
Metic men	(included in above calculation)	9,500 (hoplite soldiers and thetes)			
Metic women and children (and men)	(included in above calculation)	(9500) x 3 = 28,500	28,500 adopted from Gomme	28,500 adopted from Gomme	
Slaves	115,000	115,000	115,000 adopted from Gomme	115,000 adopted from Gomme	
Total	343,600	315,500	383,500	292,950	
Population in Athens & Piraeus		155,000			60,000 to 65,000

Table 2. Estimated population sizes of the Athenian polis. The table lists population calculations based on data provided by various historians. The numbers listed in bold have been used in the calculation of the grand totals.

Another issue of importance is what percentage of this total population would have lived inside the walled areas of Athens and Piraeus before the refugee crisis of 431 BCE. Later ancient historians such as Ian Morris estimate the population of the walled city of Athens as between 35,000 to 40,000, and the population in Piraeus as 25,000 (Morris 2005: 15). These population estimates are supported by scant published archaeological evidence of house sizes in Athens. The median 50% of houses in the mid-5th century BCE throughout Greece reported by Morris (2004) range from 110 sq. m to 180 sq. m. If we use the median of this

range, 145 sq. m as the average house size, that means that up to 8275 houses could have fit in the domestic area of 120 hectares in Athens, with 4.2 to 4.8 people per household (Morris 2004: 772). John Travlos, on the other hand, must have envisioned a larger average house size in Athens. He states that Athens held up to 6000 houses with around 36,000 occupants, resulting in 6 people per house (Travlos 1971: 72). A typical family of 6 would have included parents, an average of two children, and one or two slaves or an elderly family member.

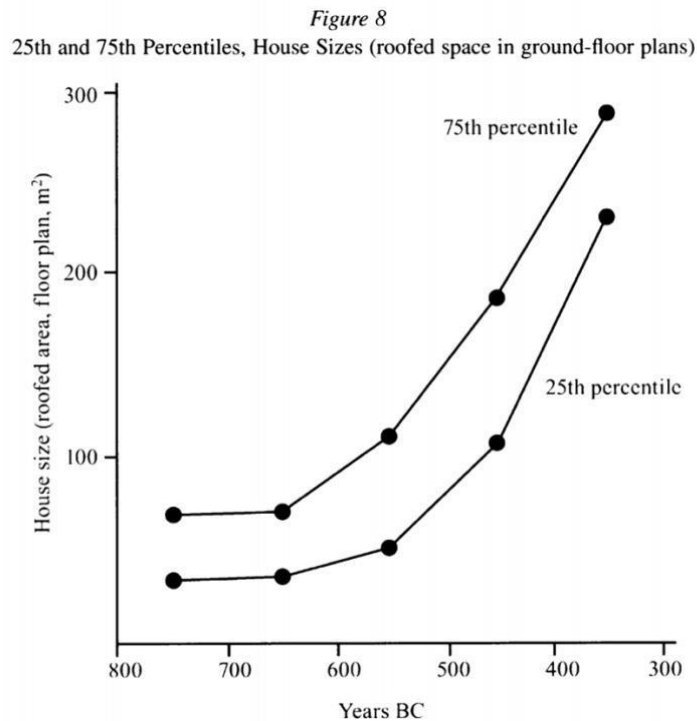


Figure 1. Evolution of house sizes in Athens. In the mid-5th century BCE the median 50% of houses ranged between 110 m² and 180 m² (Morris 2004: fig.8).

1.3.2 Area Calculation of Walled parts of Athens and Piraeus.

The population densities in the walled areas of Athens and Piraeus both before and during the outbreak of the war and the plague must be calculated. The first step is to calculate the area inhabited by both residents and refugees within Athens and Piraeus. Morris stated that the walled area of Athens encompassed 215 hectares, of which only 120 hectares was used for domestic settlement (Morris 2005: 15). My measurements show that an additional 3 hectares were occupied by the Acropolis,

and another 2 hectares can be estimated for enclosed sacred areas, which according to Thucydides were off-limit to the arriving refugees (Th. 2.17.1). This would have left approximately 90 hectares available for refugees in Athens. As for the other walled areas, Meera Patel calculated the total area of Piraeus as 473 hectares and the area between the Long Walls as 191 hectares (Patel 2017: 18). Compared to Athens, a much smaller area of Piraeus, around 60 hectares in the middle of the town, would have been used for domestic settlement, as some evidence for roads and houses has been found there (Fig. 3; Wycherley 1978: 263). In addition, we can estimate that another 80 hectares were taken up by harbor installations. The southern part of the Akte peninsula, ca. 150 ha, remained outside of the walls, however, and was therefore unguarded, so it is unlikely that refugees settled in this area. This would have left 150 hectares of open area for refugee settlement in the northern half of the Akte peninsula. In addition, refugees may have settled in other uninhabited areas in the north, which covered another 33 hectares. In total there were approximately 183 hectares available for refugees in Piraeus.

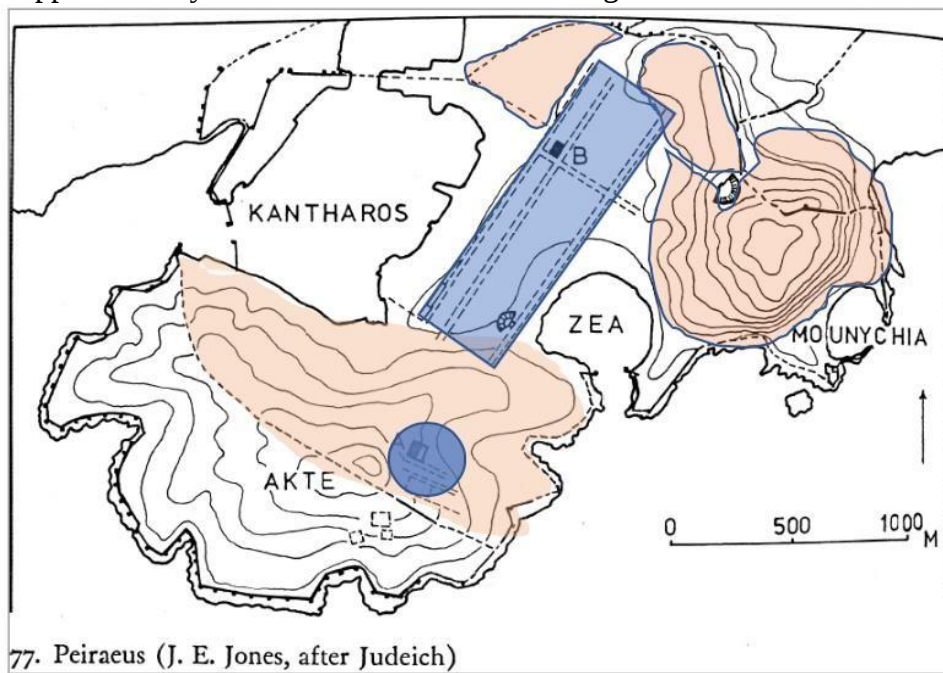


Figure 2. Map of Piraeus with the estimated area of habitation. The areas in orange represent those available for refugees, and the areas in blue represent those used for domestic settlement (Wycherley 1978: 264).

The total area between the Long Walls that ran between Athens and Piraeus was measured as 191 hectares, but since there was a deme called Xypete located in this area, I roughly estimate that only 90%, or 172 hectares, were open for refugee

settlement (Traill 1975: Map 1). These numbers (90+183+172) give a total of 445 hectares available for refugee settlement in Athens and Piraeus, and between the Long Walls. In all those areas, 199 hectares (120+60+19) would have been used for domestic settlement.

According to Morris' estimate, before the refugee crisis, around 65,000 people occupied an area of 644 hectares in Athens and Piraeus. This gives a population density of 101 people per hectare. In order to estimate the population densities of the walled areas during the refugee crisis we will use the minimum and maximum population estimates of 300,000 and 400,000, and make calculations for two scenarios: one that assumes that 50% of the Attic population entered the walled area and one that assumes that 75% of the population came into the urban areas. The estimates are provided in Table 5 below.

	Domestic Settlement	Refugee Settlement	Other buildings	Total Area
Athens	120 hectares	90 hectares	5 hectares	215 hectares
Piraeus	60 hectares	183 hectares	80 hectares- harbor 150- unoccupied	473 hectares
Long walls	19 hectares	172 hectares		191 hectares
Total Area	199 hectares	445 hectares	235 hectares	879 hectares

Table 3. Area calculations of domestic and refugee settlements at Athens and Piraeus (after Morris 2005: 15; Wycherley 1978: 264).

	Area	Population	Density
Athens Piraeus Long Walls	199 ha settlement	65,000	326 people/ha
Athens Piraeus Long Walls	644 ha total	65,000	101 people/ha

Table 4. Domestic population density in the walled areas of Athens and Piraeus before the war.

Population increase	Area	Population	Density
50% increase of 300,000	445 ha	117,500 people	264 people/ha
75% increase of 300,000	445 ha	176,250 people	396 people/ha
50% increase of 400,000	445 ha	167,500 people	376 people/ha
75% increase of 400,000	445 ha	251,250 people	564 people/ha

Table 5. Refugee population density during plague years. The minimum population is based on an estimate that 50% of the Attic population of 300,000 entered the walled area of Athens-Piraeus. The maximum population is based on an estimate that 75% of the Attic population of 400,000 entered the walled area of Athens-Piraeus.

If we accept the maximum population estimate, the population density more than quadrupled during the war and plague. With such a large population density, it is likely that diseases with fast transmission rates would have spread much too quickly. On top of the overcrowding, another potential factor involved in the spread of the disease is the fact that Athens was dependent on a simple sanitation system that was overtaxed during the population influx.

1.4 Athens' Strained Sanitation Systems during the Outbreak of the Plague.

Thucydides tells us that as the plague entered Attica, it first affected the residents of the port of Piraeus. The Athenians at first believed that the water reservoirs had been poisoned (Th. 2.48.2). Ancient Athens relied on structures such as wells, cisterns, and aqueducts to supply water, and they used cesspools for waste disposal (Wycherley 1977: 240). Although simple, these systems worked well for the population of Athens before the war. However, these systems were strained under the influx of refugees at the beginning of the Peloponnesian war, as they had not been constructed to support such a large population.

Thucydides clearly states that the plague was the worst in the most populated areas, indicating that the refugees from the rural countryside must have suffered more than the urban population (Th. 2.52.1). Unlike the citizens of the city,

most refugees did not have access to houses and had to live in crowded huts and shacks (Th. 2.17.2-4). These overcrowded conditions must have taxed the water supply systems. Athens relied mostly on wells and cisterns for the private residential supply of water (Camp 1977: 106). The Athenians used channels cut into rock to bring water from mountains to the city and terracotta pipes to run water to public buildings (Wycherley 1978: 250). These water supply systems were sufficient for the pre-war population of the walled areas, and would have been strained by overcrowding.

Not only did the people have to bring water into homes, but they also needed a system to remove waste from their homes. Athens during this time had a crude sanitation system consisting of cesspools to dispose of liquid wastes (Wycherley 1978: 240-41). Athenians also tended to leave garbage behind in the streets, aggravating disease outbreak, as wastes are a breeding ground for disease-causing organisms (Adorni and Giannelli 1970: 39). It was not uncommon for people to use a garden or the street as a toilet, further risking the spread of disease through feces, which harbor bacteria (Wycherley 1978: 251; Adorno and Giannelli 1970: 47). Also, the refugees from the rural countryside likely did not follow the same sanitation etiquette as those from the city. Moreover, Thucydides says that people became careless during the plague, and some patients even jumped into cisterns to seek relief from their hot fevers (Th. 2.49.5; 2.52.3).

Another factor that would have strained sanitary conditions during Athens' refugee crisis was the burial of human bodies. By law, Athenians conducted all burials outside of the city walls. This rather sanitary system must have weakened during the war, as it was difficult for Athenians to travel outside of the walls to dispose of the dead bodies while under siege by the Spartans (Wycherley 1978: 253). Before the war and the plague, Hansen estimates that the yearly mortality rate in Athens was around 2.5% (Hansen 1988: 21). If we use an estimated population size of 65,000 in Athens and Piraeus before the war, a mortality rate of 2.5% would mean that around 135 people died each month. This number increased enormously during the first three years of the plague, when the total estimated mortality was 25-33% of the population within the walls (Hansen 1988: 21, Sherman 2017: 55). If we estimate the total population within the walls during the refugee crisis as averaging 280,000 and the average mortality rate as 29%, that means that around 2,255 people died per month, an overwhelming increase from 135 people per month before the plague.

Normally, Athenian burial practices involved much contact with the dead. These customs increased the risk of infection, as both typhoid fever and smallpox can still be carried and spread by a dead body (Kurtz and Boardman 1971: 144). During the time of the plague, extensive contact with both the home of the deceased and the body of the deceased could easily have spread the pathogen causing the plague. Proper burial would have removed the body from the area of living, limiting

the exposure to the disease carried by the body. The problem was that during Spartan invasions, Athenians could not go outside the walls to bury the dead. Moreover, Thucydides tells us that as the plague took its toll, people began to neglect burial practices because the number of dead was too high, and bodies of the plague victims were often left unburied in the buildings and streets (Th. 2.52.3).

As this brief overview has shown, the basic water supply and waste management systems as well as burial practices that existed in 5th-century-BCE Athens were insufficient to cope with the large numbers of refugees that flowed into the city at the beginning of the war. Once the plague took hold and the dead toll mounted, people began to abandon proper burial practices and left the dead in the street, creating a fertile ground for infection by the plague.

1.5 Using SIR Modeling to Study the Spread of Infection during the Athenian Plague.

The population within the walled area of the city increased enormously during the first year of the Peloponnesian war, and most refugees were crammed together without access to adequate housing. My estimated minimum and maximum population sizes in the refugee quarters are 117,500 people and 251,250 people, respectively. These were in addition to the 65,000 permanent residents of the walled areas. In present-day immunology, a widely used method for studying and predicting the spread of infectious diseases is called SIR modeling. The model can be used to create simulations of hypothetical outbreaks and design measures to prevent or mitigate the spread of disease.

SIR models can be built as complex or as simple as needed. Due to time constraints I utilized a very specific and simple formula proposed by Rhodes and Anderson (2008) to calculate the basic reproductive number (R_0) for a disease, which is the maximum number of secondary infections that can be caused by a single infected individual in a constant population. The formula used is $R_0 = \frac{8Rp\bar{v}\rho}{\pi\alpha}$, where R = the radius (in km) of the area in which an infected person can transmit a disease to another person; p = the transmission probability of infection given contact with an infected individual; $\bar{v}$ = velocity (in km/day) of the infected individual passing through throughout the space inhabited by the population; ρ = the population density (in people/km²), and α = the infectious period (in days). This value of R_0 only gives the hypothetical number of infections that can be caused by one single individual; since the transmission of an infectious disease involves many complex parameters, R_0 does not accurately model the true exponential spread of the disease.

I focus my SIR model on three of the most likely identifications of the Athenian plague: typhoid fever, measles, and smallpox. I use as much as possible data from before the 20th-century worldwide vaccination campaigns, which considerably reduced infection rates. For typhoid fever, a (p) of 10% was adopted from a report on the medical history of the South African Anglo-Boer War from 1899-1902. During this war, a typhoid fever outbreak occurred within a static camp during the war. Out of the 556,653 men who served in the British Forces, 57,684 were infected by typhoid fever. (Villiers 1981). Due to the variability in the length of typhoid fever symptoms and infectivity, for the purposes of the model, an infectious period of 7 days, or the average length of the first stage of symptoms, will be used (CDC 2017).

As for measles, the CDC states that the transmission rate for unvaccinated people is as high as 90%. The mean infectious period for measles is reported as 8 days by the Mayo Clinic. For smallpox, data are used that were collected during a smallpox eradication campaign during the 1960's. The transmission rate in rural Afghanistan in 1969 was 50% in the case of a susceptible patient living in the house, which is applicable in ancient Athens, where there were no hospitals and the ill were treated at home (Meltzer et al. 2001). The WHO reports that most infectious period for smallpox is during the first week, which is why a period of seven days is used (2016).

For the susceptible population (S), the numbers used are those calculated previously in the section about population densities, but these are converted to people/km² (by multiplying by 100) to fit the model. Although the absolute minimum estimate that was obtained was 264 people/ha which represents an influx of only 50% of the rural population assuming a minimum population size for the entire polis of Athens, this was not included here because it is lower than the density of the residential area, and thus unlikely; for Thucydides explicitly mentions that the refugee areas were more crowded than the residential areas (Th. 2.17.1).

A contact radius (R) of 0.002 km, and an average velocity of an infected person of 2 km/day are used for the SIR calculation; both have been adopted from Rhodes and Anderson (2008), who do not provide a specific rationale for those values. In my own experience of volunteering at a hospital in the U.S., a radius of 6 feet is considered as the radius for infectivity, which corresponds to 0.002 km. The table below shows my calculations using the equation by Rhodes and Anderson, which gives a numerical value to represent the maximum number of secondary infections that can be caused by one infected individual.

Residential area (65,000 people in 199 hectares)						
	P- transmission probability	ρ - population density (people/km)	α - infectious period (days)	R- radius to become infected (km)	v- velocity (km/day)	R_0 (people)
Typhoid	0.1	32600	7	0.002	2	4.74373077
Measles	0.9	32600	8	0.002	2	37.3568798
Smallpox	0.5	32600	7	0.002	2	23.7186538
Refugee area (176,250 people in 445 hectares)						
	P- transmission probability	ρ - population density (people/km)	α - infectious period (days)	R- radius to become infected (km)	v- velocity (km/day)	R_0 (people)
Typhoid	0.1	39600	7	0.002	2	5.76232326
Measles	0.9	39600	8	0.002	2	45.3782957
Smallpox	0.5	39600	7	0.002	2	28.8116163
Refugee area (251,250 people in 445 hectares)						
	P- transmission probability	ρ - population density (people/km)	α - infectious period (days)	R- radius to become infected (km)	v- velocity (km/day)	R_0 (people)
Typhoid	0.1	56400	7	0.002	2	8.20694525
Measles	0.9	56400	8	0.002	2	64.6296939
Smallpox	0.5	56400	7	0.002	2	41.0347263

Table 6. Secondary rate of infection caused by a single infected person. Rhodes and Anderson's (2008) proposed equation $R_0 = \frac{8Rpv\rho}{\pi\alpha}$, used to model the transmission of typhoid, measles, and smallpox at various population estimates.

The resulting values can be used to analyze the transmission of the plague. The basic reproductive numbers are significantly higher when comparing the minimum and maximum population density estimates in the refugee areas. The model used above highlights the stark difference made by refugee influx in the disease transmission, as Thucydides described it (Th.2.17.1).

The numbers for R_0 in the figure above only give the numbers of infectious cases produced by one infected person. To illustrate the devastation of the plague, whichever of the three proposed diseases it was, it is useful to construct a hypothetical scenario. Imagine a ship carrying ten men infected with typhoid fever. Once they landed in Piraeus, where Thucydides tells us the plague started, each infected man would have been capable of infecting on average 6 other people (R_0). Although R_0 is not a function of time, we can assume that these infections occurred over the total infectious period of seven days. This is known as a generation. After one generation, the original ten men have infected sixty others. After 6 weeks the infection would have reached 466,560 people if allowed to spread without impediment. These hypothetical calculations indicate that typhoid fever would have reached epidemic proportions in ancient Athens in a span of 4-6 weeks. In the case

of measles, the disease would have reached epidemic proportions in just over two weeks. In the case of smallpox, this would have occurred in three weeks.

The reproductive numbers of typhoid fever compare well to Thucydides' description of the long, large-scale devastation wreaked by the Athenian plague. The reproduction rates of measles and smallpox seem much too high to continue a three-year long epidemic. Thus my SIR modeling shows that typhoid fever is a much more likely candidate than measles or smallpox as the cause of the Athenian plague, supporting the identification obtained through DNA analysis.

1.6 Advice from Modern Immunology: Measures to Prevent the Spread of the Athenian Plague.

Even though people with medical training, such as Thucydides, noticed increased infection rates in areas with the greatest population densities (Th. 2.52.1), ancient Athenians did not understand the principles of infection or the ways of controlling infection that we know today, and this led to an increased infection rate from person-to-person. Had the Athenians understood that infectious diseases spread through bacteria or viruses, they could have taken various measures to slow down or stop the spread of the disease.

Typhoid fever is the most likely candidate on the basis of the archaeological evidence, as it was actually found in the dental remains of potential plague victims excavated at Athens. Typhoid fever, measles, and smallpox have different methods of transmission, therefore different factors must be considered when studying the spread of the disease. Thucydides tells us that the plague was the worst in the most densely populated areas and that those who nursed the sick experienced the highest level of mortality (Th. 2.51.4). Nowadays, organizations like the WHO and the CDC highlight infection control protocols for disease outbreaks. In case of an incident of typhoid fever for example, a Rapid Response Team would investigate the patient's history to find the source of infection, search for any other potential cases and carriers, and quarantine the patient until the fever has disappeared (WHO: 2011).

Another factor is patient waste disposal, as bacteria like *S. typhi*, the causative agent of typhoid fever, is excreted through feces. This contamination can occur both with patients who are alive and with dead bodies as well. This poses a problem when fecal matter is not properly disposed and comes into contact with food and water. It can also lead to contamination of water sources when rainfall comes into contact with bodies and the runoff leads into water sources. One of the symptoms of smallpox is the development of sores and scabs, which actually contain the virus. The virus can spread through items such as bedding or clothing that has been contaminated by these scabs (CDC: *Smallpox* 2016).

People who interact with the bodies of plague victims have a high risk of contracting disease. We know from literary evidence that Athenian burial customs involved heavy contact with the bodies of the dead (Kurtz and Boardman 1971: 144). The WHO outlines safety protocols for those who handle the bodies of the dead, such as undergoing training and using protective equipment such as gloves and masks (2016). In contrast, in ancient Athens, a large number of the bodies of people who had just died from the plague were not properly buried, in part because the Kerameikos cemetery was outside the walls and inaccessible during the Spartan invasions, and in part because the overwhelming number of dead caused people to abandon proper burial rites (Th. 2.52.4). If the dead had been buried in hygienic fashion, the disease would likely not have spread as quickly as it did.

Another problem must have been difficulty of access to clean water for the many refugees who had poured into the walled areas of Athens and Piraeus. The WHO has outlined simple purification techniques which could have been used by Athenians during the plague if they had a better understanding of infectious diseases. One method is to filter drinking water by means of media such as porous rock and sand. Another simple step that could have been taken is to boil water, which can kill most, if not all, waterborne pathogens.

Although they had limited technology, there are several protocols that the Athenians could have adopted to slow down the spread of the plague. A quarantine of any infected individuals and anyone who had been in contact with them would have slowed down the rate of person-to-person transmission, as infected individuals would have been able to spread the disease before they displayed any symptoms. Furthermore, all water should have been boiled to kill any contaminants from the plague or waterborne pathogens. In addition, the bodies of the victims should have been disposed of in a way that avoided any possible contamination. This included not washing the body, not touching the body with bare hands, and burying the body at least 30 m away from groundwater sources, as recommended by the WHO. By implementing these measures, the spread of the disease would have been slowed down enormously, or may even have been stopped entirely.

1.7 Conclusion

In this thesis, I sought to better understand the circumstances in the overcrowded areas as well as the identification of the disease that was responsible for this epidemic, using Thucydides' description of the symptoms as well as archaeological evidence. I then estimated possible population densities, which allowed me to apply a mathematical model to study the spread of the disease. Finally, I proposed some modern measures that could have been adopted by the ancient Athenians to contain the plague.

By creating an SIR model, I was able to compare the reproductive number of the different diseases at different population estimates. The SIR model showed that typhoid fever infection spread much more slowly than smallpox and measles. In fact, one person infected with measles was capable of infected over eight times as many people as one person infected with typhoid fever. While such rate of spread for typhoid fever compares well with Thucydides' description, the rate for smallpox and measles seems too high in order for the disease to last for three years. These calculations make it seem more likely that typhoid fever, and not measles or smallpox, was the identification of the Athenian plague. One thing is certain--a higher population density exacerbated the spread of the disease. In view of the available technology at the time, quarantine and some simple improved sanitation practices would have slowed down the spread of disease significantly.

Much remains unknown about the exact identification of the Athenian plague. Although the scientific analysis of the teeth excavated from the Kerameikos cemetery have shown that those specific individuals had been infected by typhoid fever, this does not necessarily mean that this was the one and only cause of the epidemic, as symptoms described by Thucydides do not match known symptoms of typhoid fever. More analysis of the plague victim remains is necessary to determine *all* the likely causative agent(s) of this devastating disease. If repeating the DNA analysis of the teeth, scientists should extract and amplify the entire non-human DNA genome and insert this sequence into the database in order to analyze all possible agents instead of only a few, as has been done up to now. Such a comprehensive analysis, which is expensive and out of the reach of archaeological budgets, would allow for a more definitive solution for the question of the identification of the plague of Athens, and this in turn would enable researchers to model the spread of the disease with greater accuracy.

References

Ancient Scholarship

Aristophanes, *Wasps*, ed. Transl. J. Henderson. Rev. ed. Loeb Classical Library. 1946.

Thucydides, *The History of the Peloponnesian War, Books I and II*, transl. C.F. Smith. Rev. ed. Loeb Classical Library. 1934.

Modern Scholarship

Adorno, F. and Giannelli, G., 1970. *The World of Classical Athens*, New York: Macdonald & Co.

- Andersen, K. et al., 2015. Clinical Sequencing Uncovers Origins and Evolution of Lassa Virus. *Cell*, 162(4), pp. 738–750.
- Beloch, J., 1886. *Die Bevölkerung der griechisch-römischen Welt*, Leipzig: Duncker & Humblot.
- Camp, J.M., 1977. *The Water Supply of Ancient Athens from 3000 to 86 BC*, Ph.D. dissertation, Princeton University.
- Camp, J.M., 1986. *The Athenian Agora: Excavations in the Heart of Classical Athens*, New York: Thames and Hudson.
- CDC. 2018. *Ebola (Ebola Virus Disease)*.
<https://www.cdc.gov/vhf/ebola/index.html>.
- CDC. 2016. *Glanders*. <https://www.cdc.gov/glanders/index.html>.
- CDC. 2018. *Measles (Rubeola)*. <https://www.cdc.gov/measles/about/index.html>.
- CDC. 2016. *Smallpox*. <https://www.cdc.gov/smallpox/index.html>.
- CDC. 2018. *Typhoid Fever and Paratyphoid Fever*.
<https://www.cdc.gov/typhoid-fever/index.html>.
- Curran, K.G. et al., 2016. Cluster of Ebola Virus Disease Linked to a Single Funeral—Moyamba District, Sierra Leone, 2014. *Morbidity and Mortality Weekly Report*, 65(8), pp. 202.
- de Villiers, J.C., 1984. The Medical Aspect of the Anglo-Boer War, 1899-1902 Part II, *Military History Journal*, 6(3).
<http://samilitaryhistory.org/vol063jc.html>.
- Demeny, P., and Coale, A.J., 1966. *Regional Model Life Tables and Stable Populations*, Princeton: Princeton University Press.
- Dodge, P. and Dodge, H., 1998. *The Ancient City: Life in Classical Athens & Rome*, Oxford: Oxford University Press.
- Garland, R., 2001. *The Piraeus: From the Fifth to the First Century BC*, Bristol: Bristol Classical Press.

- Gomme, A.W., 1933. *The Population of Athens in the Fifth and Fourth Centuries B.C.*, Oxford: Blackwell.
- Hansen, M.H., 1988. *Three Studies in Athenian Demography*, Copenhagen: Kgl. Danske Videnskabernes Selskab.
- Hanson, V.D., 1998. *Warfare and Agriculture in Classical Greece*. Rev. ed., Berkeley: University of California Press.
- Hornblower, S., 2009. Thucydides of Athens, *Brill's Encyclopedia of the Ancient World New Pauly*, Koninklijke Brill, Leiden, The Netherlands, pp. 632-637.
- Kurtz, D.C. and Boardman, J., 1971. *Greek burial customs*, Ithaca: Cornell University Press.
- Lederberg, J. et al., 2009. *Microbial Evolution and Co-Adaptation: A Tribute to the Life and Scientific Legacies of Joshua Lederberg: Workshop Summary*, Washington, DC.
- Littman, R.J., 2009. The Plague of Athens: Epidemiology and Paleopathology, *Mt Sinai J Med* 76, pp. 456-467.
- Martcheva, M., 2015. *An Introduction to Mathematical Epidemiology*, Boston: Springer.
- Mayo Clinic. n.d. *Measles*.
<https://www.mayoclinic.org/diseases-conditions/measles/symptoms-causes/syc-20374857>
- Mayo Clinic. n.d. *Smallpox*.
<https://www.mayoclinic.org/diseases-conditions/smallpox/symptoms-causes/syc-20353027>
- Meltzer, M. et al., 2001. Modeling Potential Responses to Smallpox as a Bioterrorist Weapon. *Emerging Infectious Diseases*, 7(6), pp. 959–969.
- Morens, D.M., Littman, R.J., 1992. Epidemiology of the Plague of Athens, *Transactions of the American Philological Association* 122, pp. 271-304.
- Morgan, T.E., 1994. Plague or Poetry? Thucydides on the Epidemic at Athens, *Transactions of the American Philological Association* 124, pp. 197-209.

- Morris, I., 2004. Economic Growth in Ancient Greece. *Journal of Institutional and Theoretical Economics JITE*, 160(4), pp. 709–742.
- Morris, I., 2005. The Growth of Greek Cities in the First Millennium BC, *Princeton/Stanford Working Papers in Classics Paper No. 120509*. <https://ssrn.com/abstract=1426835>
- Papagrigorakis, M.J., Yapijakis, C., Synodinos, P.N., Baziotopoulou-Valavani, E., 2006. DNA examination of ancient dental pulp incriminates typhoid fever as a probable cause of the Plague of Athens, *Int J Infect Dis* 10, pp. 206–214.
- Patel, M., 2018. *The Athenian Plague and the Peloponnesian War Through Thucydides' History of the Peloponnesian War*, Honor's thesis Classics, The University of Tennessee.
- National Center for Biotechnology Information. *Polymerase Chain Reaction (PCR)*, n.d., U.S. National Library of Medicine, www.ncbi.nlm.nih.gov/probe/docs/techpcr/.
- Pruss-Üstün A., Bos R., Gore F., Bartram J. 2008. *Safe Water, Better Health: Costs, Benefits and Sustainability of Interventions to Protect and Promote Health*, Geneva: World Health Organization.
- Rhodes, C.J., Anderson, R.M., 2008. Contact Rate Calculation for a Basic Epidemic Model. *Mathematical Biosciences*, 216(1), pp. 56–62.
- Rhodes, P.J., 1988. *Thucydides: History II*, Warminster: Aris & Phillips.
- Riedel, S., 2005. Edward Jenner and the history of smallpox and vaccination, *Proc (Bayl UnivMed Cent)* 18, pp. 21-25.
- Selendy, J.M.H., 2011. *Water and Sanitation Related Diseases and the Environment : Challenges, Interventions and Preventive Measures*, Somerset: Wiley-Blackwell.
- Sherman, I.W., 2017. *The Power of Plagues*, Second ed., Washington, DC: ASM Press.

- Traill, J.S., 1975. *The Political Organization of Attica; A Study of the Demes, Trittyes, and Phylai, and Their Representation in the Athenian Council*, Princeton: American School of Classical Studies at Athens.
- Trvlos, I.N., 1971. *Pictorial Dictionary of Ancient Athens*, New York: Thames and Hudson.
- Van Wees, H., 2004. *Greek Warfare: Myth and Realities*, London: Duckworth.
- Van Zandt, K.E., Greer, M.T. & Gelhaus, H.C., 2013. Glanders: an Overview of Infection in Humans. *Orphanet Journal Of Rare Diseases*, 8(1), p.131.
- Vigne, J., 1994. Rats, Mice, and Relatives III: Old World Rats and Mice (Murinae), in: Hutchins, M. (Ed.), *Grzimek's Animal Life Encyclopedia*, 2 ed., Detroit: Gale.
- Wain, J., House, D., Parkhill, J., Parry, C., Dougan, G., 2002. Unlocking the Genome of the Human Typhoid Bacillus, *Lancet Infect Dis* 2, pp. 163-170.
- WHO. 2016. *Management of dead bodies: Frequently asked questions*. <https://www.who.int/hac/techguidance/management-of-dead-bodies/ganda/en/>.
- WHO. n.d. *Smallpox*. <https://www.who.int/biologicals/vaccines/smallpox/en/>
- WHO. n.d. *Typhoid Fever. International travel and health*. <https://www.who.int/ith/diseases/typhoidfever/en/>.
- Wycherley, R.E., 1977. *The Stones of Athens*, Princeton: Princeton University Press.