Racism, Anti-scientific Prejudice, and the History of Bubonic Plague: Two Plagues Separated by 5,000 Years

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1. Introduction

When I wrote "Lessons from History" for the previous COVID-19 volume (Hoffman et al 2021), I had hopes that with the early discovery of a vaccine, the pandemic would be over soon. After all, we had conquered smallpox and polio through vaccination. How wrong I was! I knew about the anti-vax movement with regard to the MMR vaccine for measles, mumps, and rubella, but not the rise of Q-Anon, leading millions of U.S. citizens to believe that Democrats are pedophiles who eat children and use COVID vaccinations to murder innocent people. Moreover, I did not foresee that vaccine and mask mandates would bring out the worst in people around the world, lead to armed protests, and be framed in the U.S. as unconstitutional infringements on individual rights.

Recently, however, I did a deep dive into the records of two plague epidemics not covered in depth in our previous article. (Hoffman et al, 2021). The 1901-1904/1907 Bubonic Plague epidemic in San Francisco was recently covered in new depth in a PBS special (PBS, 2022) and a book (Randall, 2019). I had not devoted much space to that plague epidemic in our previous article, largely because the details were similar to other plague epidemics in Asia. Often referred to as the Third Plague Epidemic, even though we now know there was one about 3,000 BC, it started in China in 1855 and spread widely in India. The death toll in India is estimated to have been at least 10 million people, and the total death toll about 15 million. I knew it had spread to San Francisco, but the death rate in San Francisco appeared to be much lower than the death rate in India. What I had thought was notable about the Third Plague Epidemic was the discovery and isolation of the bacterium that causes plague, Yersinia Pestis, named for Swiss-born French bacteriologist Alexandre Yersin, who made the discovery in 1894 in Hong Kong. In addition, there was a vaccine available shortly thereafter (1897). Four million Indian citizens were vaccinated.

While the outline of the story of the San Francisco Plague epidemic has been well known in the historical public health literature for more than a century, the plague in bronze-age Europe is little known and is still being explored using modern DNA sequencing techniques. The picture that emerges is forcing us to rethink the story we tell about the settlement of Europe and who our ancestors were. What ties this pre-historical plague and the San Francisco plague together is the history of racism told by the evidence from both epidemics' histories.

The San Francisco epidemic was first viewed as only affecting the Chinese immigrants who lived in Chinatown. Racism among White San Franciscans frustrated attempts by public health officials to fight the plague with contemporary medical technology. Racism also affected the historical narrative of those who had settled in northern Europe and Scandinavia, leading to the

Nazi story of a master race and the need to rid the world of Jews, Gypsies, and dark-skinned people. The new paleogenetic evidence that has emerged from the genomic revolution of the 21st century has forced a complete rethinking of the genetic history of northern Europeans and Scandinavians. A plague pandemic on the contemporary scale of the Black Death likely played a central role.

Unfortunately, racism and anti-scientific bias still poison our responses to the coronavirus epidemic, even as it wanes.

2. The San Francisco Plague of 1900-1907

When I started reading about the San Francisco plague epidemic, I discovered that the history of the last plague epidemic in the U.S. had many features I observed during the past three-plus years of the COVID-19 epidemic. Beginning with the first signs in Honolulu in 1899, the bubonic plague first appeared among the Chinese immigrant workers living in Honolulu's Chinatown. The white population believed it was a disease only affecting the Chinese and burned down Chinatown.

The plague arrived in San Francisco's Chinatown in 1900. Dr. Joseph J. Kinyoun was a young bacteriologist sent to San Francisco by the U.S. Marine Hospital Service to identify and work to stop the spread of the disease. He quickly identified the tell-tale swollen lymph glands and buboes and confirmed his diagnosis by studying the bacteria under his microscope. He recommended mass immunization with the vaccine, quarantine, and general cleaning of unsanitary conditions. The link to rats and fleas was known but still not widely understood.

Not surprisingly, people were terrified of the vaccine, which was about 50-85% effective (Hawgood, 2007). Their fear was understandable since vaccination entailed being injected with a long needle containing an unknown substance that made those who took it quite sick for a day or two. Who wanted that? Moreover, the white population convinced themselves it was not their problem if they could quarantine Chinatown. The governor of California, Henry Gage, seeing the plague as a menace to the California economy, declared there was no plague, and demonized Dr. Kinyoun, who responded by playing the scientist knows more than the average person card. Californians turned on Dr. Kinyoun[2], who was quickly demoted and transferred to Detroit.

Dr. Kinyoun was replaced by Dr. Rupert Blue, who had graduated near the bottom of his medical school class and who thus did not have an inflated opinion of his medical skill. Dr. Kinyoun had lived on Angel Island, the quarantine island in San Francisco Bay, and only entered the city proper when he had to diagnose a patient or dispose of a body. Dr. Blue set up shop in the middle of Chinatown. He also employed a Chinese interpreter, who had assisted a fact-finding committee from the Public Health Service, to help him speak frankly with residents of Chinatown.

As Dr. Blue gained the trust of the residents of Chinatown, dead bodies started coming out of the proverbial woodwork. Dr. Blue and his assistants were all publicly vaccinated, encouraging residents of Chinatown to also become vaccinated. Moreover, Governor Henry Gage was defeated in 1902 by George Pardee, who was a physician and an M.D. Governor Pardee worked

with Dr. Blue to eradicate the plague. Dr. Blue observed that plague appeared to follow mass death by rats. Part of his campaign was a campaign to kill as many rats as possible. By 1904, the plague appeared to be over.

Then, at 5:18 AM, April 18, 1906, the great San Francisco earthquake struck. Estimated more than 30 years later to have been approximately 7.9 on the Richter scale, the quake ran for 500 miles down the San Andreas Fault and lasted 42 seconds, an almost impossibly long time for an earthquake to last. The devastation was unparalleled, with the possible exception of the recent (2023) earthquakes in Turkey and Syria. Much of San Francisco in 1906 was built of wood or masonry and on soft soil. There were no earthquake-resistant building codes such as we have today. The old newsreels we can still watch today show buildings collapsing throughout the city, particularly in the Market Street area, where the soils are particularly soft. Much of the city was completely leveled.

As terrible as the earthquake was, the greatest damage was done by the great fire which followed the earthquake. The fire was caused both by ruptured gas lines catching fire and by the city dynamiting destroyed buildings. It burned for four days and nights, ultimately causing approximately 90% of the damage and creating 300,000 refugees. Some of the refugees fled to Oakland and Berkeley by ferry, but most were housed in hastily constructed tent cities in the Presidio and the outskirts of San Francisco. The crowded and unsanitary conditions of a 1906 tent city were again ripe for the proliferation of rats, and plague reappeared. This time, however, white San Franciscans were vulnerable, as well. They were forced to confront the reality that this disease was not a “China Plague” but bubonic plague.

Dr. Blue, who had been sent back east after the seeming eradication of the plague before the earthquake, returned to wage a second war against rats and plague. By that time, he had read the research on the rat-flea connection and knew the real culprit was not rats but fleas. He also found that other rodents, such as ground squirrels, can spread the disease through their fleas. Today we know that rodents of the Sierra Nevada and Rocky Mountains still spread the plague, and a few humans get sick every year. Thankfully, we also now know that Yersinia Pestis is no match for modern antibiotics!

One final mystery about the San Francisco plague remained. Why were the spread and mortality rates so much lower than in other bubonic plague epidemics? Recent research may have uncovered a fortunate difference between the fleas of California and the fleas that lived on the brown and black rats of Eurasia. Apparently, the Californian fleas did not inject as much plague-infected blood into their human victims as the Eurasian fleas! Who would have thought? As a result, Californian fleas did not make human victims as sick, on average, and a higher proportion survived. This difference, while fortunate, also contributed to the persistence of the fake news that the disease was not bubonic plague. David Randall (2019) writes, “The slow spread of the disease—a phenomenon that led the city to doubt Dr. Kinyoun’s warnings and call the epidemic a fake ploy by corrupt health officials—had hinged on the stomach of a flea, a lucky quirk that spared an untold number of lives….”

3. The Bronze-age Plague

Historians of the Bronze Age (Drews, 1993; Kline, 2021; Norrie 2016) have long known that many of the great civilizations of that age, such as the Hittites[3] and Minoans[4], collapsed unexpectedly in the second millennium B.C. Robert Drews (1993) gives several possible reasons for the well-documented collapse, including earthquakes, mass migrations, drought, systems collapse, raiders and changes in warfare. Others add: volcanoes, economic factors, climate change, internal social upheaval, invasion from outside the Aegean World, and general systems collapse. In a recent book, Philip Norrie (2016) argues that the real culprit was disease: smallpox and bubonic plague, in particular.

The discovery that there likely was a plague pandemic during the Bronze Age was only possible in the 21st century. The 2003 completion of the human genome project (NIH, 2003) and the technological changes following that initial feat allow inexpensive human genetic sequencing today. First, it was necessary to find out how to detect DNA from bones and teeth left in Bronze Age burial sites and from Egyptian mummies. Second, it was necessary to identify the genetic signatures of diseases left in these burial items. A 2015 article in *Cell* (Rasmussen, et al, 2015) outlines a study of burial sites in central Asia, Anatolia, Poland, and Sweden that show remnants of *Y. Pestis* in the teeth of skeletons dated from the second and third millennia B.C. Moreover, these remnants do not contain a particular genetic marker that allows *Y. Pestis* to be transmitted by fleas. Thus, it is not bubonic plague but must be pneumonic or septicaemic plague, both of which can be spread from human to human. The recent literature on plague during the Bronze Age draws on the research outlined in the *Cell* article.

The story the bones tell is that not only was there a deadly pneumonic/septicaemic plague pandemic about 3,000 B.C., but also that mass migrations accompanied this pandemic. We know from research on the Justinian and fourteenth-century plague pandemics that plague is endemic in the central Asian steppes and Himalayan foothills. We also know that cooling temperatures can set off conditions conducive to the spread of plague (Green and Symes, 2014). The historians of the Bronze Age did note that there were volcanic eruptions that led to cooler temperatures, crop failures, and mass migrations. They can be excused for not seeing the link to a plague epidemic since genetic research on plague was not possible until the 21st century, and the link between plague and cooling temperatures was only discovered in the same recent time frame (Hoffman et al., 2021: Green and Symes, 2014).

Additional pieces of the puzzle of the genetic makeup of northern Europeans from about 3,000 B.C. going forward come from genetic research showing the following disparate events: the migration of homo sapiens from Africa to Eurasia, the extinction of the Neanderthal and the genes they left behind, the origin of blue eyes, the spread of horses and wheels, and the source of Indo-European languages. Taken together, the histories of these events result in nothing less than an upending of the white supremacist view that the Vikings were a blond, blue-eyed master race that took over the known world in the 11th century A.D., spreading their genes throughout northern Europe and as far south as the Black Sea.

One of the first discoveries using mitochondrial DNA is that all homo sapiens are descended from Africans who walked north and then east and west when they reached Anatolia. (Fleagle et al. 2010). At some point, they lost the melanin that makes skin black, probably because lighter skin is better at absorbing vitamin D in the short and cold winter days of northern climates. Along the way, homo sapiens encountered Neanderthals, a separate humanoid species. The genomic revolution also allowed scientists to sequence Neanderthal DNA and compare it to homo sapiens' DNA. The result is that homo sapiens' DNA contains about 3-4% Neanderthal DNA, but African DNA contains less than 0.5% Neanderthal DNA, indicating interbreeding between homo sapiens and Neanderthal after the African diaspora. We still do not know why the Neanderthals died off, but we do know that modern Eurasians inherit small remnants of Neanderthal DNA (Teague and McRae, 2022).

A recent article in National Geographic (Curry, 2019) summarizes the paleogenetic history of modern Europeans. If we follow the migration of homo sapiens north as the glaciers receded, the early settlers in what was to become northern Europe are direct genetic descendants of early fertile crescent farmers and ice-age mammoth hunters of southern Europe. The DNA evidence also tells us that most of the farmers had light skin and dark eyes, while many of the hunter-gatherers had darker skin and lighter eyes. The genetic and graveyard evidence also suggests that these two populations did not comingle but rather maintained their separate lifestyles and diets. These two migrations take us to about 4,000 B.C. Sometime around 3,500 B.C., the paleogenetic evidence indicates a dramatic change in the people living in northern Europe and Scandinavia. First, villages disappear, suggesting a major negative demographic event. Then, about 500 years later, burial mounds dedicated to warriors (Chechushkov and Epimakhov, 2018: Gibbons, 2017: Anthony, 2007) and decorated with "Corded Ware" pottery appeared all over Europe (Olalde, 2016). The Neolithic farmers and herders they replaced left more egalitarian, less hierarchical grave sites.

Corded Ware burials are so recognizable that archaeologists rarely need to bother with radiocarbon dating. Almost invariably, men were buried lying on their right side and women lying on their left, both with their legs curled up and their faces pointed south. In some of the Halle warehouse's graves, women clutch purses and bags hung with canine teeth from dozens of dogs; men have stone battle axes. In one grave, neatly contained in a wooden crate on the concrete floor of the warehouse, a woman and child are buried together (Curry, 2019).

The genetic evidence indicates the Corded Ware people were distinctly different from the Neolithic farmers and herders. In fact, they are more similar to Native Americans and residents of the Asian steppes than to the previous European populations. We now know they were the Yamnaya (Gibbons, 2017), a population of horseback-riding nomads who used wheeled vehicles and spoke proto-Indo-European languages. They appear to have originated in the Central Asian steppes and spread east to Siberia and across the Bering Strait land bridge to North America, west to Great Britain, south to Spain and Morocco, and north to Scandinavia. By 2500 B.C., 70% to 100% of the skeletons now available contain Yamnaya DNA, but most indicate male Yamnaya and female farmers or herders. The picture that emerges is a migration of male warriors who raped (or married) the farmer and herder women, leaving behind a new population.

The fact that they also left language and burial mounds behind suggests that they stayed (Anthony, 2007: Gibbons, 2017: Big Think, 2022).

The question still remains, however: Why were they able to take over the genetic makeup of Europe in just 500 years? The answer appears to be that they also brought plague but had a level of acquired immunity sufficient to allow their genes to survive. Skeletons dated before 3,000 B.C. in Europe do not show evidence of *Y. Pestis*. Skeletons dated from the 4th millennium B.C. do. Moreover, this is the strain of *Y. Pestis* that lacks the gene allowing flea-born transmission (Calloway, 2015; 2022: Rasmussen, et. al, 2015).

This emerging evidence suggests that the Yamnaya were on the move about 3,000 B.C., bringing pneumonic and septicaemic plague, which decimated populations the Yamnaya encountered, but not before the invading male Yamnaya left their DNA in the children they fathered. The combination of horseback-riding and wheel-using warrior tribes who killed men, raped women, and brought plague would have been sufficient to topple Bronze Age civilizations. Moreover, this picture conforms both to the historical record of the fall of Bronze Age civilizations and the paleogenetic record (Anthony, 2007; Chechushkov and Epimakhov, 2018; Gibbons, 2017). Interestingly, there is no evidence of *Y. Pestis* in Egyptian mummies, providing an answer to the historical puzzle of why the civilization of the Pharaohs did not suffer the fate of the Hittites (Rascovan, et. al, 2018).

The reasons why the Yamnaya left their Central Asian homeland may be the link between the historical evidence of massive volcanic eruptions and earthquakes disturbing the agricultural climate and the genetic evidence of plague. Volcanic eruptions, in particular, can lead to cooling temperatures around the world as the ash moves with the prevailing winds, lowering the amount of solar energy on the ground. Even a minor reduction in temperature could lead to crop failures wherever the ash plume passes. In addition, it may disrupt the delicate balance between *Y. Pestis* and its hosts (Green and Symes, 2014).

The final pieces of the genetic puzzle of the origins of the blue-eyed, blond-haired Aryan who became the Nazi symbol of a master race are the origins of blond hair and blue eyes. The origin of blond hair is similar to the origin of light skin (Norton, et. al, 2007). Blond hair and light skin have less melanin than dark hair and dark skin and allow people living in climates that allow less sunlight over the year to absorb more vitamin D (Reich and Reich, 2018; Carlberg and Hanel, 2020). Blue eyes also have less melanin, but the source of blue eyes appears to be the result of a single sudden mutation that took place about 10,000 years ago in one individual who then passed on a specific gene for blue eyes to all subsequent blue-eyed humans (Eiberg, et. al, 2008). This startling result was discovered by a team of geneticists at the University of Copenhagen who studied a large sample of apparently unrelated Danes who all had pure blue eyes. Their DNA was compared to a sample of apparently unrelated blue-eyed individuals from Jordan and Turkey and a sample of unrelated individuals with brown eyes from the same places. They discovered that all the blue-eyed individuals from Denmark, Jordan, and Turkey had genetically identical eyes. The brown-eyed individuals were also genetically distinct (Eiberg, et. al, 2008). This startling result, combined with the evolutionary fitness of light hair and light skin, shows clearly that the Vikings/Aryans were not from some master race. They evolved in response to

environmental pressure, just as Darwin predicted, and benefitted from a random mutation that added blue eyes to the genetic mix. Moreover, the blue eye mutation appears to have originated in the central Asian or Eurasian steppes, not in Scandinavia (Reich and Reich, 2018).

Some scholars have added that Neolithic hunter-gatherer males likely suffered higher mortality than Neolithic hunter-gatherer females because big-game hunting with hand-held spears and stone points was even more dangerous than childbirth. Since a higher adult male mortality would mean a higher proportion of adult females than males, surviving adult males could be more choosy about their mates. Thus, a male preference for blond hair and blue eyes would reinforce the vitamin D benefit of that combination. This was the pre-genomic explanation. Since we cannot interview Neolithic hunter-gatherers and they left only burial grounds and cave paintings, scientists appear now to favor an emphasis on random mutation and natural selection.

4. Conclusions and Final Thoughts

These two seemingly unrelated plague epidemics share important characteristics despite the differences in time and medical knowledge. They are both parts of international pandemics, both of which originated on the vast, sparsely populated steppes of central Asia. They were both caused by the same bacterium, *Yersinia Pestis*, although the earlier plague still lacked the gene present in the later plague that allowed *Y. Pestis* to be transmitted from rodents to humans by fleas. This bacterium wreaked havoc on Homo sapiens for millennia, beginning with the Bronze Age and ending in San Francisco in 1907.

What I find most interesting and disturbing is the racial response to both plagues. Why did San Franciscans decide it was only a Chinese disease, despite the knowledge of plagues in Europe? Why would a U.S. president lead his followers to believe they should not get vaccinated against a disease destined to kill a million Americans in two years? Why would people with white skin believe they were better than people with dark skin when their skin color is a genetic adaptation to winter day length? Moreover, despite their white skin, their genetic ancestors are black Africans and Central Asian nomads whose progeny includes Native Americans. Why would they storm the U.S. Capitol on January 6, 2021, and try to undo a U.S. election because they believed the people who won would allow darker-skinned homo sapiens to replace them?

The answers to the above questions are embedded in the racist mindset that still permeates the thinking of many Americans and is having a terrifying resurgence in book bans, laws banning the teaching of "critical race theory," which is actually just a ban on teaching uncomfortable truths about African American history, and the demonization of drag queens. Demonizing the Chinese is as embedded in our racist mindset as is demonizing those with dark skin or alternative gender or sexual preferences. This was especially true in the West and was at its height in the 1870s and 1880s, culminating in the U.S. with the Chinese Exclusion Act of 1882. The act banned the immigration of Chinese to the United States for 10 years. Official representatives of the Chinese government had to present papers from the Chinese government. The Chinese Exclusion Act was extended in 1892 by the Geary Act and only partially repealed in 1943. Canada followed with the Chinese Immigration Act of 1923. It was otherwise known as the Chinese Exclusion Act. Only 100 Chinese were admitted to Canada for the next 24 years.

While I hope the stories I tell in this article will help some of our fellow homo sapiens rethink their assumptions about our origins, I fear that recent, post-pandemic political developments are taking us in the opposite direction.

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