



ARCHAEOLOGICAL PERSPECTIVES ON THE STUDY OF OUTBREAKS DURING THE COVID-19 PANDEMIC

COEVOLUTION, EMERGENCE, AND RESURGENCE OF PATHOGENS THROUGH HISTORY

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The COVID-19 pandemic has dominated our world for the past year, and it has heightened interest in epidemic diseases. In June 2020, together with Maria Spyrou of the Max Planck Institute for the Science of Human History, we presented a webinar for the SAA with the same title as this article. Our goal was to provide an overview of important epidemics in the past and to explore how archaeology and genomics contribute to knowledge about those epidemics. Personal commitments prevented Maria Spyrou from participating with us on this article; therefore, we decided to narrow our focus to the New World and the introduction and spread of smallpox, one of the diseases that repeatedly ravaged Indigenous Americans for centuries after the arrival of Europeans. We believe that archaeologists can contribute to a broader knowledge of pandemics by revisiting the social factors behind ancient epidemics, gathering data from archaeological sites associated with specific outbreaks, and analyzing skeletal collections to better understand the complex dynamics and interactions of ancient diseases in changing ecosystems, demographics of human populations, and cultural practices. Improved methodologies and techniques, including molecular analysis of ancient DNA (aDNA), assisted with polymerase chain reaction (PCR) and new sequencing methods like isothermal amplification, offer key insights (Spyrou et al. 2019; see also Hofman and Warinner 2019; Sedig 2019). Past epidemics have impacted large sectors of the population in affected regions, disrupted economic activities, and increased social unrest. A pandemic like COVID-19 demands that we assess our weaknesses and strengths as a global society, or as societies across the world. The present pandemic encourages us to pause and to remember that, despite dwelling in artificial environments provided by powerful social and technological systems, we are still subject to ecological checks. It is precisely during moments of crisis when we can better observe structural inequalities within our home communities and our global community more broadly. These are the right moments for taking

political action to attack the root causes that produce and maintain vulnerabilities and inequalities.

Preparing This Article within the Context of the COVID-19 Pandemic in the United States

During the first two weeks of March 2020, many universities around the United States decided to stop in-person classes and quickly moved onto online platforms. Both students and professors found themselves struggling to adapt to the new circumstances. During February 2020, the SARS-CoV-2 virus had spread quickly from “ground zero” in Wuhan, China, to most of the metropolises of Europe and Asia, and from there to the rest of the world, including the United States. By the end of March, almost 186,000 people had been infected in the United States alone (Figure 1), and the U.S. death toll reached 3,602 (Figure 2). Things worsened in subsequent weeks, and by the end of April, the United States reached 1,061,637 infections and 58,804 deaths. Economic impacts quickly followed, with almost 37 million Americans temporarily unemployed due to uncoordinated quarantines that nevertheless managed to keep the infection rate flat during May and June, as well as reducing the number of deaths from 41,990 in May to 22,903 in June. Several police cases involving abuses of Black Americans in late May ignited civil protests and riots against police brutality in the metropolitan areas of the country during the summer. The month of July saw a new peak of infection with 1,922,685 infected individuals, followed by a steady rise in deaths that reached 25,571 that month and another 30,180 in August. The number of infections across the United States was slightly reduced in September (1,212,702 people), but during October the infection peaked again to more than 1,910,331 people. During November, as we completed the first draft of this article, the United States counted an unprecedented 4,416,616 infected people. While working on editorial corrections for the article, we looked with dismay at how the total number of infections during the month of December reached 6,335,233 people, while the number of absolute deaths more than doubled from November (37,324 people)

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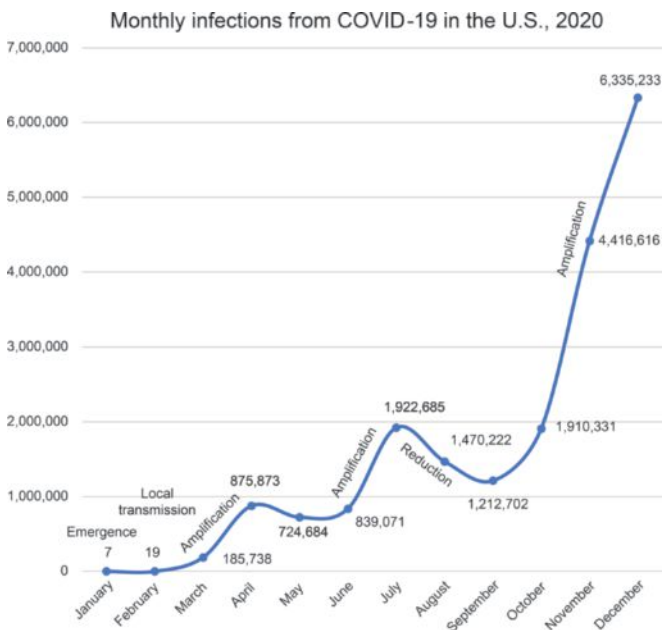


Figure 1. Monthly infections from COVID-19 in the United States from January 1 to December 31, 2020. Note that January and February of 2020 include the first phase of the outbreak, (1) the emergence and introduction of a new pathogen; (2) local transmission took place in many areas between February and March; (3) amplification occurred during three waves of infection in March–April, July, and October–December. Meanwhile, there were slight reductions in infections during May–June and August–September of 2020. Estimates based on reported daily and weekly cases of COVID-19 (<https://www.cdc.gov/nchs/nvss/vsrr/covid19/index.htm>; <https://covid.cdc.gov/covid-data-tracker/#trendsdailytrendsescases>).

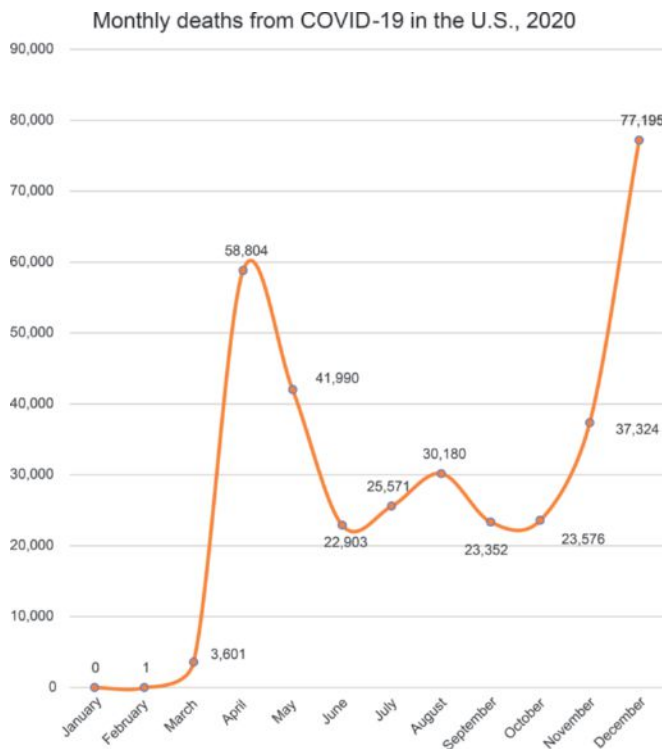


Figure 2. Monthly deaths from COVID-19 in the United States from January 1 to December 31, 2020. Estimate based on reported daily and weekly cases (<https://www.cdc.gov/nchs/nvss/vsrr/covid19/index.htm>; https://covid.cdc.gov/covid-data-tracker/#trends_dailytrendsescases).

to reach 77,195 in December. Notably, from April to December, we have seen a steady decline in the case fatality rate (CFR = number of deaths/number of infected × 100), from a high 6.71% in April to 1.22% in December (Figure 3). This is most likely due to the experience gained by medical professionals in the treatment of patients who developed strong immunological responses to the virus. We expect that the CFR will continue fluctuating in lower numbers during 2021 as vaccines become available.

During the unfolding of the COVID-19 pandemic in the first week of August, we discovered that there were segments of the population with heightened vulnerabilities to the disease: the death rate is higher for Black American (2.8 times), Hispanic (2.8 times), and Native American (2.6 times) groups than among White non-Hispanic people (see <https://www.cdc.gov/coronavirus/2019-ncov/covid-data/investigations-discovery/hospitalization-death-by-race-ethnicity.html>). What lies behind these disparities? Some would like to believe the primary reason for this differential death rate is due to genetics, an explanation that retrofits racial beliefs of Caucasian superiority. Instead,

multifactor correlations need to be investigated to understand historically constructed inequities in which Native American, Hispanic, and Black communities are poorer in general than Caucasians, forcing them to live in more densely populated neighborhoods and in smaller houses shared with extended families; these groups tend to have less access to healthy diets, they perform more jobs that pose greater health risks, and they have limited and sometimes no access to medical insurance, when compared with the White population of our country.

The first round of vaccines began in December 2020, but it will take months to vaccinate enough people to reach the threshold of so-called herd immunity, which, in the case of other infectious diseases, typically requires between 80% and 95% of the total population to be vaccinated. The human costs that we still need to endure through the winter and spring of 2021 are unknown, but they will certainly pile on the more than 350,000 deaths and almost 20 million infected people in the United States that accumulated from January 2020 to December 31, 2020. All social science disciplines are reflecting on the impacts of the COVID-19 pandemic and are examining archaeological and historical cases to find guidance as

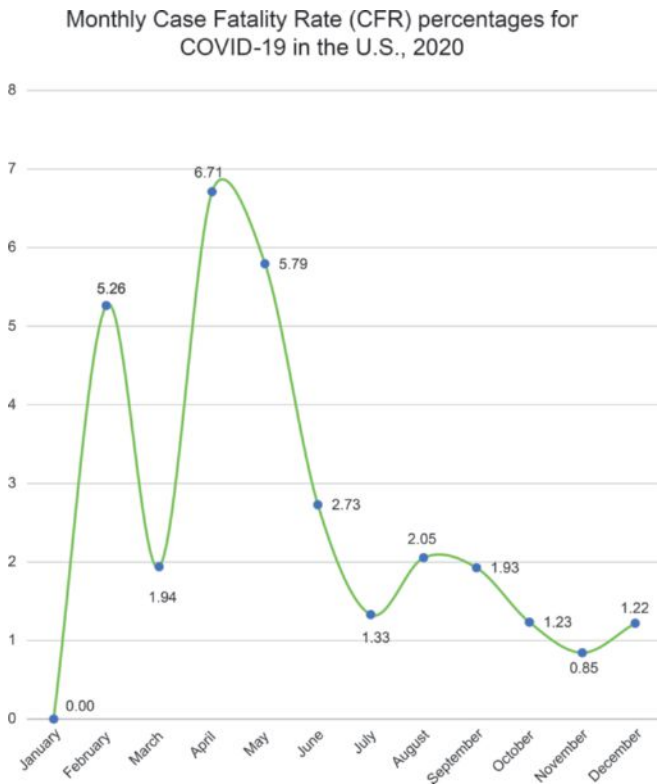


Figure 3. Variation of case fatality rates (CFR) from COVID-19 in the United States by month, from January 1 to December 31, 2020. Estimate based on reported daily and weekly cases (<https://www.cdc.gov/nchs/nvss/vsrr/covid19/index.htm>; https://covid.cdc.gov/covid-data-tracker/#trends_dailytrendscases).

to possible scenarios based on past epidemiological experiences and trends. How did one of the most powerful and wealthy nations in the world get to this level of exposure so quickly? How can the study of ancient epidemics help us to understand the current pandemic and mitigate future outbreaks of other infectious diseases?

Despite medical advances, we have learned the hard way that epidemics are as much a social problem as a medical one. Germ theory and biology alone cannot solve epidemics without political compromises, awareness of socioeconomic inequities, identification of hazards and vulnerabilities, and fair access to affordable medical attention (Blaikie et al. 1994). An epidemic can be controlled if a society has invested in developing protections long before the outbreak through risk assessment, identifying and mapping hazards, projecting human and economic losses, mitigating by way of a strong hospital infrastructure, promoting and funding research on infectious diseases, training human resources in the medical sector, maintaining epidemiological surveillance, stockpiling aid, providing universal access to healthcare, acquiring insurance to

maintain economic stability, and promoting healthy lifestyles. The creation of emergency plans will provide early epidemiological alerts and a strong response system capable of quick deployment of medical supplies and trained personnel with sufficient personal protection equipment (PPE). Unfortunately, during the current pandemic only a handful of developed countries were prepared. What is more worrisome is that a vast majority of countries have underfunded and even dismantled their health systems under the neoliberal economic principles promoted by the United States and the United Kingdom since the 1980s. Such dismantling of welfare systems has also prevented social and institutional changes to reduce historical conditions of vulnerability. The income gap between rich and poor has grown exponentially, and many people in the United States and worldwide do not have access to proper housing and formal employment, while the rural and urban poor have been disenfranchised and left behind. These social factors have produced the conditions in which some 85.7 million people worldwide have been infected and about 1.85 million people have died from COVID-19 in the first 12 months of the current pandemic (a CFR of 2.17%). Even worse is the realization that we are unprepared to face another pandemic in the next decade.

Archaeological Methodologies for Studying Ancient Epidemics

The study of ancient disease has been a relatively small and specialized field, but with the COVID-19 pandemic, interest has grown. Experts from many fields are involved in these studies, including bioarchaeologists, paleomicrobiologists, and archaeologists. Scholars, especially those working with aDNA, are transforming what we know about prehistoric and historic diseases (Spyrou et al. 2019). Archaeologists are key because before the biomolecular scholars can begin work, archaeologists must find, identify, date, and interpret human remains (Fornaciari 2017). Archaeologists play fundamental roles in gathering data that other scholars use to build the case for an ancient pandemic.

Mass graves are often the first clue that an epidemic disease had occurred (Duday 2008; Fornaciari 2017). Archaeologists must distinguish between a mass grave where many people died and were buried within a short period of time, as opposed to a graveyard where families buried their dead over many years. As with COVID-19, victims of disease die over a period of days or weeks. People may be buried in single graves or with two or three people in one grave. In other cases, those preparing graves during an epidemic might excavate large pits or long trenches. These burial areas may have been used over a period of weeks or months with each new batch of bodies covered over with dirt as they were placed in the ground. Careful excavation and attention to stratigraphy is needed to distinguish these sorts of mass graves from those that result, for example, from warfare. Warfare creates many dead within short periods—just days or even single days—and burials would likely take place in single episodes of varying durations, in contrast to the sequential placing and covering of bodies of those who died from disease. Sometimes when epidemics occur, those

preparing graves excavate more graves than required or leave half-empty trenches—such empty graves are another possible clue that an epidemic has occurred. Other clues include bodies carelessly tossed into graves or bodies encased in lime to slow decomposition (Fornaciari 2017).

Epidemic diseases have been present in the Old World since the development of agriculture over 10,000 years ago. Agriculture allowed people to live in large sedentary populations and caused intimate interactions between humans and domestic animals with resulting exposure to zoonotic pathogens (Armelagos et al. 1996). The absence of large domestic animals in most parts of the New World reduced the rate of zoonotic diseases there. Indigenous populations in the New World had not been free of disease prior to the European conquest, but they suffered from chronic and episodic diseases, rather than the acute and epidemic diseases of the Old World (Martin and Goodman 2002). The arrival of Columbus in the Caribbean Islands in 1492 immediately started the transmission of disease organisms between Europeans and Indigenous Americans. One of the most devastating of these diseases was smallpox.

Introduction of Smallpox into the Spanish Domains

Second in death toll only to tuberculosis, as horrible as the bubonic plague, and perhaps one of the most painful diseases in human history, smallpox killed millions of people and scarred the bodies and faces of survivors (Figure 4). Smallpox spread from person to person mainly by contact with droplets of saliva, nasal discharge, or by touching the scabs of infected people. Smallpox is the only serious disease that has been eradicated from human populations since the late 1970s through a successful program of vaccination and quarantine. The species that attacks humans is *Variola virus* (VARV). VARV belongs to the genus *Orthopoxvirus*, present in many hosts and in multiple strains. It is unclear which animal was the natural host of the human strain—most likely a rodent—but VARV became specific to humans at one point in its evolution. Although the human strain has been eradicated, we can still be infected by other animal strains. Orthopoxvirus infections, passed from animals to humans, have been growing in number since we ended the smallpox vaccination program, and there is always the fear that one of these animal strains could mutate and create a new strain of the virus specific to humans. Luckily, all orthopoxviruses have a pronounced cross-immunity between different animal hosts, and that is why the cowpox virus has been used to produce immunity in humans through vaccination since 1796.

There is some debate about the age of the human-specific VARV (see Biagini et al. 2012; Duggan et al. 2016; Thèves et al. 2014; cf. Babkin and Babkina 2015) and about which variant was brought by Crusaders to Europe between the tenth and thirteenth centuries AD—either the more lethal Asian VARV with a 10%–30% CFR

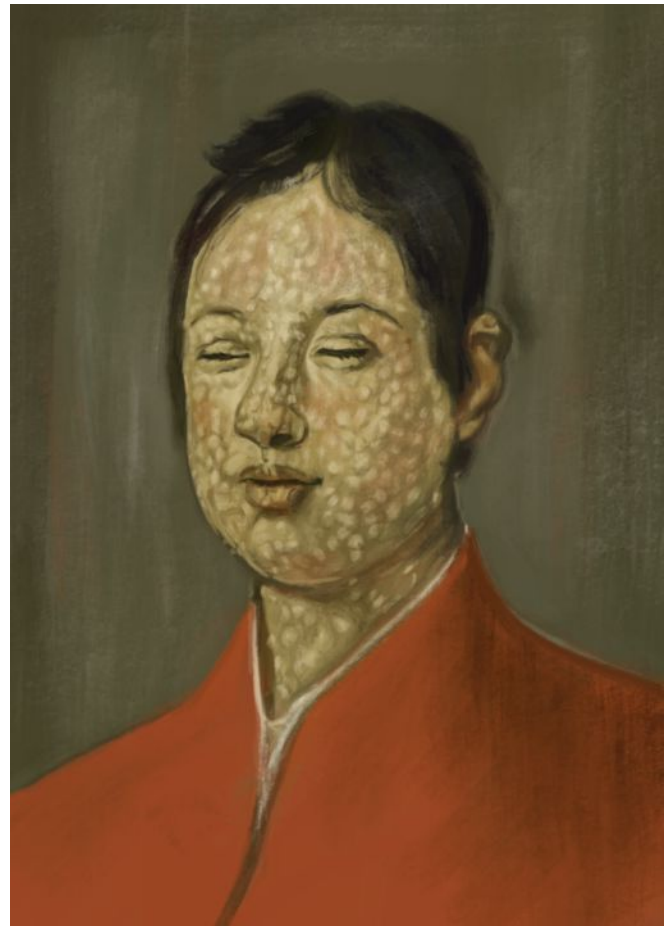


Figure 4. Oil painting of Ferdinando II de Medici on the ninth day of his smallpox infection in 1626, as painted by Justus Suttemans, Galleria Palatina, Florence, Italy. Redrawn by Gerardo Gutiérrez after Lippi and others (2015:258).

or the West African VARV with an 8%–12% CFR. Genetic studies have shown that the West Africa strain was introduced to the Caribbean Islands between 1507 and 1518 by infected people from Portuguese slave trading enclaves in West Africa.

In 1520, Pánfilo de Narváez sailed from Cuba to Veracruz under orders to arrest Hernando Cortés. He brought with him a contingent of Caribbean (Taíno) natives and at least one African slave, Francisco Eguía, infected with smallpox. After defeating Narváez in Veracruz, Cortés brought some of these slaves to central Mexico as porters. By the time Cortés returned to Tenochtitlan, the city had rebelled against his host, Moctezuma, and the Spaniards because of a massacre at the main Aztec temple led by Cortés's lieutenant, Pedro de Alvarado. Cortés was forced to abandon Tenochtitlan in the summer of 1521. The Spanish-Tlaxcalan army

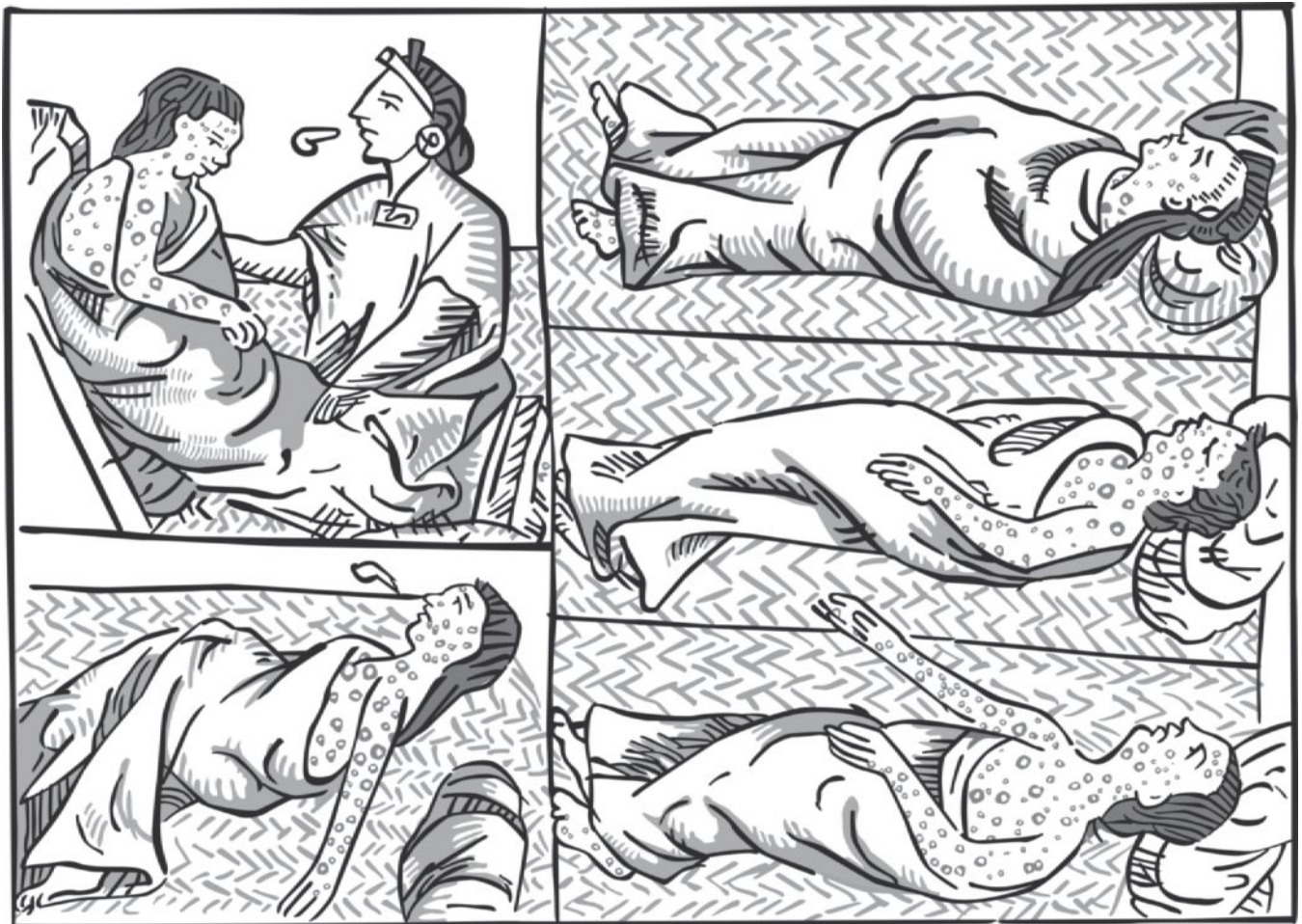


Figure 5. Images of individuals with scab-lesions on their skin, probably suffering from VARV, during the summer of 1521 in Tenochtitlan, as depicted in the Florentine Codex, Book 12, Chapter 29. Image redrawn by Gerardo Gutiérrez after Sahagún (1975 [1575/1577]:Illustration 114).

left the city in disarray, with corpses filling the street of Tacuba. It is at this very moment that the smallpox outbreak became evident among the inhabitants of Tenochtitlan, and it quickly spread to other cities. Depictions of smallpox created in Mexico during the sixteenth century show scab-lesions like those seen for the VARV infection in Europe (Figure 5). The *Relación de Michoacán* (Alcalá 2000 [1539–1541]:658) mentions that a group of Aztec ambassadors may have brought the disease to the Tarascan empire, causing an outbreak that killed the *cazonzi* Zuangua, the primary ruler of the Tarascan political system. Smallpox also reached Panama—there were frequent expeditions to find the place known as Biru or Piru along the Pacific shores of Colombia, Ecuador, and northern Peru, and one or more of these expeditions brought the disease to South America as early as 1524. The powerful Inca Huayna Capac died from this early outbreak

(Figure 6), an event that started the civil war in the Inca realm between Huascar and his brother, Atahualpa.

In the following centuries, smallpox epidemics erupted throughout the Americas, moving through Mesoamerica, Central America, and South America during the 1500s, the Atlantic coast of North America during the 1600s, and the rest of North America through the end of the seventeenth and into the eighteenth century (Alchon 2003; Carlos and Lewis 2012; Esparza 2017; Fenn 2001; Swedlund 2015). Some of these epidemics were continent-wide; for example, the smallpox epidemic of 1775–1782 (Fenn 2001). Smallpox devastated Indigenous communities, but large numbers of European-Americans also succumbed, and myriad other factors impacted Indigenous peoples in addition to disease outbreaks. In the next section, we question the common

assumption that disease was the primary cause of the dramatic decline of Indigenous peoples in the Americas after European contact and point to the multitude of other factors involved.

Reevaluating the “Virgin Soil” Hypothesis in the Spanish Borderlands and in Anglo and French Domains

The “virgin soil” hypothesis (first coined by Alfred Crosby [1976]) posits that the dramatic drop in Indigenous populations after European contact was a result of their lack of immunity to European diseases. This appealing idea sees Europeans as innocent bystanders in the decimation of Indigenous peoples. The true culprit was not the colonizers, but the Indigenous’ lack of immunity and resistance to these diseases. The term “virgin soil epidemic” has been used to describe populations hit by diseases that they had never experienced before. It is a simple idea and has become widely accepted, but the truth is far more complex (Alchon 2003; Feinman and Drake 2021; Hutchinson 2016:40; Jones 2003, 2015). It falsely implies that Indigenous immune systems were somehow weaker than those of Europeans. *In fact, Indigenous peoples have the same capable and effective immune systems as other human populations.* When epidemics struck the New World, Europeans also died in significant numbers (Lippi et al. 2015). Immunity to infectious disease is *acquired*, not inherited. If a parent has smallpox and survives, their child will be just as susceptible to smallpox if another epidemic occurs. The virgin soil hypothesis has been reconsidered and has been found to be quite problematic (Jones 2003). It was not *just* disease but numerous social factors that made mortality within Indigenous communities so much greater than it was for European colonists (Cameron et al. 2015; Feinman and Drake 2021; Gamble et al. 2021; Jones 2003, 2015).

The brutality of colonization during the European conquest of the New World played a significant role in the high death toll among Indigenous communities. Indigenous peoples were attacked, enslaved, overworked in industries like mining, their crops and land were appropriated, their communities were either co-opted or dispersed, or they were herded into mission settlements (Reséndez 2016). For these reasons, Indigenous immune systems were often compromised, and the break-up of Indigenous communities destroyed local medical knowledge and dispersed caregivers, all factors rendering any disease episode more disastrous.

Two case studies illustrate the impact of structural violence on Indigenous populations in North America. One of the most comprehensive studies of the effects of colonialism is Clark Spencer Larsen’s study of the Indigenous people of La Florida (Larsen 1994, 2015). In what is now northern Florida and coastal Georgia, the Spanish built a network of missions beginning in the early 1500s. They put in place policies that forced native populations into crowded and unsanitary mission settlements. Larsen’s study of human remains showed clear evidence of health declines for these Indigenous peoples. Crowded living conditions increased

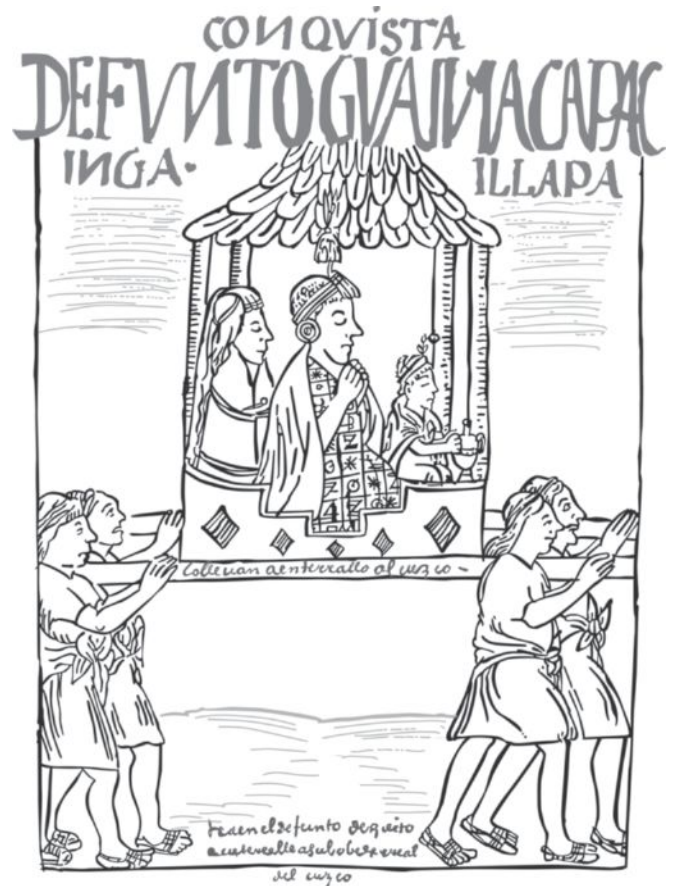


Figure 6. Funeral procession of the mummy of the Inca Huayna Capac from Ecuador to the Inca capital of Cuzco, Peru. The Inca emperor died from a disease that produced skin lesions that match descriptions of smallpox lesions. The Inca Garcilaso de la Vega claimed to have seen smallpox lesions on the mummy of Huayna Capac in Cuzco. Drawing by Gerardo Gutiérrez after Guamán Poma de Ayala (1992 [1610/1615]:350).

the prevalence of tuberculosis and syphilis. Anemia increased dramatically, indicating that they had parasites, drank contaminated water, and suffered other effects from crowded living conditions. Native peoples of La Florida were forced to do very heavy labor, especially carrying large loads of agricultural products over great distances, causing arthritis and bone damage. Their diet became largely corn based, and they had much less access to meat and seafood, resulting in numerous dental caries and enamel hypoplasia that marked periods of food stress. Clearly their health was seriously compromised by European colonization. Larsen (2015:87–90) has demonstrated that not only the health but also the social fabric of Indigenous communities in the American South was profoundly disrupted as they experienced intense structural violence. They were certainly in a situation of socially

constructed vulnerability that made it difficult for them to fight off epidemic disease when it arrived.

In northeastern North America, epidemics, warfare, and the intrusion of English settlers also combined to cause a dramatic drop in the Indigenous population. Alan C. Swedlund (2015) describes these processes in the Connecticut Valley during the 1600s. There were some 100,000 Native Americans living in New England in 1600, but that population had decreased by at least 60% by 1700. Epidemics in the early 1600s killed enough Native Americans that the English were able to establish settlements throughout the region. But warfare also lowered the native population dramatically. The Pequot War in the mid-1630s was particularly devastating. Those Pequots who did not perish were enslaved by the English, and many were sold off to distant places. Warfare, captive-taking, and enslavement continued through the rest of the century, including during the enormously destructive King Phillip's War in the 1670s (Swedlund 2015). The same was true in adjacent areas of New France (now Canada). Frequent warfare produced many Indigenous slaves who were sold to French colonists. During epidemics, these slaves died in numbers far greater than those of the French. Historian Brett Rushforth (2012:336) observes that mortality was not just a result of disease; violent beatings, unending hard labor, and deficiencies of food and clothing all contributed to excessive death rates.

Conclusion

Many factors need to be considered to understand the introduction of Old World pathogens into the New World. Importantly, the capitals of Indigenous empires in central Mexico, Michoacán, and Peru had the large population densities necessary to sustain and to amplify the biological effects of smallpox and other density-dependent diseases. Even in North America, where Indigenous population densities were lower, epidemics still made significant impacts at various points in time. Domestic animals and rats were also introduced into the New World, and these mammals acted as hosts for many pathogens. Indigenous landscapes and ecosystems experienced processes of dramatic alteration and degradation due to changing economic and political systems. Deforestation increased to create grazing land for European animals. Structural abuse of colonized populations took a major toll and promoted differential rates of death between Europeans and Indigenous peoples not only in the presence of pathogens but also deaths resulting from this abuse. Every 5–10 years, new epidemics ravaged different regions of Mexico and North America that together with deaths from warfare, malnutrition, and the destruction of Indigenous communities and their livelihoods conspired to prevent demographic recovery of the Indigenous population that undoubtedly lost a significant percentage of its original size.

We emphasize that the “virgin soil” idea that has been used to explain the decline in Indigenous populations in the New World

requires reassessment. These declines resulted more from the violence and structural injustices of colonialism than epidemic disease. As David S. Jones (2003:742) so aptly summarizes,

Although unprecedented in their widespread severity, virgin soil epidemics may have arisen from nothing more unique than the familiar forces of poverty, malnutrition, environmental stress, dislocation, and social disparity that cause epidemics among all other populations. Whenever historians describe the depopulation of the Americas that followed European arrival, they should acknowledge the complexity, the subtlety, and the contingency of the process. They need to replace homogeneous and ambiguous claims of no immunity with heterogeneous analyses that situate the mortality of the epidemics in specific social and environmental contexts. Only then can they overcome the widespread public and academic appeal of immunologic determinism and do justice to the crucial events of the encounter between Europeans and Americans.

We urge archaeologists to abandon terms like “virgin soil” and “lack of immunity” no matter how simple and appealing they may seem, and instead embrace the full complexity of colonial encounters. It is perhaps most important to emphasize that, even with significant population losses, Indigenous people continued to be the largest demographic group in the New World until the nineteenth century, and they are still here today. This endurance demonstrates the resilience of Indigenous Americans even through the most adverse moments of their histories.

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