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Virgin Soils Revisited

The decimation of American Indian populations that followed European arrival in the Americas was one of the most shocking demographic events of the last millennium. Indian populations declined by as much as 95 percent in the first century after the arrival of Christopher Columbus, prompting one historian to conclude that “early America was a catastrophe—a horror story, not an epic.” This collapse established the foundation for the subsequent social and political developments of American history. Since the earliest encounters of colonization, colonists and their descendants have struggled to explain how and why depopulation occurred. They have debated the role of race, politics, and even genocide. All have concluded that infectious diseases, introduced by Europeans and Africans, played a decisive role. American Indians suffered terrible mortality from smallpox, measles, tuberculosis, and many other diseases. Their susceptibility led to American Indian decline even as European populations thrived.

Discussions of the epidemiological vulnerability of American Indians rose to prominence with the work of William McNeill and Alfred W. Crosby in the 1970s. Both argued that the depopulation of the Americas was the inevitable result of contact between disease-experienced Old World populations and the “virgin” populations of the Americas. As Crosby defined them in 1976, “Virgin soil epidemics are those in which the populations at risk have had no previous contact with the diseases that strike them and are therefore immunologically almost defenseless.” His theory provided a powerful explanation for the outcomes of encounter between Europeans and indigenous groups, not just in the Americas but throughout the world. Since Crosby’s analysis of virgin soil epidemics appeared in the *William and Mary Quarterly*, countless writers have cited his definition and attributed the devastation of American Indian populations to their immunologic inadequacy. As argued in Jared Diamond’s Pulitzer Prize-winning *Guns, Germs, and Steel*, “The main killers were Old World germs to which Indians had never been exposed, and against which they therefore had neither immune nor genetic resistance.” Such assertions, which apply the intuitive appeal of natural selection to the demographic history of the Americas, dominate academic and popular discussions of depopulation.

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Even as Crosby's model of virgin soil epidemics remains a central theme of the historiography of the Americas, it has been misunderstood and misrepresented. Crosby actually downplayed the "genetic weakness hypothesis" and instead emphasized the many environmental factors that might have contributed to American Indian susceptibility to Old World diseases, including lack of childhood exposure, malnutrition, and the social chaos generated by European colonization. Subsequent historians, however, have often reduced the complexity of Crosby's model to vague claims that American Indians had "no immunity" to the new epidemics. These claims obscure crucial distinctions between different mechanisms that might have left American Indians vulnerable. Did American Indians lack specific genes that made Europeans and Africans, after generations of natural selection, more resistant to smallpox and tuberculosis? Did they lack antibodies that their Eurasian counterparts acquired during childhood exposure to endemic infections? Were their immune systems compromised by the malnutrition, exhaustion, and stress created by European colonization? These different explanations, blurred within simple claims of no immunity, have very different implications for our understanding of what was responsible for this demographic catastrophe.

It is now possible to revisit the theory of virgin soil epidemics and reassess the many possible causes of American Indian susceptibility to European pathogens. The confusion can be untangled by surveying and resynthesizing diverse research about Indian depopulation. A review of the literature of colonization shows the prevalence of simplistic assertions of no immunity and their possible ideological appeals. It also demonstrates the importance of defining the specific claims contained within the theory of virgin soil epidemics and evaluating each of them separately. Recent immunological research has clarified the different mechanisms that can compromise human immunity. Parallel work by biological anthropologists, archaeologists, and historians has elucidated the details of the mortality of specific Indian populations. Taken together, this work suggests that although Indians' lack of prior exposure might have left them vulnerable to European pathogens, the specific contribution of such genetic or developmental factors is probably unknowable. In contrast, the analyses clearly show that the fates of individual populations depended on contingent factors of their physical, economic, social, and political environments. It could well be that the epidemics among American Indians, despite their unusual severity, were caused by the same forces of poverty, social stress, and environmental vulnerability that cause epidemics in all other times and places. These new understandings of the mechanisms of depopulation require historians to be extremely careful in their writing about American Indian epidemics. If they attribute depopulation to irresistible genetic and microbial forces, they risk being interpreted as supporting racial theories of historical development. Instead, they must acknowledge the ways in which multiple factors, especially social forces and human agency, shaped the epidemics of encounter and colonization. . . .

Taken as a whole, recent immunological research offers many clues about the state of Indian immunity. American Indians could certainly mount immune responses to European pathogens. Perhaps their "naïveté" left them

without protective genes, making them incrementally susceptible. Perhaps their homogeneity left them vulnerable to adaptable pathogens. Research about these questions continues on the cutting edge of immunology. It is possible that definitive evidence of demographically significant resistance genes will emerge. The historical experiment, however, has run its course. European and American populations mixed for over five hundred years before scientists could study them adequately. The opportunity for further research on first contact populations remains remote. As a result, the state of virgin immunity will forever remain contested. This leaves the literature on genetics and immunity promising, but unsatisfying. Genetic arguments of population-wide vulnerability must therefore be made with great caution. Other immunological mechanisms remain plausible, but problematic. Initial lack of adaptive immunity likely left American Indian societies vulnerable to certain pathogens, but certainly not to all of them, and adaptive immunity does not seem to have been relevant for the dominant causes of mortality in developing societies.

Furthermore, the mechanisms of adaptive immunity, along with the impact of simultaneous and successive synergistic infections, emphasize the importance of the disease environment, and not only the population itself, in shaping a population's susceptibility to infection. Other features of the environment, defined broadly, also have profound effects on immunity. A population's physical, social, economic, and political environments all interact to create patterns of vulnerability, regardless of its genetic substrate.

Such vulnerabilities have long been recognized. Even as observers began asserting racial arguments of disease susceptibility in the nineteenth century, they saw that a wide range of social factors created susceptibility to epidemic disease. After studying an outbreak of measles among the indigenous populations of Fiji in 1875, W. Squire concluded, "We need invoke no special susceptibility of race or peculiarity of constitution to explain the great mortality." He blamed social conditions, especially "want of nourishment and care." In 1909, anthropologist Aleš Hrdlička reached a similar conclusion about American Indians: "Doubtless much of what now appears to be greater racial susceptibility is a result of other conditions." Sherburne Cook came to believe that disease amongst indigenous populations worldwide "acted essentially as the outlet through which many other factors found expression."

Malnutrition provides the most obvious, and prevalent, demonstration of the links between social conditions, environmental conditions, and disease. In addition to causing deficiency diseases, such as rickets and pellagra, malnutrition increases susceptibility to infection. Some vitamin deficiencies cause skin breakdown, eroding the first barrier of defense against infection. Protein deficiencies impair both cellular and humoral responses. Malnutrition during infancy and childhood has particularly devastating effects on subsequent immune function. Certain diseases have more specific connections to nutrition. Malnutrition, especially vitamin A deficiency, increases mortality from measles. Malnourished children are more likely to die from chicken pox. Such interactions create "a vicious circle. Each episode of infection increases the need for calories and protein and at the same time causes anorexia; both of these aggravate the nutritional deficiency, making the patient even more

susceptible to infection.” Understanding these relationships, scientists have realized that malnutrition “is the most common cause of secondary immunodeficiency in the world.”

Historians have thoroughly documented the impact of malnutrition on disease susceptibility. Such connections have clear importance for American Indians, who faced both disease and social disorder following European colonization. As Cronon describes, villages disrupted by disease and social breakdown “often missed key phases in their annual subsistence cycles—the corn planting, say, or the fall hunt and so were weakened when the next infection arrived.” This would have been particularly damaging for the many populations that eked out only a precarious subsistence before European arrival. Although some writers have described American Indians living in bountiful harmony with their environment, archaeologists and physical anthropologists have shown that many groups were terribly malnourished. The accomplishments of the Mayan civilization might have been undone by climate change, crop failures, and famine. Disease, malnutrition, and violence made Mesoamerican cities as unhealthful as their medieval European counterparts, with life expectancies of 21 to 26 years. The Arikaras had life expectancies as low as 13.2 years. Careful study of skeletal remains has found widespread evidence of nutritional deficiencies, with health conditions worsening in the years before contact with Europeans. Baseline malnutrition, especially in the large agricultural societies in Mexico and the Andes, left American Indians vulnerable—at the outset—to European diseases. When the conditions of colonization disrupted subsistence, the situation only grew worse.

Malnutrition may be the most obvious factor, but it was only one of many. Environmental historians have shown how physical environments can leave populations susceptible to disease. Lowland Ecuadorians, weakened by endemic parasites and intestinal diseases, were more vulnerable to European infections than their highland compatriots. After Spanish arrival in Mexico, a “plague of sheep” destroyed Mexican agricultural lands and left Mexicans susceptible to famine and disease. Colonization introduced a host of damaging changes in New England. Deforestation led to wider temperature swings and more severe flooding. Livestock overran Indian crops and required pastures and fences, leading to frequent conflict and widespread seizure of Indian land. Europeans also introduced pests, including blights, insects, and rats. All of these changes fueled rapid soil erosion and undermined the subsistence of surviving Indian populations. More dramatic environmental events also wreaked havoc. Drought, earthquakes, and volcanic eruptions undermined resistance to disease in Ecuador in the 1690s. A devastating hurricane struck Fiji in 1875, exacerbating the measles outbreak there. As one observer commented, “Certainly for the last 16 years there has been experienced no such weather, and nothing could be more fatal to a diseased Fijian than exposure to it.”

Historians and anthropologists have also documented many cases in which the varied outcomes of specific populations depended on specific social environments. The Lamanai Mayas, heavily colonized by the Spanish regime, had higher mortality than the more isolated Tipu Mayas. While much of Peru

suffered severely, the region of Huamanga lost only 20 percent of its population between 1532 and 1570, the result of “a high birth rate, the relative immunity of remote high-altitude areas to disease, shrewd politics, and good luck.” The Pueblos suffered when “the endemic problems of drought and famine were superimposed upon the economic disruption caused by the Spanish drain on food and labor.” Severe outbreaks of smallpox and erysipelas in Peru from 1800 to 1805 reflected a combination of drought, crop failures, famines, mining failures, and economic collapse. The introduction of specific epidemics reflected specific historical events. Dauril Alden and Joseph Miller traced outbreaks of smallpox from West African droughts, through the middle passage of the slave trade, to Brazil. Measles raced down the political hierarchy in Fiji in 1875 as a series of conferences carried news of a treaty with the British empire, along with the virus, from the royal family to regional and local leaders throughout the island. Local variability and contingency led Linda Newson to conclude that “levels of decline and demographic trends were influenced by the size, distribution, and character of populations, especially their settlement patterns, social organization, and levels of subsistence.” Even in the late twentieth century, specific social factors left isolated indigenous populations vulnerable to European pathogens. Magdalena Hurtado, who has witnessed first-contact epidemics in South America, emphasizes the adverse consequences of “sedentism, poverty, and poor access to health care.”

Studies of North American tribes in the nineteenth and twentieth centuries have found similar local variability. Geographer Jody Decker shows how a single epidemic among the northern Plains tribes had disparate effects, “even for contiguous Native groups,” depending on “population densities, transmission rates, immunity, subsistence patterns, seasonality and geographic location.” Drought and famine left the Hopis particularly susceptible to an epidemic in 1780. The Mandans suffered severely from smallpox in 1837: famine since the previous winter had left them malnourished, and cold, rainy weather confined them to their crowded lodges. When smallpox struck, they had both high levels of exposure and low levels of resistance. As Clyde Dollar concludes, “It is no wonder the death rate reached such tragically high levels.” Once North American tribes came under the care of the federal governments in the United States and Canada, they often suffered from malnutrition and poor sanitation. Mary-Ellen Kelm, who has studied the fates of the Indians of British Columbia, concludes that “poor Aboriginal health was not inevitable”; instead, it was the product of specific government policies.

Comparative studies have particular power for demonstrating the local specificity of depopulation. Stephen Kunitz has shown that Hawaiians suffered more severely than Samoans, a consequence of different patterns of land seizure by colonizing Europeans. The Navajo did better than the neighboring Hopi because their pastoral lifestyle adapted more easily to the challenges imposed by American settlers. In these cases similar indigenous populations encountered similar colonizers, with very different outcomes: “The kind of colonial contact that occurred was of enormous importance.” Kunitz’s cases demonstrate that “diseases rarely act as independent forces but instead are shaped by the different contexts in which they occur.”

Paralleling this work, some historians have begun to provide integrated analyses of the many factors that shaped demographic outcomes. Any factor that causes mental or physical stress—displacement, warfare, drought, destruction of crops, soil depletion, overwork, slavery, malnutrition, social and economic chaos—can increase susceptibility to disease. These same social and environmental factors also decrease fertility, preventing a population from replacing its losses. The magnitude of mortality depended on characteristics of precontact American Indian populations (size, density, social structure, nutritional status) and on the patterns of European colonization (frequency and magnitude of contact, invasiveness of the European colonial regime). As anthropologist Clark Spencer Larsen argues, scholars must “move away from monocausal explanations of population change to reach a broad-based understanding of decline and extinction of Native American groups after 1492.”

The final evidence of the influence of social and physical environments on disease susceptibility comes from their ability to generate remarkable mortality among even the supposedly disease-experienced Old World populations. Karen Kupperman has documented the synergy of malnutrition, deficiency diseases, and despair at Jamestown, where 80 percent of the colonists died between 1607 and 1625. Smallpox mortality, nearly 40 percent among Union soldiers during the Civil War, reflected living conditions and not inherent lack of innate or adaptive immunity. Mortality among soldiers infected with measles, which exceeded 20 percent during the United States Civil War, reached 40 percent during the siege of Paris in the Franco-Prussian War. Poverty and social disruption continue to shape the distribution of disease, generating enormous global disparities with tuberculosis, HIV, and all other diseases.

Is it possible to quantify the variability, to delineate the relative contribution of potential genetic, developmental, environmental, and social variables? Detailed studies have documented “considerable regional variability” in American Indian responses to European arrival. Many American Indian groups declined for a century and then began to recover. Some, such as the natives of the Bahamas, declined to extinction. Others, such as the Navajo, experienced steady population growth after European arrival. More precise data exist for select groups. Newson, for instance, has compiled data about die-off ratios, the proportion of those who died to those who survived. While die-off ratios were as high as 58:1 along the Peruvian coast, they were lower (3.4:1) in the Peruvian highlands. In Mexico they varied between 47.8:1 and 6.6:1, again depending on elevation. They ranged from 5.1:1 in Chiapas to 24:1 in Honduras and 40:1 in Nicaragua. Mortality rates from European diseases among South Pacific islanders ranged between 3 percent and 25 percent for measles, and 2.5 percent to 25 percent for influenza. Such variability among relatively homogeneous populations, with die-off ratios differing by an order of magnitude, most likely reflects the contingency of social variables. But most of these numbers are, admittedly, enormous: a 4:1 die-off ratio indicates that 75 percent died. Why did so many populations suffer such high baseline mortality? Does this reflect a shared genetic vulnerability, whose final intensity was shaped by social variables? Or does it reflect a shared social experience, of pre-existing nutritional stress

exacerbated by the widespread chaos of encounter and colonization? Both positions are defensible.

The variability of outcomes reflected in the different fates of different Indian populations provides powerful evidence against the inevitability of mortality. It undermines popular claims, made most influentially by Henry Dobyns, that American Indians suffered universal mortality from infectious diseases. Noble David Cook, for instance, argues that the vulnerability was so general that Indians died equally whatever the colonial context, "no matter which European territory was involved, regardless of the location of the region. It seemed to make no difference what type of colonial regime was created." Such assertions, which reduce the depopulation of the Americas to an inevitable encounter between powerful diseases and vulnerable peoples, do not match the contingency of the archaeological and historical records. These, instead, tell a story of populations made vulnerable.

One could argue that the differences in American and European disease environments, the nutritional status of precontact Americans, and the disruptions of colonization created conditions in which disease could only thrive. Only a time traveler equipped with a supply of vaccines could have altered the demographic outcomes. But it is also possible that outcomes might have been different. Suppose Chinese explorers, if they did reach the Americas, had introduced Eurasian diseases in the 1420s, leaving American populations two generations to recover before facing European colonization. Suppose smallpox struck Tenochtitlan after Cortés's initial retreat and not during his subsequent siege of the city. An epidemic then might have been better tolerated than during the siege. Or suppose that the epidemics of 1616–1617 and 1633–1634 struck New England tribes during the nutritionally bountiful summers and not during the starving times of winter (or perhaps it was because of those starving times that the epidemics tended to appear in winters). The historic record of epidemic after epidemic suggests that high mortality must have been a likely consequence of encounter. But it does not mean that mortality was the inevitable result of inherent immunological vulnerability.

Consider an analogous case, the global distribution of HIV/AIDS. From the earliest years of the epidemic, HIV has exhibited striking disparities in morbidity and mortality. Its prevalence varies between sub-Saharan Africa and developed countries and between different populations within developed countries. Few scientists or historians would argue that these disparities between African and Europeans or between urban minorities and suburban whites exist because the afflicted populations have no immunity to HIV. Instead, the social contingency of HIV on a local and global scale has long been recognized. We should be just as cautious before asserting that no immunity led to the devastation of the American Indians.

Historians and medical scientists need to reassess their casual deployment of deterministic models of depopulation. The historic record demonstrates that we cannot understand the impact of European diseases on the Americas merely by focusing on Indians' lack of immunity. It is certainly true that epidemics devastated American Indian populations. It is also likely that genetic mechanisms of disease susceptibility exist: they influence the

susceptibility of American Indians—and everyone else—to infectious disease. What remains in doubt is the relative contributions of social, cultural, environmental, and genetic forces. Even when immunologists demonstrate that a wide variety of genes contribute to susceptibility to infectious disease, it will likely remain unknown how these factors played out among American Indians in past centuries. Demographic data, meanwhile, provide convincing evidence of the strong impact of social contingency on human disease. This uncertainty leaves the door open for the debates to be shaped by ideology.

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.