

The Question of Racial Immunity to Yellow Fever in History and Historiography

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The belief that West and Central Africans and their descendants in the New World enjoy an innate immunity or resistance to yellow fever persists in the writings of many historians. They offer three arguments that such a racial immunity actually exists: first, that a consensus on the matter prevailed among historical observers of the disease; second, that patterns of lethality during yellow fever epidemics demonstrate it to be true; and third, because a heritable resistance to malaria is known to have spread within these populations, a similar resistance to yellow fever must have developed as well. But in fact there was never a consensus among medical observers that black immunity to yellow fever actually existed, the evidence from epidemics indicates that in fact it did not, and the analogy to the very real and well-documented evolutionary consequences of endemic malaria is not apt. As there is no evidence supporting the belief of black immunity to yellow fever, it is time for historians to discard it.

William Crawford Gorgas, the US Army sanitarian who oversaw the elimination of yellow fever from the Panama Canal Zone during the construction of the waterway, frequently declared that the success of the project demonstrated that the tropics could be made safe for habitation by “the white man” (see, e.g., Gorgas 1915: 289). Later observers found this statement profoundly racist. Fred L. Soper, the US epidemiologist, sanitarian, and head of the Pan American Health Organization, noted how he and his colleagues looked back on Gorgas’s comment with deep disapproval. “Now,” Soper wrote in his diary in 1965, “we should be ready to make the tropics safe for the native of the tropics no matter what the color of skin may be” (Soper 1965). To be sure, Gorgas was the product of a profoundly racist era, and his view that white settlement of the tropics was necessary for the region to reach its full productive potential reflected this fact (see, e.g., Gorgas 1915: 289–92). But the basis of Gorgas’s point was not, as Soper’s comment supposed, a disregard for the health and well-being of people of other races. It was the widespread belief that black people enjoyed an innate immunity—or at least a very high resistance—to yellow fever, and so the tropics were already safe for them.¹

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1. Contemporary observers understood. The editors of the *New York Times*, e.g., lauded Gorgas’s accomplishment in Panama but disagreed with his conclusion regarding its significance. The success of the fight against yellow fever during the construction of the canal, they opined, demonstrated only that the tropics could be made safe for white men and their families for a limited period of time: “For permanent

This belief persists in the writings of many historians. The work of Kenneth Kiple over many years played a leading role in spreading the view that genetic immunity to yellow fever existed in Africans and their descendants, whom he argued at worst suffered only mild cases of the disease (see, e.g., Kiple 1984, 2001; Kiple and Conèe Ornelas 1996; Kiple and Higgins 1992; Kiple and King 1981; Kiple and Kiple 1977). Todd Savitt similarly maintained, “Though blacks did contract yellow fever, they rarely died of it” (Savitt 1978: 241; see also Savitt 1988). Jo Ann Carrigan also believed that black people were naturally resistant to the worst effects of the disease, as did K. David Patterson (Carrigan 1970: 576–78; 1988: 62–63; 1994: 253–59; Patterson 1992: 861–62). The economic historians Philip Coelho and Robert McGuire argued that the widespread adoption of slavery to meet the labor needs of the British sugar-producing colonies of the Caribbean was the product of blacks’ innate immunity to yellow fever (Coelho and McGuire 1997). J. R. McNeill contended that “West Africans and people of West African descent almost certainly carry an inherited partial immunity to yellow fever whether or not they carry conferred immunity” from previous infection (McNeill 1999: 178–79). In his recent, award-winning book, McNeill moderated this position somewhat, but still insisted that such heritable immunity “probably does exist” (McNeill 2010: 46). Even this assertion is made all the more remarkable by his admission that “the historical evidence does not resolve the question of heritable immunity to yellow fever” and that “[g]enetic immunity (or resistance) to yellow fever is not necessary for the arguments in this book” (McNeill 2010: 45–46). He sought to justify his position by claiming, “If yellow fever in its present form has been present in West Africa for at least 3,000 years as the genomic evidence implies, it would be astonishing to evolutionary biologists if human populations in its midst did not evolve some form of resistance to such a virulent disease” (McNeill 2010: 45).²

An unheeded few have dissented. Philip Curtin, the pioneering historian of the roles of disease in the slave trade and in European imperialism in the tropics, acknowledged the outside possibility of genetic immunity but insisted that the acquired immunity to yellow fever gained through exposure during childhood, when cases are typically very mild, was by far the more important factor when considering the lesser mortality due to the disease for blacks compared to whites in the Caribbean during the seventeenth and eighteenth centuries (Curtin 1968). More recently, Sheldon Watts argued strenuously against the existence of any black genetic immunity to yellow fever. His work, too,

residence the tropics still are, and probably always will be, for black and brown men alone” (New York Times 1914). Although malaria could and did also claim many lives—as its toll during the construction of the Panama Canal illustrates—the threat of the tropics in the popular imagination in the United States was dominated by the fear of yellow fever (see, e.g., Espinosa 2009; Humphreys 1992).

2. It is perhaps worth underscoring that McNeill explicitly limits his claims to black people of West and Central African descent on the basis that genetic immunity could only be “a result of the disease environment of one’s ancestors—not a matter of race or skin color” (McNeill 2010: 46), but as nearly all of the black population of the Americas shared such ancestry (on the historical evidence, see, e.g., Curtin 1969; Klein 2010; for confirmation in recent mtDNA analyses, see Salas et al. 2004), a point that McNeill (2010: 45) concedes, this does not actually constitute a matter of disagreement with the other authors listed.

has had little apparent effect (Watts 2001).³ The belief that black people, at least those of West and Central African descent, enjoy—or “almost certainly” enjoy—an innate immunity or resistance to yellow fever remains dominant among historians of the topic. This belief is almost certainly wrong.

An Absence of Historical Consensus

Proponents of a genetic immunity to yellow fever among black people point to, as supporting evidence for their view, a consensus on its existence among historical observers of the disease. One of the earliest and most cited studies claimed that the “medical literature from the eighteenth century onward is replete with tracts on yellow fever that never fail to speak of black immunity” (Kiple and Kiple 1977: 434). In fact, however, many doctors of that period rejected the claim that black people enjoyed an innate resistance to the disease.

In one of the best-selling volumes on tropical diseases of the nineteenth century, James Johnson attributed Africans’ “general exemption from bilious and hepatic diseases” such as yellow fever not to some inherent characteristic but instead to their keeping their skin soft, and so avoiding excessive perspiration, by regularly rubbing themselves with oil (Johnson 1818: 166).⁴ One prominent reviewer mocked Johnson’s assertion, but not to argue instead for racial immunity. John Eberle, the editor of the *American Medical Recorder*, wrote:

A native of Africa, or of Asia, who has been “inured from his infancy to the high temperatures of his climate,” is more exempt from bilious and hepatic diseases, not because he puts oil on his skin, but for the very same reason that a native West Indian, who does not oil his skin, is less subject to the disease of his climate than

3. Unfortunately, part of the ineffectiveness of Watts’s effort can be attributed to weaknesses in the arguments he mustered. Watts argued that black immunity does not exist on the grounds, first, that doctors who depended on slaveholders for patronage (and, of course, the slaveholders) had an economic and ideological stake in the idea that blacks were not affected by yellow fever; second, that yellow fever originated in the Americas, and so Africans and their descendants had not been exposed to the disease over the many generations necessary for any genetic immunity to appreciably spread; and third, that yellow fever epidemics occurred in late-twentieth-century West Africa demonstrates that blacks are susceptible to the disease. The first of these arguments, while patently true, does not establish that those doctors were wrong when they claimed that black people did not suffer from yellow fever, or at least did not suffer as much as white people did. The second argument has been undercut by genetic studies of the yellow fever virus conducted since Watts wrote that demonstrated that the disease is definitely of Old World origin (see, e.g., Sall et al. 2010). Watts’s third argument is much stronger, though a skeptic might insist that the West African epidemics only disprove an absolute immunity held uniformly by all, rather than just some, black people (an extreme position that the writings of Kiple, among others, nevertheless often appear to advocate) and provide little proof against black resistance to the disease. As it turns out, these counterarguments have little merit: case mortality rates of these recent West African epidemics were similar to those of epidemics in the United States during the nineteenth century. I will discuss black mortality in yellow fever epidemics in depth in the following text.

4. Johnson’s *Influence of Tropical Climates* went through six British editions and was published in New York as well. The volume was so popular that, after Johnson’s death, his co-author for the sixth edition, James Ranald Martin, adopted the title for an entirely new work (see Martin 1856).

a stranger.—They are habituated, from infancy, to the combined impression of atmospheric heat and miasma. (Eberle 1821: 542–43)

According to Eberle, lifelong exposure to the disease environment was the source of immunity to yellow fever, not race (or, for that matter, oiled skin).

In fact, many well-known writers on the subject concluded, like Johnson and Eberle, that yellow fever immunity among black people was not inherent but had to be acquired. Robert Jackson, a surgeon in the British army and author of three widely read books on the fevers of the Caribbean, observed that yellow fever never seized black people coming “immediately from the coast of Africa,” but black people coming from “Europe, or the higher latitudes of America, are not by any means exempted from it” (Jackson 1791: 250).⁵ Another author similarly noted that blacks “transported directly from Africa to the Antilles” did not contract yellow fever, but “negroes born in the north do not enjoy this immunity” (Hester 1850: 82).

Laurent-Jean-Baptiste Bérenger-Féraud, chief doctor of the French Navy, repeatedly warned his readers against the belief that black people had an inherent immunity to yellow fever. Their immunity, he insisted, like that of anyone else had to be acquired through previous infection. He noted that whenever yellow fever had been absent from a location for many years, upon its return blacks and mulattoes were as susceptible to it as whites. He pointed to the case of Gorée, the island trading post a mile offshore from Dakar in Senegal. When yellow fever arrived in 1830 after an absence of more than half a century, many of the island’s mulatto and black inhabitants were struck down by the disease. During the next epidemic, just seven years later, it was true that their immunity seemed complete, but this was only the result of their previous exposure. The experience in Cayenne, French Guiana, in 1850 was similar, he wrote: in the first yellow fever epidemic in fifty years, the inhabitants—whites, mulattoes, and blacks alike—paid a heavy price. Neither race nor native residence mattered when they were exposed to the disease for the first time (Bérenger-Féraud 1878: 272–73; see also Bérenger-Féraud 1874: 300–302).

It is true that many, perhaps even most, nineteenth-century writers, and particularly those in the United States, repeated the story that black people were immune to yellow fever, but many others examined the evidence and came to the opposite conclusion. There was no consensus among well-informed observers on this topic.

The Evidence from Epidemics

Patterns of yellow fever mortality in the Caribbean during the time when yellow fever was endemic to the region do not shed much light on the question of black genetic immunity. Nearly all newly arriving black people were brought from parts of West Africa where yellow fever was also endemic and so had acquired immunity to the

5. The work was also published in Philadelphia, see Jackson (1795: 162–63).

disease from prior infection.⁶ Nearly all newly arriving whites, by contrast, had no previous exposure to the disease. The dramatic differences in the deaths caused by yellow fever across races can be explained by either acquired immunity or black genetic immunity and so provide little leverage on the question. Epidemics elsewhere are more revealing, however, and they indicate that black people have no innate resistance to the disease.

In 1793, Philadelphia was struck by yellow fever for the first time in more than three decades. In his search for more information on the disease, prominent physician (and signer of the Declaration of Independence) Benjamin Rush came across a published letter by Dr. John Lining describing an epidemic in Charleston in 1748. Lining had written, “There is something very singular in the constitution of the Negroes, which renders them not liable to this fever; for tho’ many of these were as much exposed as the nurses to the infection, yet I never knew one instance of this fever amongst them” (Lining 1756: 374). Rush had this quote reprinted in one of the city’s newspapers and urged the city’s black population to aid the sick.⁷

But perhaps unlike the black people of Lining’s Charleston, the black residents of Philadelphia had not been previously exposed to the disease, and they proved just as susceptible to it as the city’s white residents. Absalon Jones and Richard Allen, the two black ministers who helped lead efforts to care for yellow fever patients and bury the dead, wrote with a barely contained fury:

The public were informed that in the West-Indies and other places where this terrible malady had been, it was observed the blacks were not affected with it. Happy would it have been for you, and much more so for us, if this observation had been verified by our experience. When the people of colour had the sickness and died, we were imposed upon and told it was not with the prevailing sickness, until it became too notorious to be denied, then we were told some few died but not many. (Jones and Allen 1794: 15)

Working from burial records, Jones and Allen suggested that about 240 black people had died of yellow fever in 1793; the first US Census, conducted three years before, had counted 2,489 blacks in Philadelphia (Jones and Allen 1794: 15–16). The total number of yellow fever deaths in 1793 was given as 4,041 out of a population that totaled 54,391 in 1790 (Carey 1794: 121). Calculations of absolute mortality rates are problematic for reasons discussed in the following text, but it is safe to say that these figures do not demonstrate a dramatic difference in mortality across races: white or black, the disease had killed roughly one in ten of the total population. Rush

6. Nearly all, but not all: Rana Hogarth (2012: ch. 1) documents the story of a shipload of black troops recruited from Sierra Leone for the British West India Regiments who suffered a full-blown yellow fever epidemic upon arriving in Barbados in 1815.

7. Rush’s notice was printed over his pseudonym as Benezet (1793). Mathew Carey, the Philadelphian printer who became a widely read chronicler of the epidemic, again repeated Lining’s claim in his book—first hastily published while the fever still raged—with the caveat that blacks in Philadelphia “did not escape the disorder; however, the number of them that were seized with it, was not great” (Carey 1793: 78). Rush admitted his authorship of the original notice (Rush 1794: 95–96).

acknowledged his terrible mistake, writing that black people “took the disease, in common with the white people, and many died of it,” but he also noted that he had treated many black patients and had found that “[t]he disease was lighter in them, than in white people” (Rush 1794: 97). Despite all evidence to the contrary, belief in black immunity—of some sort—survived in Rush’s contradictory remarks.⁸

During the nineteenth century, New Orleans was the US city most often and most severely plagued by yellow fever, and historians who have advocated black immunity have often pointed to the infamous 1853 epidemic as providing strong evidence for their claims (see, e.g., Kiple and King 1981: 42; Kiple and Kiple 1977: 424; Patterson 1992: 861). A systematic examination of deaths in New Orleans during this epidemic, however, supports the opposite conclusion. As Jonathan Pritchett and Insan Tunali wrote, absolute mortality rates—the ratio of victims to population—are misleading when discussing yellow fever. The population at risk, the denominator of the absolute mortality rate, is hard enough to determine in ordinary circumstances given high rates of immigration such as those seen in New Orleans (and, earlier, Philadelphia), but virtually impossible during epidemics, when panic drove many to abandon the city. Instead, the proportional mortality ratio—the ratio of yellow fever deaths to deaths from all causes—is the more informative statistic, and given the profiles of the disease and of other killers, age at time of death must also be considered. In their analysis of the city’s burial records that also took into account the age and gender of the deceased, Pritchett and Tunali found that the probability that yellow fever was the cause of death for the city’s African American population was not statistically distinguishable from the probability that yellow fever caused deaths among white New Orleans natives. In contrast, deaths among whites born elsewhere in the United States or abroad were five to eighteen times more likely to have been the result of yellow fever than those of whites native to the city (Pritchett and Tunali 1995).⁹ Migrant status, not race, explained the differences in the risk of dying from the disease.

As we have seen, in the Philadelphia yellow fever epidemic of 1793, similar proportions of the city’s black and white populations were killed by the disease. In the New Orleans epidemic of 1753, among native residents, yellow fever accounted for a similar share of black and white deaths from all causes. But if black people were particularly likely to fall ill in these epidemics—due perhaps to greater exposure to the homes of the sick, through nursing or overcrowding—then these figures are not necessarily inconsistent with claims of an innate resistance. That is, if proportionally many more black yellow fever cases yielded an equivalent number of deaths, then Rush’s (1794: 97) comment that “[t]he disease was lighter in them, than in white people” and its echoes by historians could be correct after all. The records for these

8. For more on the Philadelphia epidemic, see Powell (1949) and Coleman (1987). For a recent treatment of the exchanges among Jones and Allen, Rush, and Carey as literature and as literary influences, see Otter (2010: 25–58).

9. That these authors apparently felt compelled to repeat the potential for genetic yellow fever resistance among black people despite the results of their analysis again demonstrates the pervasiveness of this narrative; see Pritchett and Tunali (1995: 528).

epidemics, however, do not provide information on the numbers of blacks and whites who were sickened, which would allow us to calculate what is known as the case mortality rate. This information is available for recent epidemics.

Although the knowledge that the disease is transmitted by mosquitoes and the availability of an effective vaccine make yellow fever epidemics much less of a threat now than in the past, several major epidemics and a number of serious outbreaks have occurred in West Africa over the last few decades. As yellow fever is endemic to the forests of West Africa and the region was the origin of most slaves imported to the New World, these epidemics provide valuable evidence on the possibility of black immunity to the disease in the Caribbean and surrounding areas. Major epidemics in the United States typically had case mortality rates of about 20 percent (Duffy 1968), although there was considerable variation: for example, the massive 1878 Mississippi Valley epidemic was on the whole actually relatively mild in its lethality, killing only about one in six of the 120,000 who suffered symptoms (see Chaillé 1881: 73).¹⁰ If the West African epidemics took a similar toll among the sick, then we should conclude that black immunity advocates are mistaken in their claim that blacks who contract the disease suffer much less from it than whites.

The record of these epidemics is unambiguous. From 1977 to 1983, a series of yellow fever outbreaks occurred in Ghana; of the 1,195 people who exhibited symptoms, 394—about one in three—were killed by the disease. During the 1978 and 1979 epidemics in the Gambia, yellow fever caused approximately 1,600 deaths, taking the life of one in five of those stricken with the disease (World Health Organization 1989: 41). An epidemic of yellow fever struck Burkina Faso in 1983: some 2,500 to 3,500 people developed symptoms, and approximately a third of them died (Baudon et al. 1986: 880). An outbreak of roughly 1,500 cases of yellow fever occurred in southwestern Mali in 1987, and half of the afflicted were killed (World Health Organization 1989: 41). In Guinea during 2000 and 2001, 833 cases of yellow fever were reported with 246 deaths, a case mortality rate of just less than 30 percent (World Health Organization 2005: 51).

The largest of the recent West African epidemics were in Nigeria. In 1986 and 1987, the country was ravaged by two severe epidemics of yellow fever, sickening an estimated thirty thousand individuals and killing roughly ten thousand (World Health Organization 1989: 40–41). Detailed surveys of three villages in the affected area confirmed a case mortality rate of at least 20 percent (Nasidi et al. 1989).¹¹ Epidemics recurred from 1988 to 1991, causing a reported 14,826 illnesses and 3,004

10. The very high case mortality rates implied by the near total mortality of various military expeditions to the Caribbean in the seventeenth and eighteenth centuries are not appropriate points of comparison because, first, these expeditions were composed almost exclusively of men in their late teens through thirties, the ages at which the disease is most fatal; second, lack of medical care and shortages of food and water exacerbated the condition of the sick (with scurvy also a possible cause of death or a contributing factor for yellow fever victims); and third, the expeditions were probably hit with multiple epidemics simultaneously (with typhus, typhoid, malaria, and dengue all possible contributing factors as well as causes of death in their own right).

11. This figure was considered low, however, because the largest of these surveys was conducted by government officials and there was considerable evidence that “those interviewed feared reprisals for not

deaths, a case mortality rate of just more than one in five (World Health Organization 1992: 246–48; 1995: 66). Another outbreak occurred in 1994, with 1,227 cases and 415 deaths; that is, yellow fever killed more than one in three of those it attacked. The evidence against any inherent black immunity to yellow fever was sufficient to convince the Nigerian government to follow up its emergency vaccination campaigns with an effort to vaccinate the country's entire population under age thirty against the disease (World Health Organization 1998: 355–57).

Yellow Fever and Evolutionary Pressure

The strongest argument offered for the claim that West and Central Africans and their descendants in the Caribbean had and continue to have a genetic immunity to yellow fever is that provided by the example of malaria (see, among others, Kiple and Kiple 1977: 420, and McNeill 2010: 44–45). The resistance to malaria provided by the “sickle cell” gene, *HbS*, which is much more common in people whose ancestors lived in the malarial zones of Africa, the Mediterranean, the Middle East, and India, is well documented (see, e.g., Piel et al. 2010).¹²

The sickle cell trait provides impressive protection against even the most deadly malaria parasite, *Plasmodia falciparum*. The red blood cells of individuals who carry one copy of the *HbS* gene have weaker outer membranes than those of people without this trait. When invaded by *P. falciparum*, these weaker cell walls exhibit greater damage that is more readily apparent to the body's immune system, and so infected cells are more quickly removed from the bloodstream, in turn reducing the density and growth rates of the parasites (see, e.g., Ayi et al. 2004). As a result, having a single copy of the sickle cell gene reduces the risk by half that infection by *P. falciparum* will cause a fever episode and the risk of a potentially fatal severe case of the disease by approximately 90 percent (Ackerman et al. 2005; Hill et al. 1991). People with this gene are therefore better able to survive their contact with the plasmodia that cause malaria, and consequently, where the disease is present, they are able to have—and pass on their genes to—more children than those without it, notwithstanding the fact that having two copies of the *HbS* gene causes sickle cell anemia, which is frequently fatal in childhood. With the evolutionary pressure of malaria continuously applied over many generations, the *HbS* gene became more and more common: in many parts of Africa with endemic malaria, the gene is carried by more than 20 percent of the population (Flint et al. 1998). In fact, the selective pressure of malaria is so strong that mutations that offer some protection against it are responsible for at least seven other genetic diseases that are found in various malarial regions around the world (Kwiatkowski 2005).

having officially reported deaths in their families”; even so, the resulting case mortality rate in this village was a far from trivial at 11.3 percent (Nasidi et al. 1989: 404).

12. It likely bears noting that the prevalence of even the sickle cell trait does not actually coincide with race: the trait is three times more common among (white) Greeks than among black Americans, who in turn are twice as likely to carry the trait as indigenous South Africans (see Graves 2010: 46–47).

Given that yellow fever is a more deadly ailment than even *P. falciparum* malaria, it is possible that genetic resistance to yellow fever may have similarly developed in those parts of Africa where that disease was long endemic. Possible, but not at all likely. There are crucial differences between malaria and yellow fever that suggest very different levels of evolutionary pressure at work. Malaria can infect and reinfect a single individual many times, and it generally causes milder symptoms on each occasion as the victim's immune system grows better equipped to fight the invading pathogens. It is much more likely to kill people who are suffering their first infection than those who have had previous experience with the disease; among populations that have long lived within malarial areas that means it is children who are most frequently struck down.¹³ This aspect of malaria causes it to exert a strong environmental pressure on human populations. The differences in the mortality rates between those with and without genetic resistance to the disease occurs primarily among children, that is, people who have yet to reach their reproductive years. When a genetic mutation occurs that offers some resistance to the disease, those who carry it will more often survive childhood to have children of their own than those who do not, and the mutation will spread through the population as a result. This explains the many otherwise harmful genetic traits that, although rare elsewhere, are relatively common in populations inhabiting various parts of the world where malaria has long been endemic: any advantageous mutation that arises in a human population exposed to malaria will become more and more common with the passage of time.

Yellow fever, however, generally presents extremely mild symptoms in children.¹⁴ In contrast to the staggering harm the virus does among adult victims, cases in children are very rarely fatal and often go unnoticed among the mundane sniffles, fevers, and colds of childhood, and anyone who survives the illness thereafter enjoys a complete and lifelong immunity. Where populations have lived for generations with endemic yellow fever, then, the disease does not generally kill people before they reach reproductive age, regardless of whether they may carry a genetic mutation that offers some resistance to the disease. In fact, where it is endemic or frequently epidemic, yellow fever rarely kills anyone at all other than newly arrived adults: longtime residents are immune due to mild cases they contracted during childhood. It was for this reason that yellow fever was known around the Caribbean as the stranger's disease during the centuries that it was endemic to the region.

This means that any person who carried a genetic resistance to yellow fever would have a reproductive advantage over those who did not in only two circumstances. The

13. In 2010, it is estimated that 65 percent of those killed by malaria worldwide were less than fifteen years of age, and 54 percent were less than five years of age; where the extent of malaria's endemicity is larger, so is the proportion of its victims who are children (Murray et al. 2012).

14. Although several of the West African epidemics described in the preceding text primarily affected children, this pattern was the result of older populations enjoying protection from colonial-era vaccinations (Meegan 1994: 115). Data on mortality by age during yellow fever epidemics before the introduction of vaccination, however, clearly shows how adults are much more susceptible to the ravages of the disease. During the 1870 yellow fever epidemic in Barcelona, e.g., children between 1 and 10 years of age suffered an absolute mortality rate of less than one-tenth that of adults aged twenty-one to fifty (Canela Soler et al. 2009: 296–97).

first is when migrating, as previously unexposed adults, into an area with endemic yellow fever. A stranger carrying a trait that ensured only a mild case of yellow fever would enjoy greatly increased chances of going on to have additional children compared to his or her companions. Migration and resettlement in West and Central Africa was certainly sufficiently common to allow this to have occurred with some frequency. But once the migration was completed, the offspring of the migrants—genetically advantaged or not—would safely contract yellow fever as children, and the evolutionary pressure would dissipate before genetic resistance became appreciably more common in the population.

The second situation in which genetic resistance would convey a reproductive advantage is at the extreme fringes of the disease's range, in locations where yellow fever epidemics struck only rarely. In such places, over several decades, an adult population could grow that had not been exposed as children during the previous epidemic. When yellow fever returned, a person with genetic resistance would more often live to reproduce another day than his or her neighbors, and the mutation could be expected to grow more common in the next generation. Here the issue is that the time between epidemics must fall within a relatively narrow window. If the next epidemic comes too soon, it will strike almost exclusively among children, killing few if any and having little impact on the relative rates of reproduction of people with and without genetic resistance. If it comes too late, those without genetic resistance will have already passed on their genes; they may be struck down, but their children will survive. In either case, the prevalence of the protective gene in the next generation would remain unaffected. Further complicating the picture is that to speak of "generations" is nearly always misleading; people virtually never are born, raised, find mates, and procreate on identical timetables. For much of any population subject to periodic epidemics, yellow fever always arrived either too early or too late to constitute a selective pressure.

Even the most frequent and devastating epidemics are quite ineffective at generating evolutionary pressure, as research on the rapid spread of the CCR5- Δ 32 mutation across Europe demonstrates. The CCR5 allele provides the blueprint for a particular receptor on the surface of the T cells of the human immune system; the Δ 32 variant deletes thirty-two base pairs from the gene and so, in individuals with copies from both parents, eliminates this receptor entirely. The trait became a topic of intense scientific investigation when it was discovered that one copy provides some resistance—and two copies very nearly complete immunity—to HIV-1, the virus that causes AIDS. Differences in surrounding alleles indicate that CCR5- Δ 32 originates from a single mutation event that took place at most a few thousand and perhaps just several hundred years ago, probably in Northern Europe (Libert et al. 1998). Today, the frequency of CCR5- Δ 32 is much higher than would be expected from random genetic drift of a mutation that did not affect reproductive chances: allele frequencies of CCR5- Δ 32 average about 10 percent across Europe, ranging from 16 percent in Finland to 4 percent in Italy (the frequency of CCR- Δ 32 is 1 to 2 percent in northern Africa, but the variant is virtually unknown in East Asian, Sub-Saharan African, and Native American populations). The rapid spread of CCR5- Δ 32, which, of course, cannot

be attributed to the recent threat posed by AIDS, led some scientists to suggest that the sporadic pandemics and recurring epidemics of plague across medieval Europe created a selective pressure for the mutation (Stephens et al. 1998).

The claim that the Black Death left a lasting mark on the human genome was highly publicized and became a popular example of evolution in action, but unfortunately this claim was entirely incorrect.¹⁵ Researchers using simulation models soon demonstrated that even severe epidemics of plague that killed 10 percent of the people of Europe once every decade, punctuated by the two pandemics that killed 40 percent and 20 percent of the population, would have only driven the frequency of CCR5- Δ 32 to at most 0.8 percent over the centuries that the disease existed in Europe (Galvani and Slatkin 2003). Subsequently, it was confirmed that modern bubonic plague does not enter immune cells through the CCR5 receptor (Mecsas et al. 2004).¹⁶ Additional simulations demonstrated that, although the dramatic but episodic lethality of the Black Death was unable to account for the rapid spread of CCR5- Δ 32, an endemic disease that disproportionately struck down children over several hundred years—as smallpox did in Europe during that time—could easily account for the rapid spread of the mutation (Galvani and Slatkin 2003).

Just as the widespread devastation caused by the Black Death necessarily had a much smaller evolutionary effect in Europe than the incessant toll of children that smallpox collected, the selective force of epidemic yellow fever on people at the fringes of its range was much weaker than the constant pressure on the genomes of populations suffering from endemic malaria. The argument that long histories with malaria increased the prevalence of genes that offer some protection to that disease in populations from which Caribbean slaves were drawn—therefore long exposure to yellow fever likely had a similar effect—is simply wrong. Because yellow fever is mild in childhood and provides lifelong immunity, any inherited resistance to yellow fever would provide a reproductive advantage only in comparatively rare circumstances, and it would therefore be unlikely to consistently become more common over time in the manner that traits offering malaria resistance do. The fact that geneticists have not identified any genes that confer an inherited resistance to yellow fever should come as no surprise.

Conclusion

There is no support for the claim that continues to be put forward that black people enjoy immunity or substantial resistance to yellow fever. But if there is no evidence

15. For an example of the publicity given the claim, see Kolata (1998). An episode of the PBS series *Secrets of the Dead* entitled “Mystery of the Black Death,” first aired in 2000, presents the claim as the solution to a murder mystery, and teaching materials for the episode are still available online as of the date of this writing; see http://www.pbs.org/wnet/secrets/previous_seasons/case_plague/index.html.

16. Although there had once been reason to doubt that bubonic plague is the same disease that caused the Black Death and the subsequent European plague epidemics (see Cohn 2002), recent genetic testing of samples collected from a Black Death burial ground in London has confirmed that it was indeed responsible (Schuenemann et al. 2011).

for an innate resistance to yellow fever among black people, where did this argument come from, and what explains its persistence?

If the fact that many blacks newly arrived to the Caribbean from Africa were in actuality immune due to previous exposure to the disease was not alone sufficient to account for the origins of the claims that black people are innately immune to yellow fever, historiography on the experiences in India and the Philippines provides another possible explanation. More than a decade ago, Warwick Anderson and Mark Harrison revealed that arguments for racial immunity arise concomitantly with the creation of racial hierarchies (Anderson 1996; Harrison 1996). In the early years of British colonial rule of India and US colonial rule of the Philippines, the health of white populations was closely monitored, while that of natives was neglected. In these circumstances, the ravages of tropical diseases on the white colonizers were at the forefront of the minds of doctors and colonial administrators, while the suffering of others was most frequently only an afterthought, if considered at all. Because white doctors rarely attended the cases and deaths of nonwhites from these diseases, they often simply assumed such deaths did not occur. This contrast between the visible deaths of whites and the unseen, unknown deaths of Indians and Filipinos led to pervasive arguments that these subject populations had innate immunities to the diseases that were so harmful to the newly arrived colonial forces. Whether considering dysentery, smallpox, typhoid, or malaria, colonial doctors viewed whites as particularly vulnerable. And as Anderson notes, this conclusion then only worked to further justify and reinforce the neglect of nonwhite populations (Anderson 1996: 102).¹⁷

This would seem to correspond to claims of black yellow fever immunity. Like native populations under colonial rule, slaves and free blacks in the Caribbean and beyond, of course, were subordinate to whites, and their health only rarely a matter of great concern to white doctors. As witnesses to the horrors of a yellow fever epidemic among their white patients, but removed from the similar horrors these epidemics visited upon black people, these doctors could easily surmise that the latter enjoyed an inherent immunity. Moreover, death rates among the underfed and overworked slaves were frequently staggering even in the absence of yellow fever epidemics (see, e.g., Higman 1984; Tadman 2000). Considering all of these factors, it is not surprising that the lethality of yellow fever among black people was often overlooked.

Comparison to colonial experiences elsewhere also suggests a potential explanation for the persistence of the unsupported story of black immunity to yellow fever until the present. By the start of the twentieth century, arguments for racial immunities were beginning to decline. As the understanding of the immune system began to advance in the last decades of the nineteenth century, evidence had begun to mount that

17. More recent work has reinforced this point by demonstrating that the phenomenon is not limited to contexts of white domination. Qing dynasty subjugation of “tribal” peoples in Yunnan in the eighteenth and nineteenth centuries was soon accompanied by the conviction among Han Chinese that the tribal peoples enjoyed an innate resistance to malaria and that they were uniquely vulnerable to the disease (see Bello 2005).

immunity was the result of specific changes in the body that resulted from exposure to disease, which undermined the idea that it could be inherited (Anderson 1996: 106–7). In a complete turnabout, medicine in racially hierarchical contexts began to focus not on nonwhites' supposed racial immunities but instead their susceptibility to contracting diseases due to their poor standards of hygiene—diseases that would then be transmitted onward to whites. In the Philippines, for example, the “threatening microbial pathology that lurked within native bodies” was the justification for US colonial policies aimed at completely reforming Filipinos' personal conduct and social relations (Anderson 1996: 115).

With regard to the idea of black immunity to yellow fever in the Caribbean and surrounding areas, this transformation in racialized medical understandings was stopped short before it even began by the elimination of yellow fever from the region. Beginning with the US campaign in Cuba, led by none other than army sanitarian William Crawford Gorgas, the disease was pushed out of the region until its only remaining foothold in the New World was deep in the Amazon rainforest, where it continues to be sustained today in populations of wild monkeys.¹⁸ By destroying the conditions in which the disease's mosquito vector thrived, these efforts ended the threat of yellow fever epidemics. But with the end of the threat, there was no reason to recast black people from beneficiaries of racial immunity into a reservoir of infection that demanded public health supervision. As it was not superseded by the new interpretation of the relationship between race and disease, the theory of black immunity to yellow fever could remain intact.

It is not surprising that the theory was then adopted wholesale by many historians decades later. As David Jones (2003: 710–11) has observed in the context of the demographic collapse suffered by American Indian populations after 1492, there is an enduring appeal to arguments that people whose ancestors lived in different parts of the world possess innate differences in disease susceptibility: they provide an elegant explanation for complex historical phenomena and can even suggest that particular outcomes were inevitable. No matter how appealing such claims may be, they must—like all others—be judged against the entirety of the evidentiary record. Even the most elegant explanations must be abandoned when contradicted by the weight of documentary or scientific evidence.

The belief that black people have an innate resistance to yellow fever is old and historically important. It certainly helped legitimate slaveholding, and it may even have played a role in the initial adoption of African slavery across the Caribbean. It shaped the strategies of both Britain and France for defending their Caribbean colonies through the nineteenth century. But there was never a consensus among medical observers that it actually existed—the evidence from epidemics indicates that in fact it did not—and the analogy to the very real and well-documented evolutionary consequences of endemic malaria is not apt. The belief in black immunity to yellow fever is misplaced, and it is now long past time that historians discard it.

18. On the US campaigns against yellow fever in Cuba and beyond, see Espinosa (2009).

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