

ANOTHER DIMENSION TO THE BLACK DIASPORA

DIET, DISEASE, AND RACISM

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CHAPTER 1

THE BLACK MAN'S CRADLE AND THE WHITE MAN'S GRAVE

Africa, the oldest home of man, is the home of the most dangerous of man's diseases.

C. D. Darlington (1969)¹

Epidemiology at any given time is something more than the total of its established facts. It includes their orderly arrangement into chains of inference which extend more or less beyond the bounds of direct observation.

W. H. Frost (1936)²

I

The shores of West Africa were the point of embarkation for the bulk of the 10 million or so blacks who unwillingly left Africa to labor on the plantations and in the cities and mines of the New World.³ Among these unhappy voyagers were the progenitors of today's North American black population. Yet although the black diaspora ceased long ago the West African homeland continues to exert an enormous physical influence on the descendants of the original migrants, both in terms of outward appearance and biochemical anomalies that have dictated a different black disease experience from the North American white.

West African climate, disease, and relative isolation are the three factors chiefly responsible for this influence. These factors combined and recombined over millennia to mold and shape the inhabitants. They were sheltered by the Sahara desert against easy access from North Africa and its succession of empires and emperors, while turbulent seas and contrary wind patterns discouraged intrusion from the South Atlantic. This is not to say that no caravans crossed the desert to West Africa or that no ships visited West African shores prior to the Portuguese explorations of the fifteenth century.

But contact with the outside world was limited even though regular trade routes did criss-cross West Africa and penetrate into the heart of the continent.⁴ Likewise within West Africa relative isolation was a rule rather than an exception, with individuals contained in small

self-sustaining village states, enjoying little intercourse, social or otherwise, with even nearby countrymen. As a consequence, early European visitors could marvel at such strange phenomena as two tribes residing on opposite banks of the Senegal River: On one side the inhabitants were of "high stature and black," but across the river lived persons of "low stature" and a "browne or tawne" complexion.⁵

This is not to suggest that there was no racial blending of West Africans, but it should be noted that because its peoples were relatively isolated both externally from abroad and internally from one another, West Africa had long been a kind of genetic hothouse. West Africans experienced a long period of internal evolutionary development that saw generation after generation emerging from common breeding pools. The result was that while they demonstrated a considerable variation in physical types, they nonetheless came to share many common features including an overwhelming predominance of Negroid characteristics.

Certainly pigment is the most obvious characteristic; it is probably a function of climate, for dark skin keeps the body cooler in hot regions. Indeed it is generally believed (although not proven) that black skin evolved as a defense against damage from the West African sun, that is, "a compromise between the conflicting demands of protection from skin cancer and sunburn, thermoregulation and synthesis of Vitamin D."⁶ Another characteristic is thick kinky hair that some are convinced serves as a sort of cap to protect the scalp from that same hot sun. In a similar vein, it is believed that hot humid air reaches lungs more efficiently through broad flat noses (as opposed to the thin nostrils of Caucasians that are credited with protecting lungs from frigid air), while the black's greater number of sweat glands supposedly operate to keep their bodies cooler. Other distinctly Negroid traits, such as a denser skeletal mass, narrower pelves, less muscular calves, smaller lungs, longer limbs, and shorter trunks, may well also be the product of the West Africans' long-term adaptation to their own peculiar environment—an adaptation that produced those broad characteristics usually thought of as racial.⁷ But clearly the qualifications contained in the foregoing make speculative any explanation of how the black's outward or phenotypical characteristics came to be.

More is known, however, about other characteristics encouraged by that same environment which, although disproportionately present in blacks of West African origin, are not racial in nature. Rather, they represent more recent (and less permanent) genetic adjustments to environmental circumstances, which, in almost every case, relate to disease.

West Africa lies within the tropical belt. Its high average temperatures and lush vegetation make it a paradise for insects, many of them disease-bearing organisms that abuse humans on a year-round basis

because they are free from those seasonal controls operative in more temperate climates. Indeed the tropical ambience is due to the location of most of West Africa well inside the line of forty-inch rainfall, and the interaction of these factors—high average temperature, heavy rainfall and an abundance of insects of myriad variety—gave birth to man's deadliest diseases.⁸

Yet humans continue to inhabit the region. Man has not been driven off or eliminated, because as a general rule "Nature prefers that neither host nor parasite should be too hard on the other. For Nature, survival of the species is all that counts and the norm, if there is one is that the host should not die and that his infection should be passed on to one new host individual."⁹ Hence the very deadliness of these diseases summoned the mechanics of genetic selection in the inhabitants to erect defenses against them—not absolute defenses in most cases but rather defenses that guarded against pathogenic proliferation that would kill the host. Consequently, individuals endowed with traits limiting the extent of infection lived to reproduce, passing those traits along to offspring. Those that did not would die, and over time West Africans developed innate mechanisms of resistance to the major diseases of their environment. Thus, the diseases themselves elicited defensive traits in their host that certainly are genetic, but not properly called racial.

Malaria resistance makes a fine example. Unquestionably of all the world's infectious diseases "malaria has caused the greatest harm to the greatest number,"¹⁰ and West Africa bids fair for first place as the world's malaria capital. It harbors some sixty of the world's 200 or so species of *Anopheles* mosquitoes, many of which carry and inject the malaria parasite into man.¹¹ The inhabitants of the region, therefore, seem to have been preyed upon by all four types of the disease (and their many strains) which parasitize the human species.¹²

To coexist with this disease, West Africans evolved hemoglobin defenses, which are blood abnormalities that resist malaria parasites and forestall their multiplication. Thus they grew relatively refractory to both the debilitating, but seldom fatal, types of malaria such as *Plasmodium vivax* and the frequently deadly *Plasmodium falciparum*.

West Africa is also inhabited by another small silvery pest, the *Aedes aegypti* mosquito (a free translation would have it the "unpleasant Egyptian"), the most important vector of another great killer, yellow fever. Most epidemiologists think that West African forests were the birthplace of this disease, in part because of the omnipresence of that disagreeable bug but also because of the striking, still to be explained, ability of West Africans to resist yellow fever over the centuries—an ability notably lacking in Europeans, who became the disease's chief victims.

Still another example is transmitted by the tsetse fly (genus *Glossina*),

a large biting fly that curses West Africa with twenty-two species, among them *Glossina palpalis* which imparts African trypanosomiasis—the African sleeping sickness—to humans.¹³ Today, although the disease is endemic to the region, and although no permanent immunity seems to be bestowed upon its survivors, only a relatively small portion of exposed West Africans appear susceptible in the first place. Moreover, those blacks who do contract African sleeping sickness demonstrate a resistance to at least local strains, which has prompted the medical observation that the disease tends to be more acute in whites than in blacks.¹⁴ Epidemiologists have still to account for this phenomenon, but a safe assumption would be that as in the case of yellow fever, while the mechanism of immunity is unknown, its presence is impressive testimony to the plasticity of man under environmental pressure.

High average West African temperatures also meant that little in the way of clothing or footwear was needed allowing the hookworm, which abounds in African soils, easy access to the human body. But again familiarity bred resistance, and as Rockefeller-funded researchers in the American South during the first decades of the twentieth century discovered, the black has somehow gained a relative immunity to hookworm infestations.

West Africans then were born into a world teeming with pathogens for which they developed a complex of defenses. Yet if they were equipped genetically to escape the worst of malaria, yellow fever, or hookworm disease, a bout with one or all was nonetheless something children could expect, and therefore child mortality was always tragically high as the cruel process of selection for protection continued.

There were, however, other diseases in West Africa to which the blacks seem to have been extraordinarily susceptible. Youngsters would normally contract yaws, for example, a disease transmitted by skin-to-skin contact that thrived among a people who required few clothes. In fact, if the child did not take the affliction at an early age, a fairly standard practice was for parents to "borrow the yaws" from an infected person—a process of inoculation which insured adult immunity.¹⁵ European observers, who seldom suffered from the affliction themselves, believed yaws and also leprosy (which Europeans rarely saw anymore) to be "Negro diseases." The distinction between the two illnesses was never clear-cut.¹⁶ West Africans were particularly susceptible to pneumonia, which proved inordinately fatal, and tetanus, which claimed countless lives, especially those of children.

Africa also harbored diseases that plagued that continent as they did Europe, treating black and white impartially. Smallpox put in frequent epidemic appearances, occasionally wiping out whole villages. Childhood diseases such as mumps and chickenpox made life as miserable for African youngsters as they did for European children. Tuberculosis

and syphilis were well-known to Europeans but apparently strangers to West Africans until contact between the two facilitated their importation. Because of a lack of historical contact each of these two illnesses tended to run a more severe course in blacks than in whites.

To battle with this pestilential host in West Africa was the task of the African medicine men, who were the guardians of remedies handed down from generation to generation. Some of their cures took the form of magical rites. But for common ailments, they possessed quite an effective command of the healing properties of roots, herbs, and other vegetation. Indeed, recent work on West African healing practices leaves little doubt that African materia medica has important significance for today's medical world.

African and European doctors alike, however, have always labored under a serious disadvantage in West Africa, for their patients have invariably been malnourished and consequently more difficult to cure than healthy individuals. The most superficial accounting of West African nutrition shows a ledger heavy with starches on the debit side but light in protein credits. One reason for this is that most varieties of cattle (and horses and asses for that matter) did not develop the resistance to African trypanosomiasis that man did (it may be that the human variety is less deadly, representing an accommodation between host and parasite), and as a result those species of the bloodsucking tsetse fly, especially *Glossina morsitans*, sought out large animals so efficiently inside the forty-inch line of rainfall that cattle raising was usually unprofitable, if not always impossible.¹⁷

Hence, West Africans were limited to raising a few goats, chickens, dogs, and sometimes a pig, animals so scarce and highly prized that they were only slaughtered on special occasions. Animal protein then did not constitute an important item in the diet. Naturally bovine milk was excluded from that diet along with the other dairy products, and because of taboos against them, eggs were not normally eaten. Additionally, depending on the people and their customs, fruit consumption was frequently frowned upon as was the use of many vegetables.¹⁸ Thus, in many cases, there were few dietary supplements to the starchy core of the West African diet, a core built around the principal crops of the region.

West Africa's most important cultivated plants have been imported, with its high-yield plants arriving only in the last few centuries from America. Prior to the sixteenth century (which saw the beginning of this introduction), the African diet centered on lower-yielding Asian imports of bananas, taro (often called the bull yam) and the small African yam, along with millet and rice.¹⁹

Because of the relatively poor yield of these crops, researchers have come to believe they barely sustained life, largely because of the tremendous multiplication of the West African peoples following the in-

roduction of American plants, despite the heavy population drain of the slave trade. Cassava (manioc) and maize were the most crucial of these population stimulants.²⁰ Although high in carbohydrates, these plants offer little in the way of protein, and with very little animal protein in the diet, West Africans must have suffered from serious protein deficiencies. Moreover, this deficiency was even further compounded by the acidic character of some of West Africa's soils and the nitrogen deficient quality of most of them that meant a reduced protein yield of the vegetable crop. Additionally, those same soils were severely leached by heavy rains leaving them with a low mineral (especially phosphorus and calcium) content.²¹

Finally Africa's principal crops, which probably delivered a sufficient amount of some nutrients, such as vitamins A and B₆, would nonetheless have left consumers deficient in most of the other B-complex vitamins, vitamin C, calcium, and iron. Not incidentally these deficiencies, coupled with a want of animal protein, remain glaringly apparent in the West African diet of this century.²²

Today poor nutrition in West Africa is considered that region's principal problem.²³ It is not so much that West Africans are undernourished as that they are badly nourished. The diet still tends to focus on a single starchy crop (cassava or maize) that provides carbohydrates in sufficient quantity to sustain life but is supplemented with little else.²⁴ Meat is still seldom eaten; the little that is eaten has a uniformly poor quality because West African animals, like their human owners, are also poorly nourished.²⁵ In addition, taboos remain that limit egg consumption, while milk products, vegetables (other than the principal crop), and fruits are not eaten with any regularity.²⁶ In short, the diets of yesterday and today seem to approximate.

The consequences of this regimen in the twentieth century are widespread anemia, kwashiorkor, and pellagra, which are all nutritional illnesses in their own right but also illnesses that leave the population susceptible to intercurrent diseases, such as pneumonia, West Africa's foremost killer today. Hence, all available information suggests that the nutritional status of those soon-to-be Afro-Americans who made the Middle Passage was determined by a diet overloaded in carbohydrates but, under the best of circumstances, low in proteins as well as many of the essential vitamins and minerals.

Moreover, West Africans had quantitative as well as qualitative nutritional problems to contend with. All too often rainy seasons alternated with droughts to make year-round agricultural activity difficult; famines therefore were not unusual occurrences. Consequently, not until the importation of cassava and maize from the Americas did a potential for resolving the quantitative difficulties even exist.²⁷

Empiric evidence to confirm that blacks reaching the Americas were severely undernourished has been generated by research on the height

of imported Africans as opposed to Creole-born slaves in the Caribbean. Data concerning the height of some 25,000 slaves in Trinidad reveal that new arrivals were significantly shorter than those born on the island.²⁸ Manual Moreno Fragnals²⁹ of Cuba has found the same to be true for that island. First generation Creole slaves were significantly taller than *bozales* (the freshly imported Africans). Not that taller is necessarily better, but this apparent rapid growth of American slaves over the course of a generation or so does suggest that even a slave diet in the New World was more protein-laden and probably of better overall quality than the West African diet.

Thus, at the risk of belaboring the point, West African nutrition was (and still is) poor. Moreover, the African was even further impoverished physically by his disease environment and paradoxically very possibly weakened as well by his defenses against that environment. By way of illustration, malaria creates anemia among its other depredations, but some of the blood abnormalities that protect against the disease are also suspected of producing other anemias, which may have aggravated that lack of protein inherent in the black diet. Likewise, the many worm-related diseases of the region must have eroded already depleted iron stores while also interfering with the metabolism of other nutrients.

Worms also share the host's protein intake which, if inadequate to begin with, means that he will have even fewer reserves for the production of substances crucial in the formation of antibodies. The result is a heightened susceptibility to invasion by other pathogens. Clearly, malnutrition and pestilence formed a grim partnership in winnowing West African lives.

But just as the West African evolved genetic protection against the diseases of his environment, he also seems to have made genetic adjustments to help him live with malnutrition. For example, some nutritionists believe today that blacks require less iron than do whites.³⁰ And, perhaps because of a greater constitutional ability to absorb calcium, blacks seem to have a lower requirement for this mineral than whites.³¹

A grave difficulty with specialized adjustments to an environment, however, is that genetic assets can quickly convert to liabilities if that environment changes rapidly or if its inhabitants relocate to radically different geographic regions as was the case with West Africans suddenly bound for North America. In their homeland a more or less year-round supply of sunlight to activate the production of vitamin D was a crucial catalyst in the black's metabolic machinery. In a more temperate North America not annually blessed with abundant sunshine, however, pigment, protective against West African sun, now screened out much of that radiation that was so important to the black nutritionally. Several studies testify to the black's ability to better withstand heat than whites.

Conversely the latter respond more efficiently to cold; their metabolic rate is far ahead of blacks' when both are identically exposed. Hence, blacks are more susceptible than whites to cold injury and to some diseases common to cold climates (see Chapter 9).

Another trait harmless in West Africa, but one that would become troublesome in a temperate climate with a milk-drinking culture, is the high frequency of lactose intolerance that characterizes West Africans and their descendants, leaving them unable to consume much milk (see Chapter 6). This condition occurs when high levels of lactase enzyme (essential for breaking down milk sugars) fail to fully develop. In West Africa, of course, there was little encouragement for the enzyme to develop first because (unlike northern Europe) there was always abundant sunlight, and thus vitamin D, to facilitate the metabolism of calcium, and second because the tsetse fly made dairying unprofitable, therefore creating an historic situation of low milk usage.

The point here is that long-term adjustments to a West African environment held the potential for creating problems of health for blacks suddenly relocated in North America and in like manner for their descendants. Yet ironically, some of those adjustments were a major reason for that relocation with West African genetic and epidemiologic factors stimulating a lively demand for Africans in the New World.

That demand was due initially to the failure of American Indians to survive European diseases against which they had little or no resistance.³² These included smallpox, usually regarded as their biggest killer, as well as "mild" diseases, such as measles and mumps, childhood afflictions for Europeans, but deadly plagues for the previously unexposed Indians.³³ Yet West Africans, already familiar with many of these Old World diseases, proved as resistant to them as whites. Consequently, as New World Indian populations declined, demand for black labor increased.

Its fulfillment, however, brought a second wave of disease to the Indians as African diseases such as falciparum malaria and yellow fever joined European pestilence in further diminishing the Indian ranks; once more the demand for black labor increased. Moreover, black refractoriness to these illnesses that struck as hard at whites as at Indians killed off any lingering possibility that Europeans might serve as a significant source of plantation labor. It was the African's ability to resist tropical as well as European disease that helped convince slaveholders, already impressed with his ability to labor in intense heat, that the black slave was a priceless gift from a thoughtful Creator.

Thus, those mechanisms for survival with which Mother Africa endowed her sons and daughters worked cruelly against her progeny by encouraging their enslavement. Exactly how potent these mechanisms were can best be appreciated by contrasting the experience of blacks and whites with yellow fever and malaria in Africa.

II

"Beware and take care of the bight of Benin. For the one that comes out there are twenty stay in." Although hyperbolic, this old chantey from the days of the slave trade suggests something of the price that Africa extracted from its European invaders, a price that established West Africa's reputation as a "White Man's Grave."³⁴

Today we know men were not struck down by a hot West African sun but instead by the tiniest of organisms living under that sun: helminthic, protozoan, bacterial, and viral parasites. Yesterday, however, the sun got the blame. Since antiquity physicians had focused on the systems of patients with an eye to maintaining their "humors" (body fluids) in balance.³⁵ When those humors were thrown out of balance, as the African sun was said to do, disease resulted. Whites sickened and died; blacks did not, for obviously they could not suffer severe humoral dislocation caused by their own homeland. They were "acclimated."

Because of this static state of medical thought, the first centuries of Europe's presence on the coast saw little attention paid to precisely what was killing whites. It did not matter much anyway. Regardless of the symptoms, the cures—tinkering with the humors—were the same. Hence there was little incentive to sort out diseases, much less to distinguish what seemed to be uniquely African afflictions from that great body of illnesses that had traditionally bedeviled Europe. For this reason, although accounts of Europeans on the African coast assure us that they died in droves, the cause of mortality as well as the number and proportion of those who succumbed went largely unrecorded. It was a fatalistic age when no one bothered to keep score on "God's will."

But by the late seventeenth century, the Royal African Company started keeping score. Many slaving concerns did the same in the eighteenth century, and British army mortality records and accounts of exploratory journeys to the interior are available for the nineteenth century. These ledgers leave no doubt of incredible white mortality. The Royal African Company data indicate that during the late seventeenth and early eighteenth centuries fully *half* of the English merchants, soldiers, and officials connected with the company were losing their lives *within their first year of residence* on the coast, and only one out of ten ever made it back to England.³⁶

Throughout the eighteenth century white crews on slaving vessels fared better only because their exposure to Africa was brief when compared with resident Royal African Company officials. A study of French slavers sailing from Nantes over a forty-year period during the last half of the eighteenth century reveals that crew mortality was upwards of an average 200 per 1,000 per voyage.³⁷ Late eighteenth-century British sail-

ors out of Bristol and Liverpool encountered more dismal odds against survival with estimates of their mortality exceeding 200 per 1,000 per voyage.³⁸ In fact despite the well-known horrors of the Middle Passage for slaves, death claimed proportionately more of the white crew members topside than the black cargoes crowded below.³⁹

Nineteenth-century statistics on British troop mortality in West Africa continued to reflect the same awful toll. The British discovered that the annual death rate per 1,000 soldiers stationed in the United Kingdom was 15.3. It ranged, however, between an appalling 483 and 668 per 1,000 mean strength annually on the African coast.⁴⁰ This deadly portrait of West Africa's disease environment was also painted by independent observers. One in Sierra Leone asserted that "the average tenure of office of the [British] officials, from the Governor downwards, was . . . less than twelve months; death or invaliding quickly claiming them."⁴¹ A physician traveling in Africa published data that revealed that 10 out of every 16 whites in Gambia and 10 out of every 27 in Sierra Leone died annually during 1822–30.⁴²

This spectacular rate of European demise was normally attributed to "fevers," although some physicians felt a professional call to be more precise and subsumed the bulk of the deaths under the rubric of "bilious remittent fever."⁴³ In reality, this "bilious remittent fever" for the most part described two diseases: yellow fever and falciparum malaria. That they were new diseases to Europeans against which they were relatively defenseless is well-illustrated by the way West Africans thrived in the midst of European death. An English physician marveled that despite "bilious fevers, of the most malignant kind . . . the natives appear to enjoy good health."⁴⁴ Then, with typical British understatement, he added that "the climate of Africa . . . has been generally found to be extremely prejudicial to the health of newly-imported Europeans."⁴⁵

The full extent of this prejudice is reflected by the markedly different disease experience of nineteenth-century black and white troops serving the British Crown in West Africa. While whites were dying at a rate that skyrocketed between 483 and 668 per 1,000 mean strength annually, their black counterparts died at a rate of 31 per 1,000. Practically all the white deaths were a result of "fever," but few black deaths were attributed to this cause, even though the blacks themselves were recruited from all over West Africa and were not necessarily serving in their own region.⁴⁶

This is not to say that being black automatically exempted one from the "fevers" under consideration, particularly if one were not born in Africa. Blacks, for example, recruited in the Americas for missionary work in West Africa reportedly perished from "fevers" at a very high rate.⁴⁷ Black immigrants from the United States to Liberia also sustained a high incidence of mortality from "fevers," the bulk of which

may have been malaria.⁴⁸ Yet these are exceptions that remain to be explained. More typical is the story of Sierra Leone where African "fevers" harvested a heavy toll of whites, but were not major killers of liberated Africans.⁴⁹ Or again a near absolute black refractoriness to African fevers as well as a terrible white susceptibility can be seen in Europe's nineteenth-century effort to penetrate the interior of the continent, forging inland on what Michael Gelfand has termed the African "Rivers of Death." Of the 44 Europeans, for example, who accompanied Dr. Mungo Park on his second (1805) expedition to the Niger, 39 died of disease. Viewed in this light, Captain James Kingston Tuckey's expedition to the Congo River in 1816 was a salubrious outing, with only 48 percent of its members lost to "fevers."⁵⁰

In addition to highlighting the deadliness of Africa, the expeditions served to remind whites that they were truly victims of disease discrimination. The great Niger expedition of 1841, for example, while not exacting a particularly heavy toll of whites (relatively) vividly demonstrated the superior ability of Africans to survive in their homeland. Among the 145 Europeans in the expedition, 42 (29 percent) died from fever. By contrast, all 158 of the "African and coloured men" survived.⁵¹

One obvious question arises. Why did Europeans continue to venture to West Africa in large numbers, given the suicidal odds against their survival? The answer is that the public was kept deliberately uninformed. During the seventeenth and eighteenth centuries, companies such as the Royal African Company were not anxious for it to be publicized that recruits for the coast had "three chances in five of being dead within a year" of their arrival.⁵²

Therefore, as one student of the question has observed, "it is a moral certainty that the Company's recruiters kept quiet about them [the odds]."⁵³ Sailors, of course, were more aware of Africa's lethal reputation. Yet accustomed to braving exotic diseases in exotic places, they were on the whole a fatalistic lot. Finally, not all who reached the shores of West Africa did so voluntarily. Many sailors were crimped, while soldiers frequently "were recruited by allowing them to serve on the Coast in commutation of punishment in Britain."⁵⁴ Thus, West Africa was not lacking in a steady stream of potential fever victims.

III

Europeans who reached the coast were no strangers to malaria. They knew their "ague" intimately with its periodic visits of chills and fevers, nausea and sweating and aching. Indeed malaria was so prevalent in much of England that it was scarcely regarded as a disease. It was a nuisance, it made one "shake" from time to time, but seldom killed—seldom, but sometimes, as the ghosts of both James I and Oliver Cromwell could testify.