The African Connection: Slavery, Disease and Racism

The purposes of this study are threefold: first, to indicate the role that disease immunities in blacks played as both a stimulus to and a rationale of slavery; second, to show how disease susceptibilities in blacks resulted in a nineteenth-century medical perception of black people which contributed significantly to American racism; and third, to point out briefly how the pages of history might be used profitably by epidemiologists searching for the etiologies of diseases related to blacks today.

Disease Immunities in Blacks and Slavery

The first task, that of relating disease immunities in blacks to the growth of Negro slavery, has been made relatively easy because of scholars such as Philip Curtin and Peter Wood, who already have called attention to the relationship. Endemic in West Africa, falciparum malaria, by far the most deadly type of malaria for man, by the very fact of its deadliness encouraged genetic defenses against it. These defenses took the form of blood anomalies such as sickle trait in its many variants and blood enzyme deficiencies; all of which discourage the high and frequently fatal level of parasitization so typical of *P. falciparum*. Moreover, malarialogists seem confident that other defenses against falciparum malaria will also be discovered; but even with only present information, we can say that around half the blacks reaching the Americas possessed known defenses against this frequently fatal malaria type.

In addition, most West Africans and persons of West African descent have proven refractory to another kind of malaria, vivax malaria, which,
although not so deadly as *P. falciparum*, is nonetheless debilitating. Malarologists have been aware of this refractoriness for decades, but it has been explained only recently. It seems that those resistant to it possess red blood cells which have been labeled "Duffy group negative" — a condition extremely rare in other racial groups without black admixture, yet a condition whose frequency reaches 100 percent among some African peoples. Experiments seem clearly to indicate that individuals who are "Duffy group negative" do not contract vivax malaria.

Finally, although the mechanism or mechanisms of protection are still not known, statistical studies of yellow fever epidemics carried out by Uttley for the island of Antigua and by Kiple and Kiple for the ante-bellum South make it clear that blacks were also very resistant to the ravages of yellow fever. It is not that blacks did not contract the disease; but rather, in the words of a turn-of-the-century New Orleans physician, while "Negroes are about as liable to contract the disease as whites... they usually have it in a remarkably mild form." To underscore this with just one example, the homicidal 1878 epidemic which ravaged Memphis infected 78 percent of the city's blacks and close to 100 percent of the whites who did not flee; however, only 9 percent of the infected blacks died, as opposed to 70 percent of the stricken whites.

Thus blacks were resistant to the two great tropical killers which mowed down whites. The implications of these immunities were far-reaching. Philip Curtin and Kenneth Davies, among others, have presented mortality statistics which revealed Europeans in West Africa from the seventeenth through the nineteenth centuries dying at a rate ranging from 300 to 700 per 1000 per year from yellow fever and malaria. This incredible mortality, as Curtin has suggested, dissuaded Europeans from locating plantations in tropical Africa, close to a seemingly inexhaustible source of cheap labor. Instead, they found the expense of transporting African workers across the Atlantic preferable to challenging the odds against their own survival in Africa.

Europeans soon discovered to their dismay, however, that Africans and their fevers were not that easily divorced, and the slave trade which

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8Curtin, "Epidemiology and the Slave Trade," pp. 197 ff.
carried the former also transplanted the latter. The American Indian population, initially viewed by the Spaniards in particular as a solution to their New World labor problems, had already been decimated by European disease and now was set upon by African disease as well. Still more Indians died, and Negroes with resistance to most European, in addition to African, diseases became even more highly prized. By contrast, whites succumbed to the newly relocated African diseases on this side of the Atlantic as easily as on the other—a phenomenon which negated any possibility that Europeans might serve as a significant source of plantation labor. It is therefore that blacks were nominated for labor in the tropics quite literally by the process of elimination, and the notion that only Africans could perform labor in hot climates was born. This differential disease experience gave enormous encouragement, of course, to the African slave trade.

In addition, these same disease immunities in Negroes which served as a rationale for the slave trade later also provided a rationale for the peculiar institution, as antebellum Southerners found themselves forced to justify their slavery to a Western world increasingly hostile to the practice. Thus one Southern physician spoke for many of his colleagues when he wrote, “white men will never be able to be substituted for negroes as field laborers; for . . . overseers invariably get sick— their children also . . . when the negroes around them are perfectly healthy.”

And during the last days of slavery, Southern agricultural and medical journals cried out that “the white man will never raise . . . can never raise a cotton or sugar crop in the United States. In our swamps and under our sun the Negro thrives, but the white man dies. Without the productive power of the negro whom an all-wise Creator has perfectly adapted to the labor needs of the South its lands would have remained a howling wilderness.”

Susceptibility, Dissimilarity and Racism

As disease immunities in blacks were twisted and turned back on them, so were their disease susceptibilities. By the second decade or so of the nineteenth century, antebellum physicians had identified a number of diseases to which blacks seemed far more susceptible and from which they were far more likely to die than were whites. Adults, by way of illustration, suffered from a high incidence of tetanus, while their infants perished at an awful rate from what was termed “trismus nascentium.” Slave children had far more fatal difficulties than whites with diseases diagnosed as “teething,” convulsions and “fits and seizures,” and they died by the thousands from worms, diphtheria and whooping cough. Child and adult alike succumbed to catarrh, pneumonia, bowel com-

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10 This composite of declarations which appeared in Southern journals during the late 1850s was put together by Kenneth Stampp, The Peculiar Institution (New York 1956), p. 7.
plaints and “scrofula” with more frequency than whites, while physicians found themselves alternately fascinated and repelled by the slave habit of consuming dirt and swilling swamp water.

Now, early in the nineteenth century, even though it was customary to think and talk in terms of “Negro diseases,” the etiologies of these diseases were conceived of essentially in climatic and environmental, as opposed to racial, terms. For example, blacks were troubled more with pleurisies and other respiratory sicknesses than were whites because their lungs were accustomed to the “torrid zone” rather than to North America’s more temperate climate. Many infant illnesses were attributed to problems of cleanliness, while the Negroes’ chronic bowel disorders were blamed on improperly cooked food, bad water and so forth.

Yet by midcentury this essentially environmental explanation for diseases among Negroes had been supplanted by one distinctly racial in orientation. Among the factors responsible for the radical change in the medical perception of the blacks, three were local and immediate. The first was the formation of the American school of ethnology that was challenging older notions of immutable species which held that men might differ, but they were more alike than different and certainly all of the same race. The American school, as exemplified by the work of Josiah Nott and George Glidden, held that Africans belonged to a separate class of men, the lowest class, with a gulf of important physiological and intellectual differences separating them from the highest class—the Caucasians. Second was the dilemma of a South imbued with a faith in democracy, yet dedicated to a peculiar institution under heavy attack from abolitionists both at home and abroad. One neat way, and indeed perhaps the only way, of resolving that dilemma was a demonstration that Negroes were inherently inferior—so inferior that they were helplessly incapable of survival as free persons. The third factor has to do with the empirical observations of Southern physicians, whose practice brought them into intimate daily contact with slaves—observations which certainly were influenced by the first two factors, yet doubtless would have wrought some reinterpretation of the causes of a differing disease experience among blacks and whites, regardless of the outside intellectual climate.

By way of illustration, and once more employing the example of the susceptibility of blacks to pneumonia, it was becoming increasingly difficult to place the blame for this susceptibility on lungs unaccustomed to a temperate climate—not when those lungs now dwelled in the chests of slaves many generations removed from the tropics. Thus the old environmental explanation gave way rather easily to new ones with a decided racial base—blacks were innately endowed with “weak” lungs.

Josiah C. Nott and George R. Glidden, eds., Indigenous Races of the Earth; or New Chapters of Ethnological Inquiry, (1857); Josiah C. Nott, The Negro Race: Its Ethnology and History (1866) and Instincts of Races (1866).
Then impelled by this “discovery” as well as the American school findings and their own mission of demonstrating the inferiority of blacks physicians turned their attention to other parts of the body. Their findings not only had the lungs of blacks smaller than those of Caucasians, but their hearts and brains also diminutive by comparison and their nervous systems less well-developed. On the other hand, their livers, kidneys, and “glands” were larger and more active than Caucasian counterparts, and their skin threw off heat more rapidly than that of whites.12

As these dissimilarities were “discovered” and commented upon, the notion naturally grew that a direct relationship existed between the blacks’ anatomical peculiarities and their disease susceptibilities. Certainly the smaller brain and less developed nervous system seemed to account for the blacks’ yellow fever and malaria immunities, because fevers were regarded by many physicians as “excitements” of the brain and nervous system. A smaller brain and retarded nervous system apparently held these excitements to a minimum. Some would tack on to this explanation the notion that black skins threw off heat better than white; thus blacks had a better ventilation system for getting rid of fevers.

The relationships between the anatomy of blacks and their disease susceptibilities were more elusive to “discover” than the above “dissimilarities.” True, weak lungs meant more pneumonia, but what did larger livers mean? Or kidneys or glands? Some experimentally inclined physicians wreaked considerable wear and tear on slave patients in an effort to find out. (This, incidentally, was thought to be excusable because of still another medical “discovery”; blacks, it was asserted, did not experience pain to the same degree as whites—again because of their nervous systems.) Yet even though a black bore up better under surgery, doctors felt that “Negro peculiarities” usually made medical practice more difficult by posing tricky problems of treatment. Foremost among these problems was the conviction that large quantities of blood should not be taken from them nor large doses of medicine given to them. In fact, to treat a slave as one would a white patient might kill him, all of which implied a substantially different approach to the sickbed of a black patient. Bereft of their old standbys (venesection and massive dosing), regular physicians must have viewed a black as an odd and trying patient indeed. Stemming in part from this frustration came the call during the 1850s for a separate branch of medicine in the South—Negro medicine.

The man who led this call and spoke for many in the medical profession was Dr. Samuel Cartwright of New Orleans, a self-styled expert on diseases in Negroes—whose writings on the subject elaborated substantially on the “discoveries” of the physiological differences of Negroes made by his predecessors and contemporaries. The Negroes’ blackness according to Cartwright was more than skin deep, and in fact “darkness” pervaded every area of the Negro body from the brain to muscles, tendons, and membranes. However, the Negroes’ major problem, according to Cartwright, was a “hebetude of intellect,” stemming from an improper “atmospherization” of their blood—the latter in turn due to weak lungs and other anatomical defects. It was this “defective hematosis,” Cartwright explained, that caused Negroes to lack in courage and mental energy, and in tandem with their smaller brain bred not only their “indolence and apathy” and childlike behavior but also their absolute dependence on whites.13

Following Cartwright’s lead, a Georgia physician summed up the findings of the antebellum medical establishment. “Look,” he commanded, “at [the black’s] small brain, his deficient intellection, his small lungs and defective atmospherisation, his increased liver, his black blood and sluggish circulation, his obtunded nervous sensibility, his enlarged glandular system all pointing to a physical character, animal in its inclination and wanting in those attributes of intelligence which elevates the white man and places him far above the brute.”14

Thus blacks became lazy, childlike, sluggish, dull in mind, weak in will at the hands of antebellum physicians, who found answers to disease immunities and susceptibilities that once more made blacks pay twice for their disease experience. In the process antebellum physicians, in the words of George Fredrickson, elevated “prejudice to the level of science, thereby giving it respectability.”15

Doctrine of the Degeneracy of Blacks and the Fears of Whites

During the postbellum years, a combination of epidemiological factors, increased mobility and the shift of many from rural to urban residence added two more diseases to the list of those to which blacks revealed a special susceptibility—these were syphilis and tuberculosis. Additionally rickets was so prevalent among black children that doctors in Baltimore, Washington, Memphis and Richmond were convinced that all young Negroes had to suffer from the disease as a kind of rite of passage. Finally, as before the War, infants and youngsters were dying of teething, worms, fits and seizures, convulsions, tetanus and trismus nascentium, while adults were still succumbing in large number to pneumonia.16

14 Ramsay, Typhoid Fever, p. 17.
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Considered together, a high frequency of stillbirths, an appalling level of infant mortality and the near epidemic assaults of pneumonia, tuberculosis and syphilis on the blacks convinced many postbellum physicians that the African race was doomed to extinction in America. However, this was regarded as something not necessarily bad; indeed, viewed within the prevailing social Darwinistic context of the period, it was seen as inevitable by physicians for whom blacks presented living or, perhaps better, dying proof of the Darwinian “survival of the fittest” doctrine. When races were thrown together in a competitive situation, the least fit race was always doomed. The peculiar institution had provided a snug, womblike non-competitive world for Afro-Americans; but now they had to think for themselves, care for themselves and compete with the whites. That the blacks’ future looked so grim was seen as proof that they could do none of these things, and the doctrine of the degeneracy of blacks was born.

Physicians could accept the end of this “degeneration” process calmly enough, but the process itself alarmed many who feared the consequences for the white community. Some complained that since the War blacks had not only managed to double their mortality rate but their crime rate as well. Many worried about syphilis in Negroes which was seen as “a very real menace to our white boys and through them after marriage to our innocent daughters also. For despite our best efforts many boys are going to sow wild oats.” Equally worrisome was tuberculosis, because “if our cooks, nurses and other servants have the disease, there is danger of our families becoming infected.”17 On a larger scale, others fretted about the hundreds of thousands of blacks employed at tasks ranging from vegetable handlers to restaurants personnel to boot blacks, who might infect whites with their diseases.

The spectre of the threat of blacks to the health of whites was not confined only to the susceptibilities of blacks; immunities as well came under venomous medical scrutiny. The spread of hookworm among whites was blamed on blacks (blacks have a natural resistance to hookworm infection) whose unsanitary habits made them “soil polluters.”18 They were also branded as the “great reservoir” of malaria in the South, placing whites who lived near them under a “hygienic handicap.”19 Finally, the historically-minded among the profession completed the indictment of blacks as propagators of disease, recalling that they were also responsible for the introduction of leprosy, filariasis and yellow fever.20

The prevalence of syphilis among blacks also triggered another kind of fear, because it was taken as proof of the licentiousness of blacks.

17 Dr. Seale Harris quoted by H. L. Sutherland, “Health Conditions of the Negro in the South: with Special Reference to Tuberculosis.” Journal of the Southern Medical Association 6 (1909) : 405.
18 Charles W. Stiles, “Soil Pollution as Cause of Ground Itch, Hookworm Disease (Ground-Itch Anemia), and Dirteating,” Rockefeller Commission Pub. No. 1 (1910), p. 35.
19 Ibid., pp. 35, 36.
and as one more indication that they were "rapidly reverting to barbarism" — a barbarism which posed a sexual threat to white females and children.21 “A rape by a negro of a white woman,” it was typically declared, “was almost unknown” during slavery. But now emancipation had left blacks “free never to labor, and sleep in hovels where God’s sunlight and air could not penetrate — absolutely free to gratify [their] every sexual impulse; to be infected with every loathsome disease and infect [their] ready and willing companions, and [they] did it, [they] did it all.”22

Hence pneumonia, tuberculosis, syphilis, and an alleged propensity for raping white women and children all became part of the lens through which the postbellum physician viewed Negroes, and the health problem of blacks became part of the larger "Negro problem" — a problem which some physicians argued ought to be entrusted “to the science of medicine.” One solution offered by that science was “the sterilization of the entire male negro population by vasectomy” which would "bravely and humanely [resolve] the greatest race problem of the ages.”23

Fortunately, as the twentieth century matured, virulent racism of the kind mentioned above within the medical profession faded; unfortunately, the crisis of the health of blacks did not. During the years 1929-31, the death rate of blacks from pellagra was fourteen times that of whites; tuberculosis was removing 199 blacks annually from every 100,000 of their number and pneumonia 254 (the death rates of blacks in both cases were more than three times those of whites.)24 Today these diseases have at least been brought under control, and although tuberculosis and pneumonia still prefer blacks to whites at better than a three to one rate, they simply are not the great killers they once were. In the case of tuberculosis, for example, while the mortality of blacks from the disease is about five times that of whites, we are nonetheless only speaking of the deaths of eight blacks per 100,000 population compared with the 250 to 300 per 100,000 of yesterday. Largely because these old enemies are collectively far from the nemesis of the Afro-American community they once were, the death rate of blacks which in 1900 stood at 30 per 1000 (as opposed to 17 per 1000 for whites) is now for the first time in our history virtually the same as the white with the exact figures 9.4 per 1000 for blacks and 9.3 for whites.

Infant Mortality, Maternal Nutrition, and Vitamin D

Today's overall death rate when calculated without reference to age conceals one last age-old enemy of blacks: that enemy is infant mortality.

For today, although the death rates of blacks and whites are the same, many more blacks than whites are dying at a younger age, with black babies having an 80 percent greater chance than their white counterparts of making the journey from cradle to grave in less than a year. Thus infant mortality among blacks is almost twice that of whites. Certainly the infant mortality of blacks in 1850 was substantially more than twice that of whites; clearly then, the comparative disadvantage of black infants in terms of survival remains a century-old problem, whereas the comparative disadvantage of black adults has decreased considerably.

Today the major cause of the differential between blacks and whites is thought by many to stem from the low birth weights of black babies, for it is axiomatic that the lower the birth weight, the greater the risk of perinatal death. Of the 50,000 infants in the United States who do not survive their first week of life, 75 percent are LBW babies, meaning they weigh less than 2500 grams or 5½ pounds. The odds against LBW babies living out a full year of life are seventeen times greater than those against an infant weighing over 2500 grams.

Unhappily blacks are inordinately represented in this risky LBW category. In 1960 an Afro-American had a 90 percent greater chance of dying during his first year than a white American; significantly he also had a 90 percent greater chance of being a LBW baby. Black infants arrive in the world weighing on the average 200 grams less than whites, reflecting the fact that, in percentage terms, blacks have twice as many LBW babies in every LBW category than do whites.

Although many factors ranging from the age of the mother to cigarette smoking can influence birthweight, maternal nutrition seems unquestionably to be the most important. By way of illustration, studies conducted in famine-ridden parts of Europe during and after World War II discovered a significant increase in the frequency of LBW babies; yet as nutritional plenty returned, birth weights also returned to normal. In the United States two extensive surveys, the Department of Health, Education and Welfare's Ten-State Nutritional Survey (carried out during the years 1968-70) and the National Center for Health Statistics, Health and Nutrition Examination Survey, as well as literally scores of

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smaller efforts, have found evidence of massive malnutrition among blacks today. Blacks, for example, receive less than half of the calcium and iron than do whites, while their diets are also dangerously low in vitamins A and C, magnesium, the vitamin B complex and protein.

In addition, there are certain physiological traits of blacks, which most persons of European descent do not possess, and which in some cases cause, and in other cases exacerbate, these nutritional deficiencies. One is pigment which, it has been hypothesized, protected the skin from sun damage in West Africa. In the more temperate United States, where abundant year-round sunshine is not the norm, however, pigment means that blacks receive about one-third less ultraviolet radiation than whites — radiation which triggers the mechanisms producing vitamin D. Vitamin D is essential to the metabolism of calcium and magnesium, and the diet of blacks is extremely deficient in these minerals. The major dietary source of D for most white Americans today is milk, which is fortified with that vitamin; yet about three-fourths of the nation's blacks are lactose intolerant, meaning that they cannot drink milk because they possess low levels of the lactase enzyme, which breaks down milk sugars. Lactose intolerance is not unusual but rather the norm for most people not of West European descent, and in other cultures the problem is handled either by the inclusion of some other high calcium-yielding staple in the diet, or by the consumption of milk in soured form such as yoghurt. In our milk drinking culture, however, good nutrition is made difficult for persons who are lactose intolerant.

Maternal vitamin D deficiency, calcium deficiency, and magnesium deficiency have all been individually linked to low birth weight babies and doubtless go far today to account for the differentials between blacks and whites of today. Yesterday these differentials were probably even more pronounced. The nutritional diseases pellagra and rickets, for example, were “discovered” to be serious problems of health in the United States at about the same time, just after the turn of the century, and found to be particularly severe among black people.

Indeed rickets, as mentioned previously, was thought by physicians in urban areas to afflict nearly every black child, meaning of course that vitamin D deficiency (and thus calcium and magnesium deficiencies) were extraordinarily widespread among the black community. Almost equally widespread were low birth weights, according to a Georgia doctor with access to the birth weights of “thousands” of whites and blacks, who found in 1934 those of blacks to be significantly less;
while in the late nineteenth century, although no scientific studies of birth weights have come to our attention, a look at the medical literature of the period indicates that postbellum physicians took it for granted that blacks would have "smaller infants."  

Moving backward once again to the antebellum period, one might assume that slaves spent more time outdoors and thus had less of a problem with vitamin D deficiency than their postbellum city dwelling descendants. Nonetheless, evidence of that deficiency among slaves abounds. As a nutritional disease rickets was not well understood by physicians, and because it was seldom fatal it was not high on the list of diseases antebellum physicians concerned themselves with. Still, of the 25 deaths diagnosed as caused by rickets in America's seven largest slaveholding states during the year 1849-50, 24 of the victims were black, emphasizing the disease's racial prejudice. Evidence of its high frequency among blacks comes from runaway slave advertisements culled from newspapers ranging geographically from New Orleans to Charleston to Memphis that contain numerous identifying characteristics of a runaway such as "bowlegged," "slightly bandy-legged," "very knock-kneed," "slightly knock-kneed" and "much knock-kneed" — indeed, these descriptions appear so often that one wonders how they could have been actually useful for purposes of identification. Bowlegs and knock-knees, of course, are the most obvious adult signs of a bout with childhood rickets.

Additionally, there is another possible method of gauging the extent of calcium, magnesium and vitamin D deficiencies on the plantation. These deficiencies, singly and in combination, play an important part in the etiology of a long misunderstood children's disease called tetany — an affliction characterized by hyperirritability of the neuromuscular system, whose symptoms include convulsions and spasms of the voluntary muscles. Calcium is important to the proper contraction of these muscles, magnesium to their relaxation, while vitamin D is essential to calcium's absorption and very possibly to that of magnesium as well; thus a severe deficiency of any one may produce tetanic symptoms, the most obvious being convulsions which can lead to death.

A sample of the seven largest slaveholding states during 1849-50 reveals that about 2500 youngsters died of convulsions, fits and seizures, etc., all of which are suggestive of tetany. Significantly, by race these "diseases" killed 204 black children nine-and-under per 100,000 live population as opposed to only 74 whites in the same age cohort — significantly, because tetany today is a far more serious problem for black  

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youngsters than white, given the problems of blacks with calcium and magnesium already discussed. This by itself of course is hardly over-whelming proof of tetany on the plantation; but in combination with the apparent prevalence of rickets it does argue strongly for it, because as a rule those nutritional circumstances which encourage rickets will also encourage tetany, and a people are seldom afflicted with one but not the other.

If it is true that blacks in the antebellum South, like their postbellum and present day descendants, were calcium, magnesium and vitamin D deficient, then it means that in addition to those youngsters whom tetany claimed, slaves too had more than their share of LBW babies whose reduced chances of living out a year of life contributed heavily to the high rate (relative to whites) of slave infant mortality.

Conclusion

As soon as epidemiologists put all of the above genetic, nutritional and climatic factors together — in other words, link LBW babies with lactose intolerance, black skin, northerly climate, improper maternal diets and an historic situation of vitamin D, calcium and magnesium deficiencies — one suspects that a great deal will be done to bring black infant mortality today into line with the white rate. Yet an important reason this has not been done already is that the same racism which we saw in the past indicting blacks for their own disease experience — twisting their heavy incidence of morbidity and mortality from certain diseases into notions of their inferiority — ironically since the Second World War has become in itself twisted to the disadvantage of blacks. Since the War it has been unfashionable, because of our efforts to purge ourselves of racism, for medical investigations to distinguish by race, and only in the last decade or so have such studies which are crucial to the solution of the problem of black infant mortality once more begun to appear in any great number. Of course, studies by race are always potentially danger-ous as antebellum doctors, postbellum doctors and their modern day IQ testing descendants have so forcefully demonstrated. But the ethnic factor in disease susceptibilities is so vitally important to the health of black Americans that we must guard against such racist nonsense driv-ing these studies once more back into the shadow.