Diets Versus Diseases: The Anthropometrics of Slave Children

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What were the living standards of American slaves? According to Robert W. Fogel and Stanley Engerman in their monumental study, *Time on the Cross*, the material standard of living of slaves compared favorably with that of other nineteenth-century agricultural laborers. More recently, utilizing anthropometric data that allow them to construct age-height profiles for slaves, economic historians have cast doubts upon this view as it applies to particular age cohorts. They question the validity of the earlier assessment of living standards as it applies to slave newborns, infants, and children.

The proponents of anthropometrics argue that the average height of a population is a good proxy for net nutrition, and imply that the nutritional status of a population is a better and less flawed measure of overall living standards than per capita income. As a result, the slave height research has led to revisionist conclusions about the diet and nutritional intake of antebellum slave infants and children. Although this literature has made important contributions to our knowledge of antebellum slavery, we have serious reservations about its conclusions. We believe that anthropometricians underappreciate the significant biological and historical role that parasitic diseases played in the physical growth of antebellum slaves. A recognition of the role that parasitic diseases played in the plantation South leads to different conclusions about the way in which we look at the material living standards of antebellum slaves, especially those of slave infants and children. In support of this, we offer estimates of the quantitative impact of two parasitic diseases on the anthropometric measurements of slave newborns and children.

Richard H. Steckel, a leading proponent of anthropometrics, has estimated distributions of slave heights from coastwise slave manifest tests. Relying on relationships between height-for-age and weight-for-age of twentieth-century populations, Steckel derives slave birthweights from his height estimates. Employing twentieth-century data on U.S. stillbirths and

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1 Richard H. Steckel contends "... research momentum has shifted to alternatives or supplements that address shortcomings in GNP as a welfare measure or that indicate living standards in time periods for which conventional measures cannot be calculated ... Less well known are the relationship between stature and conventional measures such as per capita income, and the ways that stature addresses certain conceptual inadequacies in GNP as a welfare measure" (Steckel, "Stature," p. 1903). John Komlos maintains that "This discovery [of a decline in the heights of Americans born in the late 1830s], as well as the subsequent finding of a similar trend in life expectancy, called into question the common wisdom that the rapid expansion of the U.S. economy during the antebellum decades brought about an unambiguous and monotonic improvement in the human condition" (Komlos, "Shrinking," p. 779).

2 For a synthesis of the anthropometric slave findings, see Steckel, "Work."

3 We have an extended discussion of the role of parasitic diseases in the economic history of southern slavery, including the impact of diseases on the efficiency and productivity of slavery and slave labor (see Philip R. F. Coelho and Robert A. McGuire, "Biology"). Space constraints prevented us from offering the additional evidence contained therein in the present essay.
on the relationship between birthweights and first-month deaths among the poor of Bombay, Steckel estimates stillbirth and neonatal and infant mortality rates from his birthweight estimates. These calculations indicate extraordinarily short slave children, extremely low slave birthweights, and very high neonatal and infant mortality rates. The hypothesized reasons for these findings are the work intensity of pregnant slaves, maternal diseases, poor diet of pregnant slaves, specific dietary deficiencies of pregnant slaves, and poor diet of slaves during infancy and childhood.

Steckel contends that the more important contributors to low birthweights and high neonatal mortality were work intensity during pregnancy and (possibly) maternal diseases, the less important contributors were maternal diet and dietary deficiencies, and other factors contributed even less. By implication, work becomes the primary factor in low birthweights and neonatal mortality. The typical slave worked 54 hours per week standing and stooping, and pregnant women had little time off before their fifth month. Seasonal patterns in stillbirths and neonatal mortality are cited as evidence of the effects of intense work on fetal development. But the discussion on seasonal deaths, emphasizing the timing of nutritional deprivation, appears to interchange deprivation, nutritional status, and undernutrition with diet or food intake. Consequently, the role of diet, disease, or work in seasonal deaths is not apparent. Steckel does recognize that another factor might have contributed to lower birthweights: African women with falciparum malaria had newborns that weighed less than those without it. Poor diets are said to have been the principal cause of excessive infant mortality, and diseases secondary contributors. On the causes of extraordinarily short slave children, Steckel concludes that poor infant and childhood diets were primary, and that neither work intensity, nor diseases explain the short stature. But there is no direct evidence on the diets of slave infants and children. The hypothesis for the importance of diets rests entirely on indirect evidence. The evidence is primarily the relatively late growth spurt that allowed slaves to attain nearly the same adult height as northern whites.

Steckel ("Birth Weights," p. 175) does not employ direct data on slave birthweights, as the data do not appear to exist. Steckel does relate the derived mortality estimates to mortality data from plantation records but acknowledges that plantation records notoriously underestimate deaths, especially deaths within the first month (neonatal mortality) and deaths during early infancy.

Low birthweight (less than 2,500 grams) is considered "the greatest single risk factor for neonatal and early infant mortality" (Steketee et al. "Effect," p. 33).


These conclusions are drawn from data from a sample of plantation records.

Steckel, "Work," p. 496. A major contention of the anthropometric and demographic history is that the terms "nutrition" and "nutritional status" mean food consumption minus all claims (work, disease, climate, and any other claims) on the body. According to this usage, nutrition or nutritional status do not refer to food intake or diet only. See Fogel, "Nutrition," pp. 446-47.

Steckel ("Work," p. 493) citing a 1968 study, indicates that malarial newborns weighed 263 grams less. Disease is given a less important role in infant deaths based on regressions employing proxy variables for the disease environment because "the data available do not permit separate measures of the importance of care versus the disease environment for children." Ibid., p. 496.

Although little direct evidence exists on illness by age, Steckel ("Work," pp. 500-01) suggests that his estimates of age-specific mortality rates from plantation records show that sickness declined during childhood until about age seven, concluding that disease cannot explain the short stature of slave children. Whether these mortality data capture the effects of disease is debatable, especially disease during infancy.

For anthropometric findings on northern whites, see Margo and Steckel, "Heights of Native-Born Whites."
The prevailing explanation for the anthropometric deficits of slave newborns, infants, and children is that pregnant slaves were overworked and slave infants and children were severely underfed. Certainly, both diseases and inadequate diets can have adverse health effects that manifest themselves on the human physique. And certainly it is not easy to separate the effects of one from the other. Yet there are ways of distinguishing between the diet versus disease hypotheses but they involve close attention to the details. For example, genetic factors, chronic disease, or inadequate diet may cause clinical anemia. Ignoring genetic factors, it is difficult to distinguish between diet and disease as the cause. In populations in which human skeletal remains have been examined, deficient diets are reputed to have caused porotic hyperostosis. (Porotic hyperostosis is a term used to describe bone lesions in the skull, skeletal evidence of anemia.) But recent studies suggest that, in many populations with this condition, chronic diseases, rather than dietary deficiencies, caused it. In living populations, one way to distinguish between diet-induced and disease-induced anemia is checking blood samples; in disease-induced anemia the level of serum ferritin is normal or elevated, whereas in dietary iron-deficiency anemia it is lower than normal. (A second way is taking actual bone marrow biopsies.) Another way to distinguish between the diet versus disease hypotheses is recognizing that many diseases related to specific nutritional deficiencies in the diet frequently cause body abnormalities that are symptomatic: thus, the bowed legs of a victim of rickets, the sallow complexion and "angel wings" associated with pellagra, and the loose teeth and purple gums of scurvy. So if one were to assign poor diets as the primary cause of abnormally short slaves, they should either expect to find other symptoms of nutritional deficiencies or believe that the slave victims were fed diets with adequate micronutrients but very few calories. We do not take either position, nor do we assign primacy to diets and nutritional intake in explaining the stature of American slaves. One reason for this stand is that we are unaware of evidence indicating that physical deformities were widespread in substantial proportions in slave populations. We believe that the roles of diet during infancy and childhood and work intensity during pregnancy have been overemphasized while the role of the disease environment has been underemphasized. It was the combination of parasitic diseases and the southern plantation system that caused much of the anthropometric deficits. In what follows, we provide evidence on the southern plantation disease environment and on the health effects of parasitic diseases, concentrating upon two primary debilitating antebellum southern diseases--hookworm and malaria. We then discuss the effects of hookworm and malaria infections as they relate to the antebellum South. A short discussion of the current obstetrical and gynecological research on the health effects of work and physical activity is included. We next estimate the quantitative impact that hookworm could have had on the height deficit of slave children and the quantitative impact that malaria could have had on slave birthweights. Reasonable lower bound estimates are hookworm infection alone could account for 31 percent of the slave height deficit and malarial infection alone could account for between 14 and 24 percent of the slave birthweight deficit."
See Atack and Passell, Now Economic View, chap. 12; and Harris, "HealthN" pp. 306-09. See Stuart-Macadam and Ken@ Diet 16 Steckel CBirth Weights", pp. 18749) recognizes the debilitating effects of both hookworm and malarial infections in the antebellum South but does not include any further analysis of their effects upon birthweights, mortality, or stunting. In summarizing his own work, while disease is explicitly discussed his view is still fil the work intensity of pregnant slaves appears to have been the primary factor in lower birthweights and high neonatal mortality, and that infant and childhood diets played the major role in the stunting of slave children. See Steckel ("Work," pp. 491-95, 500-0 1, and 503A5).

Diets versus Diseases

THE SOUTHERN PLANTATION DISEASE ENVIRONMENT

A number of parasitic diseases afflicted the American South. Some of these diseases pre-existed the Voyages of Discovery, and others came to the Americas after contact as part of the interchanges between the Old and New Worlds. The Africans and Europeans who came to the New World descended from peoples who had lived in fundamentally different disease ecologies for millennia. Consequently, the epidemiological experiences, both innate (genetic) and acquired (by exposure), were markedly different for Africans and Europeans. Given the regional differences in the climate and topography of the Americas, the migrations of Africans and Europeans started evolutionary processes that changed the regional disease environments of colonial America. Over time, many of the Old World diseases became endemic in the southern disease ecology. The result was a more pestilential American South. Warm-weather diseases dominated the southern disease environment and economy in the antebellum period. Of these, the best documented and studied are: malaria, yellow fever, dengue fever, dysentery, various geohehminths [flatworm, hookworm, Ascaris lumbricoides (roundworm), tapeworm, Strongyloides stercoralis (threadworm), and Trichiurus trichiura (whipworm)], schistosomiasis, and the ubiquitous smallpox. While at least two of these diseases (roundworm and whipworm) pre-dated contact, all of the diseases were (and are) found in tropical West Africa, with the geohehminth diseases being particularly widespread and abundant there."

The climate and sandy soils of the South, as well as the plantation system, were conducive to the introduction, maintenance, and spread of many parasitic diseases. The antebellum plantation system consisted of relatively dense rural black populations. Dense rural populations and the southern climate and soils: provided malarial reservoirs that allowed for its maintenance and diffusion, provided reservoirs for numerous geohehminths that allowed for their maintenance and diffusion, and increased the exposure of slaves to many parasitic diseases. The majority of blacks living in the antebellum rural South lived in large plantations of 20 or more slaves. Plantation slave quarters were typically built poorly and closely together. Infants and small children born in these plantations were Rely to be kept together in slave cr@ches or "nurseries." Plantations that concentrated children facilitated the diffusion of diseases. The concentration of infants and small children to this day contributes to disease. In the 1990s, "The rate of diarrhea for non-toilet-trained infants in daycare centers in urban arm of the United States is comparable to the rare of illnesses seen in Third World countries."
nurseries were particularly conducive to the spread of hookworm and other intestinal nematodes, as was the practice of allowing children to go barefoot. Slave infants and children, diaper-less and barefoot, wearing only shirts, defecated randomly. Diarrhea made the situation even more favorable to the transmission of numerous geohelminths that spread widely among slave children during the warmer months of the South. While plantation slavery harbored many disease reservoirs, we concentrate on hookworm and malaria because they are especially debilitating, are well studied, and are parasitic diseases that were more heavily concentrated in the South than other diseases.21 Hookworm and malaria require animal hosts to complete their life cycle. Without a critical mass of both animal hosts, these diseases could not have become endemic to the American South. The concentration of human beings into close proximity with one another on southern slave plantations insured the diseases' survival and transmission.11 The poor construction of slave quarters allowed the mosquito vectors access, the high human density of plantations allowed easy transmission from man to mosquito and the reverse. The concentration of slave infants and children in cr@ches facilitated the transmission of pathogens between and among children and their caretakers.

Hookworm larvae in the soil penetrate the skin of their host.11 The penetration is frequently through the feet of people walking barefoot, or through the hands and arms of people in contact with the ground. After penetration, the hook-worms enter the lungs, and when they are coughed up, if they are swallowed, they have their route to the human intestine. The female hookworm, once attached in the intestine and fertilized, commences egg laying. Wherever the human host goes, hookworm goes, and when the human defecates, eggs are deposited along with the feces. Given the primitive state of sanitation in the rural South, hookworms and other intestinal nematodes were widespread. Sanitary privies were rare in the antebellum rural South; facilities in the rural South typically consisted of bushes or open holes or ditches.11 Free-roaming coprophagous animals (chickens, dogs, ducks, pigs, and some wildlife) could eat the feces and spread hookworm eggs by passing the eggs (unaffected) through their own feces.11 Hookworm spread wherever infected people went and, because of its life expectancy and fertility, it was endemic in and around southern plantations.
The spread of malaria takes place when the female anopheline mosquito, seeking a blood meal, bites a human whose blood harbors infectious malaria gametocytes, which reproduce and undergo several stages inside the mosquito, and are eventually introduced into another human." The malaria protozoa depend on human hosts for their survival because nonhuman animals cannot serve as a malarial reservoir, as they do not contract malaria." In places with killing frosts, most mosquitoes die over the winter; their larvae survive, but they are free of malaria. A mass of infected humans must be available to start the cycle again.

In the nineteenth century, the growth of large southern slave plantations and the reduction in transportation costs and time facilitated the spread of malaria throughout the South. The native anopheline mosquitoes served as vectors and incubators of malaria. Where conditions were right malaria flourished. The "right" conditions would be a climate and geography conducive for harboring many mosquitoes and a relatively dense population of infected and noninfected people. Many mosquitoes were necessary because the mosquitoes must harbor the malarial pathogens for a number of days before the vector becomes infectious and there is a quite low rate of prevalence of malaria in mosquitoes.' A relatively dense population is necessary because if the number of infectious vectors is low and, if malaria is to remain an endemic disease, there has to be a relatively large number of people around to propagate the infection. In the antebellum South, the slave labor force was kept close together, and animals

1 Once endemic to the plantation South the diseases spread to nonplantation populations. Discussion of the life cycle of hookworm is contained in Chandler, Hookworm Disease, pp. 91-98. For further discussion, see Dock and Bass, Hookworm Disease, pp. 86-93.

"[In the typical county privy] ... [t]he animals root and scratch the feces, and then scatter them about over the ground. Not only this, but they often eat feces. If the feces eaten by such animals contain hookworm ova, they pass undigested and are thus widely distributed" (Dock and Bass, Hookworm Disease, pp. 86-87).

21 An attached hookworm has a life expectancy of one to five years in the human intestine. And the female hookworm is a prodigious egg layer: estimates of egg production range from 9,000 to 25,000 eggs per day. See Schad, "Parasite." A description of the malarial life cycle is contained in Oaks et al., Malaria, pp. 25-30. There is an exception. Plasmodium ovale is believed to have avian hosts in its African homeland. Anderson and May, Infectious Diseases, pp. 387-88.
were infrequently stabled. This is important: recall that nonhuman animals do not harbor the malaria protozoa. Consequently, a mosquito that carries the protozoa and feeds (after infection) exclusively on nonhumans stops the chain of infection. In the slave South, mosquitoes would have been more likely to bite humans than animals compared to other situations in which animals were stabled and humans dispersed. A constant supply of unexposed infants in slave compounds assured the survival of malaria on large-scale plantations.

THE HEALTH EFFECTS OF HOOKWORM AND MALARIA ON HUMANS

Hookworms (and other geohelminths) cause a number of problems for humans. The symptoms of hookworm infection are: anorexia, abdominal pain, geophagy, loss of appetite, nausea, headache, rash, weakness, fever, vomiting, diarrhea, dysentery, and intestinal bleeding. The impact of hookworms depends on how many are attached, the nutritional and health status of its victims, the host's ability to fight off hookworm, and the rate of infection, which is affected by living conditions, climate, and cultural practices. The same number of hookworm in children is more deleterious to their health than to adults because of their smaller size. The symptomatic effects can contribute significantly to anemia and hypoalbuminemia (inadequate protein) in children. Hookworm reduces the net nutrition available to its victim. A hookworm-infected human has a smaller intake of nutrients because of symptomatic anorexia as well as the likely actual suppression of appetite because the human body produces various cytokines in response to parasitic infestation. These cytokines can act directly on the brain to depress appetite and are known to increase metabolic rate, decrease activity, and produce cachexia and fat and protein catabolism. The food that is ingested has its nutritional value reduced by the symptomatic diarrhea and vomiting. Hookworms claim red blood cells and other fluids, further reducing the net nutrition available.

The effects of hookworm infection on human growth are well documented (as are the effects for other intestinal nematodes). In a study of school children in Malaysia in the 1980s, the data indicate significant differences in anthropometric measurements between those children moderately infected with hookworm and those not infected, ceteris paribus. Children infected with hookworm were, on average, over 6 percent lighter (1.25 kg) and over 2 percent shorter (3.12 cm) than those without hookworm, ceteris paribus. In other studies, the growth of worm-infected children in Kenya was measured after a treatment program that significantly decreased the prevalence and intensity of hookworm and whipworm infections. Anthropometric measurements increased significantly more in treated children than in controls. The effects of hookworm infection on human growth are well documented (as are the effects for other intestinal nematodes). In a study of school children in Malaysia in the 1980s, the data indicate significant differences in anthropometric measurements between those children moderately infected with hookworm and those not infected, ceteris paribus. Children infected with hookworm were, on average, over 6 percent lighter (1.25 kg) and over 2 percent shorter (3.12 cm) than those without hookworm, ceteris paribus. In other studies, the growth of worm-infected children in Kenya was measured after a treatment program that significantly decreased the prevalence and intensity of hookworm and whipworm infections. Anthropometric measurements increased significantly more in treated children than in controls.
children than in placebo groups. In one study, treated schoolboys grew significantly faster than a placebo group. Only four months after treatment, they gained on average 167 percent more in weight (1.6 kg versus 0.6 kg) and 43 percent more in height (2.0 cm versus 1.4 cm) than the placebo group and showed significant improvements in energy levels and appetite as well.34 In another study, hookworm, malaria, and other tropical worms "... were all associated with worse iron status; the association with hookworm was strongest by far."35

Malaria incapacitates by causing recurring fever, chills, vomiting, weakness, and other physical maladies. The malaria protozoa attack the human host's red blood cells and various organs. As with hookworm, malaria is associated with symptomatic anorexia, loss of appetite, and anemia. Leonard Jan Bruce-Chwatt states that the term: "chronic malaria" is sometimes applied to the condition seen in children who in highly endemic areas suffer from many attacks, often untreated, of malaria ... and results in 'malarial cachexia' characterized by stunting of growth, wasting, anemia and much enlargement of the liver and spleen."36 Malaria has differential impacts upon specific segments of a population. Pregnant women and, especially, women pregnant for the first time are particularly vulnerable to malaria.37 Malarial women have smaller babies, and a greater frequency of low-birthweight (LBW) babies, because malarial infection during pregnancy causes both preterm delivery and fetal growth retardation.38 Babies born to malarious women are subject to anemia and other illnesses, and LBW babies are at a greater risk of death. Because some acquired immunities exist, the incidence and severity of malaria disease decline with age in highly malarious environments. Infants and younger children, lacking the acquired immunities and smaller in size, accordingly also are relatively more vulnerable to malaria.

The effects of malarial infection on human growth are documented. In a study of Kenyan children, their growth increased significantly during the first six months of the 16-month study after treatment for schistosomiasis. But growth, as indicated by weight and height gains, was much slower in the last 10 months of the study. The authors determined "... the most likely explanation for this phenomenon, of the variables we measured, is the dramatic increase in malarial infection ... that probably began soon after Exam 2 [the sixth month of the study]."39 A study of malaria and fetal growth in Zaire indicates that malaria in pregnant women had significant effects on newborns.40 Over 18 percent of expectant mothers diagnosed with malarial-related pathologic findings had LBW babies; only 6.5 percent of women without any malarial findings did. The head circumference and body mass of newborns of malarious women also were significantly less than were those of newborns of nonmalarious women. In another study, babies born to Malawi women diagnosed with placental malaria, were, ceteris paribus, 1.7 times more likely to be LBW.41 Among firstborns, the incidence of LBW to malarious women was 31.6 percent; for nonmalarious women, it was 24 percent. Among second borns, the incidence of LBW to malarious women

"growth effect" of 0.3 cm growth over one year for a treated group, it typically means that one year after the study began the subjects given the treatment dosage grew 0.3 cm more than the placebo group. The 0.3 cm should not be interpreted as the total effects of hookworm on child growth unless one believes that the effects on stature of a lifetime exposure to hookworm can be offset in one year when people have a lesser worm burden or are hookworm free. We know of no reputable expert in the field who makes that contention.

36 Bruce-Chwatt, Essential Malarialogy, p. 44.
38 Bruce-Chwatt, Essential Malarialogy, p. 44.
39 On malaria and pregnancy, see Brabin, "Risks."
41 Steketee et al., "Effect," pp. 39–40. Recall that low birthweight (LBW) is less than 2,500 grams.
43 Meurs et al., "Gestational Malarialogy."
44 Steketee et al., "Effect," p. 35.
was 20.5 percent; for nonmalarious women, it was 10.4 percent. In all other births, the incidence of LBW to malarious women was 16.5 percent; for nonmalarious women, it was 9.1 percent.42

THE EFFECTS OF HOOKWORM AND MALARIA IN THE ANTEBELLUM SOUTH

Endemic hookworm and malaria in the South affected the health of slaves. Regardless of diet, hookworm and malarial infections would have had harmful effects. These effects would have been especially severe on pregnant women, infants, and children, resulting in lower birthweights; high neonatal and infant mortality; small childhood stature; and a severe protein deficiency in children (anemia, hypoalbuminemia, and kwashiorkor). Slaveowners may have systematically deprived slave infants and children of adequate diets, but a more compelling reason for the anthropometric deficits, and a late adolescent growth spurt is endemic hookworm and malaria. Regardless of the food resources available, a malarious and hookworm- and other-gastrointestinal-infested population will have, relative to others, reduced body size, increased mortality, and especially vulnerable newborns, infants, children, and pregnant women.

Regarding the relationship between the work intensity of pregnant slaves and the health of slave newborns, the scientific assessment of the health effects of work and physical activity during pregnancy among specialists in obstetrical and gynecological research has changed over the last twenty years. The current interpretation is that there is an inconclusive relationship between the effects of maternal work and physical activity (including strenuous activities) and the health of newborns. There is much evidence supporting no relationship, as well as some evidence supporting beneficial, and other evidence supporting a deleterious relationship, between physical activity and work among pregnant women, and the incidence of preterm birth, intrauterine growth retardation, and LBW. A recent study, for example, indicates that employment among black women significantly lowers their risk of having LBW newborns.43

One must consider other consequences of slaveowners putting pregnant slaves in tasks that were less physically demanding than fieldwork. Those women who were assigned to the care of children in plantation nurseries might have been in a worse, not better, environment than in the fields. The children's lack of bowel control and the shaded environment would increase the probability of hookworm infection. If the women went barefoot, the risks of hookworm infection would be compounded. Pregnant women infected with hookworms (and other intestinal nematodes) and malaria will give birth to smaller babies and will nurse them less well than women who have neither disease. Lower birthweights and high neonatal and infant mortality are all consistent with hookworm and malarial infections, as is evidence of stunted children.44 When slave children were sent to the fields, they left

42 Ibid. p. 36, figure 2. Independent of malarial condition, firstborns are more likely to be LBW and on average have lower birthweights. Compared to the overall sample mean birthweight in the study of 2,905 grams, the mean birthweight of firstborns is 2,721 grams (ibid., p. 36, table 2).
43 See Poerksen and Petitti, “Employment.” For an excellent review of the literature on the entire epidemiology of low-weight and preterm births, including a review of studies indicating a beneficial effect of work and physical activity on birthweight, see Berkowitz and Papiunik, “Epidemiology,” especially pp. 429-30, 433-34. For a review of the literature on the effects of physical activity and employment on preterm and lowweight births that indicates an inconclusive relationship, see Simpson, “Are Physical Activity.” For a study indicating no significant relationship between employment, including the amount and level of the physical intensity, among pregnant women, and the risk of spontaneous abortion, see Bryant and Love, “Effect.”
44 People of tropical African descent are more refractory to hookworm and malarial infections than other peoples are. See Kilama, “Hookworm Infection”; and Allison, “Genetic Factors.” However, this
an environment highly conducive to the spread of diseases, especially hookworm. These slaves were then typically given shoes that helped inhibit hookworm infection. And those sent to cotton fields were sent to an environment that was inhospitable to the survival of hookworm larvae.

While hookworm was endemic throughout the South, whites typically would have been exposed to fewer of the pathogens than plantation blacks. White infants and children were less likely to be raised in conditions that were so conducive to the breeding and transmittal of disease. Few whites lived in the relatively dense population clusters that characterized the plantation system. Much of the white population also may have suffered fewer mosquito bites. The resident white population of a plantation typically was absent from the slave quarters at the times when mosquitoes' bites were most likely—early mornings and evenings. Wealthy whites adapted to existing conditions; going away from their plantations during malaria seasons, sending away the most susceptible members of their families, and purchasing relatively expensive quinine which was both a remedy and a prophylactic against malaria. Consequently, the overall impact of malaria on antebellum whites is problematic.

A QUANTITATIVE ESTIMATE OF THE IMPACT OF HOOKWORM ON SLAVE HEIGHTS

We now offer a rough estimate for the impact of hookworm on the heights of slave children and, by implication, upon the other anthropometric and demographic measurements derived from slave heights. We rely on Li Chien Foo's data on the heights of moderately hookworm-infected Indian and Malay children in Malaysia. The impact of hookworm on these children in late-nineteenth-century Malaysia is used to approximate the impact of hookworm on the height of mid-nineteenth-century slave children. There are a number of issues that affect the validity of Foo's data as a proxy. Do children of Indian, is relative. Africans are still afflicted by hookworm and malaria; and in African children, they have severe consequences. See Stephenson, Latham, Adams, et al., "Weight Gain" and "Physical Fitness."

45 Previous exposure to both hookworm and malaria would have given the young slaves some acquired immunity. Consequently, the growth spurt that young field hands experienced could be due to both fewer infections and an immune system primed to resist these pathogens.

46 While going barefoot in the rural American South was common among children and lower income people during warm-weather months, the non-use of shoes among adult slaves generally would have been more likely while not engaged in physical labor.

47 See Chandler, Hookworm Disease, p. 186. While cotton fields are prophylactic to hookworm disease, sawmill camps are hospitable to its transmission. So whether the health of slave children was enhanced by their promotion to adult work depended on where they went. Recall that an attached hookworm has a life expectancy of one to five years in the intestine. Consequently, an individual with a heavy hookworm burden who is not reinfected will be cured over time as the worms age and die.

48 Although urban dwellers generally would have suffered substantially less from malaria because the anopheline mosquito's habitat is rural not urban, slaves would have faced greater disease exposure than whites in urban areas as well. Slave children residing in tightly packed living quarters, with poor sanitation because of likely discrimination in the provision of public services, and probably going barefoot much of the year, would have been subject to constant disease infestation. Urban white children, probably wearing shoes more frequently, and with relatively better sanitation, would have suffered relatively less.

49 Recall that Steckel's birthweight estimates are derived from his height estimates, and that his neonatal and infant mortality estimates are derived from his birthweight estimates. See Steckel (1986a).

50 Foo, "Hookworm Infection."

51 Contemporary data are employed because scientific studies of the effects of hookworm do not exist for the nineteenth century, as hookworm was not discovered in the American South until early in the twentieth century.
Malay, and African ancestry react similarly to hookworm infection? Does the fact that people in the mid-nineteenth century were shorter than were late-twentieth century people systematically affect the difference in heights between infected and uninfected people? Is the twentieth-century Malay disease environment worse than the nineteenth-century plantation disease environment was? Unfortunately, data do not exist to resolve these issues.

Whether these issues have any effect is problematic. In assessing their effect on the impact of hookworm on slave heights, all three could have no effect; underestimate, overestimate, or they could work in opposite directions. But a fourth issue does bias the estimate: it underestimates the impact of hookworm on slave children. Late-twentieth-century people are healthier than American slaves were. Indians and Malaysians today can expect to live more than two decades longer than did American slaves. A sickly person who is infected with hookworm will suffer more than will a similarly infected healthy person. Because slaves were sicklier than contemporaneous Indians and Malaysians, hookworm would have had more adverse effects on the slaves. This suggests that any ignored bias from the first three issues, which we expect to be small, may be outweighed by the downward bias of this factor.

Foo estimates that hookworm-infected Indian seven-year-olds were 2.4 cm shorter and hookworm-infected Malay seven-to-nine-year olds were predicted to be 3.8 cm shorter than uninfected children. The Indian seven-year olds are 48.55 percent of the sample and the Malay seven-, eight-, and nine-year olds are 51.45 percent. Thus, the weighted mean height of the children is 7.51 years. Given those sample weights and the height deficits of 2.4 cm and 3.8 cm for infected Indian and Malay children respectively, the weighted mean height deficit from hookworm is 3.12 cm for children aged 7.51 years. The mean height for a 7.5-year-old child is 113.85 cm. The mean modern height standard for a 7.5-year-old child is 123.95 cm. The deficit in the mean height of slave children relative to modern height standards correspondingly is 10.1 cm (123.95 cm minus 113.85 cm). Hookworm infection alone could account for 30.90 percent of this height deficit (3.12 cm divided by 10.1 cm).

The question is what proportion of slave children in the coastwise manifests were infected with hookworm. Unfortunately this is unknown. But to the extent slave children shipped along the coasts of the United States were more likely to be from nearby coastal areas and from southern plantations; we would expect an extremely high hookworm prevalence rate among them. Because nineteenth-century people were shorter than twentieth-century people were, the impact of hookworm very likely would be much greater than 31 percent of any height deficit between slave children and their free contemporaries. Even restricting the comparison to modern height standards, this is an important finding. Recall that the use of late-twentieth-century data imparts a bias toward underestimating the impact of hookworm, so we are confident that nearly 31 percent is not an exorbitant number in estimating the minimum impact of hookworm alone on the height deficits of hookworm-infected slave children.

56 The mean male slave height is calculated to be 114.29 cm and the mean female slave height is calculated to be 113.41 cm from Steckel, “Health,” table 1, columns 2 and 7.
57 The male height standard is 124.4 cm and the female height standard is 123.5 cm (Steckel, “Percentiles,” tables 2 and 3).
58 Because coastal areas were more humid and had fewer killing frosts than inland areas, inhabitants of the coast lived in a much more hostile disease environment. For an extended discussion of the prevalence of hookworm in the antebellum South including evidence indicating it could have been extremely high among southern plantation slave children, see Coelho and McGuire, “Biology.”
A QUANTITATIVE ESTIMATE OF THE IMPACT OF MALARIA ON SLAVE BIRTHWEIGHTS

Our estimate for the impact of malarial infection on slave birthweights has implications upon the neonatal and infant mortality estimates derived from slave birthweight estimates. The estimate relies upon contemporary data for the impact of malarial infection on birthweights for expectant mothers in Africa. While malarial infection has deleterious effects upon many anthropometric measures, the best-documented harmful effects are its effects on birthweight. As in the analysis of hookworm, the same issues arise concerning how well late-twentieth-century malarial data can be used to proxy malaria's effects in the mid-nineteenth century. One issue, though more relevant for the hookworm analysis, is less relevant here. There are fewer differences between populations: the contemporary data on malarial infection and birthweights are for African populations, and the American slave populations were primarily descendents of tropical West Africans. The two other issues are still as valid here: whether smaller people in the mid-nineteenth century, compared to the late-twentieth century, systematically affect differences in birthweights between infected and uninfected peoples; and whether the late-twentieth-century African disease environment is worse than that of the nineteenth-century plantation South.

As before, the influence of the latter two issues is uncertain. In assessing their influence on the impact of malaria on slave birthweights, they could have no effect; underestimate, overestimate, or they could work in opposite directions. There is the same issue as in the hookworm analysis that biases the malaria estimate in a known direction. Because late-twentieth-century Africans are healthier than American plantation slaves were, the proxies used here underestimate the impact of malarial infection upon slave newborns. Sicklier plantation slaves would have had more adverse reactions to malarial infections than Africans in the 1990s.

We use the data in a study by Sylvain Meuris, Bokumu Piko, Peter Eerens, et al. to evaluate the impact of malaria on American slaves. The purpose of their study "was to evaluate the consequences of malaria on the anthropometric characteristics of babies born to mothers living in conditions of year-round transmission of malaria." They report that over 73 percent of expectant mothers studied in Zaire had one or more of three malaria-associated pathologic findings: circulating malaria parasites (active malaria), malarial placental lesions (suggesting prior malarial infection), or low hemoglobin levels (a chronic malarial outcome). The study reports significant differences in birthweights based on the existence of one or more of these pathologies. The mean birthweight of babies born to women without any of the pathologic findings is 3,050 grams. The mean birthweight of babies born to women with one pathologic finding is 2,841 grams, for women with two of the three pathologies, 2,727 grams, and for women with all three pathologies, 2,625 grams.

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37 Meuris, Piko, Eerens, et al., "Gestational Malaria"; and Steketee et al., "Effect." Contemporary data are employed because scientific studies of the effects of malaria do not exist for the nineteenth century, as malaria was not scientifically identified until the late in the nineteenth century.

38 Meuris, Piko, Eerens, et al., "Gestational Malaria."

39 Ibid., p. 604.

40 The mean birthweight among all women in the study is 2,924 grams (Meuris, Piko, Eerens, et al., ibid., p. 605, table 3). This figure is consistent with other birthweight and malaria studies conducted in sub-Saharan Africa. Steketee et al. ("Effect," p. 35), for example, report a mean birthweight of 2,905 grams among Malawi women.

41 These mean birthweights are calculated from Meuris, Piko, Eerens, et al. ("Gestational Malaria," pp. 605-06, table 3 and figure 1). The mean birthweights for each specific pathologic finding are 2,797 grams for women with only circulating malaria parasites (16 percent of the pathologic findings); 2,865 grams for women with only malarial placental lesions (48.4 percent of the pathologic findings); and 2,825 grams for women with only low hemoglobin levels (35.6 percent of the pathologic findings).
Employing the modern standard for birthweight of 3,450 grams, the mean birthweight for the Zaire women with no malaria pathology is 400 grams (11.6 percent) less than the modern birthweight standard. The difference in the mean birthweight for the women with one of the three pathologies is 609 grams (17.65 percent) less; for two of the three pathologies, it is 723 grams (20.96 percent) less; and for all three pathologies, it is 825 grams (23.9 percent) less than the modern standard. The difference in the mean birthweights between babies whose mothers had no findings and whose mothers had all three pathologic findings is 425 grams (13.9 percent) less than the modern standard. Conversely, comparing Steckel's slave birthweight estimate to the Zaire birthweights, his slave birthweight estimate of 2,320 grams is only 305 grams (11.62 percent) less than the mean birthweight for the women with all three pathologies. It is 407 grams (14.92 percent) less than the mean birthweight for the women with two of the three pathologies. It is 521 grams (18.34 percent) less than the mean birthweight for the women with one of the three pathologies.

These comparisons suggest a possible range of about 14 to 24 percent of the shortfall in slave birthweights relative to modern standards could be accounted for by malarial infections alone. Conversely, the comparisons suggest that as much as 58 percent of the 730-gram difference between the nonmalarious Zaire birthweights (3,050 grams) and the slave birthweights (2,320 grams) could be accounted for by malaria infection alone. (This is calculated as the 425-gram shortfall between newborns of the Zaire women with no malaria finding and the women with all three pathologic findings divided by the 730-gram difference.) Similar to the hookworm analysis, the question here is what proportion of pregnant slaves in the coastwise trade were infected with malaria, which of course is unknown. As with hookworm, we would expect an extremely high malaria prevalence rate among slaves shipped along the coasts to the extent they were more likely to be from hostile coastal disease environments and southern plantations.

We expect that a more accurate assessment of the impact of malaria on birthweight deficits of slave newborns is likely to be toward the upper end of the estimated range. This is because contemporary data are likely to bias downward the impact of malaria on slave newborns, and current research into the effects of malaria indicates "the consequences of each pathologic condition have a cumulative effect on fetal growth." Malaria causes LBW, which is regarded as the greatest risk factor leading to neonatal and early infant death, and modern research findings "now suggest that the mortality risk linked to malaria-associated LBW may be substantially higher than previously thought." These suggest an important role for malaria in understanding the health and death of slave newborns.

CONCLUSIONS

The disease environment in which African-American plantation slaves found themselves adversely affected their growth relative to modern height standards. An alternative interpre-

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62 There are likely genetic differences in birthweights between Africans and persons of African descent (blacks) and Europeans and persons of European descent (whites). Birthweights for U.S. blacks persist in being significantly less than for U.S. whites even after controlling for socioeco-nomic differences. And sub-Saharan African birthweights are persistently reported as less than U.S. birthweights.

63 Steckel ("Birth Weights," p. 186) actually reports two mean birthweight estimates for slave newborns—one is 2,320 grams and the other, reported in note 6, is 2,330 grams.

64 For evidence on the prevalence of malaria in the antebellum plantation South indicating it could be extremely high among pregnant slaves, see Coelho and McGuire, "Biology."


66 Steckete et al., "Effect," p. 40. The explanation for this conclusion concerning increased malaria-associated mortality risk is that malarial infection is shown to cause both preterm low birthweight as well as intrauterine growth retardation low birthweight, contrary to the view that malaria causes only growth retardation. The findings of Meuris, Piko, Ercens, et al. ("Gestational Malaria," pp. 606-07) indicate primarily intrauterine growth retardation but they also indicate that preterm low birthweight played a role.
tation of the anthropometric data is to hypothesize as Stockel does that slaveowners systematically deprived infants and children of resources because it was profitable to do so. It is interesting to note that Stockel ("Nutritional Status," p. 48) acknowledges that taller slaves were worth more than were shorter slaves. Consequently, a rational slave owner would consider the decline in the market value of slaves, approximately "... 1.5 percent per inch of height," in any decision to reduce expenditures on food.

Even if all these conditions were met, the disease environment would still extract its toll. The importance of the disease environment is shown in the data analyzed by Howard Bodenham, John Komlos, and Komlos and Peter Cochrans. These data show that living in the South close to the Atlantic coast made populations shorter. The reason is that the coastal areas are more humid and have fewer killing frosts. The human inhabitants of the coast were subject to pathogenic onslaughts more frequently than were their inland contemporaries. These data are consistent with the disease hypothesis; we do not know how to reconcile them with the diet hypothesis.

Slavery was a morally repugnant institution. Moral repugnance does not mean that slaveowners starved infants and children, and overworked pregnant women. The biologic and epidemiological evidence offers an unambiguous, simpler, and less complex explanation: the disease environment adversely affected American slaves, especially pregnant women, newborns, infants, and children.


Bodenham, "Troublesome Caste"; Komlos, "Toward and Anthropometric History"; and Komlos and Cochrans, "Nutrition."

The slaves in the coastwise manifests that Stockel employs may have been disproportionately from nearby coastal regions. This suggests that the heights of shipped slaves may be biased downwards on account of the hostile coastal disease environment. Because the manifests do not list slave residence, this hypothesis has not been specifically tested. Stockel ("Slave Height Profiles," p. 372), though, maintains that the slave height distributions do not indicate a bias, either way.

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