

Racial Differences in Disease Susceptibilities: Intestinal Worm Infections in the Early Twentieth-Century American South

Philip R. P. Coelho* and Robert A. McGuire†

Summary. While the use of ancestry, ethnicity or race in contemporary medical and scientific research is controversial and the subject of debate in the United States, a hypothesis of 'racial' differentials in susceptibilities to disease has utility in the American historical context. This study employs a dataset of 542 residents of Marion County, South Carolina, collected in 1922 by medical teams to investigate the prevalence of hookworm in the American South. An examination of data for Marion County is useful because it is representative of counties in the rural agricultural South where hookworm was endemic throughout at least the early twentieth century. The results of a multivariate regression indicate a large, statistically significant difference in hookworm infection between African-Americans (blacks) and European-Americans (whites). Controlling for other demographic factors, an otherwise average white was 2.8 times more likely to be hookworm infected compared to an otherwise average black in Marion County. The predicted probability of testing positive for hookworm was 56.1 per cent for an average white and 20.3 per cent for an average black. The findings are consistent with other evidence on racial differences in hookworm infection, and have implications for understanding important historical issues concerning economic development in the American South. They also suggest that historical datasets contain important information when ancestry, ethnicity or race indentify people whose heritage is predominately from specific disease ecologies.

Keywords: race; ethnicity; disease susceptibilities; differential disease susceptibilities; racial differences; hookworm; intestinal worms; parasitic diseases; American South; 1920s

It is well known that certain diseases differentially affect specific ethnic groups.¹ Less well known is that some medications are more effective in particular ethnic groups than others.² Yet the value of using ancestry, ethnicity or race when conducting health related research is controversial and subject to debate in the contemporary medical

*Department of Economics, Ball State University, Muncie, IN 47306, USA. E-mail: 00prcoelho@bsu.edu

†Department of Economics, The University of Akron, Akron, OH 44325, USA. E-mail: rmcguire@uakron.edu

¹Some differentially afflicted peoples and their diseases are peoples of north-western European ancestry and skin cancers, tropical West African ancestry and sickle cell anaemia, and central European Jewish ancestry and Tay-Sachs disease.

²Iressa is a pharmaceutical that appears to be effective in treating lung cancers in patients of East Asian ancestry (Japanese and Chinese), and ineffective in treating people of other ancestries. See Zamiska and Whalen 2005. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic blood condition that makes some common treatments for diseases extremely hazardous. The combination of the genetic condition and some diseases affect the choice of medication. See Chan *et al.* 1974, pp. 41–8.

and scientific communities in the United States.³ The debate over racial or ethnic identification is wide ranging, with supporters both for and against its use. Yet both sides of the debate have one feature in common. Both sides neglect extant historical data that might shed light on a primary issue in the debate: are there innate differences in susceptibilities to diseases across people of different races or ethnicities? Bamshad recently argued that using an individual's race in health-related research might be valuable, but only to the extent that race accurately measures the biological, genetic ancestry of an individual, contending that to what extent race captures different gene variants and genetic effects on health among sub-populations is ultimately an empirical issue.⁴

The present paper accepts the contention that whether 'race' captures biological, genetic ancestry is an empirical issue. Accordingly, it employs a unique historical data set to test the hypothesis of differential susceptibilities to disease between individuals of different races. The relationship between race and human susceptibility to an especially widespread and debilitating parasitic disease, hookworm infection, is examined.⁵ The data come from early twentieth-century medical inspections that report the results of microscopic examinations of the stools of 542 residents of Marion County, South Carolina for the presence of hookworm infection. The hookworm report includes data on age, gender, residence, race (whether black or white), and the presence of hookworm and other parasitic worm infections for each individual. The microscopic examinations were conducted in 1922 by medical teams under the auspices of the International Health Board, a Rockefeller Foundation-sponsored organisation.⁶

Hookworm is a helminthic infection that typically spreads by skin coming into contact with soil infected with the larvae of hookworm. Because the larvae cannot survive killing frosts, hookworm is classified as a 'warm-weather' disease. Under the right conditions, however, it can become endemic in areas where it normally does not exist.⁷ It was an endemic disease in much of the rural agricultural South in the United States at least through the early twentieth century. It was highly endemic in the coastal and sandy soil areas of the South; it was never found to be endemic in most urban areas or other regions of the United States. The county examined, Marion County, South Carolina, has climatic and soil conditions suitable for the in-soil transmission of hookworm (unlike the northern United States) and it was primarily agricultural and rural in the early twentieth century. The county appears to have been a fairly typical agricultural county of coastal South Carolina and typical of the southern counties heavily infested with hookworm.

³Bamshad 2005; Cooper *et al.* 2003; Editorial 2001; Kolata 2002; Owens and King 1999; Risch *et al.* 2002; Schwartz 2001; Wade 2002a, 2002b; Wilson *et al.* 2001; Yancy 2002.

⁴Bamshad 2005, p. 945.

⁵Hookworm is estimated to currently infect 20 per cent of the world's population, primarily in the tropical and subtropical regions of the world. See Hotez and Pritchard 1995, p. 68; Sabin Vaccine Institute 2005.

⁶Other studies have documented that differences in innate immunities between ethnic groups to parasitic diseases are consistent with, and explained by, evolutionary biology. See Coelho and McGuire 1997, 1999, 2000.

⁷Rampant hookworm infestations were found in labourers working in the Alpine tunnels in the mid-nineteenth century, and in mineworkers in Europe and the United States in the nineteenth and early twentieth centuries. The mild constant temperatures found in deep mines and tunnels were conducive to the survival (and subsequent spread) of hookworm larvae.

Consequently, Marion County is representative of the early twentieth-century hookworm region of the United States, the non-Piedmont rural agricultural South.

This paper presents the results of a multivariate statistical examination of the relationship between an individual being hookworm infected and an individual's race (ethnicity) for Marion County, controlling for other potentially confounding demographic factors. The findings indicate there was a substantially greater probability of hookworm infection among European-American (white) residents of Marion County than among African-American (black) residents. This result is consistent with other evidence of racial differences in infection rates found in early twentieth-century hookworm examinations of US Army soldiers stationed in the American South, other International Health Board hookworm inspections conducted during the early 1920s in the South, and earlier studies, less rigorous than the present, that have examined southern hookworm infection by race.⁸

This study is relevant to a range of historical and contemporary issues. Some of the historical issues are: the extent of racial differences in susceptibilities to parasitic diseases in the early twentieth-century United States; the impact of racial differences in disease susceptibilities upon public policy; and the role of biological and genetic factors related to susceptibility in understanding historical changes in demographic and geographic disease patterning. Some contemporary issues are: the genetic basis for the differential resistance to disease; the greater effectiveness of some pharmaceuticals within specific ethnic groups; and the efficacy of categorising people in broad groups to assess their susceptibilities to diseases and medications.

Race, Disease, and History

This study takes an ambivalent position about the identification of population groups by certain broad categories, such as race. This ambivalence is reflected in the contemporary medical and scientific literature.⁹ Although the concept of race is devoid of any logically consistent and coherent scientific or medical meaning, in certain contexts it provides useful information. In the United States, through at least the first half of the twentieth century, people who identified their race as black or 'negro' were overwhelmingly of tropical West African ancestry, and people of this ethnic heritage have differential immunities, resistances, and susceptibilities to various maladies. Historical United States data exist that identify people by medical condition and race or colour. When these data refer to 'blacks', 'coloured', or the 'negro race', it can be confidently assumed the data apply to people of predominately West African ancestry. When the historical data refer to 'whites' or the 'Caucasian race', it can be confidently assumed the data do *not* apply to people of predominately West African ancestry. It may be intellectually more appealing to use the terms 'ethnic background', 'ethnicity' or 'ancestry' rather than race. However, the appeal does not negate the utility of existing data. Analysis and application of this kind of historical data can be used to form inferences about various groups within the overall population.

⁸See Siler and Cole 1917, Frick 1919, Knowlton 1919, Lucke 1919, Kofoid and Tucker 1921; 'Resurveys, Southern States' 1920–3; Smillie and Augustine 1925, Martin 1972*b*.

⁹Bamshad 2005; Cooper *et al.* 2003; Editorial 2001; Kolata 2002; Owens and King 1999; Risch *et al.* 2002; Schwartz 2001; Wade 2002*a*, 2002*b*; Wilson *et al.* 2001; Yancy 2002.

The question remains whether ancestry, ethnicity or race accurately captures an individual's biological, genetic ancestry. In addition to capturing biological differences, race may highlight environmental differences between groups. The exact living conditions of the African-American and European-American populations in Marion County are unknown but we are certain that the socio-economic status of the African-Americans was substantially lower than that of the white population. Although the exact provision of public health services in South Carolina is not known, in the 1920s (and indeed up to the civil rights acts of the 1960s and 1970s) blacks in South Carolina were systemically discriminated against in the provision of public services.¹⁰

In the present study, we are certain that the relevant environmental differences between southern blacks and whites (for example, living standards, socio-economic status, provision of public services, etc.) would have worked *against* blacks, making them *more* likely to have acquired hookworm and other parasites. In terms of the correlation between race and genetic ancestry, the extent of admixture among sub-populations would matter. In the specific case here, there is strong reason to presume less rather than more admixture relative to contemporary populations. In short, for particular places and times, ancestry, ethnicity or race is likely to capture the biological, genetic ancestry of members of various population groups.

Because the black population of South Carolina in 1922 overwhelmingly traced its origins to people who had been born in tropical West Africa, enslaved and transported to North America, it is hypothesised that blacks in Marion County would be relatively resistant to diseases that are endemic to tropical West Africa. Human hookworm is an old world disease endemic to tropical West Africa.¹¹ Evolutionary theory indicates that innate resistance is the product of selection. Human populations become accommodated to the disease environments in which they reside generation after generation. The genetic characteristics that make an individual more resistant to the onslaught of pathogens will spread among members of the population as the individuals with that characteristic survive longer and have more surviving and successfully reproducing children. Yet genes typically have more than one effect on the body. Some of these phenotypic effects have positive survival value, others negative, and others none. Genes that confer resistance to prevailing disease organisms, and whose phenotypic effects do not offset their survival advantages, will spread in the relevant populations as people reproduce. Accordingly, the human populations of tropical West Africa over countless

¹⁰In 1910, South Carolina spent 19 cents on the education of black children for every dollar spent on a white child's education. In 1935, that ratio was 28 cents per black child for every dollar spent on a white child in South Carolina's educational system. See Margo 1990, table 2.51, p. 21. In studying the provision of public water and sewer services in cities during the early twentieth century, Troesken 2004, however, does not find systematic discrimination against blacks in his sample. In another study, Costa and Kahn 2003 find ambiguous evidence on the discrimination against blacks in the provision of public health investments in the United States in the early decades of the twentieth century. However, neither of these studies focuses on South Carolina or the American South, nor do they offer evidence on rural populations. The provision of public services to urban blacks during the early twentieth century in the American South was problematic at best; in the rural South, the provision of public services to whites was primitive; it was worse to blacks.

¹¹Coelho and McGuire 1997, pp. 90–4, 105–9.

generations would have been evolutionarily selected for innate characteristics that endow relative resistance to hookworm infection.¹² Evolutionary theory suggests the possibility of significantly *different* susceptibilities to hookworm infection between African-American and European-American populations in the United States, with blacks being less susceptible and suffering fewer deleterious effects. Similarly, because the white population of South Carolina in 1922 overwhelmingly traced its ancestral heritage to people who had been born in north-western Europe, it is hypothesised that whites in Marion County would be relatively susceptible to diseases that were not endemic to the prehistoric environment in which the ancestral populations of north-western Europeans were selected.

The examination of historical differences in susceptibilities to hookworm between southern blacks and whites provides important public health lessons. The differential impact of hookworm on two distinct populations, identified as 'black' and 'white' living in the same rural area, can be quantified using available data. Knowledge of the differential impact of hookworm disease (and, indeed, any disease that has a human host) is important in controlling the disease. If some infected individuals are asymptomatic to hookworm and, as a result, are left untreated, then the disease may reappear in spite of efforts to suppress it among other sub-populations.¹³

There are other aspects of differential immunities that affect public health. If blacks were more resistant to hookworm infection and/or their infections were less burdensome, then, as a result of the systematic discrimination against blacks in the provision of public health services, the black population would serve as a disease reservoir for hookworm, facilitating the infection of people of non-West African ancestry. The discrimination in the provision of public health services against blacks and in favour of whites would effectively preclude the extinction of hookworm as a menace. On the other hand, the systematic discrimination against blacks more generally, and the resultant segregation in the American South, would have lessened the public health impact of the hookworm disease reservoir as the hookworm parasites would have been more concentrated in the segregated black areas of the South.

The concept that different racial (ethnic) groups have differing innate (genetic) characteristics is emotionally charged. The history of supposed genetic differences among racial or ethnic groups is singularly unfortunate in the United States and elsewhere. Regardless of its history, science does recognise that there are genetic differences among ethnic population groups whose ancestors come predominately from specific locales, and that these differences may have implications for the susceptibility, incidence and severity of various diseases. Genetic disorders are widely recognised as being concentrated among people of specific ancestries. In the United States, the incidence of Tay-Sachs is concentrated among those whose ancestry can be traced to Jews from Central Europe.

¹²The exact scientific basis of the relative resistance to hookworm infection of people of West African ancestry is not known. But evidence has emerged suggesting that genetic factors may partially account for human susceptibility to hookworm infection. See Williams-Blangero *et al.* 1997, pp. 201–8.

¹³A famous case of an asymptomatic carrier of disease is Mary Mallon, known to history as 'Typhoid Mary'. She was an asymptomatic carrier of typhoid fever who was institutionalised because of the threat she posed to public health. See Brooks 1996, pp. 915–16.

The genetic conditions of sickle cell anemia and glucose-6-phosphate dehydrogenase (G6PD) deficiency disproportionately affect people of tropical West African ancestry, as does the lack of the Duffy blood group antigens, Fy^a and Fy^b. Because the transmission of genetic diseases is relatively well understood, these observations are uncontroversial.

There are also diseases, whether genetic or not, that have statistically disproportionate effects upon identifiable populations. Before the late nineteenth century, tropical West Africa earned the sobriquet of the ‘white man’s grave’ because death-rates for British soldiers there averaged between 20 and 70 per cent per year during the early to mid-nineteenth century.¹⁴ Conversely, in the eighteenth century, tropical West Africans died at substantially higher rates in the North American colonies of Pennsylvania and Massachusetts than did people of British ancestry; and people of north-western European ancestry died at substantially higher rates than African slaves in the British Caribbean—from 3.5 to 5.0 times as high.¹⁵ These disparities in death rates are a result of evolutionary selections. In specific locales where ‘warm-weather’ diseases (malaria, yellow fever, dengue fever, intestinal nematodes, etc.) predominate, natives of that locale are more resistant to that specific disease ecology than are natives of areas with different disease ecologies.

The resistance to a disease from a particular ecology may be acquired, innate or both. However, people born in the United States whose ancestors came from tropical West Africa will not have inherited any acquired resistance to tropical West African diseases. Their acquired resistance will be virtually identical to those of other Americans born in the United States who have the same contemporaneous disease exposure as the African-Americans but whose ancestries trace back to non-tropical regions. This means that the Marion County experience is relevant for determining whether there are innate differences between populations in their resistance to hookworm infection.

Hookworm

Hookworm disease is caused by an infection of parasitic nematodes (genus *Strongyloidea*).¹⁶ There are two major hookworm species that afflict humans, *Ancylostoma duodenale* and *Necator americanus*.¹⁷ Hookworm was not identified as a problem in the United States until 1903 when population samples were drawn from the southern United States. Hookworm was then discovered to be endemic in the American South.¹⁸ In response, the Rockefeller Sanitary Commission for the Eradication of

¹⁴Curtin 1968, tables 1–3, pp. 203, 205–6; Davies 1975, pp. 93–5; Steckel and Jensen 1986, pp. 60–2. European death-rates in Africa are summarised in Coelho and McGuire 1997, table 1, pp. 95–7, and note 61.

¹⁵On African death-rates in the northern North American colonies, see Klepp 1994, p. 477 and note 13, p. 479, table 5, p. 489. These rates are summarised in Coelho and McGuire 1997, table 2, pp. 98–9, and note 71. On European death-rates in the Caribbean, see Curtin 1968, tables 1–3, pp. 203, 205–6 and Rogoziński 1992, pp. 167, 172. These rates are summarised in Coelho and McGuire 1997, table 1, pp. 95–6.

¹⁶This section borrows freely from Coelho and McGuire 1999, pp. 158–63.

¹⁷Hotez *et al.* 2004, p. 799.

¹⁸The presence of endemic hookworm in the southern United States was given wide publicity in a hypothesis presented by Charles Wardell Stiles at a scientific conference in the early 1900s. In reaction to Stiles, ‘[n]o less than eight investigators surveyed all or part of the southern lowlands . . . In every instance, the

Hookworm Disease was organised in late 1909 and lasted until March 1914. The Sanitary Commission determined that more than 43 per cent of all Southerners were infected with hookworm at the time, or 7.5 million people out of a population of approximately 17.5 million.¹⁹ Compounding the error of not recognising the disease earlier, the species of hookworm infecting southern Americans was identified as native to America and named *Necator americanus* (American killer). The scientific appellation remained even after it was discovered that *Necator americanus* was endemic to tropical West Africa, parts of the Middle East and Europe, and the Indian sub-continent.

Heavy hookworm infection causes morbidity and reduced energy levels, which had such negative consequences for human productivity that within a few years of hookworm's discovery in the American South it became known as the 'germ of laziness'. Hookworm disease had a significant impact on economic development in the southern United States.²⁰ Hookworm, though, is not considered as common in the southern United States today as it was in the early twentieth century.²¹ However, in the less developed regions of the world, hookworm continues to be 'one of the most common chronic infections' and is 'among the most important tropical diseases in humans' with important consequences for current economic development.²² An estimate derived from UNESCO has approximately 1.3 billion people infected worldwide.²³

Hookworms attach themselves to the human intestine and obtain nourishment by sucking their host's blood and other nutrients. The hookworm arrives in the small

results were the same; each researcher uncovered numerous cases of the disease and provided concrete evidence that hookworm was indeed a condition from which many in the South suffered'. See Marcus in Savitt and Young (eds) 1998, p. 91, footnote omitted.

¹⁹See Brinkley 1994, pp. 134–43.

²⁰See Brinkley 1994, 1997; Bleakley 2003.

²¹But it might not be as rare today as often thought, at least in the rural South. As recently as the early 1970s, 14.8 per cent of schoolchildren in a sample from rural Kentucky were found to be hookworm infected. See Haburchak 2005. Martin 1972a, in a 1969 survey of rural Georgia, found a 12 per cent hookworm infection rate in a sample from the coastal sandy soil areas of Georgia. However, he found only a 4.6 per cent overall infection rate for the rural areas of the state. With respect to urban areas of the United States, Pierz *et al.* 1973 found a minor incidence of hookworm in low income Puerto Rican and black children in Hartford, Connecticut, and only among those born in, or who had recently visited, Puerto Rico. None of the black children was found to have hookworm. Interestingly, among the blacks, only those who were born in the southern United States were found to be infected with other intestinal helminthes. Pierz *et al.* also discuss several studies appearing in the 1950s and 1960s which indicate that hookworm infections (as well as infections with other intestinal helminthes) in the north-eastern United States were overwhelmingly among recent immigrants or migrants from hookworm endemic locations (Puerto Rico or the southern United States). They also cite a study showing a similar result for England. In the 1960s, hookworm and other geohelminth infections were found only among children whose families had recently immigrated from India and Pakistan. Hubbard *et al.* 1974 report only one case of hookworm infection (that of a recent immigrant from Honduras) in a study of 887 five year-old 'negro' school children in New Orleans, Louisiana in the 1969–70 school year. The latter studies indicate that hookworm is not a significant urban disease and that its transmission in the north-eastern United States is problematic. None of the studies, however, can be considered definitive because the US Center for Disease Control no longer collects data on hookworm.

²²Hotez *et al.* 2004, p. 799.

²³Sabin Vaccine Institute 2005.

intestine by a circuitous route. Hookworm larvae in the soil penetrate the skin of their host.²⁴ The penetration is frequently through the feet of people walking barefoot, or through the hands and arms of people in contact with the ground such as farmers and miners. The host's body reacts to this penetration by 'causing dermatitis labeled "ground itch" or "dew poison" in the southern United States'.²⁵ After penetration, the hookworms snake their way through the blood stream into the lungs. The worms are then coughed up, and if the mucosa containing them is swallowed, the hookworms have their route to the small intestine. Once attached they live one to five years, grow, and mate (hookworms are sexual). The hookworm eggs produced by the female are passed with the faecal matter of their host.

The female hookworm lays eggs prodigiously. 'Frequently cited estimates of egg output range from 9000 to 25,000 eggs/female per day'.²⁶ The deposited eggs produce larvae that moult twice before they can infect another host. Depending on circumstances, the hookworm larvae can live for a few days to months before finding a host.²⁷ The optimal conditions for long-term larvae survival are protection from direct sunlight, a moist (but not liquid) environment, and a temperature between 15° and 35°C.

The life-cycle of the hookworm is fairly straightforward.²⁸ The hookworm eggs deposited in the faeces typically consist of four cells and are not infectious. The eggs mature into an embryonic stage (under favourable conditions this takes over 48 hours), and then into a larval stage (approximately another 48 hours). Before the larvae can become infectious they must mature further, typically an additional four to five days. Consequently, under normal conditions, from the time the eggs are passed in the faeces to the time hookworm larvae are infective is about eight to nine days.

Once attached and fertilised inside the human gut, the female hookworm begins to lay eggs. Wherever its human host (victim) goes, it goes, and when the human defaecates, large numbers of eggs are deposited along with the faeces. Given the primitive state of sanitation in the rural American South, hookworm was common. Sanitary privies were rare in the rural South prior to the twentieth century. Toilet facilities in rural areas were frequently just secluded bushes.²⁹ Free-roaming coprophagous animals (pigs, chickens, ducks, dogs, and some wild life) could eat the faeces and pass viable hookworm eggs out in their own faeces.³⁰ Once established, hookworm became permanent in the rural South because of the climate favourable to hookworm that existed throughout

²⁴*Ancylostoma duodenale* can be transmitted by nursing mothers to their children. This route of infection is not available to *Necator americanus*. Autoreinfection of hookworm can occur under circumstances of poor hygiene. See Revista de la Soc. Arg. de Biología 1925, p. 1263.

²⁵Ettling 1993, p. 784.

²⁶Schad in Gilles and Ball (eds) 1991, p. 33.

²⁷Some hookworm larvae under laboratory conditions, with constant temperature and humidity, are remarkably long lived. Some larvae survived more than two months. See Smith in Schad and Warren (eds) 1990, pp. 97–9.

²⁸Discussion of the life-cycle of hookworm is contained in Chandler 1929, pp. 91–8.

²⁹For more discussion on sanitary conditions in the rural American South, see Dock and Bass 1910, pp. 86–93.

³⁰'[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.' See Dock and Bass 1910, pp. 86–7.

much of the year. This was also due to sandy soils throughout much of the agricultural South, lack of sanitary privies, and the non-use of shoes in much of the rural South in the warm-weather months. The African slave trade introduced hookworm into North America, and it spread wherever infected people went. Because of its life expectancy (one to five years when attached to the human intestine) and fecundity, the result was endemic hookworm throughout much of the rural South. Hookworm infection causes a number of medical problems. Depending upon the number of worms attached and sucking, the signs of infection that are commonly associated with hookworm are anorexia, abdominal pain, nausea, headache, rash, weakness, fever, vomiting, diarrhoea, dysentery and intestinal bleeding. The clinical impact of hookworm disease depends upon how many hookworms are attached, the nutritional and health status of its victims, the host's ability to fight off hookworm and the rate of infection. Living conditions, climate, cultural practices and other diseases also affect the rate of hookworm infection.

It is instructive to observe that hookworm disease can cause anorexia, diarrhoea and vomiting, which contribute further to the malnutrition of its victims. '[A]norexia caused by hookworm infections in children may be an important cause of loss of weight and failure to thrive. All of these early manifestations of hookworm infection can cause a great deal of morbidity but with no anemia.'³¹ Because of their smaller size, the same number of hookworm in children is more deleterious to their health than to adults. The hookworm symptoms can contribute significantly to anaemia and hypoalbuminemia (inadequate protein).³²

Part of the reduced energy and productivity of hookworm-infected humans may be attributed to the symptomatic anorexia, diarrhoea, loss of appetite and vomiting that are associated with hookworm and which reduce the net nutrition available to its host. Hookworm-infected humans typically have a smaller nutrient consumption because of the anorexia and loss of appetite. The food intake that the host does ingest has its nutritional value reduced by the diarrhoea and vomiting associated with hookworm infection. Finally, the blood-sucking hookworms reduce the net nutrition available to the host by the loss of red blood cells and other fluids that the hookworms claim.

The Hookworm Data

The hookworm data employed in this study are from archival records housed at the Rockefeller Archive Center, Sleepy Hollow, New York. In 1909, John D. Rockefeller funded and organised the Rockefeller Sanitary Commission for the Eradication of Hookworm Disease to determine hookworm prevalence in the American South. The Sanitary Commission, in conjunction with local health providers, canvassed homes in 11 southern states from 1911–15 to estimate hookworm prevalence and home exposure risk, to treat discovered cases, and to prevent (re)infection. In the early 1920s, the International Health Board, organised by the Rockefeller Foundation in 1913 as the successor organisation to the Sanitary Commission, conducted follow-up hookworm inspections in selected counties throughout the American South to determine the continuing presence

³¹Nelson 1990, p. 420.

³²Migasena and Gilles 1991, p. 186.

of hookworm. Marion County, South Carolina, was one county that had data suitable for modern statistical analysis.

Diagnosis for hookworm involved microscopic examination of faecal specimens. Prior to 1915, the initial hookworm inspections were completed for nearly 600 southern counties. For these inspections, the percentage of 6–18 year-old children, who were infected (positive tests), were recorded at the county level in Sanitary Commission annual reports.³³ During the years 1920–23, the International Health Board conducted follow-up hookworm inspections in several dozen southern counties that again screened and microscopically examined the stools of school-age children, 6–18 years-old, and of samples of individuals of all ages, who were identified as taken at random from the country. The data employed here come from this second round of hookworm inspections.³⁴

One of the primary objectives of the Rockefeller medical teams was to draw samples of residents from southern counties that would be representative of the hookworm prevalence rates among inhabitants in the sampled counties. The present study makes the explicit presumption that the Rockefeller hookworm data for Marion County are representative of the presence of human hookworm infection in Marion County in 1922. For each individual in the Marion County sample, the hookworm inspection recorded the name, county, community, address, age, gender, ethnicity (whether white or black), whether the individual tested positive or negative for hookworm, and whether the individual tested positive or negative for other intestinal parasites (*ascaris*, *trichuris* or *strongyloides*). For relevant individuals in the sample, the records contain marginal notes describing various conditions about the individuals' specimens (for example, 'dried', or 'poor' or 'no' specimen). Because seven of the 542 individuals are recorded with a question mark under tested positive or negative for hookworm and have a marginal note indicating 'no specimen', the number of useful cases was reduced to 535. (There are seven other individuals with a marginal note indicating 'no specimen' but each of these seven is recorded as either testing positive or negative for hookworm and thus is included in the primary sample analysed.) One individual included in the primary sample is recorded with a question mark under 'age' and was assigned the average age of the other 534 individuals.

Table 1 contains the descriptive statistics for the 535 individuals in the primary sample. There are 291 males and 244 females, 242 blacks and 293 whites, 81 adults and 454 children, and an average age of 14.6 years. Of the 535 individuals, 215 tested positive for hookworm infection, 67 tested positive for the presence of other parasites (*ascaris* only), and 61 have a marginal notation concerning the condition of their specimen.

Potential Confounding Factors

While the Marion County hookworm report includes data on the presence of hookworm infection and simultaneously reported data on each individual's age, ethnicity, gender and residence, the records do not include data on other potential confounding factors, such as an individual's income, wealth, living standards, or social status (for example,

³³Rockefeller Sanitary Commission for the Eradication of Hookworm Disease, *Annual Reports 1910–14*.

³⁴'Hookworm Disease Survey, Marion County, South Carolina' 1922.

Table 1. Descriptive statistics of hookworm survey for Marion County, South Carolina, 1922

Variable	Mean	Standard Deviation	Minimum	Maximum
Gender	0.544	0.499	0	1
Race	0.548	0.498	0	1
Age	14.566	11.453	1	72
Hookworm	0.402	0.491	0	1
Other parasites	0.125	0.331	0	1
Specimen	0.114	0.318	0	1

Note: Number of observations is 535. Gender, Race, Hookworm, Other parasites, and Specimen are dichotomous categorical variables that are coded as a 1 if an individual has the characteristic, 0 otherwise. The mean of a dichotomous variable thus indicates the proportion of individuals in the sample with the characteristic. The characteristics are male (Gender), white (Race), tested positive for hookworm infection (Hookworm), tested positive for other parasitic infection (Other parasites), and condition of the individual's specimen was noted (Specimen).

Source: 'Hookworm Disease Survey, Marion County, South Carolina' 1922, Box 79, Record Group 5, Series 3, International Health Board, Rockefeller Foundation Archives, Sleepy Hollow, NY: Rockefeller Archive Center.

education or literacy). However, we are confident that the potential impact of any omitted factors would have been to increase the probability that the black population of Marion County would be more prone to hookworm infection than the white population. The aetiology of hookworm requires an infected person to defaecate somewhere that would allow other persons to be infected by the larvae contained in the contaminated faeces. Hookworm, then, is a disease of poverty (going barefoot and lacking sanitary latrines), children and manual labourers who are continually exposed to the soil, and ignorance of the factors that cause hookworm infection. The socio-economic environmental differences between southern blacks and whites in the early 1920s—low incomes, poverty, poor diet and high illiteracy of blacks, and the systematic discrimination in the provision of public services against blacks—all acted to increase the black hookworm infection rate relative to whites.

Table 2 presents 1920 United States Census data for various demographic and socio-economic variables for Marion County, for South Carolina, and for selected southern states in the hookworm-endemic agricultural South. These data show that the black population of Marion County was systematically disadvantaged relative to the white population. Marion County was not an outlier in South Carolina. It was demographically and economically representative of similar counties in South Carolina, and South Carolina is demographically and economically representative of similar southern states in which hookworm was known to be endemic in 1920.

In 1920, Marion County was a rural, agricultural county with nearly 50 per cent of its land in farming. The county had slightly more blacks and was less literate. It also had somewhat lower population density and slightly more tenant farming than that of the state as a whole. (The state's density is increased by including the city of Charleston.) Marion County produced an agricultural output valued at \$8,525,757; the value of manufacturing products was \$3,152,000. Blacks in Marion County had nearly five times the rate of illiteracy (indicative of poverty and discrimination in the provision of

Table 2. Demographic and socio-economic characteristics for Marion County, South Carolina, and Selected Southern States, 1920

	Marion County	South Carolina	Alabama	Georgia	Mississippi	North Carolina
Total population	23,721	1,683,724	2,348,174	2,895,832	1,790,618	2,559,123
Male	11,840	838,293	1,173,105	1,444,823	897,124	1,279,062
Female	11,881	845,431	1,175,069	1,451,009	893,494	1,280,061
Total white	10,563	818,538	1,447,032	1,689,114	853,962	1,783,779
Male	5,310	415,823	733,039	854,109	433,396	899,031
Female	5,253	402,715	713,993	835,005	420,566	884,748
White population (% of total)	44.5	48.6	61.6	58.3	47.7	69.7
Total black	13,147	864,719	900,652	1,206,365	935,184	763,407
Male	6,522	422,185	439,779	590,443	462,829	373,965
Female	6,625	442,534	460,873	615,922	472,355	389,442
Black population (% of total)	55.4	51.4	38.4	41.7	52.2	29.8
Total illiterate (%) ^a	22.5	18.1	16.1	15.6	17.2	13.1
White illiterate (%) ^a	7.5	6.5	6.4	5.4	3.7	8.2
Black illiterate (%) ^a	35.4	29.3	31.3	29.1	29.3	24.5
Area in square miles	529	30,495	51,279	58,725	46,362	48,740
Population density ^b	44.8	55.2	45.8	49.3	38.6	52.5
Total number of farms	2,579	192,693	256,099	310,732	272,101	269,763
Farms operated by owners	820	67,724	107,089	102,123	91,310	151,376
White owned	580	44,859	89,887	86,081	68,131	129,099
Black owned	240	22,759	17,202	16,042	23,179	22,277
Farms operated by tenants	1,749	124,231	148,269	206,954	179,802	117,459
White tenants	620	38,132	70,395	93,016	41,954	63,542
Black tenants	1,129	86,068	77,874	113,938	137,848	53,917
Farms operated by managers	10	738	741	1,655	989	928
White managers	—	555	614	1,448	797	832
Black managers	—	183	127	207	192	96
White owner operated farms as % of all farms	22.5	23.30	35.10	27.70	25.00	47.90
Black owner operated farms as % of all farms	9.3	11.80	6.70	5.20	8.50	8.30
White tenant operated farms as % of all farms	24.0	19.80	27.50	29.90	15.40	23.60
Black tenant operated farms as % of all farms	43.80	44.70	30.40	36.70	50.70	20.00

Continued

Table 2. *Continued.*

	Marion County	South Carolina	Alabama	Georgia	Mississippi	North Carolina
White manager operated farms as % of all farms	—	0.29	0.24	0.47	0.29	0.31
Black manager operated farms as % of all farms	—	0.10	0.05	0.07	0.07	0.04

Note: Major areas of the four selected southern states (Alabama, Georgia, Mississippi and North Carolina) were part of the coastal, sandy-soil hookworm-infested agricultural South in 1920.

^a10 years of age and over.

^bpopulation per square mile.

Source: United States Bureau of the Census, Fourteenth Census of the United States taken in the Year 1920, vol. 3. 'Population 1920: Composition and Characteristics of the Population by States', Table 1, pp. 54, 202, 528, 730, 924; Table 4, pp. 56, 204, 530, 732, 926; vol. 6, 'Pt. 2. Agriculture: Reports for States with Statistics-the Southern States', County Table I, pp. 232, 276, 279, 304, 488, 522 (Washington, DC: Government Printing Office, 1921).

public schools) as whites. Although there are no direct data on black–white differences in income or wealth, the 1920 census data indicate that Marion County blacks were much less likely to be farm owners (240 'negro' versus 580 white owner-operated farms) and much more likely to be farm tenants (1,129 'negro' versus 620 white tenant-operated farms). While there is no black–white breakdown for farm managers at the county level, there is for the state of South Carolina, which had 183 'negro' versus 555 white manager-operated farms.

In Marion County, the average owner-operated farm was 105 acres with a value of farm land and buildings of \$5,938; the average tenant-operated farm was 44.4 acres with a value of farm land and buildings of \$3,228; the average manager-operated farm was 359 acres with a value of farm land and buildings of \$21,460. These data were not broken down by ethnicity, but tenant farmers were overwhelmingly black while owner farmers were overwhelmingly white in Marion County. Presumably, managers were overwhelmingly white as well. For South Carolina as a whole, the 1920 census has a breakdown by ethnicity for the average value of all farm property. White farmers had property worth \$7,305, while 'coloured' farmers had \$3,135 in property. The demographic and socio-economic characteristics of South Carolina in 1920 were in the range of those of the four nearby hookworm-infested southern states. Table 2 indicates that South Carolina was most similar to Mississippi, then Georgia and Alabama, and least similar to North Carolina.

The census data all confirm the general historical impression that blacks were an impoverished, socially disadvantaged population in the early twentieth-century American South, and that Marion County was not exceptional. Consequently, any omitted or unmeasured variables that may be confounding factors would indicate an increased rate of hookworm infection in the black population relative to the white population. As a result, if the observed rate of hookworm infection among blacks in Marion

County is lower than that of the white population, it was in spite of their lower (and deplorable) socio-economic living conditions.

Statistical Findings

A multivariate logistic regression of hookworm infection (the dependent variable), regressed on the demographic factors (the independent variables), was estimated. The estimated logistic regression equation includes the demographic data from the International Health Board hookworm inspections (race or ethnicity, gender and age) and an age squared variable to capture the non-linear effects of age.³⁵

Table 3 reports the logistic findings for the 535 individuals.³⁶ The estimated regression indicates that the predicted probability of hookworm infection for the 'average' individual in the sample, an individual with average values for all independent variables, is 0.381, very close to the actual hookworm prevalence rate of 40.2 per cent in the sample. More importantly, the findings indicate a highly statistically significant, and large, positive relationship between hookworm infection and being white.³⁷ In terms of the magnitude of the relationship, the findings indicate that an otherwise average white was nearly 2.8 times more likely to be infected with hookworm, *ceteris paribus*, than an otherwise average African-American. The predicted probability of testing positive for hookworm was 56.1 per cent for an otherwise average white and just 20.3 per cent for an otherwise average black; an incremental effect of race (ethnicity) of 35.8 percentage points.³⁸ The findings also indicate a statistically significant positive relationship between hookworm infection and being male. For an otherwise average individual, the predicted probability

³⁵Logistic regression techniques are designed to analyse data where the dependent variable is qualitative (or dichotomous) rather than continuous. The dependent variable here is a dichotomous variable, coded 1 or 0, measuring the presence or absence of hookworm infection, respectively. The estimated logistic regression equation, similar to ordinary least squares regression, is given by the form $Y = a + b_1X_1 + b_2X_2 + b_3X_3 + b_4X_4$, where Y is the dependent variable, X_1 through X_4 are the independent variables, a is the intercept, and b_1 through b_4 are the logistic regression coefficients. The regression equation shows how the dependent variable (hookworm infection) is statistically explained by the independent variables (race or ethnicity, gender, age and age squared) for all individuals in the estimated sample.

³⁶The descriptive statistics and logistic estimates employing the same data but with several modifications to the number of cases analysed are reported in an unpublished appendix. First, all 14 individuals with a marginal note indicating 'no specimen' are deleted from the sample. Second, all 68 individuals with any marginal note about their specimen are deleted. Third, all 67 individuals who tested positive for other intestinal parasites are deleted. Fourth, the 132 individuals with either a marginal note or who tested positive for other parasites are deleted (three individuals both had a marginal note and tested positive for other parasites). The appendix is available from the authors on request.

³⁷This result holds for all logistic regressions estimated, independent of the sample used. In fact, the sample used has no impact on the magnitude or precision (standard error) of the estimated coefficient of the race (ethnicity) variable. The sample size, though, does have some impact on the magnitude and precision of the coefficients of the other variables.

³⁸Moreover, recall the earlier discussion indicating that Marion County blacks in 1922 were an impoverished, socially disadvantaged population, especially relative to whites. This fact suggests that estimation procedures that included any omitted or unmeasured socio-economic variables in an examination of hookworm infection would *strengthen* the influence of race (ethnicity) on the probability of being hookworm infected relative to estimates where the socio-economic variables were omitted. This is because the influence of any omitted socio-economic variables would be captured by the included race variable with which they are strongly correlated.

Table 3. Logistic regression of hookworm infection and demographic variables

Variable	Coefficient estimate	Standard error	Incremental/ Marginal effect	t-statistic	p-value
Constant	-1.8768	0.3534	—	5.3107	<0.0001
Gender	0.3186	0.1933	0.0748	1.6482	0.0993
Race	1.6156	0.2000	0.3585	8.0780	<0.0001
Age	0.0472	0.0330	0.0111	1.4303	0.1521
Age ²	-0.00103	0.000614	-0.00024	1.6775	0.0926
Predicted probability of hookworm infection for 'average' individual = 0.381					
Likelihood Ratio = 82.919					
$R^2 = 0.144$					
Number of observations is 535					

Note: Dependent variable is Hookworm, which equals 1 if individual tested positive, 0 otherwise. For dichotomous categorical variables, the incremental effect is calculated as the difference in the predicted probability of an individual testing positive for hookworm when the variable has a value of 1 and the predicted probability when it has a value of 0, with all other independent variables at their mean values. The incremental effect measures the percentage point change in the predicted probability evaluated at the means of the variables. For continuous variables, the marginal effect measures the change in the predicted probability associated with a per-unit change in the variable, with all other independent variables at their mean values. The predicted probability for the 'average' individual is calculated employing the mean values of all independent variables. R^2 is a measure of the goodness of fit of the estimated model, which indicates the extent to which the variation in hookworm infections is statistically explained by the variation in the independent variables. The logistic regression was estimated with SAS (8.0).

Source: Logistic regression estimates of data contained in 'Hookworm Disease Survey, Marion County, South Carolina' 1922.

of testing positive for hookworm was 41.6 per cent for a male and 34.1 per cent for a female; an incremental effect of 7.5 percentage points. The findings also indicate a highly suggestive positive relationship between hookworm infection and age (p -value = .1521) and a statistically significant negative relationship with age squared.

Economic and social factors, such as absence from rural agriculture and attendance in school, may have played a protective role against hookworm infection. Given whites' lower participation rate in agriculture and greater educational attainment in the early twentieth-century American South, it may be surprising that the relationship between hookworm infection and white ethnicity is so strongly positive. Conversely, it may be surprising that the relationship with black ethnicity is so strongly negative given the pervasive discrimination against African-Americans in the provision of public services, and the greater participation rate of African-Americans in agriculture, their lower educational attainment, and presumptively lesser levels of income and purchasing power. Intuition aside, the findings reported in the present paper are consistent with other evidence showing greater rates of hookworm infection and intensity in whites than in blacks in the American South.

The earliest scientific evidence of hookworm infection in the American South dates from the early decades of the twentieth century. The evidence is for individuals aggregated by race and includes aggregate rates of hookworm infection and intensity by racial category. The evidence shows that the rates of infection and intensity among people of

predominately tropical West African ancestry ('negro', 'coloured' or black) were substantially lower than people whose ancestry was not African-American. The earliest evidence indicating ethnic differences in hookworm infection comes from the examination and treatment of infections among southern men enlisted in the United States Army. In one such examination, Knowlton, a medical officer in the US Army, reports that 'white' soldiers from the Carolinas and Florida had over four times the worm burden of 'coloured' soldiers from the same states. The examinations and treatments conducted at Camp Jackson, South Carolina, involved 69 'white' soldiers and 18 'coloured' soldiers with known hookworm infections, but who were selected 'without reference to the degree of infection'.³⁹ The 'white' soldiers had an average worm count of 155.3 worms while the worm count for the 'coloured' soldiers averaged 38.3.⁴⁰ Several other early twentieth-century hookworm examinations conducted by the Army report similar findings.⁴¹

To determine the continuing presence of hookworm in the American South during the early decades of the twentieth century, the International Health Board, as noted earlier, conducted a series of hookworm inspections (resurveys) during 1920–23. Unlike earlier hookworm inspections, the resurveys reported hookworm infection among individuals by race for selected southern counties. The data in the surviving records of the county resurveys that contain results for both black and white residents of a county exist for 60 counties in 11 southern states; the records indicate that the white infection rate was greater in 57 of the counties.⁴²

In a study of hookworm in Alabama in the 1920s, Smillie and Augustine present data which show that children classified as white had substantially more hookworm infection and intensity than children classified as black.⁴³ Table 4 presents the Smillie and Augustine data, which were collected for children 6–16 years-old in Alabama and in Covington County, Alabama. The data in row 1 indicate that white children in rural Alabama had over two and a half times the hookworm infection rate of black children (62.9 per cent versus 23.9 per cent).⁴⁴ In rural Covington County, infection rates for both races were substantially higher but still different; the county samples indicate that white children had a hookworm infection rate over one and a half times the rate for black children (96.4 per cent versus 61 per cent). The means of the worm counts in Table 4 are illustrated in Figure 1.⁴⁵ The means indicate that the intensity of infection in Covington County and in Alabama among white children (330 and 187 worms,

³⁹Knowlton 1919, p. 701.

⁴⁰Knowlton 1919, p. 703.

⁴¹Siler and Cole 1917; Frick 1919; Lucke 1919; Kofoid and Tucker 1921.

⁴²'Resurveys, Southern States', 1920–23.

⁴³Smillie and Augustine 1925, pp. 1958–63.

⁴⁴The hookworm infection rate in each case is calculated by subtracting the percentage of children with no infection from 100 per cent.

⁴⁵For each column in Table 4, the mean worm count is calculated by weighting the mean of the endpoints of each range of hookworm infection (no infection, very light, light, etc.) by the percentage infected (in decimal form) for each range and summing the results for the six ranges. This procedure has the effect of assuming that the children had an equal likelihood of infection for all worm counts within each infection range.

Table 4. Prevalence and intensity of hookworm infection and ethnicity for rural Alabama and rural Covington County, Alabama, early 1920s

Level of hookworm infection	Per cent of 'white' children infected		Per cent of 'coloured' children infected	
	Rural Alabama (n = 1,284)	Rural Covington County (n = 444)	Rural Alabama (n = 555)	Rural Covington County (n = 117)
No infection	37.1	3.6	76.1	39.0
Very light (1–25 worms)	12.6	15.3	13.8	30.9
Light (26–100 worms)	16.7	24.5	6.1	20.6
Moderate (101–500 worms)	26.0	42.5	3.0	8.0
Heavy (501–1000 worms)	4.2	7.8	0.3	0.9
Very heavy (1001–3000 worms)	3.3	6.3	0.3	0.0

Note: No exact date is given for the collection of these data. But it can be inferred that the collection took place in the early 1920s. The children examined were 6–16 years- old. Source: W. G. Smillie and D. L. Augustine 1925, 'Intensity of Hookworm Infection in Alabama: Its Relationship to Residence, Occupation, Age, Sex, and Race', *Journal of the American Medical Association*, 85, 1958–63. Copyright © (1925) American Medical Association. All rights reserved.

respectively) was seven to eight times greater than among black children (48 and 23 worms, respectively).

The study of rural Covington County, similar to that of Marion County, was for a 'warm-weather' county that was predominately agricultural. Writing in the

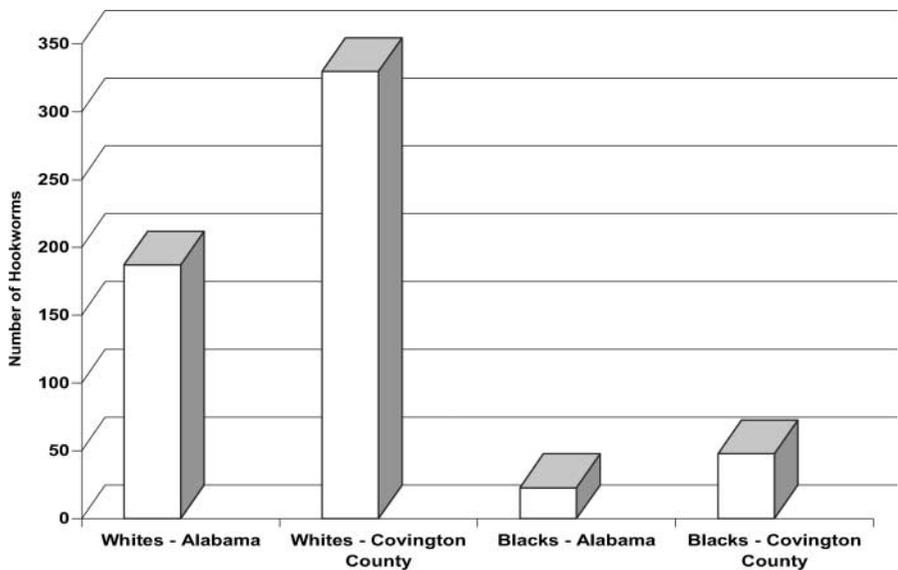


Fig. 1. Mean hookworm burden for blacks and whites in rural Alabama and rural Covington County, Alabama, early 1920s.

Source: Calculated from the data reported in Table 4.

Journal of the American Medical Association, Smillie and Augustine state that: 'Our results clearly show that when the two races [blacks and white] are living under almost identical conditions of sanitation, economic status, occupation, soil temperature, etc., the whites may have a heavy infestation [of hookworm] whereas the negroes have a very light infestation.'⁴⁶ Smillie and Augustine, however, do not provide a modern analysis of any of the potential confounding factors in hookworm infection.

Greater hookworm infection among whites than blacks in the American south has also been documented in a more recent sampling of southerners. Martin reports that in a survey of residents of rural south-eastern Georgia conducted in mid-1969, whites had an infection rate of 16 per cent while blacks had a hookworm infection rate of 8 per cent. Similar to Smillie and Augustine, Martin also does not provide any formal analysis of the potential confounding factors in hookworm infection.⁴⁷

In the non-medical literature, Brinkley found that in a sample of counties in the southern states suitable for high-intensity hookworm infections, the percentage of the population that was black had a significant positive effect on income in 1910. He attributed the differential in income to the greater susceptibility of whites to hookworm disease.⁴⁸ Brinkley's finding of an income differential in favour of blacks is an important piece of evidence because this was a time in the American South when blacks were systematically discriminated against. Likewise, Coelho and McGuire contend that in colonial America, African slaves had higher levels of productivity than European indentured servants in the American South because African slaves were more resistant to hookworm, malaria and other warm-weather diseases. Coelho and McGuire show that in the absence of differential levels of productivity in favour of African slaves, African slavery in the South would have been uneconomic because throughout most of the colonial era the annual expenses (including capital costs) of indentured servants were lower than those of slaves.⁴⁹

The ultimate explanation for these findings is one of evolutionary selection. Tropical West Africans were exposed to endemic hookworm infection for millennia; their innate resistance to hookworm was pro-adaptive. The American descendants of tropical West Africans inherited some of their ancestors' genetic defences against hookworm. The significant positive relationship between white ethnicity and presence of hookworm infection is explained by strong evolutionary selection pressures on the African ancestors of American blacks and its absence in American whites. This resulted in a significant reduction in the susceptibility of early twentieth-century American blacks to hookworm. In essence, this paper can be interpreted as a historical case study of evolutionary biology, supporting a hypothesis of innate differences in susceptibility to a particularly debilitating parasite, hookworm, across people of different racial or ethnic heritages.

⁴⁶Smillie and Augustine 1925, p. 1962.

⁴⁷Martin 1972b.

⁴⁸Brinkley 1994, pp. 113–14.

⁴⁹Coelho and McGuire 1997, pp. 102–9. See also pp. 98–101.

Conclusion and Historical Implications

Based on a sample of 542 residents of Marion County, South Carolina in 1922, which yielded 535 useful cases, the multivariate regression findings indicate there is a statistically significant and large difference between the probability of hookworm infection among African-Americans (blacks) and European-Americans (whites). Controlling for potentially confounding demographic factors, an otherwise average white was nearly 2.8 times more likely to have had hookworm infection compared to an otherwise average black living in Marion County. This finding supports a hypothesis of innate differences in susceptibility to hookworm infection, a debilitating parasitic disease, in peoples of differing racial or ethnic backgrounds, and suggests that ancestry, ethnicity or race (properly understood) can be useful predictors in understanding disease patterns among racial or ethnic groups under appropriate circumstances. In particular, they can be useful in the context in which the race or ethnic identification relates to people whose heritage is predominately from locations in which specific disease ecologies prevail. The strong statistical evidence derived from historical data should encourage other scholars to mine such data. Historical studies also have the merits of being uncontaminated by current hypotheses and are relatively inexpensive compared to clinical trials.

The finding that whites in the early twentieth-century rural South have a significantly greater probability of being infected with a parasite that reduces energy and lowers productive capabilities has significant implications for important historical issues. Two of the more important ones are the relative productivity of black and white agricultural labour in the early twentieth-century South, a time and place where productivity, and thus economic performance, was inherently linked to human physical labour, and an issue that has been debated since the nineteenth century: the economics of American 'negro' slavery.

With respect to the second issue, the currently accepted interpretation of plantation slavery is that large-scale enterprises operating with slave labour were more profitable than using free labour in smaller farms. There does not appear to be disagreement with this statement. The only issue appears to be the exact reason for the efficiency advantage of plantation slavery over free labour.⁵⁰ The available evidence suggests that hookworm and other parasites were endemic and widespread throughout much of the rural South in the nineteenth and earlier centuries. Since slave labour was black labour and free labour was white labour in the antebellum agricultural South, existing studies on the productivity of American slavery are confusing two issues: the productivity of slave labour in southern agriculture relative to free labour; and the difference in productivity between people moderately afflicted with hookworm relative to those who are more severely afflicted.⁵¹ Endemic hookworm in the antebellum South would reduce the productivity of white (all white labour was free) relative to slave labour (black). Consequently, the existing studies that estimate the productive efficiency of

⁵⁰Fogel and Engerman 1980, p. 672.

⁵¹For examples of the existing studies on slave productivity, see Fogel and Engerman 1971, 1974, 1977, 1980; David and Temin 1979; Schaefer and Schmitz 1979; Wright 1979; Field 1988; Fogel 1989; Grabowski and Pasurka 1989; Field-Hendrey 1995.

large-scale slave plantations over free farms are biased in overestimating the efficiency of slave agriculture; the question that remains is by how much it is overstated.

Acknowledgements

We wish to thank Gareth Morgan, MD, for his helpful comments and suggestions on this article. We also wish to thank the Rockefeller Archive Center, Sleepy Hollow, New York, for permission to access its holdings and use its material in this article. Coelho received support for this research from the National Science Foundation under Grant No. SES-0079179 and a summer research grant from the Miller College of Business, Ball State University, Muncie, IN. McGuire received support for this research from the National Science Foundation under Grant No. SES-0003342.

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