Side Effects of Immunities: the African Slave Trade*

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Working Paper

August 1, 2015

Abstract

The resistance of Sub-Saharan Africans to diseases that were plaguing the southern United States contributed to the establishment of African slavery in those regions. Specifically, Africans' resistance to malaria increased the profitability of employing African slave labor, especially that of slaves coming from the most malaria-ridden parts of Africa. In this paper, I first document that African slavery was largely concentrated in the malaria-infested areas of the United States. Moreover, I show that the introduction of a virulent strain of malaria into US colonies greatly increased the share of African slaves, but *only* in states where malaria could thrive. Finally, by looking at the historical prices of African slaves, I show that enslaved individuals born in the most malaria-ridden African regions commanded higher prices.

Keywords: Slavery, Malaria, African Slave Trade, Colonial Institutions.

JEL Classification: I12, N31, N37, N57, J15, J47.

*I am indebted to Matteo Cervellati for his inspiring supervision of my work. Moreover, I wish to thank Oded Galor for kindly hosting me at Brown University, and Juan Dolado and Andrea Ichino for their mentorship at the European University Institute. Finally, comments and suggestions from Scott Abramson, Fabrizio Bernardi, Mariastella Botticini, Graziella Bertocchi, Emilio Depetris Chauvin, Daniele Fabbri, James Fenske, Martin Fiszbein, Andrew Foster, Moshe Hazan, Eliana La Ferrara, Giorgio Gulino, John R. Logan, Ricardo Estrada Martinez, Stelios Michalopoulos, Omer Moav, Luca Pensieroso, Giovanni Prarolo, Louis Putterman, Dominic Rohner, Tim Squires, Paolo Vanin, Hans-Joachim Voth, David Weil and the other participants at the Unviersity of Bologna Wednesday seminar, Brown University Macro Lunch Seminar and S4 Graduate Fellow Lecture and Reception, Income Inequality Thematic Group were precious inputs for my work.

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1 Introduction

The practice of slavery compromised the long-term economic prosperity of both the enslavers and the enslaved. On the one hand, new empirical evidence documents that slavery represented a burden for societies historically relying on it as a source of labor, as it fostered long-term poverty, lower contemporary public good provision and higher inequality in these societies (Engerman and Sokoloff, 1997; Nunn, 2008; Dell, 2010).[1] On the other hand, slavery prejudiced the growth prospects of populations subject to enslavement - as in the case of Africans - by engendering social distrust and fostering ethnic stratification (Nunn and Wantchekon, 2011; Whatley and Gillezeau, 2011).[2] These findings have sparked significant interest in exploring what contributed to the historical geographical distribution of slavery, and especially in examining the factors that fostered and orientated the African Slave Trade.

This paper is the first to empirically document the role played by diseases, and notably malaria, in determining why slavery was practiced in certain American regions and not in others, and why Africans, and Africans from certain regions in particular, were transported and enslaved to the New World so numerously. I conjecture that the resistance of Sub-Saharan Africans to the disease environment prevalent in the US South made them especially attractive for employment in those regions. Specifically, I hypothesize that Africans' resistance to malaria increased the profitability of African slave labor, and primarily of African slaves from more malarial countries.

The hypothesis builds on two premises. First, malaria was absent from North America before European settlement.[3] Importantly, once Europeans settled, malaria (a disease requiring specific bio-climatic conditions for transmission) did not spread all over North America but became endemic only in regions warm and humid enough. The introduction of the disease radically modified the epidemiological environment of the southern United States, engendering high rates of mortality and morbidity among locals. The second premise is that, as a consequence of a greater historical exposure to the disease, certain African populations had developed a vast

[1] See also Acemoglu, García-Jimeno, and Robinson (2012), Bertocchi and Dimico (2014) and Bobonis and Morrow (2014).

[2] See also Nunn (2007) and Nunn and Puga (2012).

[3] The conventional hypothesis, supported by genetic and archeological evidence, is that malaria was introduced into the Americas by European immigrants or by an individual transported through the transatlantic slave trade. See Yalcindag (2012) for a recent influential contribution in favor of the conventional hypothesis.

set of resistances to malaria, granting some form of protection from the disease.

My hypothesis is that the redesigned epidemiological environment of the US South coupled with Africans' malaria resistance fundamentally affected the colonizers' labor choices. The first consequence is that in malaria-infested areas labor became scarcer and slave labor more extensively exploited. The second consequence is that, among the available laborers, more malaria-resistant workers started to be preferred. Africans had a higher stock of resistance to malaria than locals. Among them, the resistance to malaria of Africans from more malaria-ridden regions was even higher. Thus, the evolving geographical distribution of malaria explains key patterns of the African slave trade to the Americas: i) determining which American areas imported and exploited more slaves; and ii) why Africans, and those from certain African regions in particular, were more massively enslaved into those areas.

The first test of the hypothesis that I propose documents that African slavery was concentrated in malaria-infested areas of the United States. In order to exploit only the exogenous component of malaria exposure, following Kiszewski et al. (2004), throughout the analysis I primarily employ an index of malaria prevalence and stability of transmission predicted on the basis of bio-climatical characteristics.[4] The results show a strong positive cross-sectional correlation between malaria incidence and the share of *both* blacks and slaves across US counties in 1790. The relation remains statistically significant even when considering only within-state variation and when accounting for the suitability of the county's soil for sugar, cotton, rice, tea and tobacco cultivation.[5]

Despite a visually striking spatial correspondence between black/slave counties and malaria-ridden counties, in a cross-sectional framework I are unable to fully exclude that some county characteristics, such as the climate or the fertility of the soil, might be related to both African slavery and geographic suitability for malaria. Therefore, employing a panel of 12 United States colonies in the decades from 1640 to 1780, I explore the consequences of the introduction of

[4]Additionally, I exploit a recently-released index of malaria endemicity that measures malaria parasite prevalence at the beginning of the twentieth century (circa 1900), thus before the massive malaria eradication campaigns that took place after the second World War. Note that the National Malaria Eradication Program (NMEP) was launched in the US in 1947.

[5]Engerman and Sokoloff (1997, 2000) argue that the US South, unlike the North, had climates and soils suitable for crops with large economies of scale, cultivated in large plantations where slave labor was more profitable. Fogel and Engerman (1974) and Fogel (1994) also argue that the types of crop in the south, sugar in particular, favored slavery because these crops were fruitfully cultivated through gang labor, a particularly exhausting and unpleasant set of labor routines which free workers preferred to avoid.

falciparum malaria, the most deadly and virulent strain of malaria, into the colonies.

In fact, the available evidence indicates that *falciparum* malaria entered the northern American colonies in the 1680s, favored by weather anomalies that characterized the decade. As information on the timing of the actual introduction of *falciparum* malaria in each state is missing, I use the decade of the first *falciparum* epidemics as the threshold of malaria introduction for all states. Then, I further exploit across-state variation in malaria stability of transmission to proxy for the likelihood *falciparum* malaria had of striking and becoming endemic in each state.[6]

This difference-in-difference exercise allows me to compare the increase in the share of African slaves in the US states that were more suitable for malaria with states that were less suitable, following the introduction of *falciparum* malaria. My results show that the introduction of the most debilitating form of malaria sharply increased the share of Africans in regions with more favorable bio-climatic preconditions for malaria transmission.

The main threat to identification lies in the existence of other shocks occurring around the same decades as the introduction of *falciparum* malaria and correlated with malaria incidence. I address this concern by examining all the main alternative explanations for the switch to African slave labor. Most importantly, my results hold when accounting for a time-varying effect of soil suitability for rice,[7] and when accounting for variations in English wages.[8] Within the same empirical framework, I further show that the introduction of *falciparum* malaria predicts the approval of "slave codes", sets of laws placing harsh restrictions on the liberties of enslaved individuals.

The evidence presented so far is consistent with the explanation that a sudden deterioration in the health environment that followed the introduction of *falciparum* malaria into the US colonies reduced the availability of free laborers, increasing landowners' incentives to opt for slave labor. Then, given the proximity of the African coasts, Africans were possibly the closest

[6]The geographical variation in the index follows from the facts that some states had too low average temperatures and populations of mosquitoes with life spans too short for *falciparum* malaria to become a serious threat, some other states met conditions for transmission only occasionally, and the weather in some other states was warm and humid enough to fully sustain seasonal malaria transmission.

[7]Wood (1974) associates the switch from European servants to African slaves in South Carolina with the beginning of rice cultivation - rice of a particular variety that West Africans knew better how to grow.

[8]According to Galenson (1981) and Menard (2001), the switch followed a decrease in the supply of Europeans migrating as servants, which happened as a consequence of lower population growth and higher wages in England.

available population to enslave irrespective of their degree of resistance to malaria. Although, according to my hypothesis, these mechanisms were also at work, this study is interested in documenting the specific contribution that differential resistance to malaria made in tilting the labor demand of slave-owners toward African labor, and not only toward African labor in general but toward Africans from more malarial regions in particular.

In support of this hypothesis, I conjecture that if differential resistance to malaria caused a variation in productivity I would expect to observe higher prices paid for slaves more resistant to malaria. Given that resistance to malaria is higher in the regions historically most exposed to the disease, I expect to see higher prices paid for African slaves born in more malaria-ridden regions.

Using historical data from the "Louisiana, Slave Records, 1719-1820" database, I assemble a dataset of prices for over 3000 individuals born in 21 different African countries experiencing enslavement in the Louisiana plantations. I proxy resistance to malaria for each individual in the dataset with the level of malaria stability in his/her country of origin. The results show a positive and robust correlation between the selling price of the slave and his/her level of resistance to malaria. I further show that the results remain unaffected when including in the regressions a vast set of controls, including proxies for health conditions unrelated to malaria susceptibility, for production costs and for agricultural skills.

In support of the hypothesis that individuals from highly malarial countries were preferred because of their better health when facing malaria-related health distress, I look at mortality rates during the journey from Africa of individuals from various African countries. The results show that individuals from more malaria-infested countries tended to perish less.

With this paper, I aim to contribute to the vibrant empirical literature exploring the determinants of colonial institutions, and of slavery in particular. Among related works, Acemoglu, Johnson, and Robinson (2001) is a direct benchmark. By focusing on within-country - and within-US state - variation and by looking at the change in the health environment caused by the introduction of the virulent strain of malaria, I propose new strategies for identifying the effect of the epidemiological environment on settlers' exploitative behaviors. Moreover, my results suggest that Europeans colonizers established extractive institutions in malaria-infested African countries not only because of the difficulties in settlement caused by high mortality

rates, but also because of the search for malaria-resistant slaves. Within the same strand of literature, this work complements the studies of Nunn and Puga (2012) and Fenske and Kala (2014), who also explore the link between geography and the African Slave Trade. In line with their works, I find that geography contributed to determining which African regions were mostly raided for slaves.

Equally importantly, being the first quantitative analysis of the historical role of malaria in African slavery in the United States, this work contributes to the historical literature exploring the role of diseases in the peopling of the Americas. Indeed, for decades historians have debated and strongly disagreed over the role played by tropical diseases in the development of African slavery in the New World.[9] Through the development and formal testing of a systematic set of hypotheses, this work aims to integrate historical evidence and insights from the works of Curtin (1968), Coelho and McGuire (1997), Kiple and King (2003), McNeill (2010) and Mann (2011).

More broadly, this study complements the stream of economic literature exploring the relationship between health, infectious diseases and economic growth, both historically and today. See, among others, Gallup, Sachs, and Mellinger (1999), Acemoglu and Johnson (2006), Weil (2007), Bleakley (2007, 2010), Cervellati and Sunde (2011), Voigtländer and Voth (2012), Depetris-Chauvin and Weil (2013) and Alsan (2015).[10]

The paper is organized as follows. In the next section, I provide an epidemiological and a historical background. The cross-county analysis is introduced in Section 3.1. Section 3.2 presents the empirical framework exploring the effect of the introduction of *falciparum* malaria into the US colonies. I turn to slave prices in Section 3.3. The final section concludes.

[9]On the one hand, there is the position of the historian Philip Curtin (1968), who wrote "[o]n the American side of the ocean, planters soon found that both the local Indians and imported European workers tended to die out, while Africans apparently worked better and lived longer in the 'climate' of tropical America". On the other hand, scholars such as the celebrated historian of slavery Kenneth Stampp fiercely opposed the hypothesis, rejecting the idea that black people fared better than whites in the sickly lowlands of the US South as a myth (Stampp, 2011).

[10]Even more generally, by conjecturing an interplay between the geographic environment, climatic events and broadly-defined historical institutions, my work shares the same conceptual framework as Michalopoulos (2012), Alesina, Giuliano, and Nunn (2013) and Ashraf and Galor (2013), to name just a few.

2 Background

2.1 Malaria: the Great Debilitator

Malaria is a parasite transmitted to humans by mosquitoes. How threatening the disease is to humans depends on three key variables in the malaria transmission process: the parasite, the mosquitoes and the weather. The single-cell parasite, the *plasmodium*, exists in different strains and, among these strains, *vivax* malaria and *falciparum* malaria are the most widespread.[11] *Vivax* malaria is a milder form of the disease, rarely fatal, whereas *falciparum* malaria is the most virulent and lethal form. The mosquitoes that transmit malaria are the females of the *Anopheles* genus.[12] Certain *Anopheles* species, for instance the primary malaria vectors in Africa, prefer to feed on humans rather than on any other vertebrate, favoring the process of malaria transmission.[13] The weather is the third key variable for malaria transmission. On the one hand, higher temperatures reduce the duration of the development of the parasite within the mosquito, aiding malaria transmission. On the other hand, mosquitoes require enough water and hot enough temperatures to reproduce, develop and survive.[14] On top of this, the two major strains of malaria require different climatic conditions, with *falciparum* malaria needing higher temperatures than *vivax* malaria to become infectious.[15]

The classic clinical symptoms of malaria attacks are fever, chills, nausea and aches. Of all the existing strains, *falciparum* malaria is responsible for the most serious malaria symptoms, as it can lead to impaired consciousness, psychological disruption, coma and even death (cognitive malaria).[16] Even though after repeated infections malaria virulence and the mortality risk are

[11] Other strains are the Plasmodium malariae, Plasmodium ovale and Plasmodium knowlesi.

[12] More precisely, of the 430 Anopheles species that we know, only 30-40 transmit malaria.

[13] Another characteristic of mosquitoes that affects their ability to transmit the disease is their average life span, mosquitoes living longer have higher chances of transmitting the infection.

[14] More specifically, malaria transmission intensity is a complex function of temperature, as temperature affects several aspects of the transmission process. It affects the number of available mosquitoes per human, mosquito feeding rates, daily vector survival and time required for sporogony (the development of the parasites ingested by the mosquito). See Gething, Van Boeckel, Smith, Guerra, Patil, Snow, and Hay (2011) for an accurate modelling of the effect of temperature on the intensity of *vivax* malaria versus *falciparum* malaria transmission.

[15] *Vivax* malaria can continue development with temperatures as low as 9 degrees C; *falciparum* reproduction stops below 18 degrees C (Humphreys, 2001). For this reason, in the hot season *vivax* malaria used to reach even coastal northern European regions, such as Scotland and Finland.

[16] The untreated mortality rate of *falciparum* malaria can range between 20 and 40% in a susceptible population, whereas *vivax* does not kill more than 5% of infected individuals (Rutman and Rutman, 1976). For example, on the west coast of Africa in the early 1800s, mortality rates for Europeans often exceeded 50% per year (Curtin, 1989). After the introduction of quinine (late 1800s) the mortality rate fell to about 25%, indirect

reduced, the disease does not stop being a burden. In fact, continual infections deteriorate general health conditions, decreasing the ability to resist other diseases.[17] Precisely because it tends to weaken the immune system and drain energies, malaria has been named the 'the great debilitator' (Dobson, 1989).

The best proof of the health burden that malaria represented is written in the genetic code of a share of the world's population. In fact, over the last millennia a vast range of genetic adaptions have arisen to protect humans against the disease, to the point that malaria is considered the 'strongest known force for evolutionary selection in the recent history of the human genome' (Kwiatkowski, 2005). Blood cell abnormalities are the most well-known and studied genetic resistances to malaria.[18] However, current research has shed light only on the tip of the iceberg since a vast set of protective mechanisms remain unexplored and genetic factors seem to account for many more than the sole protective effects of blood cell disorders (MacKinnon et al., 2005).

Acquired immunities represent the second big category of protective resistance.[19] While resistance to the severe life-threatening consequences of infection is acquired relatively fast (Doolan et al., 2009), clinical immunity to milder symptoms is acquired slowly and requires repeated infections (Stevenson and Riley, 2004).[20] Importantly, a recent stream of research has pointed out that innate and acquired immunities are likely to interact, so that infections can trigger innate responses that might facilitate the acquisition of acquired immunities.[21] In other words, innate resistance to malaria can engender better adaptive responses once an individual faces an episode of infection.

evidence that the majority of deaths were caused by *falciparum* malaria (Hedrick, 2011).

[17]Rutman and Rutman (1976) report the result of a study documenting that for every ascertained malaria death, 5 additional deaths are caused by malaria indirectly, which acts by worsening the virulence of other diseases.

[18]Malaria is the evolutionary force behind genetic variation such as the Duffy blood group antigen, sickle cell disease, thalassemia, glucose-6-phosphatase deficiency, and many more. See Sirugo et al., 2006 and Carter and Mendis, 2002) for insightful reviews. In Table 27 of Appendix A, I summarize the main blood cell-abnormalities and the type of protection they grant. Importantly, different populations have independently developed specific evolutionary responses to malaria (Kwiatkowski, 2005).

[19]The key determinants of the acquired immune status of an individual are the number of malarial infections experienced and the intervals between infections.

[20]Based on available knowledge, innate and acquired resistance interact in complex ways, granting various levels of protection: i) by reducing the number of parasites, ii) once parasitized, by reducing the risk of becoming ill with fever and iii) once infected with malaria, by reducing the risk of developing severe malaria (Carter and Mendis, 2002; Kwiatkowski, 2005).

[21]See Mackinnon, Mwangi, Snow, Marsh, and Williams (2005) for the case of sickle cell trait.

Sub-Saharan Africa hosts the most debilitating strains of the disease and the species of mosquitoes most threatening to humans. Therefore, African populations have developed a particularly vast range of innate immunities to malaria. For instance, the sickle cell trait, a blood cell disorder that can reduce the likelihood of developing cerebral malaria after a *falciparum* infection by up to 90%, is widespread among several African populations. Even African populations that do not present a high frequency of the sickle cell trait, have independently developed a high frequency of other resistances, such as the HbC allele in Dogons in Mali or the high levels of antimalarial antibodies in Fulani in Burkina Faso.[22] Importantly, even across Sub-Saharan African populations I find substantial heterogeneity in the degree of resistance to malaria (Kwiatkowski, 2005).

2.2 Malaria Reaches the US Colonies

Before the European settlement, the geographical remoteness of the Americas had completely spared the continent from the major Old World diseases, which then started to be introduced into the continent.[23] On the one hand, diseases transmitted through direct human contact - i.e. through air or body fluids - immediately spread across all latitudes in the very early phase of settlement. On the other hand, the introduction of tropical diseases relying on vectors for transmission - such as malaria - took longer and, once introduced, remained largely confined to tropical and semi-tropical areas.

The delayed introduction of vector diseases, and notably malaria, is explained by the epidemiology of the disease. For the *plasmodium* of malaria to be introduced into the US Colonies, a set of conditions had to materialize together: i) an individual infected with malaria had to embark on a ship (and survive till destination);[24] ii) upon arrival in North America, the destina-

[22]On top of this, a great majority of Sub-Saharan Africans are completely immune to *vivax* malaria (thanks to the protection granted by the Duffy blood group antigen), whereas all other human populations are vulnerable to this species of malaria parasite.

[23]There are several explanations of the way diseases primarily traveled from the Old World to the New World. First, the New World had a relatively low number of animals available for domestication and thus less scope for the development of indigenous animal-born infections. Second, the relative scarcity of diseases was a direct consequence of the way the continent was populated during the migration of humans out of Africa: small bands of humans migrated to North America through the Bering Strait, so that no vector disease could complete the voyage in the cold weather of the Strait and very few human-contact diseases could sustain themselves in these small migrating bands (Diamond and Ford, 2000; Wolfe, Dunavan, and Diamond, 2007; McNeill, 2010).

[24]Given that somebody suffering from malaria paroxysms would had hardly been selected as a slave and would probably not have dared to face a long sea journey if a voluntary migrant, the individuals that carried

tion region had to host some variety of *Anopheles* mosquitoes that could transmit the infection to the local susceptible population; iii) the climate/season at destination had to be warm and humid enough for the *Anopheles* mosquitoes and the parasite to be active. Since *vivax* malaria, unlike *falciparum*, was widespread in many of the European countries where the first settlers were from, the likelihood of somebody infected with the disease embarking was higher than for *falciparum* malaria.[25] Moreover, at destination, the weather conditions compatible with *falciparum* transmission only existed during the warmest seasons and only in the warmer states, whereas we know *vivax* malaria was transmitted as far north as the state of New York.[26] For all these reasons, the conditions for the introduction of *vivax* malaria were met earlier in time than for *falciparum* (Mann, 2011).

In effect, historical evidence shows that already at the beginning of the 17th century US settlers suffered from relapsing fevers that characterize *vivax* malaria infections. On the contrary, *falciparum* malaria struck later. During the 1680s unusually virulent and deadly epidemics of *falciparum* malaria started to ravage the colonies (Wood, 1974; Childs, 1940; Rutman and Rutman, 1976), possibly as a consequence of weather anomalies associated with the El Nino events of 1681 and 1683-84. There is no way to know with certainty *who* carried *falciparum* malaria into the US colonies and *from where* he/she was traveling. At that time, *falciparum* had already been introduced in South America and in the Caribbean (Curtin 1993, Yalcindag 2012) from Africa, so that the human carrier of *falciparum* malaria into the colonies is likely to have been an African slave or a European mariner traveling from areas infested with the disease.[27]

What is certain is that in the US colonies where it took root and flourished malaria started to take a "dreadful toll" among settlers. Data for Christ Parish in South Carolina from the early eighteenth century show that 86% of the population used to die before reaching age 20,

malaria to the New World were possibly in the incubation stage or in a latent stage of the infection.

[25]It is also likely that the higher mortality rate of *falciparum* malaria versus *vivax* malaria reduced the probability of a human carrier of *falciparum* remaining alive throughout the voyage.

[26]On top of this, *vivax* malaria, unlike *falciparum*, has a long dormant phase and tend to relapse after the primary infection, so that human carriers can host the parasite a-symptomatically for several months.

[27]Interestingly, Packard (2007) points out that since "... human hosts who exhibit resistance to P. Falciparum are less efficient transmitters of the parasite to Anopheline mosquitoes than humans with no resistance, white settlers were probably more responsible for the subsequent transmission of [malaria] falciparum in South Carolina that were West Africans."

and 57% before reaching age 5.[28] Unsurprisingly, the great majority of deaths took place in the "ague and fever" months, between August and November (Packard, 2007).[29] A factor that increased the effective burden of malaria was its rural nature, which took the largest share of the malaria toll from farmers. Often hitting during harvest time, malaria caused serious losses in terms of worker time and efficiency.[30]

This paper emphasizes the health consequences of the introduction of *falciparum* malaria into the US colonies, despite the fact that other tropical diseases to which Africans also had previous comparatively higher exposure were introduced in the first decades of European settlement. The most devastating of these was yellow fever, which hit the US colonies repeatedly throughout the eighteenth century in waves of epidemics.[31] Although yellow fever possibly also played a role, there are certain characteristics of the disease that make it a less compelling explanation for African slavery. First, slavery was primarily a rural phenomenon,[32] and while malaria was to a large extent a rural disease, yellow fever mainly hit in big cities, sea-coast cities in particular. Moreover, while *falciparum* malaria was largely confined to the US South, yellow fever epidemics were frequent even as far north as New York and Philadelphia.[33]

2.3 Labor Preferences in Colonial America

For several decades, European workers had been the principal source of labor in the US colonies, where they were mainly employed as *indentured servants*. Under a contract of servitude called indenture, the emigrant agreed to work for a designated master for a fixed period of time in return for passage to a specified colony (Galenson, 1981).[34]

[28] From Packard (2007). Note that a high child mortality rate is typical of malaria-infested regions.

[29] In fact, malaria has a seasonal nature in North America, and it is expected to hit in the late summer and early autumn.

[30] Van Dine (1916) reports the results of an investigation by the Bureau of Entomology in Louisiana as late as 1913, at a time when the disease was largely under control. Still, malaria was responsible for about 15 lost days of work per adult per year, mainly concentrated in the most labor-intensive season.

[31] According to McNeill (2010) and Kiple and King (2003), yellow fever was the main determinant of patterns of African enslavement in tropical and semi-tropical America. Regarding other diseases, Coelho and McGuire (2006) present evidence in favor of descendants of Africans having a lower susceptibility to hookworm. Hookworms, however, do not cause morbidity and mortality comparable to yellow fever and malaria.

[32] Although not solely a rural phenomenon. See Goldin (1976) for a study on the presence of slaves in cities.

[33] The first ascertained yellow-fever epidemics hit Charleston and Philadelphia simultaneously, causing similar amounts of damage (Waring, 1975).

[34] It is estimated that between a half and two thirds of all white immigrants to the American colonies after the 1630s came under indenture, and that up to 75% of Virginian settlers in the seventeenth century were servants

For European servants, health at destination was one of the key variables to consider in deciding *whether* and *where* to migrate. Indirect evidence comes from the length of indentures: servants directed to less healthy locations had to serve for shorter periods (Galenson, 1981). Despite various attempts by the colonial governments to hide news of diseases from potential settlers, information on health conditions in the colonies frequently reached the home country (Wood, 1974).

Unsurprisingly, a contraction in the supply of European servants migrating to southern US colonies followed the introduction of *falciparum* malaria. According to Menard (2001), the deterioration in the health environment of certain states made these destinations unattractive. South Carolina, for instance, started to be considered "the great charnel house of the country" and had increasing difficulties in attracting new Europeans.[35]

Since the early days of settlement, colonizers had tried to satisfy their labor needs by enslaving Native American tribes. In several pre-colonial US states the phenomenon was anything but marginal. However, Native Americans were only considered partially suitable for employment in plantations. The high degree of morbidity and mortality they experienced is considered among the main explanations behind their perceived unfitness. First, the Native American population was fully susceptible to common European diseases such as measles and smallpox, which set them on a long-term trend of demographic decline. On top of this there was malaria, to which they also had no previous exposure and high susceptibility.[36]

European settlers seem to have rapidly reached the conviction that Africans were more resistant to malaria than Europeans and Native Americans. We frequently find statements such as this: "The old plantation was situated in rich lands, abounding in malaria, against which only the negro was proof."[37] Africans' lower susceptibility to malaria even attracted the inquiry of the scientific community. In the *American Journal of the Medical Sciences* in 1856, Dr. Alfred Tebault reported the results of his studies on the differential incidence of malaria between Africans and white Americans (Savitt, 2002). According to his findings, blacks suffered

(Galenson, 1981).

[35]Menard (2001) notes that after European servants started to avoid unhealthy southern destinations, they continued to flow to the newly established colony of Pennsylvania migrants.

[36]Humphreys (2001) reports the widespread conviction that Native Americans could not live in the same areas as the Africans, as they tended to die from fevers so rapidly. It is no surprise then that in the US colonies Native American slaves were sold for prices up to 50% lower than African slaves (Menard, 2001).

[37]Mallard (1892).

from about one third of the malaria attacks that struck white Americans. Importantly, slave owners' perceptions of this differential susceptibility to diseases went even further, to the point that planters' claimed to be able to discern different health susceptibilities even among Africans based on their place of origin.[38]

If the ethnic composition of the labor force is one side of the coin, coercion of workers and their legal liberties is the other. The first Africans brought to the US colonies were employed as indentured servants, just like Europeans. Unlike Europeans, their settlement was involuntary, but after a period of work they were not infrequently able to gain their freedom. In effect, in the first half of the seventeenth century African "slaves" were allowed to work independently, could buy and sell their produce, barter their free time for wages, and eventually buy their freedom.[39] For a long time the legal status of Africans brought to North America remained blurred, regulated more by customary practice than by actual laws. Moreover, it varied widely across states and over time (Wiecek, 1977). Starting in the second half of the seventeenth century, states started to approve legislation aiming at a reduction of the liberties of African workers, and at a stiffening in their status of slaves. This process culminated in the approval of "slave codes", which were a comprehensive set of laws that attempted to define slave status and sanction once and for all its elementary characteristics. Broadly speaking, all "slave codes" had in common three basic elements: slavery was as a life-long condition inherited through the mother, slave status had a racial basis and slaves were defined as property (Wiecek, 1977).

3 Empirical Analysis

3.1 Malaria and African Slavery across the United States Counties

3.1.1 Data

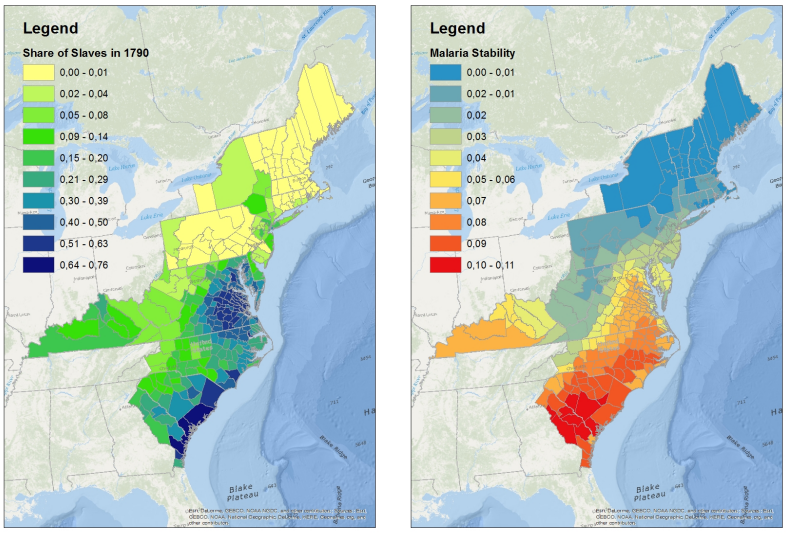
The US Census of 1790 provides county-level information on the slave status and ethnicity of the population for the 14 states in the Union.[40] As Figure 1 shows, the practice of slavery was

[38] For instance, laborers from the Congos were not appreciated because of their ill health in lowland plantations. Additional evidence is reported in Section 3.3.

[39] Indeed, Ira Berlin (2009) writes on the African slaves shipped to Jamestown in 1619 that they were "Set to work alongside a melange of English and Irish servants, little but skin color distinguished them...".

[40] The Northwest and Southwest Territories were not organized in counties and are excluded from the analysis. The final sample include Connecticut, Delaware, Georgia, Maryland, Massachusetts, New Hampshire, New

Figure 1: Malaria and Slavery across US Counties



(a) Share of Slaves (b) Malaria Stability

largely concentrated in the southern states. The northern states had a very low number or no slaves,[41] whereas in Virginia and Georgia about a third of the population were slaves. The picture was very heterogeneous even within the same state, to the point that in South Carolina there were counties with a share of slaves reaching 70%, and counties with less than 10% of slaves. The distribution of blacks followed a parallel pattern and across counties there was an almost one-to-one correlation between the share of blacks and the share of slaves.[42]

Turning to malaria distribution, ideally I would require a historical measure of the malaria incidence across the United States counties in 1790. Unfortunately, accurate measures of malaria morbidity and mortality for 1790 are unavailable and, most importantly, morbidity and mortality are themselves a consequence of living standards, agricultural productivity and other features that might be related to colonizers' labor choices. Since malaria transmission can take place only in specific climatic and biological environments, to proxy for effective historical malaria exposure I exploit an exogenous predicted measure of incidence devised by Kiszewski

Jersey, New York, North Carolina, Pennsylvania, Rhode Island, South Carolina, Vermont and Virginia.

[41] Two states - Vermont and Massachusetts - had already abolished slavery and three other states - New Hampshire, Connecticut and Rhode Island - were in the process of abolishing slavery.

[42] All Europeans and European descendants were classified as "Whites", while "non-Whites" were people of African ancestry, or mixed African ancestry. Note that until 1860 the census did not include non-taxed American Indians (i.e. living in tribal society), who composed the great majority of the Native American population.

et al. (2004): the Malaria Stability Index. This index predicts the risk of being infected with malaria is as a function of characteristics of the mosquito vector prevalent in the region - the proportion biting people and the daily survival rate - and climate - a combination of temperature and precipitation conditions.[43] Moreover, I also use a historical index of malaria endemicity measured at the beginning of the twentieth century, produced by Lysenko (1968) and digitalized by Hay S.I. (2004).[44] The index aims to measure the historical average parasitization rate at a geographically disaggregated level and offers the advantage of measuring actual malaria incidence at a time that predates large-scale public health interventions for malaria eradication.[45]

3.1.2 Estimation and Results

I begin by estimating an Ordinary Least Square (OLS) regression across US counties in 1790, where the outcomes of interest are the slave share and the black share, and malaria stability is the main explanatory variable. My preferred specifications include state fixed effects, which net out the average differences in the slave/black share across states. Looking at within-state variation is especially important because in 1790 several states were in the process of banning/had already banned slavery. Even if state legislation might itself be a response to labor market needs and - indirectly - to malaria exposure, I am interested in showing that even counties sharing the same legal and institutional features followed different labor patterns. The county-level controls include a full set of soil suitability indexes taken from FAO GAEZ, and

[43]Malaria risk is a non-linear function of both temperature and precipitation. Temperature has a hump-shaped effect on malaria risk and the risk is present only when the precipitation level in the previous month is higher than a threshold. The average risk is computed for each month of the year, and then averaged out into a cross-sectional variable. The final index has a spatial resolution of 0.5 x 0.5 degrees and ranges from 0 to 39. The climatic data employed are averages of monthly observations between 1901 and 1990. Ideally, for my application the index should rely on historical climatic data, which are, however, not available for US counties in 1790. As long as the outcome variables - share of slaves and share of blacks - did not have an influence on temperature and precipitation, the results are not driven by this aspect of the index.

[44]In a previous version of this paper, Esposito (2013), I exploited a predicted index of malaria risk devised by Hong (2007). The index by Hong (2007) is constructed on the basis of several climatic and geographic characteristics and of the share of land cleared for agriculture. Since the share of land cleared for agriculture might capture features of the county with a direct effect on the dependent variables, it is less suited for my specific application. Note, however, that the index would give virtually identical results.

[45]The measure goes from 0, no transmission, to 5 holoendemic (transmission occurs all year long). The intermediate steps are epidemic, hypoendemic (very intermittent transmission), hyperendemic (intense, but with periods of no transmission) and mesoendemic (regular seasonal transmission).

Table 1: Malaria, Slavery and Blacks across 1790 US Counties

	Share of Slaves			**Share of Blacks**		
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	5.672	4.997	4.193	5.941	5.241	4.293
Conley s.e. 100 km	(1.017)***	(0.959)***	(0.881)***	(1.062)***	(1.016)***	(0.929)***
Conley s.e. 500 km	(1.578)***	(1.289)***	(1.387)***	(1.611)***	(1.330)***	(1.409)***
Conley s.e. 1000 km	(1.265)***	(1.139)***	(1.334)***	(1.291)***	(1.166)***	(1.369)***
Standardized Coefficient	0.747	0.572	0.542	0.764	0.581	0.593
Crop Suitabilities	No	Yes	Yes	No	Yes	Yes
Distances	No	No	Yes	No	No	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
δ for $\beta = 0$		1.10	1.27		1.10	1.29
Observations	285	285	285	285	285	285
R-squared	0.601	0.716	0.793	0.593	0.712	0.800

Notes: The table reports Ordinary Least Square (OLS) estimates. The unit of observation is the US county in 1790. The dependent variable is the county share of slaves in columns 1 to 3, and the county share of blacks in columns 4 to 6. Malaria Stability is an index measuring the force and stability of malaria transmission. The "Crop suitabilities" controls include soil suitability for cotton, sugar, rice, tea and tobacco. The "Distances" controls include distance to the sea, to the closest river and to Charleston. Standardized coefficients of Malaria Stability are reported. δ for $\beta = 0$ measures the degree of selection of unobservables relative to observables which would reduce the Malaria Stability coefficient to 0, with an assumed R_{max} equal to 0.90 (Oster, 2013). Conley standard errors are reported in brackets, with cutoff thresholds for latitude and longitude at 100, 500 and 1000 km. ***, **, * indicate significance at 1, 5, and 10% levels respectively.

namely soil suitability for cotton, tea, tobacco, rice and sugar.[46] Moreover, I add three possibly relevant measures of distance: to the closest river, to the closest sea and to Charleston. Turning to statistical inference, to account for spatial correlation in the errors I compute Conley (1999) standard errors adjusted for two-dimensional spatial dependence.[47]

Table 1 reports the main results.[48] columns (1) and (4) include only state fixed effects as controls, in columns (2) and (5) I add soil suitabilities for crops, while columns (3) and (6) include both soil suitabilities for crops and distances. The results are consistent throughout the specifications and show that malaria exposure is positively and strongly correlated with the share of slaves in the county. Since exactly the same picture emerges when looking at the share of blacks, for brevity I will just comment on the former. The estimated coefficient of my favorite specification - Column 3 - implies that a one standard deviation increase in malaria

[46] I could include coffee as well. However, the available measures of coffee suitability do not present any variability across the sample of interest.

[47] I report estimates with cutoff values at 100, 500 and 1000 km.

[48] Tables 11 and 12 in Appendix A.1 provide additional specifications and robustness checks.

stability would predict an increase in the share of slaves of 0.13.[49]

Of all the other explanatory variables, the main other predictor of slave share is the distance of the county from the coast.[50] Regarding crop suitabilities, I find that a higher suitability for sugar and tea increases the share of slaves.[51] One concern that may arise is that suitabilities for crops are measured with noise and might not fully capture the heterogeneity in agricultural productivity across counties. More generally, if there is any omitted factor positively correlated with both the share of slaves and the malaria stability index, the estimated coefficient of malaria might be higher than the true one. To gain a more formal insight into the size of this bias, following Oster (2013) I compute how important the unobservable characteristics of the county should be relative to the observable ones for the estimated effect of malaria stability to fall down to 0. The results, reported in Table 1, show that for the true effect of malaria stability to be 0, there should be an effect of unobservables about 1.3 times as large as the effect of the observed set of controls (Column 3).[52]

Note that high malaria incidence counties also have a larger share of families owning slaves, and a larger black population measured in absolute terms (Appendix A.1 Table 10, Panel A). Moreover, the results are robust to the inclusion of climatic controls such as temperature and precipitation, as well as when holding constant the total county population (Appendix A.1 Table 10, Panel B). Finally, the estimated coefficients are remarkably similar to the baseline ones when using the alternative measure of historical malaria incidence and when looking only at "slave states" (Appendix A.1 Table 11, Panel C and Panel D).

[49]Interestingly, a regression including malaria stability alone would give an R-square as high as 0.366 (Appendix A.1 Table 11). Adding state fixed effects increases the R-squared to 0.60. To get a better sense of the magnitude of the coefficient, the size implies that going from a county where the index predicts virtually no malaria - like Washington in Massachusetts - to a county with the maximum malaria stability in the sample - like another Washington county, Georgia this time - leads to an increase in the ratio of slaves to the total population of about 44 percentage points, which is a substantial change given that the average share of slaves in the sample is 22%.

[50]A one standard deviation increase in this distance is associated with a 0.35 standard deviation decrease in the share of slaves.

[51]Interestingly, there is no significant and positive correlation between suitability for tobacco, rice and cotton with either the share of slaves or the share of blacks in 1790. This is not a surprise for cotton, since its cultivation in the southern United States boomed only around the 1820s. Moreover, tobacco was not traditionally associated with slavery, as it was very often cultivated in small farms by Europeans small land owners.

[52]The reported δ are computed assuming an R_{max} equal to 0.9. Note, however, that for any assumed value of R_{max}, δ is larger than 1 which Oster (2013) indicates as a reasonable heuristic value.

3.2 The Introduction of Falciparum Malaria into the Colonies

3.2.1 Empirical Strategy

The cross-sectional results may be fundamentally flawed if the areas where malaria occurred were different from other areas along dimensions that we do not observe, which could be the actual reason for greater exploitation of African slaves. To exclude this, I propose an identification strategy that exploits the timing of the introduction of the most virulent strain of malaria - *plasmodium falciparum* - into the colonies and the additional fact that, requiring specific climatic conditions to spread, *falciparum* malaria only became endemic in the states warm and humid enough to support transmission.

The first challenge of the exercise is to identify the exact timing of the introduction of *falciparum* malaria into the US colonies. Indeed, thanks to historical evidence, the introduction of the disease can be dated with a sufficient degree of accuracy. In fact, epidemiology would suggest that when a *falciparum* malaria infection hits a population never previously exposed to the parasite, violent epidemics must follow. Epidemics are expected to hit until a new equilibrium is reached, when *falciparum* malaria starts to be endemic to the region. In effect, a series of epidemics started to hit the most southern US colonies during the 1680s. The most well-known is the one that hit Charleston in 1684 (Waring, 1975). An increase in the virulence and mortality of *fevers and agues* was registered in various places, like South Carolina (Wood, 1974; Childs, 1940) and Virginia (Rutman and Rutman, 1976). The epidemic forms that the infection took at first and the sudden rise in the mortality rates that followed are consistent with the traits of *falciparum* malaria.

Exploring anecdotal evidence with these epidemiological considerations in mind leads Wood (1974) and Rutman and Rutman (1976) to date the introduction of *falciparum* malaria around the mid-1680s. I further conjecture that the weather anomalies that characterized the decade created weather conditions particularly suitable for the introduction of *falciparum* malaria. In fact, based on data that climate historians have pieced together, starting from the 1860s we observe an increase in extreme weather events.[53] Importantly, there is vast anecdotal evidence

[53]El Nino events were documented in 1681, 1683-1684 and 1687-88. For comparison, note that in the two previous decades we have evidence of only one El Nino event (1671) in the 1670s, and one event in the 1660s (1661). Extreme weather events can increase malaria risk in many ways, for instance by creating additional mosquito-breeding places. An exceptionally dry summer can increase the pools of stagnant water in a river,

Table 2: Health Changes in South Carolina

Year	Source	Opinion on the Health of the Colonies
1674	Joseph West, letter to Lord Ashley	*Our people (God be praised) doe continue very well in health and the country seemes to be very healthfull and delightsome.*
1681	Thomas Newe, letter to his father	*...most have a seasoning, but few dye of it.*
1684	Lord Cardross and William Dunlop, leaders of the Scottish contingent *Carolina Merchant*	*We found the place so extraordinarily sicklies that sickness quickley seased many of our number and took away great many...*
1737	Immigrant from Europe	*I herewith wish to have everybody warned that he should not hanker to come into this country, for diseases here have too much sway and people have died in masses.*

documenting a sudden deterioration in the health environment of the Southern colonies in the 1680s. Table 2 reports extracts for South Carolina before and after the *falciparum* epidemic that hit Charleston in 1684.

Ideally, the analysis would require information on the specific timing of the introduction of *falciparum* malaria into each US state. However, while historical analyses of the health environment of the major colonial states are vast and informative, smaller and more peripheral states have received less investigation. Moreover, the actual timing of the introduction of the disease into each state could itself be a consequence of endogenous factors, such as a larger prior importation of workers from tropical areas where the disease was already endemic.[54]

To overcome data limitations and the potential source of endogeneity that might drive the actual timing of the introduction, I use the same date of *falciparum* malaria arrival for all states. Based on the work of Wood (1974) and Rutman and Rutman (1976), I consider the decades up tp 1680 (included) as prior to introduction, and the subsequent decades as post-introduction. Moreover, I exploit the differential geographic suitability for malaria across states to predict where malaria was more likely to hit and then become endemic.

In a dif-in-dif exercise, I examine the effect of the falciparum shock on the change in the share of blacks before and after 1690, comparing the states where *falciparum* malaria could thrive

and unusually heavy rains and floods can do the same.

[54]In other words, we can conceptualize the likelihood of *falciparum* malaria arriving in the colonies as a function of two sets of factors: i) the exogenous likelihood related to the bio-climatic conditions of each US state, as the arrival of a *falciparum* malaria carrier in a cold state where conditions for transmission are not met or met only rarely was less likely to generate epidemics than the arrival of the carrier in a state where the bio-climatic conditions for transmission are frequently met; ii) the endogenous components affecting the probability of introduction, like for instance the size of the workforce from places where malaria was endemic.

with the states where it could not. To validate the correctness of the timing of introduction I follow two strategies. First, I estimate a fully flexible specification allowing for the effect of malaria stability to vary over time. As a second strategy, I show that weather anomalies in more malaria-suitable states predict the introduction of *falciparum* malaria in those states.

The main threat to this strategy is posed by shocks that differentially affected states more or less suitable for malaria and were contemporaneous to the introduction of *falciparum* malaria. Drawing on the most popular explanations provided by historians for the rapid switch towards African labor in high-malaria states, I show that the effect is not driven by the confounding effects of factors highlighted by the competing hypotheses.

According to several authors, the rapid increase in African labor in the colonies followed the introduction of a specific variety of rice, the cultivation methods of which were mastered by people from certain African regions.[55] To take into account the possible effect of a surge in rice cultivation on the share of African labor, I allow for a different effect of the state average suitability for rice before and after 1690, in an exercise which mirrors my main specification where malaria stability is the variable of interest.[56]

An alternative explanation behind the rise in African labor in the US colonies centers around the role played by English wages. While the prices of African slaves remained relatively stable in the second half of the seventeenth century, the price of servants increased notably due to a rise in wages registered in England, which pushed up the opportunity costs of Europeans willing to migrate to the colonies (Galenson, 1981). If the effect of a reduced supply of servants homogeneously affected all the states in my sample, accounting for aggregate shocks hitting all the colonies at once would eliminate this potential bias. Furthermore, to exclude the possibility that the lower availability of European servants affected certain states more than others, I allow the time series of farm wages in England to affect each state differently.

An additional concern may arise from the possibility that the *falciparum* malaria epidemics

[55] Around 1685 Captain John Thurber introduced a particular variety of rice in Charleston: 'Gold Seede' from Madagascar, which happened to prosper in the soils of South Carolina. According to other sources, bushels of rice were sent to Carolina earlier on. What we know for sure are the bushels of rice exported to England from the US colonies. The figures are available from 1698. The rice exported from the producing areas was still very little in 1698, with 10,407 pounds of rice exported. However, exports started increasing fast so that in 1700 the colonies exported 394,130 pounds of rice. In 1750, the amount of rice exported was over 27 million pounds. Source: Colonial and Prefederal Statistics, Chapter Z.

[56] As a robustness exercise, without imposing any structure, I control for a time-varying effect of the average suitability for rice of the state on the share of blacks.

observed were a consequence of the greater inflow of Africans in high malaria states, and not *viceversa*. Despite the fact that the inflow of workers from malaria-infested areas certainly increased the likelihood of epidemics, it is important to note that my exercise resembles a reduced form specification in spirit, where *only* variation in bio-climatic suitability to malaria is exploited instead of the actual measure of malaria incidence.[57] In any case, there is indeed scope for path-dependence in the mechanism which leads to the establishment of African slavery in the southern US. In fact, bringing Africans to states where malaria can take root could have increased the likelihood of acquiring malaria, which then might have further enhanced the need for African workers.

3.2.2 Data

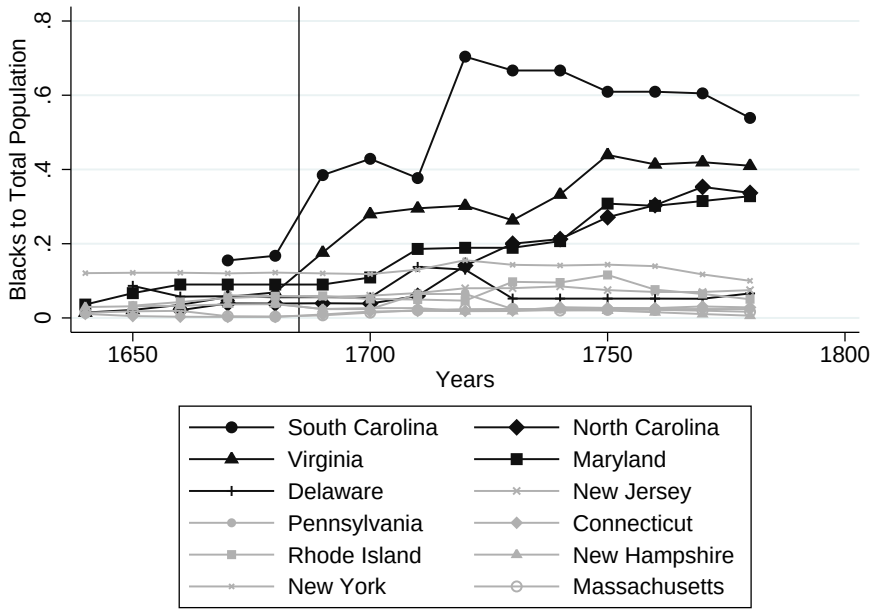
The Colonial and Pre-Federal Statistics of the US Census provide figures on the number of "Whites" and "Negroes" in each state over the decades from the early days of settlement. In addition to the ethnic composition of the state population, the analysis would require information on the coercive status of the labor force employed, which the Census does not, however, provide.[58] As a matter of fact, even the very concept of what being enslaved actually meant in the seventeenth century is rather difficult to define, as the coercive status of the African workers imported into the US colonies differed widely across states and over time. Indeed, it is extremely difficult to assemble a complete mapping of the colonial legislation ruling slave status over time, since the legal origin of slavery fed on a complex set of heterogeneous and sometimes contradictory provincial acts and customary practices (Wiecek, 1977). Overall, however, throughout the second half of the seventeenth century we observe a progressive stiffening of the legislation governing slave status, which culminated into the approval of comprehensive "slave codes" in some of the US states. Therefore, I employ the timing of the approval of a "slave code" as a proxy for the apex in the reduction of liberties experienced by Africans employed in each state.

In my final sample, where the unit of observation is the state at the beginning of each

[57] The fully-flexible estimate of the effect of malaria suitability should further reduce the scope for concerns.

[58] In other words, while it is possible to track the size of the African population entering the colonies across the seventeenth and eighteenth centuries, I cannot say whether they were enslaved or not.

Figure 2: Blacks over Total Population in US Colonies



†The graphs show the ratio of Africans to the total population in the US pre-federal states. The line is black for all states with a malaria stability index higher than the average and grey for all states with an index below the average.

decade, I observe 12 states from 1640 to 1780.[59] Figure 2 summarizes the evolution of the share of Africans over time across the 12 states in the sample.

I use the malaria stability index of Kiszewski et al. (2004) to proxy for the average geographical suitability for malaria in the state.[60] In Figure 2, states with higher than average malaria stability are reported in black and lower than average malaria-stability states are in light grey. Before 1690, the high-malaria states had average shares of blacks only slightly higher than the low-malaria states: respectively 6% and 4%. After 1690, the two groups of states took diverging paths, with the low-malaria states states maintaining the same low share of blacks (around 5%), while in the high-malaria states blacks reached on average 27% of the total popu-

[59]I exclude 1630, as information for only 4 states was available. However, the results would not change if I were to include 1630 in the sample. Moreover, as they would not contribute to the empirical analysis, I exclude states that are observed only after 1690. Moreover, to be able to compare data over time I consider Maine, Playmouth and Massachusetts as a single state.

[60]As a robustness check, I employ the historical index of malaria endemicity measured at the beginning of the twentieth century. The index contains relevant information as long as the malaria distribution at the beginning of the twentieth century is a function of bio-climatic conditions already present during the colonial times. However, if the distribution of African slaves affected the malaria endemicity rate at the beginning of the twentieth century, the results might be biased and need to be interpreted with caution.

lation. Furthermore, I find that until the 1680s no state had a slave code in force, while starting in the 1690s more than half of the states approved a slave code.

3.2.3 Estimation and Results

Figure 2 seems to suggest that after 1690 the share of blacks in the population jumped rapidly *only* in more malaria-suitable states. Turning to a more formal analysis, I propose a set of estimates based on the specification below:

$$\%Black_{s,t} = \alpha + \beta * MS_s * Post1690_t + \sum_{i=1}^{n} \gamma * \mathbf{I_{s,t}} + \mu_s + \mu_t + \epsilon_{s,t} \quad (1)$$

The main interest lies in β, the coefficient of the interaction term between $Post$-1690_t, an indicator taking value 1 for the decades following 1690 (with 1690 included), and the variable MS_s, which is a continuous index measuring malaria stability in the state. All the specifications include state fixed effects μ_s and decade fixed effects μ_t, with the aim to net out variation arising from time-invariant differences across states and shocks common to all states. The main outcome of interest is $\%Black_{s,t}$, the share of black population in the state at the beginning of the decade.[61] As an alternative outcome, I look at the likelihood of having a slave code sanctioning slavery, measured with an indicator variable taking value 1 in each state-decade during which a slave code is in force, and 0 otherwise.[62]

$\mathbf{I_{s,t}}$ is a vector of time-varying controls, of which the most important are: an interaction term between soil suitability for rice and the variable $Post$-1690_t, the lagged term of the share of blacks in the state and a state-specific effect of English farm wages.[63] Standard errors are

[61] Ideally, I would like to identify the same diverging paths in the time series of wages for free workers, which at the time were primarily European servants. In other words, I expect to observe a higher cost for free labor in states experiencing a deterioration in the health environment that followed the introduction of *falciparum* malaria. Moreover, I would expect an increase in the price of Africans in the same states. Unfortunately, there are no seventeenth century wage/price data for more than a few counties. It is nonetheless interesting to explore available evidence for Maryland, one of the areas experiencing a deterioration in the health environment caused by the introduction of *falciparum* malaria. See Appendix A.2.

[62] Since several states approved multiple versions of their codes, I also exploit a variable taking value 1 in each state-decade during which a new version of a slave code was approved.

[63] More formally, the state-specific effect of English farm wages is captured by controlling for a full set of interaction variables between the time series of wages and state fixed effects, $wage_t * \mu_c$. In a subset of specifications, I also control for a time-varying effect of average temperature in the state, captured by including a full set of interaction variables between the time-invariant average temperature in the state and decade fixed effects, $temp_s * \mu_t$.

Table 3: Malaria and the Share of Blacks: US States 1640-1780

	Share of Blacks						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability x Post-1690	4.222***	3.775***	4.140***	1.603**	3.602***	6.232***	3.091**
Cluster (State) s.e.	(0.825)	(0.971)	(0.778)	(0.651)	(0.939)	(1.625)	(1.020)
Bootstrap s.e. p-value	*0.000*	*0.042*	*0.000*	*0.004*	*0.002*	*0.024*	*0.082*
Rice Suitability x Post-1690		0.002					0.002
		(0.002)					(0.001)
Yellow Fever			0.018				-0.002
			(0.011)				(0.003)
Blacks to Total Population Lag				0.700***			0.607***
				(0.129)			(0.104)
England Farm Wage x State fixed effects					Yes		Yes
Temperature x Decade fixed effects						Yes	Yes
Decade fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	166	166	166	157	166	166	157
R-squared	0.586	0.594	0.594	0.828	0.734	0.712	0.891
Number of state	12	12	12	12	12	12	12

Notes: The table reports OLS estimates. The unit of observation is the US state in the decade. The panel includes all decades from 1640 to 1780. The dependent variable is the share of black people in the state. Malaria Stability is an index measuring the force and stability of malaria transmission. The variable Post-1690 is an indicator variable equaling 1 from 1690 onwards and 0 otherwise. All the regressions include decade fixed effects and state fixed effects. Standard errors clustered at the state level are reported in parenthesis. I report p-values for the null hypothesis (Malaria Stability x Post1690 = 0) computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

clustered at the state level and, given the small number of clusters in the sample, for each specification I report a test for the null hypothesis $\beta = 0$, computed with wild bootstrap standard errors.[64]

The main results are summarized in Table 3. The estimated coefficients consistently show that after 1690 more malaria-suitable states have a significantly larger share of blacks in the population. In terms of magnitude, according to the β coefficient estimated in the baseline specification of column (1), a standard deviation increase in malaria stability leads to a 0.11 increase in the share of blacks.[65] Accounting for post-1690 suitability for rice decreases β mildly - column (2) - just like accounting for the state-specific effects of English farm wages - column (5). In column (3) I account for the number of yellow fever epidemics in the state in the decade, which, however, leave the coefficient unaffected. On the contrary, as expected, including the lagged share of blacks in the population considerably reduces the size of β, more than halving it

[64]I employ Stata routines kindly made available by Bill Evans and Judson Caskey.

[65]Note that the estimated coefficient is very similar in size to the cross-sectional one obtained in the cross-county analysis.

- column (4).[66] Importantly, despite a reduction in magnitude, β is still positive and precisely estimated.[67] Finally, it is reassuring that the inclusion of temperature interacted with year fixed effects does not reduce, but actually increases, the size of β, indicating that the the estimated effect is not driven by pre/post 1690 differences acting along a climatic gradient.

Additional specifications, reported in Tables 16 and 17, show that the results are robust to the inclusion of further controls and are not driven by any state-specific behavior. In columns (5) to (8) of Table 19, I allow for the effect of soil suitability for rice, tobacco and tea to vary over time, interacting soil suitabilities with a full set of decade fixed effects. In column (9), I add state-specific time trends. In columns (4) to (8) of Table 17, I exclude each of the four southernmost states, while column (11) reports the coefficients for the historical malaria endemicity index.

Table 4 reports Linear Probability Model (LPM) estimations exploring the distribution of "slave codes". In columns (1)-(5) the dependent variable, Slave Code in Force, indicates whether a slave code is in force in the state-decade, whereas in columns (6)-(8) the variable, Slave Code Approval indicates whether the state approved a slave code in the decade. In all the regressions, the Malaria Stability index is normalized so as to range from 0 to 1. The results show that the *falciparum* malaria shock increases the likelihood of having a slave code in force, and of approving one, for highly malaria-suitable states when compared to low malaria-suitable states.[68]

To verify that the timing of introduction is correctly specified, I adopt two strategies. I first estimate a fully flexible time-varying effect of malaria stability on the share of blacks, showing that indeed the effect of malaria stability starts to increase in the 1690s (see Figure 3). As a second strategy, I construct a variable ($Falciparum\ Malaria$) indicating for each state whether there is historical evidence of *falciparum* malaria's appearance. Since the variable $Falciparum\ Malaria$ is measured with error, I instrument it with a variable aiming at capturing the effect of weather anomalies in more malaria-suitable states. My proxy measure

[66]Note that this effect is in part mechanical, since in a dynamic panel model the introduction of a lagged dependent variable leads to a downward bias in the estimation of other covariates.

[67]The lagged black share might be capturing path dependence in the establishment of African slavery as the main source of labor. That is, individuals from Africa might had carried strains of malaria that deteriorated the health environment, in turn leading to new demands for African workers.

[68]A standard deviation increase in malaria stability increases by 0.12 the probability of having a slave code in force.

Table 4: Malaria and Slave Codes: US States 1640-1780

	Slave Code In Force					Slave Code Approval	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability Nrm. x Post-1690	0.979***	1.301***	1.003***	0.898***	1.012***	0.265***	0.293*
Cluster (State) s.e.	(0.247)	(0.178)	(0.296)	(0.198)	(0.248)	(0.073)	(0.134)
Bootstrap s.e. p-value	*0.018*	*0.004*	*0.026*	*0.008*	*0.010*	*0.008*	*0.110*
Rice Suitability x Post-1690		-0.021***			-0.014**		-0.003
		(0.005)			(0.006)		(0.002)
Blacks to Total Pop. Lag			0.133		0.445		0.466
			(0.875)		(1.001)		(0.699)
England Farm Wage x State fe				Yes	Yes		Yes
Decade fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	166	166	157	166	157	166	157
R-squared	0.634	0.657	0.613	0.763	0.755	0.185	0.214
Number of state	12	12	12	12	12	12	12

Notes: The table reports Linear Probability Model (LPM) estimates. The unit of observation is the US state in the decade. The panel includes all decades from 1640 to 1780. The dependent variable in columns (1)-(5), Slave Code in Force, indicates whether a slave code is in force in the state-decade. In columns (6)-(8), the dependent variable, Slave Code Approval, takes value 1 if the state approved a slave code in the decade and 0 otherwise. Malaria Stability Nrm., is an index measuring the force and stability of malaria transmission normalized to range between 0 and 1. The variable Malaria Post-1690 is an indicator variable equaling 1 from 1690 onwards and 0 otherwise. All the regressions include decade fixed effects and state fixed effects. Standard errors clustered at the state level are reported in parenthesis. I report p-values for the null hypothesis (Malaria Stability x Post1690 = 0) computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

for weather anomalies (common to all states) is the number of El Nino events registered in the decade.[69] The results, summarized in Table 18, show that: i) weather anomalies in the highly malaria-suitable states significantly predict the variable *Falciparum Malaria* - First Stage, Table 18; ii) weather anomalies in the more malaria-suitable states are also associated with a sizable increases in the share of blacks - Reduced Form, Table 18; iii) instrumenting the variable *Falciparum Malaria* with weather anomalies in the more malaria-suitable states, I find instrumental variable estimates in line with the OLS ones - OLS and IV, Table 18.

3.3 Malaria Resistance and Slave Prices

Only to the unpracticed eye could all Africans look alike.

– Ullrich Bonnel Philips, *American Negro Slavery*

Evidence presented so far documents that the most malaria-infested regions resorted to

[69]More precisely, the instrument is the interaction term between El Nino events $\#El\ Nino_t$ and the malaria stability in the state MS_c.

African slave labor extensively more than other US regions. This section aims to show that these patterns did not only follow from labor scarcity in the unhealthy areas, but that the slave-owners' labor choices moved from a conscious intention to meet their labor needs with the comparatively most productive pool of laborers.

Thus, in this last section I provide evidence of slave-owners' preferences for slaves with higher resistance and immunities to malaria. In order to do so, I conjecture that among the African workers shipped to North America, individuals born in African regions with more malaria had a higher stock of acquired and innate immunities to the disease. On the premise that malaria resistance was associated with better health and higher productivity, I search for a *malaria premium* in the slave transactions occurring in the Louisiana slave market.

The possibility of detecting a *malaria premium* in prices depends on the existence of a competitive market for slaves. Fogel and Engerman (1974) claim that slave-owners were rational profit maximizers who paid a price for their slaves equal to their marginal productivity, properly discounted. Indeed, Louisiana was a very large slave market with thousands of yearly transactions. Moreover, Kotlikoff (1979) shows that selling prices reflected productivity differentials resulting from characteristics such as age, sex and skills.[70]

Crucially, a large body of anecdotal evidence documents the fact that colonizers preferred certain African groups over others, and that health played an important role in the shaping of these preferences.[71] Perceptions that individuals born in different African regions fared differently in the low land plantations of North America are present in planters' own accounts. Ibos from the Niger Delta, for instance, were considered sickly, whereas Gold Coast slaves were seen as hardy, robust and subject to little mortality (Littlefield, 1981).

Planters had limited understanding of the determinants behind these health differentials. Certain diseases had clearly identifiable symptoms, such as smallpox, while the symptoms of malaria were more difficult to identify. Notwithstanding the obvious complications in diagnosis experienced at the time, interesting insights emerge from the details of these perceived differen-

[70]Kotlikoff (1979) estimates a male premium from 23.6% to 48.8%. The polynomial he estimates for age peaks at age 22. The presence of warranties for a large share of slave transactions is another indicator of the rationality and scrutiny that characterized the trade (Kotlikoff, 1979).

[71]Peter Wood (1974), among many others, asserts that "white colonists would have marveled at the ignorance of their descendants, who asserted blindly that all Africans looked the same". Along similar lines, Wax (1973) claims that "slave preferences were apparent in all of the colonies and helped to shape the dimensions and composition of the slave trade to the mainland".

tial health susceptibilities. In the case of Congos, for instance, their higher degree of mortality was experienced in lowland plantations (Geggus, 2001), and we know that malaria was in fact the major disease of the lowlands. Furthermore, regarding the peculiar fitness of Gold Coast slaves, it is mentioned that they "were fit to work immediately". In other words, they needed to undergo a less debilitating form of 'seasoning', the dangerous process of adjustment to the local set of pathogens that each newcomer had to go through, among which malaria fevers represented the major component. Importantly, as malaria tends to weaken the immune system, individuals contracting the infection are more vulnerable to several other diseases.

For the present analysis it is important to acknowledge that the health of individuals enslaved from Africa was also influenced by the hardship of the long voyage from their African country of origin, both the inland voyage to the African embarkation port and the sea journey to the New World (Littlefield, 1981).[72]

Moreover, slave origin was considered to matter for a vast set of physical and cultural characteristics beyond health, such as body size and agricultural skills. For instance, body size was considered a direct consequence of the food availability and the vegetation in the regions of birth.[73] Moreover, Mauro (1964) points out that slaves "did not arrive naked, but brought with them a sense of sedentary life and of agriculture".[74]

3.3.1 Data

To test my hypothesis, I employ a database collecting records for a large number of individuals who came to Louisiana as slaves between 1719 and 1820: The "Louisiana, Slave Records" database, conceived and designed by Gwendolyn Midlo Hall (Hall, 2005). The database contains a rich set of biographical and genealogical details for thousands of individuals who experienced slavery in the Louisiana plantations. For the purpose of my analysis, the "Louisiana, Slave Records" database has the unique feature of documenting for individual slaves born in Africa

[72]The direction of the effect of the hardships faced during the long voyage from Africa on individual health is ambiguous. On the one hand, the hardship of the voyage might have long-lasting detrimental consequences on health; on the other hand we might expect a selection bias effect with the consequence of only the more healthy people being able to survive to the destination.

[73]Slaves from the Gold Coast, for instance, had access to a vast set of nutritious foods, whereas people living on the coast had diets relying mainly on fish, tubers and vegetables (Littlefield, 1981).

[74]Wood (1974) points to the central role of skills in the cultivation of rice. According to him, slaves from African "rice" regions were in high demand in places specializing in extensive rice cultivation, like South Carolina.

Table 5: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)					
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Ecology	0.019***	0.017***	0.034***	0.033***	0.035***	0.069**
Cluster s.e. (country)	(0.005)	(0.004)	(0.009)	(0.009)	(0.011)	(0.024)
Bootstrap s.e. p-value	*0.010*	*0.024*	*0.004*	*0.008*	*0.008*	*0.018*
Male Slave	0.194***	0.194***	0.194***	0.193***	0.193***	0.193***
	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)
Slave Age	0.048***	0.048***	0.048***	0.048***	0.048***	0.048***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Slave Age Squared	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Voyage Length		0.002**	0.002***	0.002**	0.002**	0.006*
		(0.001)	(0.001)	(0.001)	(0.001)	(0.003)
Distance Coast			0.209**	0.186*	0.200*	0.385**
			(0.081)	(0.091)	(0.102)	(0.183)
Ruggedness				-0.130	-0.167	-0.091
				(0.253)	(0.262)	(0.549)
Average Temperature				-0.013	-0.021	-0.136
				(0.046)	(0.049)	(0.084)
Distance Atlantic Markets					0.044	-0.056
					(0.077)	(0.100)
Average Rice Suitability						-0.000
						(0.000)
Land Suitability						-0.533
						(0.846)
Region fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Observations	3186	3186	3186	3186	3186	3186
R-Squared	0.446	0.447	0.448	0.448	0.448	0.449

Notes: The table reports OLS estimates. The unit of observation is the individual slave transaction. The dependent variable is the natural logarithm of the selling price of the slave. Malaria Stability is an index measuring the force and stability of malaria transmission in the country of birth of the individual enslaved. All the regressions also control for the age of the slave, the square of the age and whether the slave is a male. "Region fixed effects" are indicator variables that define African macro-regions (Upper Guinea, Bight of Benin, Western Africa and Southeastern Africa) for the country of birth of the slave. "Year fixed effects" are indicator variables for the year (1741-1820) of the document (from which the information was retrieved). "Document Language fixed effects" are indicator variables for the language of the original document (English, French or Spanish). "Document Type fixed effects" are indicator variables for the type of documents from which the information was retrieved (estate sale, mortgage, marriage contract...). Standard errors are clustered at the country level (21 clusters). Given the small number of clusters, I report p-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

both their selling price and their place of birth.[75]

Reconnecting information on the place of birth as recorded in the database to a geographical unit is a key part of my analysis. For over half of the individuals in the sample, the place of birth is defined in terms of "modern countries" or political entities largely overlapping with modern country borders.[76] For about one third of the records, the ethnicity of the slave is provided, whereas cities or geographical locations are the place of birth indicated for about 10% of individuals.[77] I aggregate all the available information at the African country level, since anecdotal evidence suggests that planters did not have detailed knowledge of the various African ethnicities, but tended to refer to broader families or larger geographical/cultural units.[78] Narratives in Wax (1973), who studies US slave-owners' preferences by place of origin, show that slave-owners referred to the origins of their slaves with terms such as as: Calabari, Gold Coast, Wydah, Gambia, Angola and Congo slaves.[79]

The link to modern countries for the majority of the reported places of origin in the sample is straightforward (for instance Gold Coast, Gabon, Coast of Senegal). I exclude individuals in the sample with an ethnicity not clearly traceable to a single modern country.[80] Moreover, I perform a vast set of robustness checks, excluding individuals whose place of origin can only imperfectly be matched to a modern country.

Among the subset of slaves born in Africa, I exclude records for individuals that contain no price information or that are sold in groups.[81] I further exclude individuals with no age

[75]Individuals born in Africa represent more than half of the individuals for whom I have details on the place of birth. Following the instructions of the author, Hall (2005), I exclude records retrieved from the Atlantic Slave Trade database, which mixes the place of birth and the place of embarkation of the slave. The choice to exclude slaves for whom I only know the port of embarkation and not the exact place of birth follows from my aim to proxy the endowment of acquired and innate immunities to malaria with the epidemiological environment of the place of birth. Note, however, that the results would not change if I included records from the Atlantic Slave Trade database.

[76]This is no surprise if we look at two historical maps drawn by Europeans in 1808 and 1829, which I provide in the Appendix. In fact, geographical areas are identified with labels that are easily linkable to modern countries and that to a large extent overlap with modern country borders.

[77]For the remaining records, I could not track the recorded place of birth to any geographical location.

[78]For instance, in the two advertisements reported in the Appendix, the slave traders refer to slaves from Sierra Leon, Windward Coast and Rice Coast.

[79]Hall (2005) claims that - wherever the ethnic origin of the slave was specified - it was an information provided by the slave him/herself.

[80]In particular, I exclude Manding and Fulani. Note, however, that including them by tracing them to the country which currently hosts the largest population would leave the results unaffected.

[81]Two types of prices are provided: sale and inventory prices. For my baseline results I use selling prices. If I included inventory prices, the results would not change. A robustness estimation including all the prices is available in the Appendix.

or sex information. For my baseline specifications, I assemble a final sample composed of 3186 individuals sold in the Louisiana Slave Market between 1741 and 1820, with places of birth spanning 21 African countries.[82] Since different slave transactions took place in different currencies, I exploit the price conversion variable constructed by Robert A. Rosenberg, which converts all prices into dollar values. The average selling price in the sample is equal to 570 US dollars. The majority of the transactions involved male individuals, and the average age was 29.

3.3.2 Estimation and Results

Turning to my empirical exercise, I propose the following baseline specification:

$$ln(price)_{i,c,r,t} = \beta_0 + \beta_1 MS_{c,r} + \beta_2 \mathbf{X_{i,c,r,t}} + \beta_3 \mathbf{Z_{c,r}} + \mu_r + \mu_t + \epsilon_{i,c,r,t}$$

where the dependent variable is the natural logarithm of the price for the individual i sold in year t, born in African country c, located in region r. The main variable of interest is the level of malaria stability in the country of origin of the enslaved individual $MS_{c,r}$. Individual controls $\mathbf{X_{i,c,r,t}}$ include age, age squared, a dummy variable taking value one if the slave is a male, type of sale transaction and the language of the document that registered the transaction. I allow for a non-linear effect of age on prices by including an age squared term. Note that document type refers to the kind of transaction - i.e. sale, estate sale, seizure for debt - and allows me to exclude any patterns in slave pricing related to the type of transaction confounding my results. Equally importantly, since French, Spanish and English slave owners are involved in the transactions, I include fixed effects for the languages of the document registering the transaction. On top of this, all the specifications include four African regions fixed effects, to avoid comparing slaves born in overly distant locations.

Finally, I add an extensive set of country-level controls $\mathbf{Z_{c,r}}$. The first set of controls aims to proxy for the hardship of the journey from the African country of origin. The second set of country controls include measures capturing differences in the production costs of slaves across

[82]The countries of origin for the individuals in my sample are Angola, Benin, Burkina Faso, Cameroon, Central African Republic, Congo, Congo Democratic Republic, Cote d'Ivoire, Gabon, Ghana, Guinea, Guinea Bissau, Liberia, Mali, Mozambique, Nigeria, Senegal, Sierra Leone, Tanzania, Togo and Zimbabwe.

countries: following Nunn and Puga (2012) and Fenske and Kala (2014), I control for ruggedness and average temperature. The third set of controls includes several measures of suitability for agriculture, and in particular for rice cultivation. Additional controls attempt to exclude malaria stability by capturing some particular suitability of the soil, climatic characteristics or vegetation. Finally, the timing of the transition to agriculture and the historical population can proxy for cultural attitudes towards sedentarianism and complex social organizations.

Table 5 reports the main results; additional specifications are available in the Appendix (Table 23 and 24). The estimated effect of malaria stability in the country of birth on the price of the individual is large in size and precisely estimated: a one standard deviation increase in the malaria stability index raises the price paid for the individual slave by about 7% (Column 1).[83] Interestingly, the only other controls that persistently show a well-estimated correlation with the selling price are the length of the sea journey from the individual country of birth and the country distance to the African coast.[84] Both controls have a positive effect on the slave price, possibly reflecting a selection effect in the subset of slaves that survived as far as the Louisiana's plantations. The results are robust to the inclusion of controls, which in fact tend to increase the size of the estimated coefficient.

I perform several robustness checks. Tables 23 and 24 report specifications including a vast list of controls. In the estimates in columns 1-2 of Table 25, I use an alternative measure of malaria exposure in the country of origin: historical malaria endemicity. In columns 3 to 8, I show that the results do not depend on slave transactions including slaves whose origin is only imperfectly attributable to a modern country. In order to do so, I exclude all slaves from ethnic groups whose geographical distribution crosses a border, even if only marginally (see Table 25, No border Groups).[85] Moreover, I show that the results do not depend on the inclusion of slaves whose place of birth is Congo, and neither do they on slaves whose place of birth is Guinea (see Table 25).

[83]To get a better sense of the magnitude, this also implies that going from the 25th to the 75th percentile of the malaria stability in the country of origin leads to a predicted price that is 10% higher, which is broadly about half of the difference between the price for a male and a female.

[84]Note that for countries that do not have access to the sea, I imputed the length of the sea journey for the closest country.

[85]Note that I have already excluded from the sample individuals from groups whose territory is almost evenly split across two (or more) locations. All the ethnicities left in the baseline specification have a predominantly larger amount of their land in one specific modern country.

Table 6: Malaria and Slave Mortality

	Share of Deaths during Middle Passage				
	(1)	(2)	(3)	(4)	(5)
Malaria Stability	-0.004***	-0.009***	-0.010***	-0.009***	-0.006***
	(0.001)	(0.002)	(0.003)	(0.003)	(0.002)
Bootstrap s.e. p-value	*0.040*	*0.004*	*0.002*	*0.018*	*0.066*
Voyage Lenght	Yes	Yes	Yes	Yes	Yes
Ruggedness	No	Yes	Yes	Yes	Yes
Temperature	No	No	Yes	Yes	Yes
Soil	No	No	No	Yes	Yes
Latitude	No	No	No	No	Yes
Region fixed effects	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes
Observations	3,951	3,951	3,951	3,951	3,951
R-squared	0.267	0.271	0.271	0.271	0.275

Notes: The table reports OLS estimates. The unit of observation is the single African Trade ship voyage. The dependent variable is the share of enslaved individuals dying during the voyage. Malaria Stability is an index measuring the force and stability of malaria transmission in the African country of embarkation."Region fixed effects" are indicator variables that define African macro-regions (Upper Guinea, Bight of Benin, Western Africa and Southeastern Africa) for the country of birth of the slave. "Year fixed effects" are indicator variables for the year of the sea voyage. Standard errors are clustered at the country level. Given the small number of clusters (17), I report p-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors. ***, **, * indicate significance at 1, 5, and 10% levels, respectively.

3.3.3 Additional Results: Slave Mortality and Quantity of Enslaved Individuals

The previous results document a positive correlation between prices of individuals enslaved in Louisiana and the malaria incidence in their countries of origin. To prove that behind this correlation lies a health differential, I would ideally need data on the morbidity and mortality that slaves from different African countries experienced in the Louisiana plantations. Since these data are not available, I exploit data on the mortality of individuals during their journey to the Americas. This approach is insightful since the transatlantic journey was undoubtedly a situation of unparalleled health distress, and malaria fevers represented one of the major source of health hazards.[86] The results, reported in Table 6, shows that ships departing from more malaria-infested countries had a lower average rate of enslaved individuals perishing during the voyage.

An additional implication of the main hypothesis is that malaria incidence in the country of

[86]Sheridan (1981) documents that fevers and dysentery were the main causes of morbidity and mortality experienced on the ships. Among the fevers, malaria fevers (intermittent and recurrent) played a large role.

origin should have exactly the same positive effect found for prices on the quantities of slaves imported. I exploit the timing of the introduction of *falciparum* malaria and look at the number of voyages registered in the Transatlantic Slave Trade database from Africa to US colonies before and after 1690. However, the scarcity of direct trips to Africa before the introduction of *falciparum* malaria reduces the power of the test. Given this major caveat, the results are in line with my expectations. Figure 4 reports a time-varying coefficient of malaria stability on the number of voyages from African countries.[87] Despite not being precisely estimated, the results show an increase in the size of the coefficient for Malaria Stability taking place in the decades following 1690.[88]

4 Conclusion

This paper has provided empirical evidence in favor of an old and debated explanation for the establishment of African slavery in the southern part of the United States. I have argued that Africans were especially attractive for employment in tropical and semi-tropical areas because they had higher resistance to many of the diseases that were ravaging those regions. In particular, Africans' resistance to malaria increased the profitability of African slave labor, especially of slaves coming from the most malarious parts of Africa. To verify the hypothesis, I have exploited the time variation arising from the introduction of *falciparum* malaria into the US colonies together with state variation in geographic suitability for malaria. By doing so, I have compared the percentage of slaves in the US colonial states that were more suitable for malaria with that of states that were less suitable before and after the introduction of *falciparum* malaria. Moreover, using the historical prices of African slaves sold in the Louisiana slave market, I have documented the existence of a malaria premium. That is, I have shown that, among slaves transported from Africa, slaves born in African regions with more malaria commanded higher prices.

[87] More precisely, the dependent variable is the number of voyages from African country c over a 5-years interval. The specification includes African country fixed effects and 5-year intervals fixed effects.

[88] As an an alternative, I look at the total number of individuals enslaved to the New World by African country, and show that countries with higher malaria stability were more massively exposed to the trade (Table 26).

References

Acemoglu, D., C. García-Jimeno, and J. Robinson (2012): "Finding Eldorado: Slavery and Long-run Development in Colombia," *Journal of Comparative Economics*.

Acemoglu, D., and S. Johnson (2006): "Disease and development: the effect of life expectancy on economic growth," .

Acemoglu, D., S. Johnson, and J. A. Robinson (2001): "The Colonial Origins of Comparative Development: An Empirical Investigation," *American Economic Review*, 91(5), 1369–1401.

Alesina, A., P. Giuliano, and N. Nunn (2013): "On the origins of gender roles: Women and the plough," *The Quarterly Journal of Economics*, 128(2), 469–530.

Allison, A. (2006): "Genetic factors in resistance to malaria," *Annals of the New York Academy of Sciences*, 91(3), 710–729.

Alsan, M. (2015): "The Effect of the TseTse Fly on African Development," *American Economic Review*, 105(1), 382–410.

Ashraf, Q., and O. Galor (2013): "The out of Africa hypothesis, human genetic diversity, and comparative economic development," *The American Economic Review*, 103(1), 1–46.

Berlin, I. (2009): *Many thousands gone: The first two centuries of slavery in North America*. Harvard University Press.

Bertocchi, G., and A. Dimico (2014): "Slavery, education, and inequality," *European Economic Review*, 70, 197–209.

Bleakley, H. (2007): "Disease and development: evidence from hookworm eradication in the American South," *The Quarterly Journal of Economics*, 122(1), 73.

——— (2010): "Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure," *American Economic Journal: Applied Economics*, 2(2), 1–45.

Bobonis, G. J., and P. M. Morrow (2014): "Labor coercion and the accumulation of human capital," *Journal of Development Economics*, 108, 32–53.

Carter, R., and K. N. Mendis (2002): "Evolutionary and historical aspects of the burden of malaria," *Clinical microbiology reviews*, 15(4), 564–594.

Cervellati, M., and U. Sunde (2011): "Disease and development: The role of life expectancy reconsidered," *Economics Letters*, 113(3), 269–272.

Childs, S. J. R. (1940): "Malaria and colonization in the Carolina low country, 1526-1696," Ph.D. thesis, Johns Hopkins Press.

Coelho, P., and R. McGuire (1997): "African and European Bound Labor in the British New World: The Biological Consequences of Economic Choices," *The Journal of economic history*, 57(01), 83–115.

Coelho, P. R., and R. A. McGuire (2006): "Racial differences in disease susceptibilities: Intestinal worm infections in the early twentieth-century American South," *Social history of medicine*, 19(3), 461–482.

Conley, T. G. (1999): "GMM estimation with cross sectional dependence," *Journal of econometrics*, 92(1), 1–45.

Curtin, P. (1968): "Epidemiology and the slave trade," *Political Science Quarterly*, pp. 190–216.

Curtin, P. D. (1993): "Disease exchange across the tropical Atlantic," *History and philosophy of the life sciences*, pp. 329–356.

Dell, M. (2010): "The persistent effects of Peru's mining Mita," *Econometrica*, 78(6), 1863–1903.

Depetris-Chauvin, E., and D. N. Weil (2013): "Malaria and Early African Development: Evidence from the Sickle Cell Trait," Discussion paper, National Bureau of Economic Research.

Diamond, J., and L. Ford (2000): "Guns, germs, and steel: the fates of human societies," *Perspectives in Biology and Medicine*, 43(4), 609.

Dobson, M. (1989): "History of malaria in England.," *Journal of the Royal Society of Medicine*, 82(Suppl 17), 3.

Doolan, D. L., C. Dobaño, and J. K. Baird (2009): "Acquired immunity to malaria," *Clinical microbiology reviews*, 22(1), 13–36.

Engerman, S., and K. Sokoloff (1997): *Factor Endowments, Institutions, and Differential Growth Paths among New World Economies*, vol. 89. Stanford University Press.

Fenske, J., and N. Kala (2014): "Climate and the slave trade," *Journal of Development Economics*.

Fogel, R. W. (1994): *Without consent or contract: the rise and fall of American slavery*. WW Norton & Company.

Fogel, R. W., and L. Engerman, S (1974): "Time on the Cross," *The Economics of American Negro Slavery*, 2, 242–243.

Galenson, D. W. (1981): *White servitude in colonial America: An economic analysis*. Cambridge University Press Cambridge.

Gallup, J. L., J. D. Sachs, and A. D. Mellinger (1999): "Geography and economic development," *International regional science review*, 22(2), 179–232.

Geggus, D. (2001): "The French slave trade: an overview," *The William and Mary Quarterly*, pp. 119–138.

Gething, P. W., T. P. Van Boeckel, D. L. Smith, C. A. Guerra, A. P. Patil, R. W. Snow, and S. I. Hay (2011): "Modelling the global constraints of temperature on transmission of Plasmodium falciparum and P. vivax," *Parasit Vectors*, 4(92), 4.

Goldin, C. D. (1976): *Urban slavery in the American South, 1820-1860: a quantitative history*. University of Chicago Press.

Hall, G. M. (2005): *Slavery and African ethnicities in the Americas: restoring the links*. University of North Carolina Press.

Hay S.I., Guerra C.A., T. A. N. A. . S. R. (2004): "The global distribution and population at risk of malaria: past, present and future," *Lancet Infectious Diseases*, 4(6), 327–336.

Hedrick, P. (2011): "Population genetics of malaria resistance in humans," *Heredity*, 107(4), 283–304.

Hong, S. C. (2007): "The burden of early exposure to malaria in the united states, 1850–1860: Malnutrition and immune disorders," *The journal of economic history*, 67(04), 1001–1035.

Humphreys, M. (2001): *Malaria: poverty, race, and public health in the United States*. JHU Press.

Kiple, K., and V. King (2003): *Another Dimension to the Black Diaspora: diet, disease and racism*. Cambridge University Press.

Kiszewski, A., A. Mellinger, A. Spielman, P. Malaney, S. E. Sachs, and J. Sachs (2004): "A global index representing the stability of malaria transmission," *The American journal of tropical medicine and hygiene*, 70(5), 486–498.

Kotlikoff, L. J. (1979): "The structure of slave prices in New Orleans, 1804 to 1862," *Economic Inquiry*, 17(4), 496–518.

Kwiatkowski, D. P. (2005): "How malaria has affected the human genome and what human genetics can teach us about malaria," *The American Journal of Human Genetics*, 77(2), 171–192.

Littlefield, D. C. (1981): "Rice and slaves," *Ethnicity and the Slave Trade in Colonial South Carolina*.

Lysenko, A.J., S. I. (1968): "Geography of malaria. A medico-geographic profile of an ancient disease," *Medicinskaja Geografija*, p. 25146.

Mackinnon, M. J., T. W. Mwangi, R. W. Snow, K. Marsh, and T. N. Williams (2005): "Heritability of malaria in Africa," *PLoS medicine*, 2(12), e340.

Mallard, R. Q. (1892): *Plantation life before emancipation*. Negro History Press.

Mann, C. C. (2011): *1493: Uncovering the New World Columbus Created*. Alfred a Knopf Incorporated.

Mauro, F. (1964): *L'expansion européenne:(1600-1870)*, no. 27. Presses universitaires de France.

McNeill, J. (2010): "Mosquito Empires: Ecology And War In The Greater Caribbean, 1620-1914," .

Menard, R. R. (2001): *Migrants, Servants and Slaves: Unfree Labor in Colonial British America*, vol. 699. Variorum.

Michalopoulos, S. (2012): "The origins of ethnolinguistic diversity," *The American economic review*, 102(4), 1508.

Nunn, N. (2007): "The long-term effects of Africa's slave trades," .

Nunn, N. (2008): "The Long-Term Effects of Africa's Slave Trades," *The Quarterly Journal of Economics*, 123(1), 139–176.

Nunn, N., and D. Puga (2012): "Ruggedness: The blessing of bad geography in Africa," *Review of Economics and Statistics*, 94(1), 20–36.

Nunn, N., and L. Wantchekon (2011): "The Slave Trade and the Origins of Mistrust in Africa," *The American Economic Review*, 101(7), 3221–3252.

Oster, E. (2013): "Unobservable selection and coefficient stability: Theory and validation," Discussion paper, National Bureau of Economic Research.

Packard, R. M. (2007): *The making of a tropical disease: a short history of malaria*. Johns Hopkins University Press, Baltimore.

Rutman, D. B., and A. H. Rutman (1976): "Of agues and fevers: malaria in the early Chesapeake.," *The William and Mary Quarterly*, 33, 31.

Savitt, T. L. (2002): *Medicine and slavery: The diseases and health care of blacks in antebellum Virginia*. University of Illinois Press.

Sheridan, R. B. (1981): "The Guinea Surgeons on the Middle Passage: The Provision of Medical Services in the British Slave Trade," *International Journal of African Historical Studies*, pp. 601–625.

Sokoloff, K., and S. Engerman (2000): "History lessons: Institutions, factors endowments, and paths of development in the new world," *The Journal of Economic Perspectives*, 14(3), 217–232.

Stampp, K. M. (2011): *The peculiar institution*. Alfred A. Knopf.

Stevenson, M. M., and E. M. Riley (2004): "Innate immunity to malaria," *Nature Reviews Immunology*, 4(3), 169–180.

Van Dine, D. (1916): "The relation of malaria to crop production," *Scientific Monthly November*, pp. 431–439.

Voigtländer, N., and H.-J. Voth (2012): "The Three Horsemen of Riches: Plague, War, and Urbanization in Early Modern Europe," *The Review of Economic Studies*, p. rds034.

Waring, J. I. (1975): "Colonial medicine in Georgia and South Carolina.," *The Georgia historical quarterly*, 59, 141.

Wax, D. D. (1973): "Preferences for slaves in colonial America," *Journal of Negro History*, pp. 371–401.

Weil, D. N. (2007): "Accounting for The Effect of Health on Economic Growth," *The Quarterly Journal of Economics*, 122(3), 1265–1306.

Whatley, W., and R. Gillezeau (2011): "The impact of the transatlantic slave trade on ethnic stratification in Africa," *The American Economic Review*, pp. 571–576.

Wiecek, W. M. (1977): "The statutory law of slavery and race in the thirteen mainland colonies of British America," *The William and Mary Quarterly: A Magazine of Early American History*, pp. 258–280.

Wolfe, N. D., C. P. Dunavan, and J. Diamond (2007): "Origins of major human infectious diseases," *Nature*, 447(7142), 279–283.

Wood, P. (1974): "Black Majority: Negroes in Colonial South Carolina from 1670 through the Stono Rebellion," *Norton Library, New York*.

Yalcindag, E., E. Elguero, C. Arnathau, P. Durand, J. Akiana, T. J. Anderson, A. Aubouy, F. Balloux, P. Besnard, H. Bogreau, et al. (2012): "Multiple independent introductions of Plasmodium falciparum in South America," *Proceedings of the National Academy of Sciences*, 109(2), 511–516.

SIDE EFFECTS OF IMMUNITIES: THE AFRICAN SLAVE TRADE

ONLINE APPENDIX

APPENDIX A: DATA SOURCES AND ADDITIONAL TABLES

Appendix A.1: Malaria and African Slavery - 1790 United States Counties

Data Sources: 1790 United States County
Share of Slaves
Ratio of slaves to total population in the county in 1790. Source: Historical U.S. census. *www.nhgis.org.*
Share of Blacks
Ratio of blacks to total population in the county in 1790. Source: Historical U.S. census. *www.nhgis.org.*
Malaria Stability
Average Malaria Stability Index in the state. Source: average Malaria Stability is constructed as the state average of the Malaria Stability index from Kiszewski *et al.* (2004).
Malaria Endemicity
Average Historical Malaria Endemicity in the state. Source: average Historical Malaria Endemicity is constructed as the state average of the Malaria Endemicity level, devised by Lysenko (1968) and digitalized by Hay (2004).
Crop Suitability Indexes
Estimated suitability index (value) for cultivating cotton, coffee, rice, sugar, tea and tobacco with Low input in a rainfed agriculture. Source: FAO/IIASA, 2011. Global Agro-ecological Zones (GAEZv3.0). FAO Rome, Italy and IIASA, Laxenburg, Austria. *http://gaez.fao.org/Main.html.*
Distance to Sea
Average county distance to seas and oceans (1000 km). Source: GSHHG - A Global Self-consistent, Hierarchical, High-resolution Geography Database, computed using ArcGIS with data in North America Equidistant Conic projection.
Distance to Rivers
Average county distance to inland water bodies, rivers and lakes (1000 km). Source: computed using ArcGIS with data in North America Equidistant Conic projection.
Distance to Charleston
Average county distance to to Charleston country (1000 km). Source: computed using ArcGIS with data in North America Equidistant Conic projection.
Average Precipitation
Average county monthly precipitation mm/month (baseline period 1961-1990). Source: CRU CL 2.0 data from New (2002).
Average Temperature
Mean annual county temperature (baseline period 1961-1990). Source: from FAO/IIASA, 2011-2012. Global Agro-ecological Zones (GAEZ v3.0). FAO Rome, Italy and IIASA, Laxenburg, Austria.
Total Population
Total population in the county in 1790. Source: Historical U.S. census. *www.nhgis.org.*
Share of Families with Slaves
Share of families owning slaves in the county in 1790. Source: Historical U.S. census. *www.nhgis.org.*
Blacks
Black People in the county in 1790. Source: Historical U.S. census. *www.nhgis.org.*

Table 8: Summary Statistics of Cross-County Analysis

Variable	Mean	Std. Dev.	Min	Max	N
Share of Slaves	0.224	0.205	0	0.762	285
Share of Blacks	0.239	0.209	0.002	0.772	285
Malaria Stability	0.043	0.029	0	0.108	285
Malaria Endemicity	2.054	0.975	0	3	285
Cotton Suitability	2469.092	1746.858	0	7486.781	285
Rice Suitability	1071.552	1422.706	0	5802.241	285
Sugar Suitability	140.035	508.068	0	2874.037	285
Tea Suitability	2142.92	2162.941	0	7170.96	285
Tobacco Suitability	4000.996	1380.764	0.305	7261.313	285
Distance Sea	0.118	0.136	0	0.713	285
Distance River	0.011	0.006	0.001	0.035	285
Distance Charleston	0.63	0.363	0.001	1.689	285
Average Precipitation	95.261	8.106	71.054	119.591	285
Average Temperature	12.588	3.225	4.625	19.337	285
Total Population	13662.926	11349.489	305	75980	285
Share of Families with Slaves	0.234	0.183	0	0.684	142
Blacks	2643.519	3886.554	16	51583	285

Table 9: Cross-correlation table

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)
(1) Share of Slaves	1.000																
(2) Share of Blacks	0.997	1.000															
(3) Malaria Stability	0.605	0.591	1.000														
(4) Hist. Malaria Endemicity	0.700	0.687	0.780	1.000													
(5) Cotton Suitability	0.437	0.427	0.712	0.562	1.000												
(6) Rice Suitability	0.046	0.037	0.479	0.208	0.382	1.000											
(7) Sugar Suitability	0.295	0.284	0.407	0.262	0.111	0.356	1.000										
(8) Tea Suitability	0.525	0.514	0.662	0.637	0.502	0.264	0.104	1.000									
(9) Tobacco Suitability	-0.042	-0.050	0.212	0.092	0.549	0.182	-0.130	0.182	1.000								
(10) Sea Distance	-0.273	-0.303	0.084	-0.143	0.173	0.204	-0.104	0.059	0.284	1.000							
(11) River Distance	-0.196	-0.205	-0.031	-0.105	0.016	-0.129	-0.076	-0.055	0.101	0.094	1.000						
(12) Distance to Charleston	-0.552	-0.535	-0.886	-0.746	-0.657	-0.393	-0.376	-0.696	-0.315	-0.210	0.067	1.000					
(13) Average Precipitation	-0.039	-0.040	0.318	0.064	0.028	0.535	0.342	0.233	-0.063	-0.082	0.023	-0.287	1.000				
(14) Average Temperature	0.630	0.623	0.920	0.750	0.688	0.514	0.457	0.670	0.268	0.033	-0.159	-0.935	0.363	1.000			
(15) Total Population	-0.269	-0.261	-0.382	-0.327	-0.293	-0.214	-0.087	-0.382	-0.025	-0.219	0.151	0.464	0.000	-0.427	1.000		
(16) Share of Families w. Slaves	0.951	0.954	0.641	0.620	0.490	0.311	0.330	0.410	-0.116	-0.295	-0.308	-0.556	0.187	0.666	-0.277	1.000	
(17) Blacks	0.615	0.617	0.321	0.365	0.229	-0.049	0.233	0.218	-0.014	-0.218	-0.048	-0.283	-0.062	0.315	0.251	0.526	1.000

Table 10: Malaria and Slavery: Additional Results

	PANEL A					
	Share of Slaves			**Share of Blacks**		
Malaria Stability	4.158	3.138	3.083	4.257	3.067	3.011
Conley s.e. 100 km	(0.873)***	(0.946)***	(0.946)***	(0.896)***	(0.951)***	(0.949)***
Conley s.e. 500 km	(1.397)***	(1.472)**	(1.518)**	(1.390)***	(1.460)**	(1.505)**
Conley s.e. 1000 km	(1.351)***	(1.500)**	(1.555)**	(1.328)***	(1.478)**	(1.532)**
Total Population	Yes	No	Yes	Yes	No	Yes
Temperature and Precipitation	No	Yes	Yes	No	Yes	Yes
Crop Suitabilities	Yes	Yes	Yes	Yes	Yes	Yes
Distances	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Observations	285	285	285	285	285	285
R-squared	0.795	0.802	0.805	0.795	0.802	0.805
	PANEL B					
	Share of Families with Slaves			**Number of Blacks**		
Malaria Stability	4.457	4.384	2.945	73,133	67,844	56,294
Conley s.e. 100 km	(0.987)***	(1.012)***	(0.909)***	(13,070)***	(13,669)***	(12,963)***
Conley s.e. 500 km	(1.162)***	(1.439)***	(1.014)***	(15,931)***	(11,618)***	(15,127)***
Conley s.e. 1000 km	(0.929)***	(1.328)***	(0.774)***	(12,679)***	(8,969)***	(13,664)***
Crop Suitabilities	Yes	Yes	Yes	Yes	Yes	Yes
Distances	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Observations	142	142	142	285	285	285
R-squared	0.647	0.704	0.781	0.241	0.326	0.344

Notes: The table reports OLS estimates. The unit of observation is the US county in 1790. The dependent variable in Panel A is the county share of slaves (columns 1-3), and the county share of blacks (columns 4-6). In Panel B (columns 1-3) the dependent variable is the share of families owning slaves, and the absolute number of blacks in the county (columns 4-6). Malaria Stability is an index measuring the force and stability of malaria transmission. The "Crop suitabilities" controls include soil suitability to cotton, sugar, rice, tea and tobacco. The "Distances" controls include distance to the sea, to the closest river and to Charleston. Average temperature and precipitation are modern climatic measures. Total population measures the total inhabitants of the county in 1790. Conley standard errors are reported in brackets, with cutoff thresholds for latitude and longitude at 100, 500 and 1000 km. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 11: Malaria and Slavery across US Counties

	Dependent Variable: Share of Slaves 1790							
	PANEL A Malaria Stability - Full Sample							
Malaria Stability	4.289	5.672	6.820	5.461	5.768	5.289	5.907	4.997
Conley s.e. 100 km	(0.550)***	(1.017)***	(1.143)***	(1.014)***	(1.029)***	(0.922)***	(0.995)***	(0.959)***
Conley s.e. 500 km	(1.047)***	(1.578)***	(1.688)***	(1.653)***	(1.635)***	(1.237)***	(1.608)***	(1.289)***
Conley s.e. 1000 km	(1.046)***	(1.265)***	(1.376)***	(1.332)***	(1.322)***	(0.932)***	(1.309)***	(1.139)***
Crop Suitability Indexes								
Cotton Suitability	No	No	Yes	No	No	No	No	Yes
Sugar Suitability	No	No	No	Yes	No	No	No	Yes
Rice Suitability	No	No	No	No	Yes	No	No	Yes
Tea Suitability	No	No	No	No	No	Yes	No	Yes
Tobacco Suitability	No	No	No	No	No	No	Yes	Yes
State FE	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	285	285	285	285	285	285	285	285
R-squared	0.366	0.601	0.621	0.609	0.648	0.615	0.658	0.716
	PANEL B Malaria Stability Full Sample				PANEL C Malaria Endemicity Full Sample		PANEL D Malaria Stability Only Slave States	
Malaria Stability	3.678	5.301	5.252	4.193			5.704	4.145
Conley s.e. 100 km	(0.803)***	(0.884)***	(1.126)***	(0.881)***			(1.022)***	(0.909)***
Conley s.e. 500 km	(1.261)***	(1.191)***	(1.586)***	(1.387)***			(1.579)***	(1.419)***
Conley s.e. 1000 km	(1.185)***	(1.057)***	(1.336)***	(1.334)***			(1.267)***	(1.394)***
Malaria Endemicity					0.126	0.050		
Conley s.e. 100 km					[0.022]***	[0.017]**		
Conley s.e. 500 km					[0.021]***	[0.010]***		
Conley s.e. 1000 km					[0.017]***	[0.008]***		
Crop Suitability Indexes	Yes	Yes	Yes	Yes	No	Yes	No	Yes
Distances								
Sea Distance	Yes	No	No	Yes	No	Yes	No	Yes
River Distance	No	Yes	No	Yes	No	Yes	No	Yes
Distance Charleston	No	No	Yes	Yes	No	Yes	No	Yes
State FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	285	285	285	285	285	285	244	244
R-squared	0.788	0.727	0.717	0.793	0.615	0.767	0.505	0.749

Notes: The table reports OLS estimates. The unit of observation is the US county in 1790. The dependent variable is the share of slaves in the county population. In panel A, B and D, the explanatory variable is Malaria Stability, an index measuring the force and stability of malaria transmission, whereas in Panel C the explanatory variable is Malaria Endemicity, an index measuring the malaria parasitization rate at the beginning of the 20th century. For the sample of "slave states", I exclude counties in Connecticut, Massachusetts, New Hampshire, Rhode Island and Vermont. Conley standard errors are reported in brackets, with cutoff thresholds for latitude and longitude at 100, 500 and 1000 km. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 12: Malaria and Slavery across US Counties

	Dependent Variable: Share of Blacks 1790							
	PANEL A Malaria Stability - Full Sample							
Malaria Stability	4.280	5.941	7.143	5.726	6.037	5.548	6.191	5.241
Conley s.e. 100 km	(0.571)***	(1.062)***	(1.205)***	(1.059)***	(1.073)***	(0.976)***	(1.029)***	(1.016)***
Conley s.e. 500 km	(1.093)***	(1.611)***	(1.740)***	(1.686)***	(1.669)***	(1.276)***	(1.646)***	(1.330)***
Conley s.e. 1000 km	(1.091)***	(1.291)***	(1.416)***	(1.359)***	(1.349)***	(0.954)***	(1.341)***	(1.166)***
Crop Suitability Indexes								
Cotton Suitability	No	No	Yes	No	No	No	No	Yes
Sugar Suitability	No	No	No	Yes	No	No	No	Yes
Rice Suitability	No	No	No	No	Yes	No	No	Yes
Tea Suitability	No	No	No	No	No	Yes	No	Yes
Tobacco Suitability	No	No	No	No	No	No	Yes	Yes
State FE	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	285	285	285	285	285	285	285	285
R-squared	0.349	0.593	0.614	0.601	0.640	0.607	0.655	0.712
	PANEL B Malaria Stability Full Sample				PANEL C Malaria Endemicity Full Sample		PANEL D Malaria Stability Only Slave States	
Malaria Stability	3.801	5.572	5.451	4.293			5.976	4.237
Conley s.e. 100 km	(0.826)***	(0.935)***	(1.187)***	(0.905)***			(1.067)***	(0.929)***
Conley s.e. 500 km	(1.261)***	(1.224)***	(1.635)***	(1.383)***			(1.612)***	(1.409)***
Conley s.e. 1000 km	(1.180)***	(1.076)***	(1.344)***	(1.313)***			(1.293)***	(1.369)***
Malaria Endemicity					0.129	0.045		
Conley s.e. 100 km					[0.025]***	[0.018]***		
Conley s.e. 500 km					[0.023]***	[0.011]***		
Conley s.e. 1000 km					[0.019]***	[0.009]***		
Crop Suitability Indexes	Yes	Yes	Yes	Yes	No	Yes	No	Yes
Distances								
Sea Distance	Yes	No	No	Yes	No	Yes	No	Yes
River Distance	No	Yes	No	Yes	No	Yes	No	Yes
Distance Charleston	No	No	Yes	Yes	No	Yes	No	Yes
State FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	285	285	285	285	285	285	244	244
R-squared	0.795	0.725	0.713	0.800	0.602	0.772	0.499	0.760

Notes: The table reports OLS estimates. The unit of observation is the US county in 1790. The dependent variable is the share of blacks in the county population. In panel A, B and D, the explanatory variable is Malaria Stability, an index measuring the force and stability of malaria transmission, whereas in Panel C the explanatory variable is Malaria Endemicity, an index measuring the malaria parasitization rate at the beginning of the 20th century. In the sample of "slave states", I exclude counties in Connecticut, Massachusetts, New Hampshire, Rhode Island and Vermont. Conley standard errors are reported in brackets, with cutoff thresholds for latitude and longitude at 100, 500 and 1000 km. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Appendix A.2: Malaria and African Slavery - Panel United States 1640-1790

Data Sources: Malaria and Slavery - Panel United States 1640-1790

Blacks to Total Population
Ratio of black population in the state in the decade. Maine, Playmouth and Massachusetts added up together. States for which we have information only after 1690 are excluded (Kentucky, Tennessee, Georgia and Vermont). Decade 1630 is excluded because data for only 4 states is available. Source: Colonial and Prefederal Statistics, Chapter Z.

Blacks to Total Population Lag
Ratio of black population in the state in the decade lagged. Source: Colonial and Prefederal Statistics, Chapter Z.

Total Population
Total population in the state in the decade. Source: Colonial and Prefederal Statistics, Chapter Z.

Post-1690
Indicator variable that equaling 1 from 1690 onwards, and 0 otherwise.

Yellow Fever
Number of yellow fever epidemics in the decade within the decade. Source: Duffy (1972).

England Farm Wage
Daily Farm Wage England, averaged over decades. Source: English prices and wages, 1209-1914 (Gregory Clark).

Malaria Stability
Average Malaria Stability Index in the state. Source: average Malaria Stability is constructed as the state average of the Malaria Stability index from Kiszewski (2004).

Falciparum Malaria
Falciparum Malaria is an indicator variable taking value 1 if in the state-decade there is historical evidence of falciparum malaria. Source: Wood (1974) and Rutman and Rutman (1976).

Malaria Endemicity
Average Historical Malaria Endemicity in the state. Source: average Historical Malaria Endemicity is constructed as the state average of the Malaria Endemicity level, devised by Lysenko (1968) and digitalized by Hay (2004).

Crop Suitability Indexes
Estimated suitability index (value) for cultivating cotton, coffee, rice, sugar, tea and tobacco at a disaggregated geographic level. Source: FAO/IIASA, 2011. Global Agro-ecological Zones (GAEZv3.0). FAO Rome, Italy and IIASA, Laxenburg, Austria. *http://gaez.fao.org/Main.html*. The suitability index employed is the one estimated for low inputs level and rain-fed conditions.

Average Temperature
Mean annual country temperature (baseline period 1961-1990). Source: state average of the mean annual temperature across grids, from FAO/IIASA, 2011-2012. Global Agro-ecological Zones (GAEZ v3.0). FAO Rome, Italy and IIASA, Laxenburg, Austria.

Slave Code in Force
Indicator variable taking value 1 in each state-decade during which a slave code was in force, 0 otherwise. Source: (Wiecek, 1977)

Slave Code Approved
Indicator variable taking value 1 in each state-decade during which a slave code was approved, 0 otherwise. Source: (Wiecek, 1977)

 # El Nino Events
Number of El Nino years in the decade. Source: *https://sites.google.com/site/medievalwarmperiod/Home/historic-el-nino-events*.

Table 14: Summary Statistics - Panel United States 1640-1790

Variable	Mean	Std. Dev.	Min.	Max.	N
Time-Varying					
Blacks to Total Population	0.12	0.15	0.003	0.704	166
Black People	11845.651	30243.983	15	220582	166
White People	52471.404	67674.448	170	319450	166
Post-1690	0.723	0.449	0	1	166
Malaria Stability x Post-1690	0.224	0.291	0	1	166
Malaria Stability Nrm. x Post-1690	0.193	0.281	0	1	192
Rice Suitability x Post-1690	5.706	8.076	0	25.466	166
Falciparum Malaria	0.241	0.429	0	1	166
Yellow Fever	0.151	0.5	0	3	166
Farm Wage in England	4.762	0.409	4.275	5.82	166
Slave Code Approval	0.072	0.26	0	1	166
Slave Code in Force	0.349	0.478	0	1	166
El Nino Events (#)	3.807	1.405	1	6	166
Malaria Stability x El Nino Events	0.1	0.112	0.001	0.522	166
Malaria Stability Nrm. x El Nino Events	1.151	1.293	0.017	6	166
Time-Invariant					
Malaria Stability	0.026	0.025	0.001	0.087	166
Rice Suitability	7.708	8.311	0.295	25.466	166
Tea Suitability	963.581	1344.649	0	4282.766	166
Tobacco Suitability	3207.936	1211.556	1040.467	4670.695	166
Historical Malaria Endemicity	1.435	0.690	0.700	2.845	166
Average Temperature	10.591	3.284	5.721	16.898	166

Table 15: Cross-correlation table

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
(1) Blacks to Total Population	1.000											
(2) Black People	0.628	1.000										
(3) White People	0.129	0.539	1.000									
(4) Post 1690	0.276	0.236	0.402	1.000								
(5) Malaria Stability x Post 1690	0.800	0.423	0.058	0.478	1.000							
(6) Falciparum Malaria	0.783	0.570	0.171	0.349	0.835	1.000						
(7) Rice Suitability x Post 1690	0.625	0.297	0.040	0.439	0.729	0.589	1.000					
(8) Slave Code Approval	0.602	0.473	0.365	0.454	0.629	0.533	0.302	1.000				
(9) Slave Code Approved	0.237	0.069	-0.035	0.173	0.281	0.278	0.175	0.381	1.000			
(10) # El Nino Events	0.152	0.162	0.267	0.568	0.272	0.198	0.249	0.200	0.005	1.000		
(11) Malaria Stability x # El Nino Events	0.719	0.370	-0.030	0.225	0.854	0.725	0.601	0.479	0.166	0.356	1.000	
(12) Malaria Stability Nrm. x # El Nino Events	0.719	0.370	-0.030	0.225	0.854	0.725	0.601	0.479	0.166	0.356	1.000	1.000

Table 16: Malaria and Share of Blacks across US States

	Share of Blacks								
Malaria Stability x Post-1690	4.222***		3.775***	3.602***	3.822***	6.145***	3.899***	5.590***	7.117***
Cluster (State) s.e.	(0.825)		(0.971)	(0.939)	(1.064)	(1.097)	(1.098)	(1.194)	(0.816)
Bootstrap s.e. p-value	*0.000*		*0.042*	*0.002*	*0.046*	*0.020*	*0.022*	*0.136*	*0.068*
Rice Suitability x Post-1690		0.009*	0.002						
		(0.004)	(0.002)						
England Farm Wage x State fe				Yes					
Rice Suit x Decade fe					Yes			Yes	Yes
Tea Suit x Decade fe						Yes		Yes	Yes
Tobacco Suit x Decade fe							Yes	Yes	Yes
Decade fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Time Trend									Yes
Observations	166	166	166	166	166	166	166	166	166
R-squared	0.586	0.432	0.594	0.734	0.683	0.745	0.639	0.793	0.919
Number of state	12	12	12	12	12	12	12	12	12

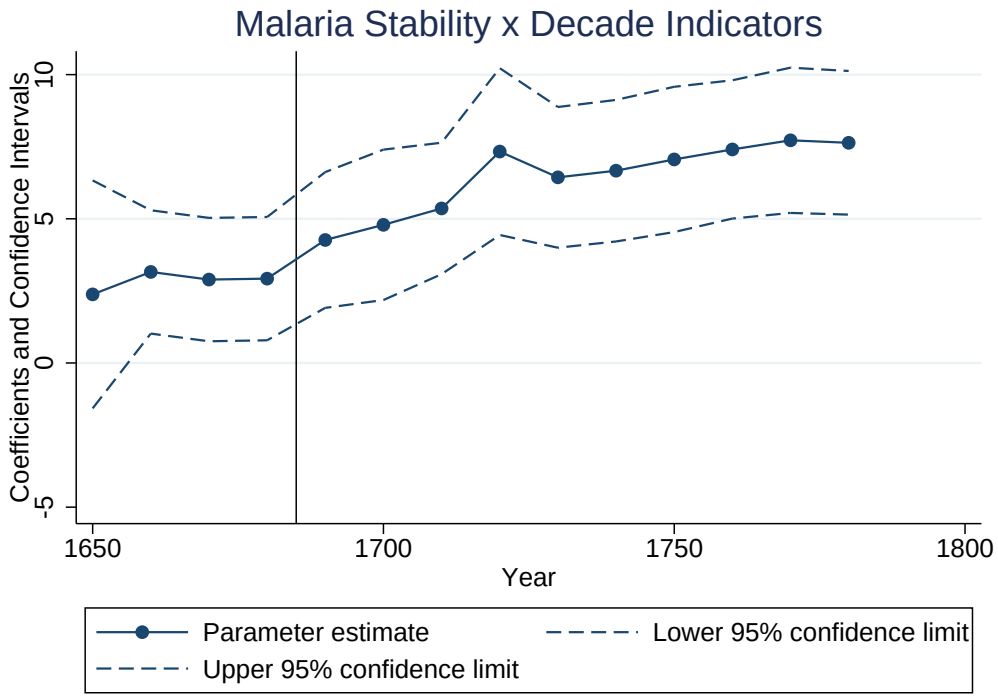
Notes: The table reports OLS estimates. The unit of observation is the US state in the decade. The panel includes all decades from 1640 to 1780. The dependent variable is the share of black people in the state. Malaria Stability is an index measuring the force and stability of malaria transmission. The variable Post-1690 is an indicator variable equaling 1 from 1690 onwards, and 0 otherwise. All the regressions include decade fixed effects and state fixed effects, except for the last Column which also include state specific time trend. Controls variable x Year FE are cross-sectional variables interacted with a full set of decade fixed effects. Standard errors clustered at the state level are reported in parenthesis. Since we only have 12 clusters, I report p-values for the null hypothesis (Malaria Stability x Post-1690) computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 17: Malaria and Share Blacks across US States

	Share of Blacks										
	Full Sample			Only 1680-1690	No S.Carolina	No N.Carolina	No Virginia	No Maryland	Full Sample		
Malaria Stability x Post-1690		4.140***	6.232***	1.847**	3.655***	3.679**	4.710***	4.182***	4.026***	1.603**	
Cluster (State) s.e.		(0.778)	(1.625)	(0.808)	(0.851)	(1.270)	(0.896)	(0.930)	(0.761)	(0.651)	
Bootstrap s.e. p-value		*0.000*	*0.026*	*0.188*	*0.008*	*0.044*	*0.014*	*0.010*	*0.000*	*0.004*	
Malaria Endemicity x Post-1690											0.140***
Cluster (State) s.e.											(0.034)
Bootstrap s.e. p-value											*0.000*
Yellow Fever	0.030	0.018									
	(0.022)	(0.011)									
Temperature x Decade fe			Yes								
Total Population									Yes		
Blacks to Total Pop. Lag										Yes	
Decade fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	166	166	166	24	151	154	153	151	166	157	166
R-squared	0.337	0.601	0.712	0.600	0.536	0.503	0.613	0.563	0.662	0.828	0.543
Number of state	12	12	12	12	11	11	11	11	12	12	12

Notes: The table reports OLS estimates. The unit of observation is the US state in the decade. The panel includes all decades from 1640 to 1780. The dependent variable is the share of black people in the state. Malaria Stability is an index measuring the force and stability of malaria transmission. The variable Post-1690 is an indicator variable equaling 1 from 1690 onwards, and 0 otherwise. All regressions include decade fixed effects and state fixed effects. Controls variable x Decade fe are cross-sectional variables interacted with a full set of decade fixed effects. Standard errors clustered at the state level are reported in parenthesis. Since I only have 12 clusters, I report p-values for the null hypothesis (Malaria Stability x Post-1690) computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Figure 3: Fully Flexible Estimation



†The graphs plot the estimated coefficients obtained by regressing the share of slave in the US state on the Malaria Stability index of the state interacted with decade fixed effects. All regressions include state and decade fixed effect.

Table 18: Malaria and Share of Blacks - Instrumental Variable Estimates

	Falciparum Malaria		**Share of Blacks**		
	First Stage		Reduced Form	OLS	IV
Falciparum Malaria				0.228***	0.223***
				(0.049)	(0.032)
Malaria Stability * # El Nino Events	2.567***		0.572***		
	(0.699)		(0.171)		
Decade fixed effects	Yes		Yes	Yes	Yes
State fixed effects	Yes		Yes	Yes	Yes
F Test Excluded Instrument	12.48				
Observations	166		166	166	166
R-squared	0.454	0.355	0.628	0.471	
Number of state	12		12	12	12

Notes: The table reports OLS and IV estimates. The unit of observation is the US state in the decade. The panel includes all decades from 1640 to 1780. Falciparum Malaria is an indicator variable taking value 1 if in the state-decade there is historical evidence of falciparum malaria. Malaria Stability is an index measuring the force and stability of malaria transmission. In columns 2-4, the dependent variable is the share of black people in the population. The variable Post-1690 is an indicator variable equaling 1 from 1690 onwards, and 0 otherwise. # El Nino Events is a variable counting the number of El Nino episodes registered in the decade. All regressions include decade fixed effects and state fixed effects. Standard errors clustered at the state level are reported in parenthesis. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 19: Malaria and Slave Codes - Instrumental Variable Estimates

	Falciparum Malaria		**Slave Code in Force**		
	First Stage		Reduced Form	OLS	IV
Falciparum Malaria				0.509***	0.670***
				(0.136)	(0.181)
Malaria Stability Nrm. * # El Nino Events	0.223***		0.150***		
	(0.061)		(0.037)		
Decade fixed effects	Yes		Yes	Yes	Yes
State fixed effects	Yes		Yes	Yes	Yes
F Test Excluded Instrument	12.48				
Observations	166		166	166	166
R-squared	0.454	0.566	0.616	0.144	
Number of state	12		12	12	12

Notes: The table reports OLS and IV estimates. The unit of observation is the US state in the decade. The panel includes all decades from 1640 to 1780. Falciparum Malaria is an indicator variable taking value 1 if in the state-decade there is historical evidence of falciparum malaria, and 0 otherwise. In columns 2-4, the dependent variable is Slave Code in Force, an indicator variable taking value 1 if a slave code is in force in the state-decade, 0 otherwise. Malaria Stability Nrm., is an index measuring the force and stability of malaria transmission normalized to range between 0 and 1. # El Nino Events is a variable counting the number of El Nino episodes registered in the decade. All the regressions include decade fixed effects and state fixed effects. Standard errors clustered at the state level are reported in parenthesis. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Appendix A.3: Slave Prices and Malaria in the Country of Origin

Data Sources: Slave Prices and Malaria in the Country of Origin

Slave Price
Sale value of an individual slave converted into a common denominator price (original prices were expressed in different currencies). Source: Louisiana Slave Database (SALEVALP).

Document Language
Language of the document or file: English, French or Spanish. Source: Louisiana Slave Database (LANGUAGE).

Document Type
Type of document or file: estate inventory, estate sale, sale which does not involve probate, criminal litigation, other litigation, mortgage, marriage contract, will, seizure for debt, confiscation in criminal proceedings, reports of a runaway, miscellaneous, list (as in census or taxation list), testimony of slaves, Atlantic slave trade. Source: Louisiana Slave Database (DOCTYPE).

Male Slave
Male dummy created on variable SEX, excluding slaves whose sex was unidentified. Source: Louisiana Slave Database (SEX).

Slave Age
Age of slave as reported in document or file. If a range of years was given, the mean age was computed: e.g., for a slave of 30 to 35 years, 32.5 was entered. Source: Louisiana Slave Database (AGE).

Malaria Stability
Average Malaria Stability Index in the country of origin of the slave. Source: average Malaria Stability is constructed as the country average of the Malaria Stability index from Kiszewski (2004) across grids, computed using ArcGIS with data in Africa Equal Area Conic projection.

Malaria Endemicity
Average Historical Malaria Endemicity in the country of origin of the slave. Source: average Historical Malaria Endemicity is constructed as the country average of the Malaria Endemicity level, devised by Lysenko (1968) and digitalized by Hay 2004, computed using ArcGIS with data in Africa Equal Area Conic projection.

Voyage Lenght
Average voyage length in days. Source: the Trans-Atlantic Slave Trade Database.

Distance Coast
Average distance to nearest ice-free coast (1000 km). Source: Nunn (2012).

Land Suitability
Average land suitability in the country of origin of the slave. Source: average Land Suitability is constructed as the country average of the land suitability index from Ramankutty (2002) across grids, computed using ArcGIS with data in Africa Equal Area Conic projection.

Mean Elevation
Average country elevation. Source: mean elevation is constructed as the country average of elevation across grids, computed using ArcGIS with data in Africa Equal Area Conic projection, with data from National Oceanic and Atmospheric Administration (NOAA) and U.S. National Geophysical Data Center, TerrainBase, release 1.0 (CD-ROM), Boulder, Colo. accessed through Atlas of Biosphere.

Average Precipitation
Average country monthly precipitation mm/month (baseline period 1961-1990). Source: average monthly precipitation is constructed as the country average of the mean monthly precipitation across 10 minute grids, computed using ArcGIS with data in Africa Equal Area Conic projection, with CRU CL 2.0 data from New (2002).

Average Relative Humidity
Average country relative humidity (%):how much water vapor is in the air. Source: average relative humidity is constructed as the country average of relative humidity across grids, computed using ArcGIS with data in Africa Equal Area Conic projection, with data from New (1999) accessed through Atlas of Biosphere.

Data Sources: Slave Prices and Malaria in the Country of Origin

Tropical Land

Share of tropical land. Source: Nunn (2012).

TseTse Fly Suitability

Average country predicted suitability for tsetse flies. Source: predicted suitability for tsetse flies is constructed as the sum of predicted suitability (0 to 1) for the presence of Tsetse groups (Fusca, Morsitans and Palpalis), computed using ArcGIS with data in Africa Equal Area Conic projection. Data produced for FAO - Animal Health and Production Division and DFID - Animal Health Programme by Environmental Research Group Oxford (ERGO Ltd) in collaboration with the Trypanosomosis and Land Use in Africa (TALA) research group at the Department of Zoology, University of Oxford.

Average Crew Deaths

Average share of the crew who dyed along the coast in the country. Source: the Trans-Atlantic Slave Trade Database.

Ruggedness

Average country ruggedness (Terrain Ruggedness Index, 100 m). Source: Nunn (2012).

Average Temperature

Mean annual country temperature (baseline period 1961-1990). Source: average temperature is constructed as the country average of the mean annual temperature across grids, computed using ArcGIS with data in Africa Equal Area Conic projection, from FAO/IIASA, 2011-2012. Global Agro-ecological Zones (GAEZ v3.0). FAO Rome, Italy and IIASA, Laxenburg, Austria.

Distance Atlantic Markets

Distance to slave markets, Atlantic trade (1000 km). Computed as "the sailing distance from the point on the coast that is closest to the countryâs centroid to the closest major market of the Atlantic slave trade (Virginia, USA; Havana, Cuba; Haiti; Kingston, Jamaica; Dominica; Martinique; Guyana; Salvador, Brazil; and Rio de Janeiro, Brazil)". Source: from Nunn (2007).

Average Rice Suitabilty

Country average of soil suitability to rice, low input rain-fed agriculture. Source: FAO GAEZ.

Historical Croplands Cover

Country average of the fraction of grid cells occupied by cultivated land in 1700. Source: average historical cropland cover across grids is computed using ArcGIS with data in Africa Equal Area Conic projection, using crop cover data from 1700 by Hall (2006) and Ramankutty (1999).

Fertile Land

Share of fertile land. Source: Nunn (2012).

Transition to Agriculture

Year of transition from reliance mainly on hunting and gathering to reliance mainly on cultivated crops (and livestock). Source: from Chanda (2007).

Ln(Population in 1400)

Natural log of population in 1400. Source: from Nunn (2012).

State Antiquity in 1700

Index measuring the presence of a supra-tribal polity within the present-day boundaries of countries. Computed adding up 50-year scores from year 1 to 1700 aC. Source: from Chanda (2007).

Table 21: Summary Statistics - Malaria in the Country of Origin and Prices

Variable	Mean	Std. Dev.	Min.	Max.	N
Male Slave	0.692	0.462	0	1	3186
Slave Age	29.455	12.301	1.8	80	3186
Slave Age Squared	1018.832	888.694	3.24	6400	3186
Ln(Slave Price)	6.177	0.644	2.303	8.102	3186
Malaria Stability	18.859	3.801	5.255	31.357	3186
Malaria Endemicity	4.171	0.432	2.459	4.899	3186
Voyage Lenght	63.248	13.684	43.235	134.444	3186
Distance Coast	0.396	0.191	0.045	1.081	3186
Land Suitability	0.363	0.135	0.111	0.635	3186
Mean Elevation	325.115	126.32	29.973	1043.994	3186
Average Precipitation	120.162	40.892	27.98	232.986	3186
Average Relative Humidity	66.868	12.606	33.756	80.578	3186
Tropical Land	85.479	25.826	8.401	100	3186
TseTse Fly Suitability	0.537	0.21	0.045	0.831	3186
Ruggedness	0.293	0.197	0.141	1.194	3186
Average Temperature	25.933	1.486	21.01	28.718	3186
Distance Atlantic Markets	4.903	0.904	3.705	10.595	3186
Average Rice Suitability	2067.258	725.617	283.201	3247.568	3186
Historical Croplands Cover	0.034	0.025	0.003	0.098	3186
% Fertile soil	28.821	25.833	4.063	74.973	3186
Transition to Agriculture	3.018	0.279	1.25	3.5	3186
Ln(Population in 1400)	13.202	1.1	11.383	15.592	3186
State Antiquity	0.27	0.207	0	0.637	3180

Table 22: Cross-Correlation Table - Malaria in the Country of Origin and Slave Price

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)	(21)	(22)	(23)
(1) Male Slave	1.000																						
(2) Slave Age	0.092	1.000																					
(3) Slave Age Squared	0.067	0.972	1.000																				
(4) Ln(Slave Price)	0.129	-0.384	-0.455	1.000																			
(5) Malaria Ecology	-0.055	0.114	0.108	-0.071	1.000																		
(6) Malaria Endemicity	-0.075	-0.092	-0.083	0.050	0.337	1.000																	
(7) Voyage Lenght	-0.029	0.011	0.004	0.018	0.154	0.582	1.000																
(8) Distance Coast	0.062	0.065	0.050	-0.025	-0.504	-0.564	0.035	1.000															
(9) Land Suitability	-0.051	0.110	0.103	-0.078	0.631	0.348	0.120	-0.528	1.000														
(10) Mean Elevation	0.005	-0.018	-0.024	0.006	-0.554	-0.017	0.103	0.400	0.135	1.000													
(11) Avg. Precipitation	-0.054	-0.109	-0.102	0.068	-0.069	0.383	0.004	-0.399	0.374	0.416	1.000												
(12) Avg. Relative Humidity	-0.049	-0.181	-0.171	0.114	-0.334	0.635	0.436	-0.184	-0.046	0.450	0.709	1.000											
(13) Tropical Land	-0.068	-0.096	-0.092	0.056	0.037	0.690	0.476	-0.312	0.434	0.496	0.784	0.854	1.000										
(14) TseTse Fly Suit.	-0.052	-0.119	-0.111	0.078	-0.006	0.566	0.287	-0.320	0.331	0.415	0.793	0.796	0.919	1.000									
(15) Ruggedness	-0.015	0.083	0.080	-0.066	0.250	-0.068	-0.434	-0.422	0.788	0.251	0.367	-0.211	0.140	0.140	1.000								
(16) Avg. Temperature	0.024	0.158	0.150	-0.092	0.607	-0.375	-0.211	0.008	0.031	-0.758	-0.666	-0.881	-0.743	-0.687	-0.028	1.000							
(17) Dist. Atlantic Markets	-0.015	-0.066	-0.070	0.050	-0.407	0.323	0.619	0.237	-0.252	0.363	-0.016	0.552	0.348	0.082	-0.434	-0.538	1.000						
(18) Avg. Rice Suitability	-0.074	0.009	0.004	-0.001	0.449	0.663	0.571	-0.371	0.694	0.249	0.560	0.528	0.857	0.738	0.255	-0.348	0.218	1.000					
(19) Hist. Croplands Cover	-0.004	0.058	0.064	-0.038	0.524	-0.039	-0.386	-0.516	0.140	-0.784	-0.365	-0.608	-0.581	-0.557	0.237	0.707	-0.450	-0.320	1.000				
(20) % Fertile soil	-0.021	0.146	0.138	-0.098	0.771	0.107	0.142	-0.261	0.321	-0.639	-0.595	-0.609	-0.397	-0.530	0.051	0.771	-0.126	0.051	0.694	1.000			
(21) Trans. to Agriculture	-0.014	-0.025	-0.018	0.025	0.389	0.163	-0.121	-0.241	0.201	-0.192	0.293	0.069	0.259	0.546	0.08 0	0.122	-0.631	0.290	-0.021	-0.114	1.000		
(22) Ln(Population in 1400)	-0.005	0.162	0.146	-0.121	0.363	-0.259	-0.137	0.262	0.205	-0.079	-0.385	-0.554	-0.385	-0.611	0.168	0.498	-0.021	-0.117	0.321	0.586	-0.365	1.000	
(23) State Antiquity	0.024	0.114	0.107	-0.103	0.202	-0.396	-0.336	0.289	-0.173	-0.333	-0.666	-0.701	-0.711	-0.826	-0.075	0.642	-0.150	-0.548	0.485	0.570	-0.320	0.843	1.000

Table 23: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)									
	Baseline	Voyage Lenght		Nutrition and Vegetation						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Malaria Stability	0.019***	0.017***	0.036***	0.023***	0.018***	0.023***	0.028***	0.026***	0.024***	0.058***
	(0.005)	(0.004)	(0.009)	(0.005)	(0.006)	(0.005)	(0.006)	(0.006)	(0.006)	(0.019)
Wild Bootstrap P-value	*(0.010)*	*(0.024)*	*(0.006)*	*(0.000)*	*(0.020)*	*(0.000)*	*(0.002)*	*(0.000)*	*(0.008)*	*(0.004)*
Voyage Length		0.002**								0.003**
		(0.001)								(0.001)
Distance Coast			0.205**							0.058
			(0.085)							(0.115)
Land Suitability				-0.138**						-0.066
				(0.064)						(0.608)
Mean Elevation					-0.000					0.001
					(0.000)					(0.000)
Average Precipitation						-0.000				0.002
						(0.000)				(0.001)
Average Relative Humidity							-0.003			-0.002
							(0.002)			(0.009)
Tropical Land								-0.001*		-0.008
								(0.000)		(0.005)
TseTse Fly Suitability									-0.072	0.364
									(0.048)	(0.314)
Male Slave	0.194***	0.194***	0.194***	0.194***	0.194***	0.194***	0.193***	0.193***	0.194***	0.193***
	(0.022)	(0.022)	(0.021)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)
Slave Age	0.048***	0.048***	0.048***	0.049***	0.049***	0.049***	0.048***	0.049***	0.049***	0.048***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Slave Age Squared	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Region fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.446	0.447	0.447	0.447	0.446	0.447	0.447	0.447	0.446	0.449
Observations	3186	3186	3186	3186	3186	3186	3186	3186	3186	3186

Notes: The table reports OLS estimates. The unit of observation is the individual slave transaction. The dependent variable is the natural logarithm of the selling price of the slave. Malaria Stability is an index measuring the force and stability of malaria transmission in the country of birth of the individual enslaved. All the regressions also control for the age of the slave, the square of the age and whether the slave is a male. "Region fixed effects" are indicator variables that define African macro-regions (Upper Guinea, Bight of Benin, Western Africa and Southeastern Africa) for the country of birth of the slave. "Year fixed effects" are indicator variables for the year (1741-1820) of the document (from which the information was retrieved). "Document Language fixed effects" are indicator variables for the language of the original document (English, French or Spanish). "Document Type fixed effects" are indicator variables for the type of documents from which the information was retrieved (estate sale, mortgage, marriage contract...). Standard errors are clustered at the country level (21 clusters). Given the small number of clusters (21), I report p-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 24: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)								
	Production Costs			Agricultural Skills				Human Capital	
	(1)	(2)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Malaria Stability	0.021***	0.021***	0.020***	0.024***	0.019***	0.019***	0.019**	0.016**	0.016**
	(0.004)	(0.004)	(0.007)	(0.006)	(0.006)	(0.005)	(0.007)	(0.007)	(0.006)
Wild Bootstrap P-value	*(0.000)*	*(0.000)*	*(0.034)*	*(0.000)*	*(0.012)*	*(0.010)*	*(0.064)*	*(0.010)*	*(0.020)*
Ruggedness	-0.112***								
	(0.034)								
Average Temperature		0.019**							
		(0.007)							
Distance Atlantic Markets			0.016						
			(0.071)						
Average Rice Suitability				-0.000					
				(0.000)					
Historical Croplands Cover					0.058				
					(0.477)				
% Fertile soil						0.000			
						(0.000)			
Transition to Agriculture							0.003		
							(0.047)		
Ln(Population in 1400)								-0.013	
								(0.013)	
State Antiquity									-0.053
									(0.053)
Male Slave	0.193***	0.193***	0.195***	0.194***	0.194***	0.194***	0.194***	0.194***	0.195***
	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)
Slave Age	0.049***	0.049***	0.048***	0.049***	0.048***	0.048***	0.048***	0.048***	0.048***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Slave Age Squared	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Region fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.447	0.447	0.446	0.446	0.446	0.446	0.446	0.446	0.447
Observations	3186	3186	3186	3186	3186	3186	3186	3186	3180

Notes: The table reports OLS estimates. The unit of observation is the individual slave transaction. The dependent variable is the natural logarithm of the selling price of the slave. Malaria Stability is an index measuring the force and stability of malaria transmission in the country of birth of the individual enslaved. All the regressions also control for the age of the slave, the square of the age and whether the slave is a male. "Region fixed effects" are indicator variables that define African macro-regions (Upper Guinea, Bight of Benin, Western Africa and Southeastern Africa) for the country of birth of the slave. "Year fixed effects" are indicator variables for the year (1741-1820) of the document (from which the information was retrieved). "Document Language fixed effects" are indicator variables for the language of the original document (English, French or Spanish). "Document Type fixed effects" are indicator variables for the type of documents from which the information was retrieved (estate sale, mortgage, marriage contract...). Standard errors are clustered at the country level (21 clusters). Given the small number of clusters (21), I report p-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

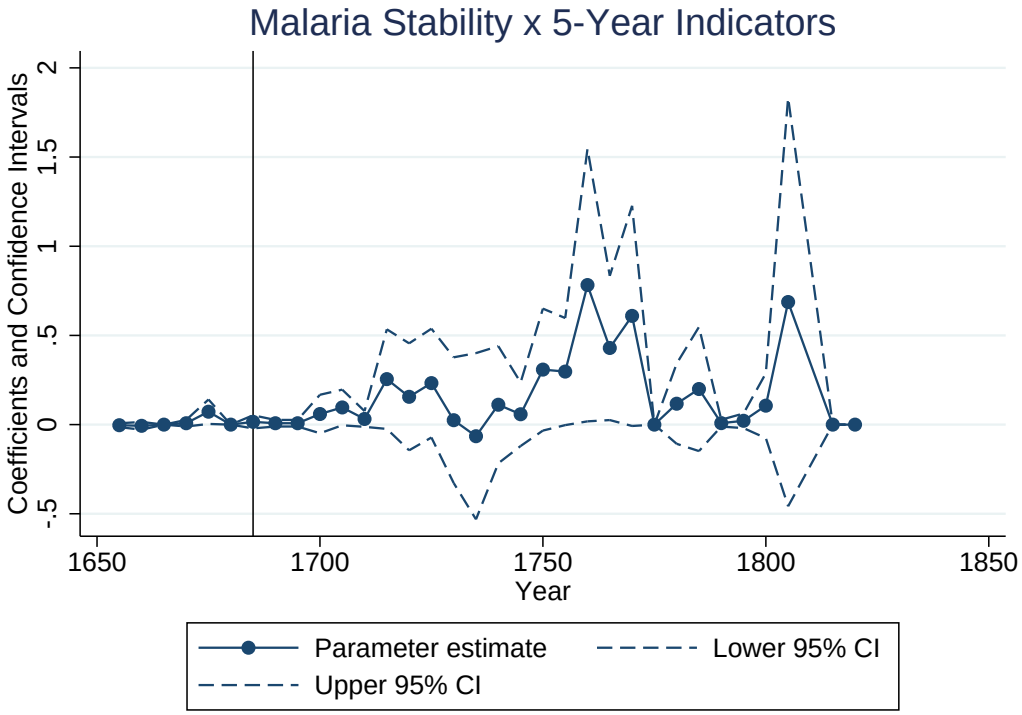
Table 25: Malaria in the Country of Origin and Slave Price

	Ln(Slave Price)					
	Endemicity		No Border Groups		No "Congo"	
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Endemicity	0.054***	0.297**				
	(0.017)	(0.123)				
Wild Bootstrap P-value	*(0.000)*	*(0.038)*				
Malaria Stability			0.020***	0.047***	0.019***	0.056***
			(0.005)	(0.015)	(0.006)	(0.018)
Wild Bootstrap P-value			*(0.012)*	*(0.076)*	*(0.014)*	*(0.002)*
Voyage Length Controls	No	Yes	No	Yes	No	Yes
Nutrition and Vegetation Controls	No	Yes	No	Yes	No	Yes
Slave Age and Age Squared	Yes	Yes	Yes	Yes	Yes	Yes
Male Slave	Yes	Yes	Yes	Yes	Yes	Yes
Region FE	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.444	0.448	0.449	0.452	0.443	0.448
Observations	3186	3186	1616	1616	1988	1988

	Ln(Slave Price)		**Slave Price**		**Ln(Slave Price)**	
	No "Guinea"		Full Sample		Sale and Inventory	
	(7)	(8)	(9)	(10)	(11)	(12)
Malaria Stability	0.020***	0.060***	9.965***	25.075***	0.010**	0.034***
	(0.002)	(0.004)	(0.022)	(0.006)	(0.005)	(0.011)
Wild Bootstrap P-value	*(0.002)*	*(0.004)*	*(0.022)*	*(0.006)*	*(0.088)*	*(0.008)*
Voyage Length Controls	No	Yes	No	Yes	No	Yes
Nutrition and Vegetation Controls	No	Yes	No	Yes	No	Yes
Slave Age and Age Squared	Yes	Yes	Yes	Yes	Yes	Yes
Male Slave	Yes	Yes	Yes	Yes	Yes	Yes
Region fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.450	0.454	0.364	0.366	0.505	0.508
Observations	2832	2832	3186	3186	5734	5734

Notes: The table reports OLS estimates. The unit of observation is the individual slave transaction. The dependent variable is the natural logarithm of the selling price of the slave in columns 1 to 8. In columns 9-10, the dependent variable is the absolute value of the selling price. In columns 11-12, the dependent variable include both selling and inventory prices. Malaria Stability is an index measuring the force and stability of malaria transmission in the country of birth of the individual enslaved. All the regressions also control for the age of the slave, the square of the age and whether the slave is a male. "Region fixed effects" are indicator variables that define African macro-regions (Upper Guinea, Bight of Benin, Western Africa and Southeastern Africa) for the country of birth of the slave. "Year fixed effects" are indicator variables for the year (1741-1820) of the document (from which the information was retrieved). "Document Language fixed effects" are indicator variables for the language of the original document (English, French or Spanish). "Document Type fixed effects" are indicator variables for the type of documents from which the information was retrieved (estate sale, mortgage, marriage contract...). Standard errors are clustered at the country level (21 clusters). Given the small number of clusters (21), I report p-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Figure 4: Number of Slave Voyages and Malaria Incidence of African Countries



†The graphs plots the estimated coefficients obtained by regressing the number of slave voyage to North America from African country j (in a 5-year interval) on the Malaria Stability index of country j interacted with 5-year interval fixed effects. All regressions include African country and 5-year interval fixed effects.

Table 26: Malaria and Slave Exports in Africa

	Ln(Slaves Exported over Total Area)				
	(1)	(2)	(3)	(4)	(5)
Malaria Stability	0.262***	0.205***	0.217***	0.162**	0.136**
	(0.030)	(0.049)	(0.044)	(0.064)	(0.065)
Ruggedness	No	Yes	No	Yes	Yes
Temperature	No	Yes	No	Yes	Yes
Humidity	No	Yes	No	Yes	Yes
Near Coast	No	No	Yes	Yes	Yes
Distances Slave Markets	No	No	Yes	Yes	Yes
Ln Pop Density 1400	No	No	No	No	Yes
Observations	57	47	57	47	47
R-squared	0.471	0.483	0.560	0.526	0.604

Notes: The table reports OLS estimates. The unit of observation is the African Country. The dependent variable is the natural logarithm of the total number of slaves exported over the total land area. Malaria Stability is an index measuring the force and stability of malaria transmission. Robust standard errors in parenthesis. ***, **, * indicate significance at 1, 5, and 10% levels respectively.

ONLINE APPENDIX B: ANECDOTAL EVIDENCE AND EPIDEMIOLOGICAL BACKGROUND

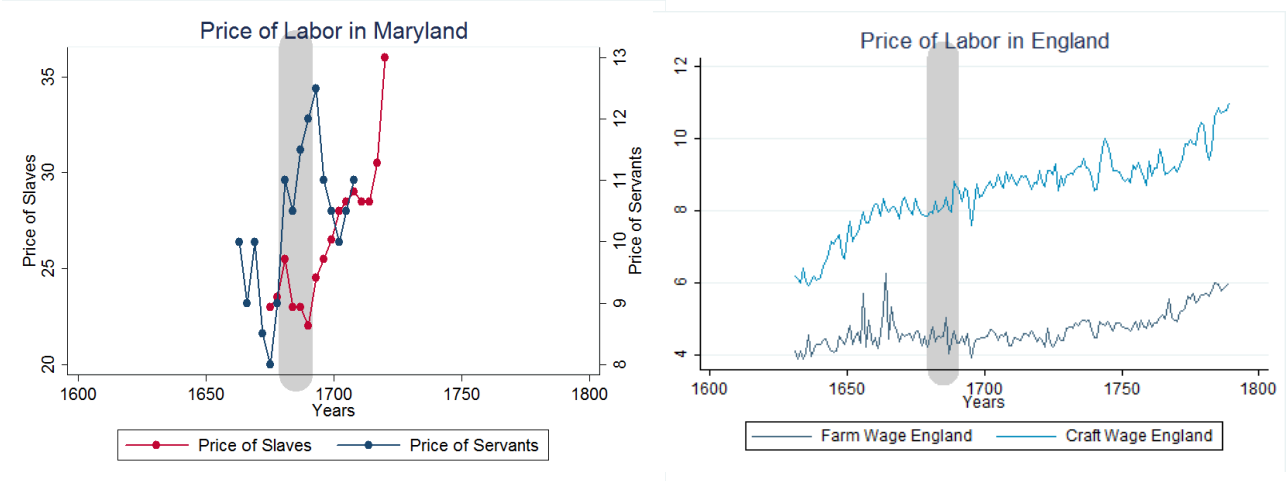
Table 27: Common Polymorphisms That Affect Resistance to Malaria

	Type of Protection	**Geographical Distribution**
Thalassemias	Approximately 50% reduction in the risk of malarial disease	High frequencies around Mediterranean sea shores, through most of Africa, Middle East, Central Asia, Arabian peninsula, Indian subcontinent, Southeast Asia, southern China, and Western Pacific Island (from Philippines to New Guinea and Melanesia)
Sickle Cell Trait	Approximately 90% protection against P. falciparum malarial mortality	In many parts of Africa, frequent at 30%
G6PD	Approximately 50% protection against severe P. falciparum malaria	same as Thalassemias
Hemoglobin C	Approximately 90% protection against P. falciparum malarial infection in the homozygote (30% in a heterozygous combination)	among certain West African population, frequent at 10%-20%
Hemoglobin E	May protect against P. vivax, and clear P. falciparum infection more rapidly	High frequencies in population across South-East Asia
Ovalocytosis	Reduced risk of P. vivax and P. falciparum infection	New Guinea (up tp 20%), Solomon Islands and Vanuatu
RBC Duffy Negativity	Complete refractoriness to P. vivax infection	More than 95% frequencies in West and Central Africa, at lower frequencies through the Arabian peninsula, across the Middle East and to the edges of Central Asia

ONLINE APPENDIX C: EVOLUTION OF LABOR COSTS IN MARYLAND

The left panel of Figure 5 plots the time series of slaves' prices and servants' wages. The grey area in the graph indicates the 1680s. First note that African slaves, the red line in the graph, cost more than European servants, blue line. This is no surprise since Europeans served for a few years (three to seven) whereas slaves were for life. Most importantly, during the 1680s the price of servants rose sharply, whereas the price of slaves swung but remained fairly at the same level. In the decades following 1680s, the price of servants tended to decrease while the price of African slaves took off. These dynamics are consistent with the hypothesis that a deterioration in the health environment decreased the supply of English and European workers willing to migrate to the Chesapeake, increasing wages. After starting to experience with African labor, slave price bolted upwards. Interestingly, over the 1680s we do not observe any substantial increase in the English farm wages, plotted in the right panel of Figure 5.

Figure 5: Labor Costs in Maryland



ONLINE APPENDIX D: COUNTRY OF BIRTH OF THE SLAVE

For a sub-sample of the slaves in the "Louisiana, Slave Records, 1719-1820" database, information on country of birth of the slave is provided. Out of the 3186 slaves composing the baseline sample (selected because reporting information on selling price, year of the sale, sex and age of the slave, type and language of the document source), for the majority of the slaves birthplace provided is a geographical region corresponding to modern countries (Angola, Benin, Coast of Senegal, Mozambique...). For more than one third we can retrace the ethnic origin of the slaves, whereas we know the city of origin for less than a tenth of slaves in the sample.

Table 28: Country of Origin: Louisiana Slave Database

Country	Entry in the Louisiana Slave Database	Total
Angola	Angola (3), Dimba (2)	5
Benin	Aja/Fon/Arada (65), Bargu (6), Benin (9), Juda, Port of (1)	81
Burkina Faso	Bobo (3), Marka (1)	4
Cameroon	Bakoko/Bacoro (1)	1
Central African Republic	Papelaou (1), Sango (1)	2
Congo	Atoyo/Atyo/Auda (2), Congo (1198)	1200
Congo Democratic Republic	Ham/Hamba (1), Louba (1), Mandongo (11), Ngala (1), Samba (1),	15
Cote d'Ivoire	Bacoy (1), Gold Coast (10)	11
Gabon	Gabon (10)	10
Ghana	Akwa (1), Coromanti (7), Fanti (7), Mina (197)	212
Guinea	Guinea/Guinea Coast (354), Kisi (26), Kouniaca (2), Soso (15), Toma (3)	400
Guinea Bissau	Bissago (1), Gabu/Cabao (1), Nalo (1)	3
Liberia	Gola (1)	1
Mali	Bamana (150)	150
Mozambique	Makwa (29), Mozambique (9)	38
Nigeria	Apa (3), Birom (1), Calabar (47), Edo (20), Ekoi (1), Esan/Edoid (1), Hausa (54), Ibibio/Moko (35), Igbo (189), Nago/Yoruba (110), Nupe (1)	462
Senegal	Coast of Senegal (1), Diola (4), Moor/Nar (59), Serer (3), Wolof (231)	298
Sierra Leone	Boke (1), Kanga (127), Koranko (2), Limba (2), Mende (2), Temne (5)	139
Tanzania	Makonde (3)	3
Togo	Cotocoli (2), Konkomba (148)	150
Zimbabwe	Karanga (1)	1

Figure 6: Map of Africa 1808

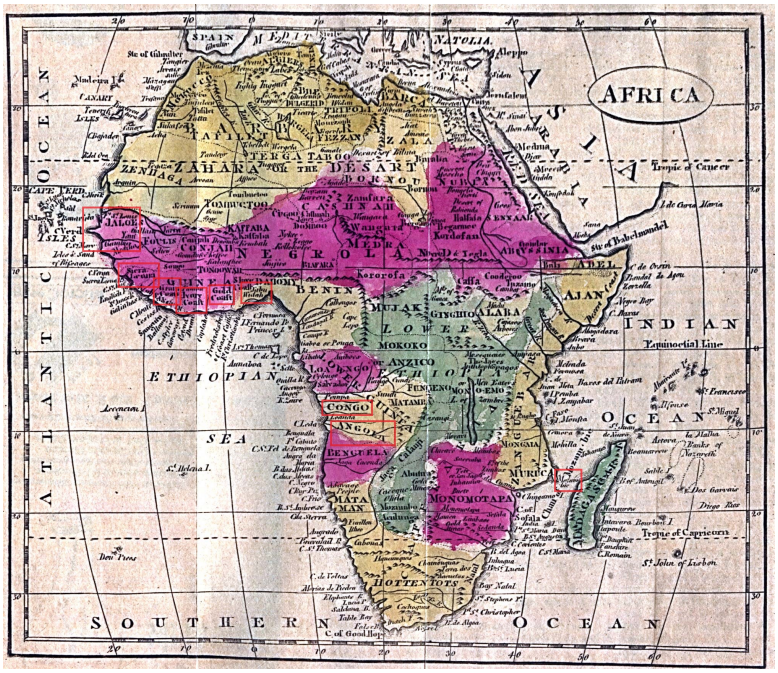
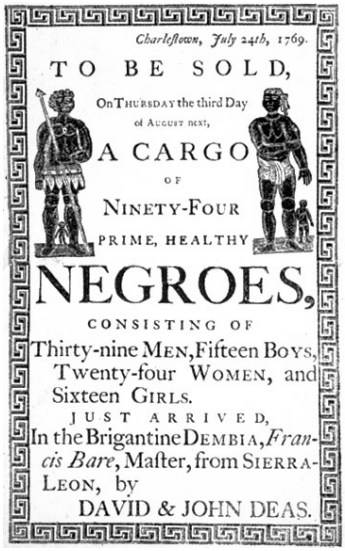


Figure 7: Map of Africa 1829

Figure 8: Advertisements



Charleſtown, July 24th, 1769.

TO BE SOLD,

On THURSDAY the third Day of AUGUST next,

A CARGO OF NINETY-FOUR PRIME, HEALTHY

NEGROES,

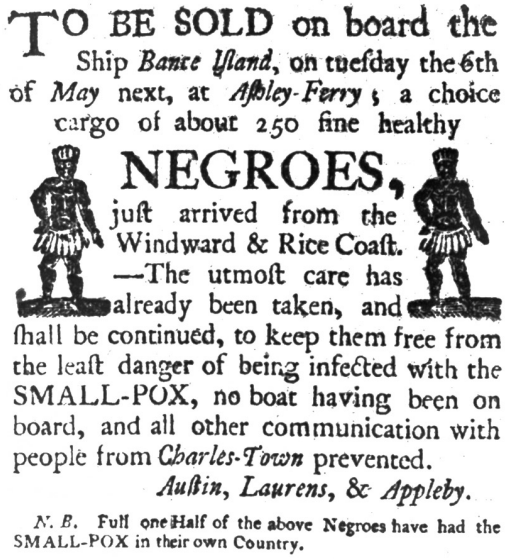
CONSISTING OF

Thirty-nine MEN, Fifteen BOYS, Twenty-four WOMEN, and Sixteen GIRLS.

JUST ARRIVED,

In the Brigantine DEMBIA, *Francis Bare*, Maſter, from SIERRA-LEON, by

DAVID & JOHN DEAS.



TO BE SOLD on board the Ship *Bance Iſland*, on tueſday the 6th of *May* next, at *Aſhley-Ferry*; a choice cargo of about 250 fine healthy

NEGROES,

juſt arrived from the Windward & Rice Coaſt. —The utmoſt care has already been taken, and ſhall be continued, to keep them free from the leaſt danger of being infected with the SMALL-POX, no boat having been on board, and all other communication with people from *Charles-Town* prevented.

Auſtin, Laurens, & Appleby.

N. B. Full one Half of the above Negroes have had the SMALL-POX in their own Country.