Side Effects of Immunities: the African Slave Trade*

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Abstract

Why was slavery concentrated in the South of the United States? And why were certain African populations extensively raided to be enslaved in those regions? The novel empirical evidence presented in this paper reveals that (i) *malaria* was a major determinant for the diffusion of African slavery in the southern United States and (ii) *malaria-resistance* made sub-Saharan Africans especially attractive for employment in these regions. I first document that African slavery was more widespread in those US counties subject to higher malaria exposure. Moreover, I show that the introduction of a particularly virulent, and previously absent, species of malaria into the United States caused a large and rapid increase in the prevalence of African slave labor. Finally, by looking at the historical prices of African slaves in the Louisiana slave market, I find that more malaria-resistant slaves, *i.e.* those born in the most malaria-ridden regions of Africa, commanded higher prices.

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

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

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1 Introduction

There is growing evidence that 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 represents a burden for societies that historically relied on it as a source of labor, as it has engendered long-term poverty, lower contemporary public goods provision and higher inequality (Engerman and Sokoloff, 1997; Nunn, 2008; Dell, 2010).[1] On the other hand, slavery also appears to have stunted the long-term growth prospects of populations subject to slave raids and enslavement - as in the case of African countries - 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 distribution of slavery, and in examining the factors that influenced and oriented the African Slave Trade.

This paper is the first to document the role played by diseases, and notably malaria, in determining why slavery spread in certain areas of the United States and not in others, and why Africans - and Africans from certain regions in particular - were transported to and enslaved in the New World so numerously. I argue that wherever in the United States malaria took root and flourished, labor became scarcer and more expensive, slavery more economically attractive, and malaria-resistant labor in high demand. In such areas, the malaria-resistance of Sub-Saharan Africans increased the profitability of African slave labor, and primarily that of African slaves from more malaria-ridden areas. Indeed, the empirical evidence I present reveals that African slavery was largely concentrated in the more malaria-infested areas of the United States and that it jumped after the introduction of a virulent malaria species. Moreover, by looking at the historical prices of African slaves in the United States, I show that slaves born in regions of Africa with higher prevalence of malaria commanded higher prices.

The hypothesis builds on two facts. First, malaria was absent from North America before European settlement. Importantly, once Europeans settled, malaria (a disease requiring specific bio-climatic conditions for transmission) did not spread all over North America but

[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).

became endemic only in regions which were 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 and making these areas extremely unattractive to migrants, who started to avoid them and prefer healthier destinations.[3] The second fact is that, as a consequence of a greater historical exposure to the disease, certain African populations had developed a vast set of resistances to malaria, granting protection from the disease.

In the paper, I argue that the radical change in the epidemiological environment that resulted from the introduction of malaria, coupled with the malaria resistance of certain African populations, fundamentally affected the evolution of labor choices in the United States. In areas subject to high malaria exposure, labor became scarcer and the practice of forced labor more widespread. At the same time, among the pool of available laborers more malaria-resistant workers started to be preferred. Africans had a higher stock of resistance to malaria than locals, and among them, the resistance to malaria of Africans from more malarial regions was even higher.

The first empirical test of the hypothesis outlined above reveals that African slavery was concentrated in the more malaria-infested counties 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-climatic characteristics.[4] The results show a strong positive cross-sectional correlation between malaria incidence and the share of African slaves across US counties in 1790. The relation remains sizable and statistically significant, even when accounting for the suitability of the county's soil for sugar, cotton, rice, tea and tobacco cultivation. Controlling for soil suitabilities for these labor-intensive crops is important, as many economists have considered this the main reason why slavery spread in certain areas of the United States and not others.[5]

[3]In particular, Europeans started to avoid unhealthy southern destinations while continuing to flow to safer northern colonies Menard (2001).

[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 United States in 1947.

[5]Engerman and Sokoloff (1997, 2000) argue that the US South, unlike the North, had climates and soils

There is a visually striking spatial correspondence between slave counties and malarial counties; a cross-sectional framework, however, cannot fully exclude that one or more unobservable time-invariant county characteristics - potentially correlated with geographic suitability for malaria, such as the temperature or the productivity of the soil - may be the actual driver of African slavery. In order to control for this, in my second empirical exercise I focus on an episode of *major* and *rapid* intensification of malaria incidence: the period surrounding the introduction of *falciparum* malaria in the United States, the most deadly and virulent malaria species. The available evidence indicates that *falciparum* malaria entered North America in the 1680s, favored by weather anomalies that characterized the decade in question.

As the actual timing of the introduction of *falciparum* malaria in each US state could be partly related to endogenous factors - such as trade intensity with places where malaria was endemic - in my baseline strategy I use the decade of the main ascertained *falciparum* epidemics, which took place in a decade of extreme weather anomalies, as the threshold for malaria introduction for all states. Then, I exploit across-state variation in malaria suitability as a proxy for the likelihood of *falciparum* malaria striking and becoming endemic in each state.[6] As an alternative strategy, I exploit exogenous variation from weather anomalies and bio-climatic characteristics to predict both where and when *falciparum* malaria was more likely to be introduced. I then use this predicted measure in an instrumental variable framework.[7]

This difference-in-difference exercise allows me to examine the increase in the share of African slaves that followed the introduction of *falciparum* malaria, comparing the US states that were more suitable for malaria with states that were less suitable. Employing a panel of 12 US states in the decades from 1640 to 1780, my results show that the introduction of the most debilitating form of malaria increased the share of Africans significantly more in regions with more favorable bio-climatic preconditions for malaria transmission (compared to others).

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.

[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] More precisely, my predicted measure combines time-varying information on weather anomalies with cross-sectional variation of malaria suitability across states.

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 my measure of malaria exposure. 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,[8] and for variations in English wages.[9]

Within the same empirical difference-in-difference exercise, I further show that the introduction of *falciparum* malaria increased the likelihood of slavery becoming institutionalized.[10] I use the date of approval of "slave codes", sets of laws placing harsh restrictions on the liberties of African individuals, as an indicator of the process of institutionalization of African slavery in the United States. My results show that after 1680 states that are more malaria-suitable are more likely to adopt "slave codes" than less malaria-suitable ones.

The third piece of empirical evidence I provide documents the specific contribution that malaria-resistance made in tilting the labor demands of slave-owners toward African labor, in particular toward Africans from more malaria-ridden regions. According to my hypothesis, the labor shortages that resulted from malaria introduction could not be filled with any type of labor, but pushed land-owners to demand for *malaria-resistant* labor. Epidemiological studies shows that malaria resistance is higher in regions historically most exposed to the disease. We would expect thus to see higher prices being paid for slaves born in such regions.

Using historical data from the "Louisiana, Slave Records, 1719-1820" database, I assembled a dataset of prices for over 3000 individuals born in 21 different African countries who experienced 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 a vast set of controls - including proxies for health conditions unrelated to malaria susceptibility, body

[8] 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.

[9] 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.

[10] In fact, at the beginning of the seventeenth century the status of African workers, who were few in numbers, was not disciplined by laws and was not dramatically different from the status of other workers, like Scottish, English or Irish. In the last decades of the seventeenth century the working conditions of African workers drastically changed and the slavery system of the US South took full shape as a condition of permanent inherited bondage.

size, slave traders' production costs, and for agricultural skills of the enslaved individuals - is taken into account.

With this paper, I aim to contribute to the vibrant empirical literature exploring the determinants of slavery and of colonial institutions more generally - such as Acemoglu, Johnson, and Robinson (2001), Bruhn and Gallego (2012), Dell (2012) and Nunn and Puga (2012) among others. It enriches this literature by fshowing how a *changing (epidemiological) environment* brought about institutional change. This represents both a step forward in the identification of the causal effect of the epidemiological environment on historical institutions, and an insightful setting for understanding the emergence of colonial institutions.

The paper also contributes to the literature investigating why African slavery emerged as a dominant labor system in the South of the United States (and not in the North). This literature includes Fogel and Engerman (1974), Goldin and Sokoloff (1981), Fogel (1994), Hanes (1996) and Wright (2003). It does so by directly addressing a longstanding regarding the switch from white European to African slave labor in the South of the United States that took place in the late seventeenth century.

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).[11]

Finally, 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.[12] Through the development and formal testing of a systematic set

[11] 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.

[12] 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).

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).

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 analysis of the introduction of *falciparum* malaria into the US colonies. I turn to slave prices in Section 3.3. The final section concludes.

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.[13] *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.[14] 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.[15] 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.[16] On top of this, the two major strains of malaria require different climatic conditions, with *falciparum* malaria needing

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

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

[15] 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.

[16] 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.

higher temperatures than *vivax* malaria to become infectious.[17]

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).[18] Even though after repeated infections malaria virulence and the mortality risk are reduced, the disease does not stop being a burden. In fact, continual infections deteriorate general health conditions, decreasing the ability to resist other diseases.[19] 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.[20] 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.[21] While resistance to the severe life-threatening consequences of infection is acquired relatively fast

[17] *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.

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

[19] 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.

[20] 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).

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

(Doolan et al., 2009), clinical immunity to milder symptoms is acquired slowly and requires repeated infections (Stevenson and Riley, 2004).[22] 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.[23] In other words, innate resistance to malaria can engender better adaptive responses once an individual faces an episode of infection.

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.[24] 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.[25] On the one hand, diseases transmitted through direct human contact -

[22]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).

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

[24]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.

[25]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).

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);[26] ii) upon arrival in North America, the destination 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.[27] 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.[28] 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

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

[27]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.

[28]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.

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.[29]

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, and 57% before reaching age 5.[30] Unsurprisingly, the great majority of deaths took place in the "ague and fever" months, between August and November (Packard, 2007).[31] 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.[32]

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.[33] 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,[34] 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.[35]

[29]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."

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

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

[32]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.

[33]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.

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

[35]The first ascertained yellow-fever epidemics hit Charleston and Philadelphia simultaneously, causing similar

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).[36]

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.[37]

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.[38]

amounts of damage (Waring, 1975).

[36]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 (Galenson, 1981).

[37]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.

[38]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).

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."[39] 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 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.[40]

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 often 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 Africans were allowed to work independently, could buy and sell their produce, barter their free time for wages, and eventually buy their freedom.[41] 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).

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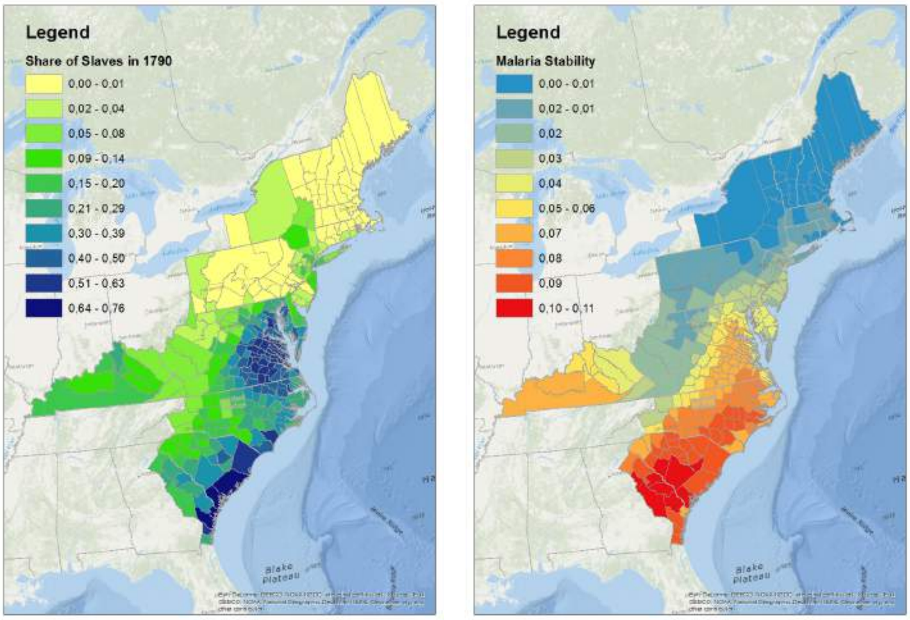
[39] Mallard (1892).

[40] 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.

[41] 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...".

Figure 1: Malaria and Slavery across US Counties



(a) Share of Slaves (b) Malaria Stability

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.[42] As Figure 1 shows, the practice of slavery was largely concentrated in the southern states. The northern states had a very low number or no slaves,[43] 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

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

[43]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.

almost one-to-one correlation between the share of blacks and the share of slaves.[44]

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 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.[45] 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).[46] 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.[47]

[44]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.

[45]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.

[46]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 of my study, it is less suited for my specific application. Note, however, that the index would give virtually identical results.

[47]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).

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.[48] The county-level controls include a full set of soil suitability indexes taken from FAO GAEZ, and namely soil suitability for cotton, tea, tobacco, rice and sugar. 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.[49]

Table 1 reports the main results.[50] 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 stability would predict an increase in the share of slaves of 0.12.[51] Armed with these estimates, we can think of a counterfactual scenario of no malaria transmission, imposing the malaria stability

[48]Specifications without state fixed effects are reported in Appendix A.1, Tables 11 and 12.

[49]I report estimates with cutoff values at 50, 100 and 500 km.

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

[51]Interestingly, a regression including malaria stability alone would give an R-square as high as 0.366 (Appendix A.1 Table 11), while adding state fixed effects increases the R-squared to 0.60. Again, in terms of explained variation, the partial R-square of malaria stability (in the full specification, Column (3)) is 0.39. 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%.

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

	Share of Slaves			**Share of Blacks**		
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	0.164***	0.144***	0.121***	0.164***	0.144***	0.121***
Conley s.e. 50 km	(0.024)	(0.024)	(0.022)	(0.025)	(0.025)	(0.023)
Conley s.e. 100 km	[0.029]	[0.028]	[0.025]	[0.031]	[0.029]	[0.026]
Conley s.e. 500 km	{0.037}	{0.032}	{0.030}	{0.038}	{0.033}	{0.031}
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 $\beta = 0$		2.04	2.12		2.05	2.16
Observations	285	285	285	285	285	285
R-squared	0.60	0.72	0.79	0.60	0.72	0.79

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 a index measuring the force and stability of malaria transmission, in order to have 0 mean and unit standard deviation. 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. 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 50, 100 and 500 km. ***, **, * indicate significance at 1, 5, and 10% levels respectively, computed with the largest of the standard errors reported.

variable to be 0 for all counties in the sample. Figure 4 in Appendix A.1 shows that in the absence of malaria transmission African slavery would had been a decidedly minor phenomena, concentrated in a few coastal counties.

Of all the other explanatory variables, the main other predictor of slave share is the distance of the county from the coast.[52] Regarding crop suitabilities, I find that a higher suitability for sugar and tea increases the share of slaves but no other significantly positive correlation with suitabilities for other crops.[53] 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 control variables are measured with noise or 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

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

[53] 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.

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 2 times as large as the effect of the observed set of controls (Column 3).[54]

Looking at other outcomes, higher malaria incidence predicts also a larger share of white families owning slaves out of all families, 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). Note that the results are identical when using the alternative measure of historical malaria incidence, with the estimated coefficients remarkably similar to the baseline ones (Appendix A.1 Table 11, Panel C). Finally, if we were to look only at "slave states", states where slavery was legally sanctioned in 1790, we would again find the same identical estimated coefficients (Appendix A.1 Table 11, Panel D).

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 looks at a change in malaria intensity, by exploiting as a *quasi-natural* experiment the introduction of *falciparum* malaria, the most virulent and deadly malaria species, into the United States.

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

[54]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 always larger than 1.5, which is reassuring given that Oster (2013) indicates 1 as a reasonable heuristic value.

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.*

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 and the epidemic forms that the infection took at first, coupled with the sudden rise in the mortality rates that followed, are consistent with the traits of *falciparum* malaria.[55]

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 1680s we observe an increase in extreme weather events.[56] Importantly, there is vast anecdotal evidence documenting a sudden deterioration in the health environment of the Southern colonies in the 1680s. Table 2, for instance, reports extracts for South Carolina before and after the *falciparum* epidemic that hit Charleston in 1684.

[55]See, for instance, Wood (1974); Childs (1940) for South Carolina and Rutman and Rutman (1976) for Virginia.

[56]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). Figure 5 plots the full time series of El Nino events. 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, and unusually heavy rains and floods can do the same.

Ideally, the analysis would require information on the specific timing of the introduction of *falciparum* malaria into each US state. However, while historical analysis 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.[57]

To overcome data limitations and the potential source of endogeneity that might drive the actual timing of the introduction in different states, in my baseline analysis 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 to 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 with the states where it could not.

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.[58] To take into account the possible effect of a surge in

[57]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, 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.

[58]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.

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.[59]

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.

As an alternative strategy, in order to get rid of the endogeneity that might drive the timing and location of *falciparum* malaria introduction, I use time and state variation in bio-climatic characteristics to predict *when* and *where falciparum* malaria became endemic. The predictor is constructed on the intuition that weather anomalies created conditions favoring the introduction of *falciparum* malaria, *particularly* in states with a higher bio-climatic potential for malaria transmission. Therefore, as a source of bio-climatic variation I exploit the interaction between a time-series of weather anomalies (common for all states) and the cross-sectional variation in malaria suitability across states.[60] As a second step, I collect all available information on the appearance of *falciparum* malaria for each state and instrument it with my predicted index.

A concern that may arise is the possibility that the *falciparum* malaria epidemics observed were a consequence of the greater inflow of Africans in high malaria states, and not *viceversa*. Note that both the identification strategies are intended to tackle this concern. First, my baseline exercise resembles a reduced form specification in spirit. In other words, the inflow of workers from malaria-infested areas certainly increased the likelihood of epidemics, but the

Source: Colonial and Prefederal Statistics, Chapter Z.

[59] As a robustness exercise, without imposing any structure, I control for a time-varying effect of the average suitability for rice (and of a set of cash crops) of the state on the share of blacks.

[60] More formally, my exogenous predictor be the term $El\ Nino_t * MS_c$, the interaction term between the number of El Nino events in the decade, which varies by decade, and the malaria stability index in the state, which varies across states.

interaction between the post-introduction variable (equal for all states) and the malaria stability index *only* exploits exogenous variation in bio-climatic suitability to malaria - i.e. it is not an actual measure of malaria incidence. Furthermore, in my second strategy I indeed use an actual measure of incidence - indicating where and when *falciparum* malaria was endemic - but I instrument this variable with a predicted measure constructed on the basis of bio-climatic information.[61]

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. As first outcome variable, I look at the average share of blacks in the total population of the state. As a second outcome of interest, I investigate *when* and *in which* US states the southern slave labor system was institutionalized. Until the last decades of the seventeenth century, in fact, Africans in the United States were treated similarly as servants from England, Scotland, Ireland and Native Americans.[62] Then, in the last decades of the seventeenth century "... Negroes did cease to be servants and became slaves": Africans became property of their owners, for life.[63] The stiffening of the legislation governing living and working conditions of Africans culminated into the approval of comprehensive "slave codes" in some of the US states. For my analysis, I employ 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 decade, I observe 12 states from 1640 to 1780.[64] Figure 2 summarizes the evolution of the share

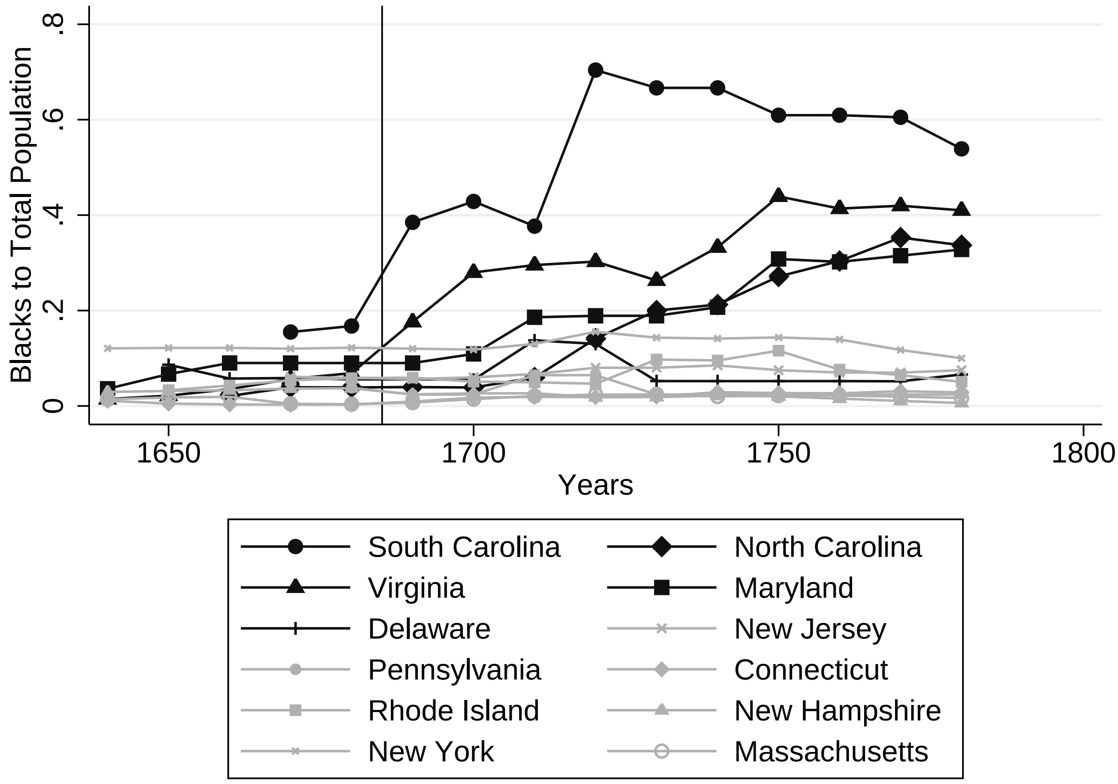
[61] Note, however, that according to my hypothesis there is indeed scope for path-dependence in the mechanism which led to the establishment of African slavery in the southern US. In fact, bringing Africans to states where malaria can take root could have increased the likelihood of acquiring malaria, which then might have further enhanced the need for African workers. Even according to this version of the hypothesis, the primary driver remains malaria.

[62] "The status of Negroes was that of servants" (Oscar and Handlin, 1950).

[63] As Oscar and Handlin (1950) put it "slavery was not there from the start...", and it was in the last decades of the seventeenth century that "Negroes did cease to be servants and became slaves, ceased to be men in whom masters held a proprietary interest and became chattels, objects that were the property of their owners. In that transformation originated the Southern labor system.".

[64] 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.

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.

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.[65] 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 population. 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.[66]

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 the more malaria-suitable states. Turning to a more formal analysis, I propose a set of estimates based on the specification below:

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

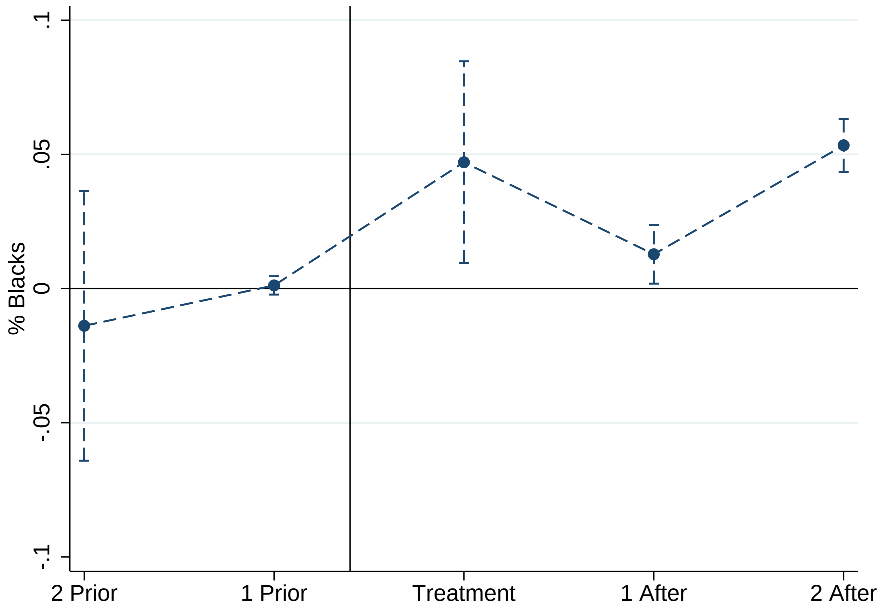
The main interest lies in β, the coefficient of the interaction term between $Post\text{-}1690_t$, an indicator taking value 1 for the decades following 1690 (with 1690 included), and the variable MS_s, which is a continuous index measuring malaria stability in the state standardized in order to have 0 mean and unit standard deviation. 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.[67]

[65] 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.

[66] Note that, given that the treatment is continuous, I cannot provide the standard difference-in-difference graphical representation mapping treated and not treated.

[67] 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

Figure 3: Malaria and Share of Blacks: Lags and Leads



†The graphs plots coefficients of the two leads (2 Prior, 1 Prior) and two lags (1 After, 2 After, 3 After) of the main explanatory variable ($MS_s * Post1690_t$).

Additionally, I look at the likelihood of having a slave code, measured with an indicator variable taking value 1 in each state-decade during which a slave code is in force, and 0 otherwise.[68]

$\mathbf{I_{s,t}}$ 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 and a state-specific effect of English farm wages.[69] Standard errors are 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.[70]

The difference-in-difference framework identification rests on the assumption of identical

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.

[68]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.

[69]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, $temp_s * \mu_t$.

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

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	0.108***	0.096***	0.105***	0.091***	0.159***	0.103***	0.127***
Robust s.e.	(0.011)	(0.012)	(0.011)	(0.013)	(0.023)	(0.011)	(0.023)
Cluster [State] s.e.	[0.022]	[0.026]	[0.021]	[0.024]	[0.043]	[0.020]	[0.041]
Bootstrap s.e. p-value	*0.000*	*0.042*	*0.000*	*0.002*	*0.024*	*0.000*	*0.058*
Rice Suitability x Post-1690		0.002					0.004*
		(0.002)					(0.001)
		[0.003]					[0.002]
Yellow Fever			0.018				0.011
			(0.010)				(0.008)
			[0.011]				[0.011]
England Farm Wage x State fixed effects				Yes			Yes
Temperature x Decade fixed effects					Yes		Yes
Total Population (1000s)						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	166	166	166	166
R-squared	0.875	0.877	0.878	0.918	0.913	0.898	0.954
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, standardized in order to have 0 mean and unit standard deviation. 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. Robust standard errors are reported in round bracket, standard errors clustered at the state level are reported in squared brackets. 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 (related to standard errors clustered at the state level).

counterfactual trends at different treatment intensity. Figure 2 seems to suggest that more and less malaria-suitable states were following similar trends before 1690. Before turning to the results, I provide more formal evidence in favor of the identifying assumptions by including leads and lags in the baseline specification. As we would wish, Figure 3 shows that all leads (the main coefficients in the periods prior to the treatment) are very close to zero.

The main results exploring the share of blacks in the state are summarized in Table 18. The estimated coefficients consistently show that after 1690 more malaria-suitable states have a significantly larger share of blacks in the population compared to less malaria-suitable states. 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.[71] Accounting for post-1690 suitability for rice decreases β mildly - column (2) -

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

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, just as controlling for the population in the state. 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. In additional specifications, reported in Tables 16, I show that the effect of stability is not driven by a spurious correlation with cash crop suitability. First, I interact cash crops suitability with a post-1690 indicator variable, second, 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 Panel B of Table 17, as a robustness exercise, I exclude each of the four southernmost states and show that the results are not driven by any specific southern state.

Table 4 reports Linear Probability Model (LPM) estimations exploring the distribution of "slave codes". In the upper panel the dependent variable, Slave Code in Force, indicates whether a slave code is in force in the state-decade, whereas in the lower panel the variable Slave Code Approval indicates whether the state approved a slave code in the decade. 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.[72] As for robustness checks, Panel A of Table 16 shows that results hold when using the historical malaria endemicity index.

As a second identification strategy, I construct a variable ($Falciparum\ Malaria$) indicating for each state when historical evidence documents *falciparum* malaria's appearance. Since the variable $Falciparum\ Malaria$ is measured with error and might be endogenously driven by spurious factors, I instrument it with a variable aiming at capturing the effect of weather anomalies in more malaria-suitable states. My proxy measure for weather anomalies (common to all states) is the number of El Nino events registered in the decade.[73] The results, summarized in Table 18, show that: i) weather anomalies in the highly malaria-suitable states significantly

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

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

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Slave Code in Force							
Malaria Stability x Post-1690	0.287***	0.381***	0.276***	0.257***	0.295	0.280***	0.283**
Robust s.e.	(0.037)	(0.037)	(0.039)	(0.037)	(0.124)	(0.037)	(0.099)
Cluster [State] s.e.	[0.075]	[0.054]	[0.073]	[0.059]	[0.243]	[0.073]	[0.123]
Bootstrap s.e. p-value	*0.018*	*0.004*	*0.016*	*0.008*	*0.072*	*0.028*	*0.086*
Rice Suitability x Post-1690		Yes					Yes
Yellow Fever			Yes				Yes
England Farm Wage x State fixed effects				Yes			Yes
Temperature x Decade fixed effects					Yes		Yes
Total Population (1000s)						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	166	166	166	166
R-squared	0.756	0.771	0.762	0.836	0.767	0.760	0.859
Number of state	12	12	12	12	12	12	12
Slave Code Approval							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability x Post-1690	0.078***	0.086***	0.077***	0.096***	0.101*	0.080***	0.139*
Robust s.e.	(0.032)	(0.031)	(0.031)	(0.043)	(0.069)	(0.033)	(0.104)
Cluster [State] s.e.	[0.022]	[0.022]	[0.022]	[0.025]	[0.050]	[0.025]	[0.064]
Bootstrap s.e. p-value	*0.002*	*0.004*	*0.002*	*0.006*	*0.030*	*0.002*	*0.022*
Rice Suitability x Post-1690		Yes					Yes
Yellow Fever			Yes				Yes
England Farm Wage x State fixed effects				Yes			Yes
Temperature x Decade fixed effects					Yes		Yes
Total Population (1000s)						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	166	166	166	166
R-squared	0.240	0.240	0.240	0.262	0.285	0.241	0.301
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 of the upper panel, Slave Code in Force, indicates whether a slave code is in force in the state-decade. In the bottom panel, the dependent variable, Slave Code Approval, takes value 1 if the state approved a slave code in the decade and 0 otherwise. 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, standardized in order to have 0 mean and unit standard deviation. 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. Robust standard errors are reported in round bracket, standard errors clustered at the state level are reported in squared brackets. 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 (related to standard errors clustered at the state level).

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 and a higher likelihood of having a slave code in force - 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 from the previous sections shows that malaria was an important determinant for the diffusion of African slavery in the southern United States. This section aims to document that it is precisely the comparative higher malaria-resistance of sub-Saharan Africans that made them especially attractive for employment in these regions. In other words, this section aims to show that slave-owners' labor choices moved from a conscious intent to meet their labor needs with the comparatively most productive pool of laborers.[74]

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. Along similar lines, Kotlikoff (1979) shows that selling prices reflected productivity

[74]In other terms, according to the hypothesis the enslavement of Africans in the US South did not only follow from labor scarcity in the unhealthy malaria-ridden areas but for a specific quest for malaria-resistant labor.

differentials resulting from characteristics such as age, sex and skills.[75]

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.[76] 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 differential 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. On top of this, it is important to keep in mind that malaria tends to weaken the immune system, so that 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.[77] 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 in-

[75]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).

[76]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".

[77]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.

stance, body size was considered a direct consequence of the food availability and the vegetation in the regions of birth.[78] Moreover, Mauro (1964) points out that slaves "did not arrive naked, but brought with them a sense of sedentary life and of agriculture".[79]

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 both their selling price and their place of birth.[80]

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.[81] 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.[82] 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.[83] Narratives in Wax (1973), who studies US slave-owners' preferences by place of origin,

[78] 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).

[79] 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.

[80] 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.

[81] 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.

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

[83] For instance, in the two advertisements reported in the Appendix, the slave traders refer to slaves from

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

	Ln(Slave Price)					
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	0.066***	0.058***	0.078***	0.073***	0.076***	0.082***
Cluster (s.e.)	(0.017)	(0.014)	(0.022)	(0.017)	(0.020)	(0.020)
Bootstrap s.e. p-value	*0.010*	*0.018*	*0.008*	*0.000*	*0.000*	*0.020*
Male Slave	0.194***	0.193***	0.195***	0.193***	0.193***	0.192***
	(0.022)	(0.021)	(0.022)	(0.022)	(0.022)	(0.022)
Slave Age	0.048***	0.048***	0.048***	0.048***	0.048***	0.049***
	(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 Lenght		0.002**				0.002***
		(0.001)				(0.001)
Distance Atlantic Markets			0.034**			-0.017
			(0.013)			(0.096)
Land Suitability				-0.082		-0.136
				(0.070)		(0.148)
Average Rice Suitability					-0.000	-0.000
					(0.000)	(0.000)
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
δ for $\beta = 0$		1.26	3.73	2.68	3.28	5.46
Observations	3186	3186	3186	3186	3186	3186
R-Squared	0.446	0.446	0.446	0.446	0.446	0.447

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 African country of birth of the individual enslaved, standardized to have 0 mean and unit standard deviation. 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 three African macro-regions (Upper Guinea, Bight of Benin and Western 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 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.

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.[84]

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.[85] 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.[86] I further exclude individuals with no age 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.[87] 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

Sierra Leon, Windward Coast and Rice Coast.

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

[85]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.

[86]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.

[87]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.

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 three 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 includes several measures of suitability for agriculture, and in particular for rice cultivation. Additional controls attempt to exclude malaria stability be capturing: (i) across-country/ethnicity differences in body height and size, (ii) heterogeneity in the slave traders' production costs across countries,[88] and (iii) agricultural skills or cultural attitudes towards sedentarianism and complex social organizations prevalent in the African country of origin of the enslaved individual .

Table 5 and 6 report the main results; additional specifications are available in the Appendix (Table 22, 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).[89] 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 major Atlantic slave markets.[90] Both controls have a positive effect on the slave price, possibly reflecting a selection effect in the subset of slaves that survived as

[88]Following Nunn and Puga (2012) and Fenske and Kala (2015), I control for ruggedness and average temperature.

[89]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.

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

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 attempt to control for differences in body size in two ways. First, I assemble the available data on historical body heights for different African countries/ethnic groups. As a second way, I construct a variable indicating whether a famine/drought took place during the first two years of life of the individual slave in his/her African country of birth.[91] My results, reported in Table 6, show that the inclusion of these controls leave the coefficient of interest unaffected. Similarly, results hold when controlling for proxy of historical agricultural skills, historical population density and state antiquity.

I perform several robustness checks. Table 22 report specifications including a vast list of geo-climatic controls. In Table 23, I use an alternative measure of malaria exposure in the country of origin: historical malaria endemicity. Finally, 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 24, No border Groups).[92] 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 24).

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

[91] Details on the variables' construction are available in the Appendix.

[92] 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.

of health hazards.[93] The results, reported in Table 25, 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 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 6 reports a time-varying coefficient of malaria stability on the number of voyages from African countries.[94] 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.[95]

4 Conclusion

In this paper, 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 more malaria-ridden 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,

[93]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.

[94]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.

[95]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).

I have shown that, among slaves transported from Africa, slaves born in African regions with more malaria commanded higher prices.

Table 6: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)										
	Height		Body Size			Production Costs		Agricultural Skills		Human Capital	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
Malaria Stability	0.063***	0.082**	0.071***	0.068***	0.067***	0.067***	0.071***	0.064***	0.081**	0.053**	0.054**
Cluster (s.e.)	(0.016)	(0.028)	(0.012)	(0.010)	(0.012)	(0.002)	(0.000)	(0.004)	(0.044)	(0.010)	(0.020)
Bootstrap s.e. p-value	*0.010*	*0.024*	*0.004*	*0.008*	*0.008*	*0.010*	*0.024*	*0.010*	*0.024*	*0.010*	*0.024*
Slave Height		0.006									
		(0.006)									
Famine in Childhood				-0.037*							
				(0.021)							
Drought in Childhood					-0.041***						
					(0.012)						
Ruggedness						-0.062					
						(0.041)					
Average Temperature							0.015**				
							(0.006)				
Historical Croplands Cover								0.346			
								(0.402)			
Transition to Agriculture									-0.043		
									(0.032)		
Ln(Population in 1400)										-0.012	
										(0.013)	
State Antiquity											-0.066
											(0.055)
Male Slave	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age Squared	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Region fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2872	2576	3170	3170	3170	3186	3186	3186	3186	3186	3186
R-Squared	0.448	0.453	0.447	0.448	0.448	0.446	0.446	0.446	0.446	0.446	0.446

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 African country of birth of the individual enslaved, standardized to have 0 mean and unit standard deviation. 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 three African macro-regions (Upper Guinea, Bight of Benin and Western 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 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.

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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.*

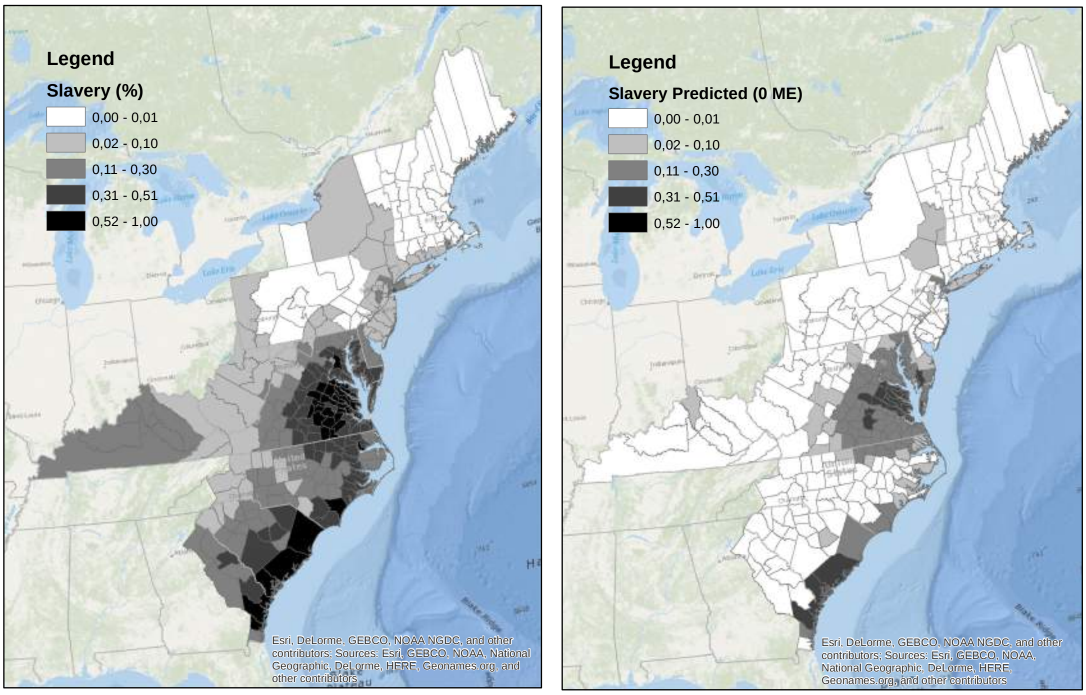
Table 8: Summary Statistics of Cross-County Analysis

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	1	-1.493	2.24	285
Malaria Endemicity	0	1	-2.107	0.97	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) Historical 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 with 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

Figure 4: Malaria and Slavery: Counterfactual Scenario



†The figure on the left map the share of slaves in the population across US counties in 1790. The figure on the right maps the counterfactual shares of slaves in the absence of malaria transmission, based on the the baseline specifications and estimates.

Table 10: Malaria and Slavery: Additional Results

	PANEL A					
	Share of Slaves			**Share of Blacks**		
Malaria Stability	0.120	0.090	0.089	0.123	0.088	0.087
Conley s.e. 50 km	(0.022)***	(0.025)***	(0.025)***	(0.023)***	(0.025)***	(0.025)***
Conley s.e. 100 km	[0.025]***	[0.027]**	[0.027]**	[0.026]***	[0.027]**	[0.027]**
Conley s.e. 500 km	{0.040}**	{0.042}**	{0.044}**	{0.040}***	{0.042}**	{0.043}**
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	0.60	0.80	0.81	0.80	0.81	0.82

	PANEL B					
	Share of Families with Slaves			**Number of Blacks**		
Malaria Stability	0.129	0.126	0.085	2,109	1,957	1,624
Conley s.e. 50 km	(0.024)***	(0.026)***	(0.024)***	(405)***	(485)***	(419)***
Conley s.e. 100 km	[0.026]***	[0.029]***	[0.028]***	[447]***	[535]***	[463]***
Conley s.e. 500 km	{0.034}***	{0.041}***	{0.029}***	{409}***	{375}***	{428}***
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	0.65	0.70	0.78	0.24	0.33	0.34

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, it was standardized in order to have 0 mean and unit standard deviation. 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 50, 100 and 500 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	0.124	0.164	0.197	0.157	0.166	0.153	0.170	0.144
Conley s.e. 50 km	(0.010)***	(0.024)***	(0.027)***	(0.023)***	(0.024)***	(0.022)***	(0.023)***	(0.024)***
Conley s.e. 100 km	[0.016]***	[0.029]***	[0.033]***	[0.029]***	[0.030]***	[0.027]***	[0.029]***	[0.028]***
Conley s.e. 500 km	{0.030}***	{0.046}***	{0.049}***	{0.048}***	{0.047}***	{0.036}***	{0.046}***	{0.037}***
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	0.648	0.609	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	0.106	0.153	0.151	0.121			0.165	0.120
Conley s.e. 50 km	(0.020)***	(0.022)***	(0.028)***	(0.022)***			(0.024)***	(0.023)***
Conley s.e. 100 km	[0.023]***	[0.025]***	[0.033]***	[0.025]***			[0.030]***	[0.026]***
Conley s.e. 500 km	{0.036}***	{0.034}***	{0.046}***	{0.040}***			{0.041}***	{0.046}***
Malaria Endemicity					0.123	0.048		
Conley s.e. 50 km					(0.016)***	(0.014)***		
Conley s.e. 100 km					[0.022]***	[0.017]***		
Conley s.e. 500 km					{0.020}***	{0.010}***		
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	0.79	0.73	0.72	0.79	0.62	0.77	0.51	0.75

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, standardized in order to have 0 mean and unit standard deviation. In Panel C the explanatory variable is Malaria Endemicity, an index measuring the malaria parasitization rate at the beginning of the 20th century, standardized in order to have 0 mean and unit standard deviation. 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 50, 100 and 500 km. ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 12: Malaria and Slavery across US Counties

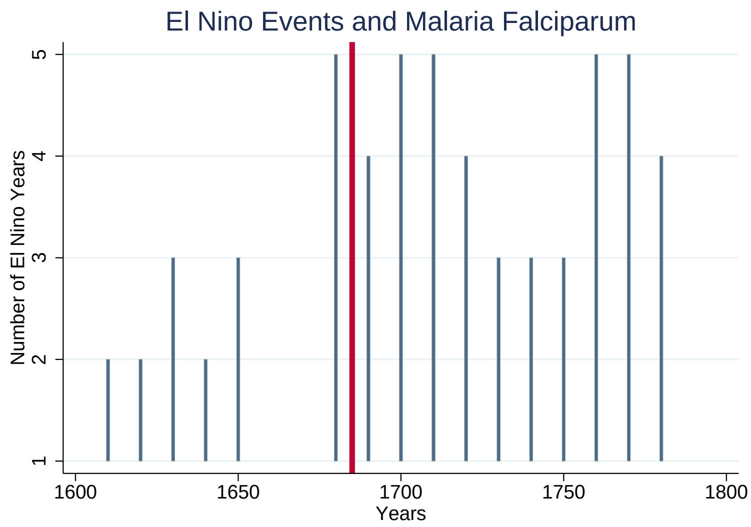
	Dependent Variable: Share of Blacks 1790							
	PANEL A Malaria Stability - Full Sample							
Malaria Stability	0.123	0.171	0.206	0.165	0.174	0.160	0.179	0.151
Conley s.e. 50 km	(0.011)***	(0.025)***	(0.029)***	(0.024)***	(0.025)***	(0.024)***	(0.024)***	(0.025)***
Conley s.e. 100 km	[0.016]***	[0.031]***	[0.035]***	[0.031]***	[0.031]***	[0.028]***	[0.030]***	[0.029]***
Conley s.e. 500 km	{0.032}***	{0.047}***	{0.050}***	{0.049}***	{0.048}***	{0.037}***	{0.048}***	{0.038}***
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		0.640	0.601	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	0.110	0.161	0.157	0.124			0.172	0.122
Conley s.e. 50 km	(0.021)***	(0.023)***	(0.029)***	(0.023)***			(0.025)***	(0.024)***
Conley s.e. 100 km	[0.024]***	[0.027]***	[0.034]***	[0.026]***			[0.031]***	[0.027]***
Conley s.e. 500 km	{0.036}***	{0.035}***	{0.047}***	{0.040}***			{0.047}***	{0.041}***
Malaria Endemicity					0.129	0.045		
Conley s.e. 50 km					(0.018)***	(0.015)***		
Conley s.e. 100 km					[0.024]***	[0.017]***		
Conley s.e. 500 km					{0.023}***	{0.011}***		
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, standardized in order to have 0 mean and unit standard deviation. In Panel C the explanatory variable is Malaria Endemicity, an index measuring the malaria parasitization rate at the beginning of the 20th century, standardized in order to have 0 mean and unit standard deviation. 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 50, 100 and 500 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 dis-aggregated 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: Historical El Nino Events. Retrived from: *https://sites.google.com/site/medievalwarmperiod/Home/historic-el-nino-events*.

Figure 5: Weather Anomalies: El Nino Events



†The graph plots the time series of El Nino events in the seventeenth and eighteenth century. The grey bars measures the numbers of El Nino years registered in the decade. The red line plots the year 1684, year of the major Charleston epidemic.

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	0.029	0.872	-0.959	2.398	166
Hist. Malaria Endemicity x Post 1690	0.018	0.868	-1.065	2.044	166
Falciparum Malaria	0.241	0.429	0	1	166
Rice Suitability x Post 1690	5.706	8.076	0	25.466	166
Yellow Fever	0.187	0.589	0	4	166
Farm Wage in England	4.762	0.409	4.275	5.82	166
Total Population (thousands)	64.317	87.747	0.185	538.004	166
Slave Code Approval	0.349	0.478	0	1	166
Slave Code Approved	0.072	0.26	0	1	166
# El Nino Events	3.807	1.405	1	6	166
Malaria Stability x # El Nino Events	0.061	4.128	-5.752	14.39	166
Time-Invariant					
Malaria Stability x # El Nino Events	0.061	4.128	-5.752	14.39	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	0	1	-1.065	2.044	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)	(13)	(14)	(15)
Blacks to Total Population	1.000														
Black People	0.628	1.000													
White People	0.129	0.539	1.000												
Post 1690	0.276	0.236	0.402	1.000											
Malaria Stability x Post 1690	0.766	0.358	-0.144	0.021	1.000										
Hist. Malaria Endemicity x Post 1690	0.721	0.345	-0.099	0.013	0.926	1.000									
Falciparum Malaria	0.783	0.570	0.171	0.349	0.768	0.846	1.000								
Rice Suitability x Post 1690	0.625	0.297	0.040	0.439	0.600	0.487	0.589	1.000							
Yellow Fever	0.355	0.089	0.014	0.197	0.224	0.167	0.181	0.226	1.000						
Farm Wage in England	0.184	0.444	0.704	0.309	0.006	0.004	0.108	0.136	-0.025	1.000					
Slave Code Approval	0.602	0.473	0.365	0.454	0.478	0.444	0.533	0.302	0.348	0.407	1.000				
Slave Code Approved	0.237	0.069	-0.035	0.173	0.229	0.203	0.278	0.175	0.109	-0.105	0.381	1.000			
# El Nino Events	0.152	0.162	0.267	0.568	0.012	0.007	0.198	0.249	-0.044	0.195	0.200	0.005	1.000		
Malaria Stability x # El Nino Events	0.716	0.340	-0.124	0.044	0.909	0.842	0.707	0.557	0.182	0.016	0.443	0.176	0.034	1.000	
Total Population (thousands)	0.316	0.760	0.957	0.391	0.012	0.043	0.328	0.133	0.042	0.696	0.444	-0.003	0.262	0.021	1.000

Table 16: Malaria and Share of Blacks across US States

	Share of Blacks									
Malaria Stability x Post-1690	0.096***	0.097***	0.163***	0.157***	0.099***	0.099***	0.117**	0.125**	0.095**	0.098*
Robust s.e.	(0.012)	(0.013)	(0.023)	(0.020)	(0.016)	(0.017)	(0.026)	(0.025)	(0.017)	(0.019)
Cluster (State) s.e.	[0.026]	[0.028]	[0.029]	[0.029]	[0.028]	[0.029]	[0.042]	[0.042]	[0.043]	[0.047]
Bootstrap s.e. p-value	*0.042*	*0.046*	*0.022*	*0.020*	*0.096*	*0.022*	*0.060*	*0.058*	*0.034*	*0.056*
Rice Suit x Post-1690	0.002									
	(0.001)									
	[0.002]									
Tea Suit x Post-1690			-0.004**							
			(0.002)							
			[0.002]							
Tobacco Suit x Post-1690					0.001					
					0.001					
					[0.002]					
Cotton Suit x Post-1690							-0.001			
							(0.002)			
							[0.003]			
Sugar Suit x Post-1690									0.008	
									(0.006)	
									[0.014]	
Rice Suit x Decade fe	No	Yes	No	No	No	No	No	No	No	No
Tea Suit x Decade fe	No	No	No	Yes	No	No	No	No	No	No
Tobacco Suit x Decade fe	No	No	No	No	No	Yes	No	No	No	No
Cotton Suit x Decade fe	No	No	No	No	No	No	No	Yes	No	No
Sugar Suit x Decade fe	No	No	No	No	No	No	No	No	No	Yes
Decade fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	166	166	166	166	166	166	166	166	166	166
R-squared	0.877	0.904	0.880	0.923	0.876	0.891	0.875	0.910	0.877	0.907
Number of state	12	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. Malaria Stability is an index measuring the force and stability of malaria transmission, standardized in order to have 0 mean and unitary standard deviation. 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. Controls variable x Year FE are cross-sectional variables interacted with a full set of decade fixed effects. Robust standard errors are reported in round bracket, standard errors clustered at the state level are reported in squared brackets. I also 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 (related to standard errors clustered at the state level).

Table 17: Malaria and Share Blacks across US States

	PANEL A **Malaria Endemicity**				PANEL B **Different Samples**				
Dependent Variable:	% **Blacks**	**Slave Code in Force**	**Slave Code Approval**		% **Blacks**				
Sample:	Full Sample				Only 1680-1690	No S.Carolina	No N.Carolina	No Virginia	No Maryland
Malaria Endemicity x Post-1690	0.097***	0.264***	0.064**						
Robust s.e.	(0.013)	(0.034)	(0.028)						
Cluster (State) s.e.	[0.024]	[0.065]	[0.019]						
Bootstrap s.e. p-value	*0.000*	*0.014*	*0.004*						
Malaria Stability x Post-1690					0.047**	0.093***	0.094***	0.120***	0.107***
Robust s.e.					(0.021)	(0.014)	(0.014)	(0.011)	(0.012)
Cluster (State) s.e.					[0.030]	[0.023]	[0.034]	[0.024]	[0.025]
Bootstrap s.e. p-value					*0.188*	*0.008*	*0.046*	*0.014*	*0.010*
Decade fixed effects	Yes	Yes	Yes		Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes		Yes	Yes	Yes	Yes	Yes
Observations	166	166	166		166	166	166	166	166
R-squared	0.862	0.749		0.236	0.922	0.887	0.762	0.898	0.879
Number of state	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. In Column (1)-(3) and (5)-(8), the panel includes all decades from 1640 to 1780, in Column (4) only 1680 and 1690. In Column (1) and (4) to (8) the dependent variable is the share of black people in the state, in Column (2), Slave Code in Force, indicates whether a slave code is in force in the state-decade, while in Column (3), Slave Code Approval, takes value 1 if the state approved a slave code in the decade and 0 otherwise. Malaria Stability is an index measuring the force and stability of malaria transmission, standardized in order to have 0 mean and unitary standard deviation. Historical Malaria Endemicity is an index measuring the malaria parasitization rate at the beginning of the 20th century, standardized in order to have 0 mean and unit standard deviation. 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. Robust standard errors are reported in round bracket. I also 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 18: Malaria and Share of Blacks - Instrumental Variable Estimates

	Falciparum Malaria	**Share of Blacks**			**Slave Code in Force**		
	First Stage	Reduced Form	OLS	IV	Reduced Form	OLS	IV
Falciparum Malaria			0.228***	0.517***		0.509***	0.920***
			(0.025)	(0.090)		(0.075)	(0.098)
Malaria Stability * # El Nino Events	0.065***	0.015**			0.044***		
	(0.012)	(0.006)			(0.014)		
Decade fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
F Test Excluded Instrument	12.48						
Observations	166	166	166	166	166	166	166
R-squared	0.809	0.805	0.888	0.662	0.711	0.744	0.709
Number of state	12	12	12	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. In Column (1), the dependent variable, *Falciparum* Malaria, is an indicator variable taking value 1 if in the state-decade there is historical evidence of *falciparum* malaria. In Column (2) to (4) the dependent variable is the share of black people in the state, in (5) to (7) the dependent variable, Slave Code in Force, indicates whether a slave code is in force in the state-decade. Malaria Stability is an index measuring the force and stability of malaria transmission, standardized in order to have 0 mean and unit standard deviation. # 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. Robust standard errors at the state level are reported in parenthesis. ***, **, * indicate significance at the 1, 5, and 10% levels respectively (related to standard errors clustered at the state level).

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.

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

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).

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.

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

Slave Height
Average height of African slaves from the area. Based on average country/ethnic group/region historical data on heights. Source: Eltis 1982.

Famine in Childhood
Indicator variable taking value 1 if a famine was registered in the two first years of life of the individual in his/her African country of birth. Source: constrcuted using historical weather information from Nicholson 2001.

Drought in Childhood
Indicator variable taking value 1 if a drought was registered in the two first years of life of the individual in his/her African country of birth. Source: constrcuted using historical weather information from Nicholson 2001.

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, from FAO/IIASA, 2011-2012. Global Agro-ecological Zones (GAEZ v3.0). FAO Rome, Italy and IIASA, Laxenburg, Austria.

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

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

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 crop cover data from 1700 by Hall (2006) and Ramankutty (1999).

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).

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.

Sickle Cell
Average predicted frequency of sickle haemoglobin alleles in the country. Source: Piel et al. 2013.

G6PD
Average predicted allele frequency for G6PD deficiency in the country. Source: Howes et al. 2012.

Mean Elevation
Average country elevation. Source: mean elevation is constructed as the country average of elevation across grids 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 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 with data from New (1999) accessed through Atlas of Biosphere.

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). 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.

Table 20: 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
Document Year	1802.053	11.846	1741	1820	3186
Malaria Stability	0	1	-3.579	3.288	3186
Voyage Lenght	63.248	13.684	43.235	134.444	3186
Distance Atlantic Markets	4.903	0.904	3.705	10.595	3186
Land Suitability	0.363	0.135	0.111	0.635	3186
Average Rice Suitability	2067.258	725.617	283.201	3247.568	3186
Slave Height	161.456	4.772	157.2	171.8	2576
Famine in Childhood	0.246	0.431	0	1	3170
Drought in Childhood	0.257	0.437	0	1	3170
Average Temperature	25.933	1.486	21.01	28.718	3186
Ruggedness	0.293	0.197	0.141	1.194	3186
Historical Croplands Cover	0.034	0.025	0.003	0.098	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
Malaria Endemicity	0	1	-3.961	1.684	3186
Sickle-Cell	0	1	-3.602	1.529	3186
G6PD	0	1	-2.5	2.277	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

Table 21: 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)	(24)	(25)	(26)
(1) Male Slave	1.000																									
(2) Slave Age	0.092	1.000																								
(3) Slave Age Squared	0.067	0.972	1.000																							
(4) Document Year	-0.003	0.071	0.068	1.000																						
(5) Malaria Stability	-0.055	0.114	0.108	-0.142	1.000																					
(6) Voyage Lenght	-0.029	0.011	0.004	0.097	0.154	1.000																				
(7) Distance Atlantic Markets	-0.015	-0.066	-0.070	0.175	-0.407	0.619	1.000																			
(8) Land Suitability	-0.051	0.110	0.103	-0.150	0.631	0.120	-0.252	1.000																		
(9) Average Rice Suitability	-0.074	0.009	0.004	0.016	0.449	0.571	0.218	0.694	1.000																	
(10) Slave Height	0.051	0.240	0.216	-0.243	0.343	-0.517	-0.504	0.377	-0.279	1.000																
(11) Famine in Childhood	-0.049	0.016	0.017	0.012	-0.137	0.002	0.065	-0.113	-0.076	-0.045	1.000															
(12) Drought in Childhood	-0.008	-0.140	-0.117	0.258	-0.383	0.186	0.396	-0.302	-0.020	-0.372	0.346	1.000														
(13) Average Temperature	0.024	0.158	0.150	-0.167	0.607	-0.211	-0.538	0.031	-0.348	0.758	-0.071	-0.395	1.000													
(14) Ruggedness	-0.015	0.083	0.080	-0.167	0.250	-0.434	-0.434	0.788	0.255	0.465	-0.084	-0.313	-0.028	1.000												
(15) Historical Croplands Cover	-0.004	0.058	0.064	-0.131	0.524	-0.386	-0.450	0.140	-0.320	0.580	-0.108	-0.378	0.707	0.237	1.000											
(16) Transition to Agriculture	-0.014	-0.025	-0.018	-0.043	0.389	-0.121	-0.631	0.201	0.290	-0.129	-0.050	-0.180	0.122	0.080	-0.021	1.000										
(17) Ln(Population in 1400)	-0.005	0.162	0.146	-0.082	0.363	-0.137	-0.021	0.205	-0.117	0.606	-0.007	-0.132	0.498	0.168	0.321	-0.365	1.000									
(18) State Antiquity	0.024	0.114	0.107	-0.078	0.202	-0.336	-0.150	-0.173	-0.548	0.580	0.023	-0.111	0.642	-0.075	0.485	-0.320	0.843	1.000								
(19) Malaria Endemicity	-0.075	-0.092	-0.083	0.085	0.337	0.582	0.323	0.348	0.663	-0.689	-0.048	0.114	-0.375	-0.068	-0.039	0.163	-0.259	-0.396	1.000							
(20) Sickle-Cell	-0.063	-0.092	-0.084	0.048	0.035	-0.024	-0.087	0.369	0.388	-0.369	0.014	0.139	-0.539	0.314	-0.269	0.213	-0.136	-0.323	0.470	1.00						
(21) G6PD	-0.055	0.033	0.031	-0.011	0.560	0.675	0.368	0.445	0.627	-0.238	-0.086	-0.065	0.049	-0.046	0.159	-0.030	0.092	-0.074	0.736	-0.086	1.000					
(22) Mean Elevation	0.005	-0.018	-0.024	0.062	-0.554	0.103	0.363	0.135	0.249	-0.179	0.088	0.290	-0.758	0.251	-0.784	-0.192	-0.079	-0.333	-0.017	0.402	-0.207	1.000				
(23) Average Precipitation	-0.054	-0.109	-0.102	0.092	-0.069	0.004	-0.016	0.374	0.560	-0.583	-0.014	0.110	-0.666	0.367	-0.365	0.293	-0.385	-0.666	0.383	0.795	-0.132	0.416	1.000			
(24) Average Relative Humidity	-0.049	-0.181	-0.171	0.207	-0.334	0.436	0.552	-0.046	0.528	-0.927	0.040	0.380	-0.881	-0.211	-0.608	0.069	-0.554	-0.701	0.635	0.550	0.179	0.450	0.709	1000		
(25) Tropical Land	-0.068	-0.096	-0.092	0.107	0.037	0.476	0.348	0.434	0.857	-0.692	-0.012	0.195	-0.743	0.140	-0.581	0.259	-0.385	-0.711	0.690	0.632	0.367	0.496	0.784	0.854	1.000	
(26) TseTse Fly Suitability	-0.052	-0.119	-0.111	0.087	-0.006	0.287	0.082	0.331	0.738	-0.661	-0.017	0.136	-0.687	0.140	-0.557	0.546	-0.611	-0.826	0.566	0.577	0.182	0.415	0.793	0.796	0.919	1.000

Table 22: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)					
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	0.080***	0.097***	0.062***	0.088***	0.088***	0.129**
Cluster (s.e.)	(0.016)	(0.023)	(0.020)	(0.019)	(0.022)	(0.062)
Male Slave	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age Squared	Yes	Yes	Yes	Yes	Yes	Yes
Average Precipitation	-0.000					0.000
	(0.000)					(0.001)
Average Relative Humidity		-0.003				-0.003
		(0.002)				(0.008)
Mean Elevation			-0.000			0.000
			(0.000)			(0.000)
Tropical Land				-0.001*		-0.001
				(0.000)		(0.004)
TseTse Fly Suitability					-0.085	0.021
					(0.050)	(0.197)
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	0.446	0.446	0.446	0.446	0.446

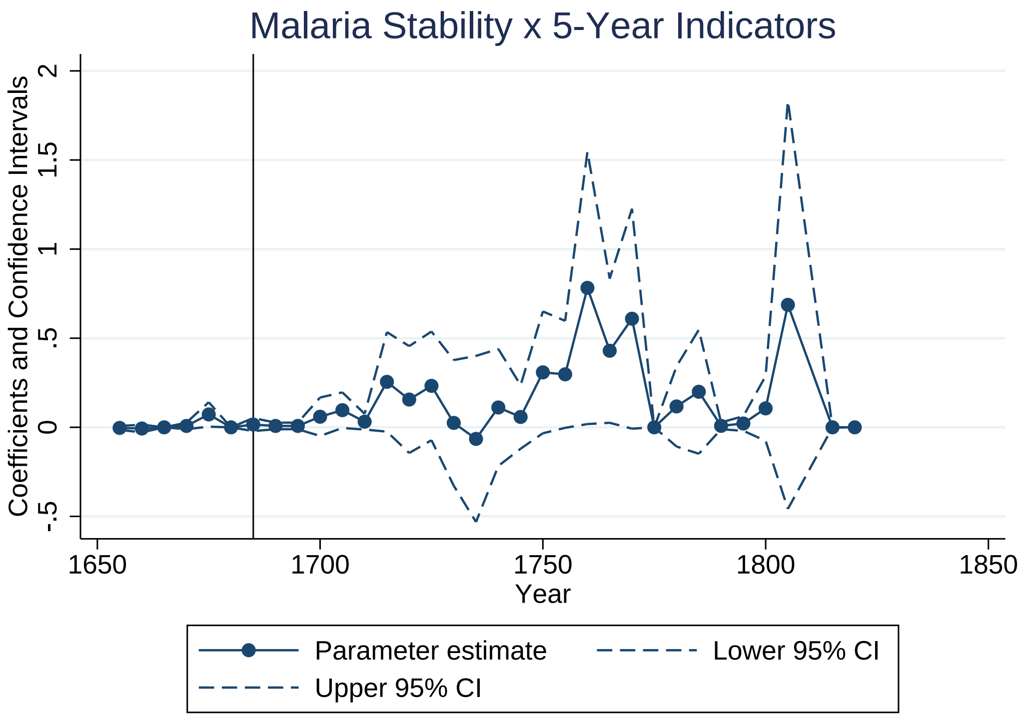
Notes: The table reports OLS estimates. The unit of observation is the individual slave transaction. The dependent variable is the natural logarithm of the selling price of the slave. Malaria Stability is an index measuring the force and stability of malaria transmission in the African country of birth of the individual enslaved, standardized to have 0 mean and unit standard deviation. 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 three African macro-regions (Upper Guinea, Bight of Benin and Western 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). ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

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

	Ln(Slave Price)					
	(1)	(2)	(3)	(4)	(5)	(6)
Historical Malaria Endemicity	0.022***	0.015**	0.024***	0.029***	0.019*	0.025***
Cluster (s.e.)	(0.007)	(0.005)	(0.007)	(0.005)	(0.010)	(0.006)
Male Slave	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age Squared	Yes	Yes	Yes	Yes	Yes	Yes
Voyage Lenght		Yes				Yes
Distance Atlantic Markets			Yes			Yes
Land Suitability				Yes		Yes
Average Rice Suitability					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
Observations	3186	3186	3186	3186	3186	3186
R-Squared	0.444	0.445	0.444	0.444	0.444	0.446

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. Historical Malaria Endemicity is an index measuring the malaria parasitization rate at the beginning of the 20th century, standardized in order to have 0 mean and unit standard deviation. 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 three African macro-regions (Upper Guinea, Bight of Benin and Western 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). ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Figure 6: 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 24: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)						**Slave Price**	
	No Border Groups		**No "Congo"**		**No "Guinea"**		**Full Sample**	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Malaria Stability	0.075***	0.074***	0.072***	0.082***	0.069***	0.082***	36.746***	40.732***
Cluster s.e.	(0.018)	(0.019)	(0.021)	(0.021)	(0.015)	(0.023)	(8.840)	(6.777)
Male Slave	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Slave Age Squared	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Region fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Language fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Document Type fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	1616	1616	1988	1988	2832	2832	3186	3186
R-Squared	0.449	0.451	0.443	0.445	0.450	0.451	0.363	0.365

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 Column (1) to (6), and the absolute value of the sale price in Column (7) to (8). Malaria Stability is an index measuring the force and stability of malaria transmission in the African country of birth of the individual enslaved, standardized to have 0 mean and unit standard deviation. 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 three African macro-regions (Upper Guinea, Bight of Benin and Western 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). ***, **, * indicate significance at the 1, 5, and 10% levels respectively.

Table 25: 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.

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

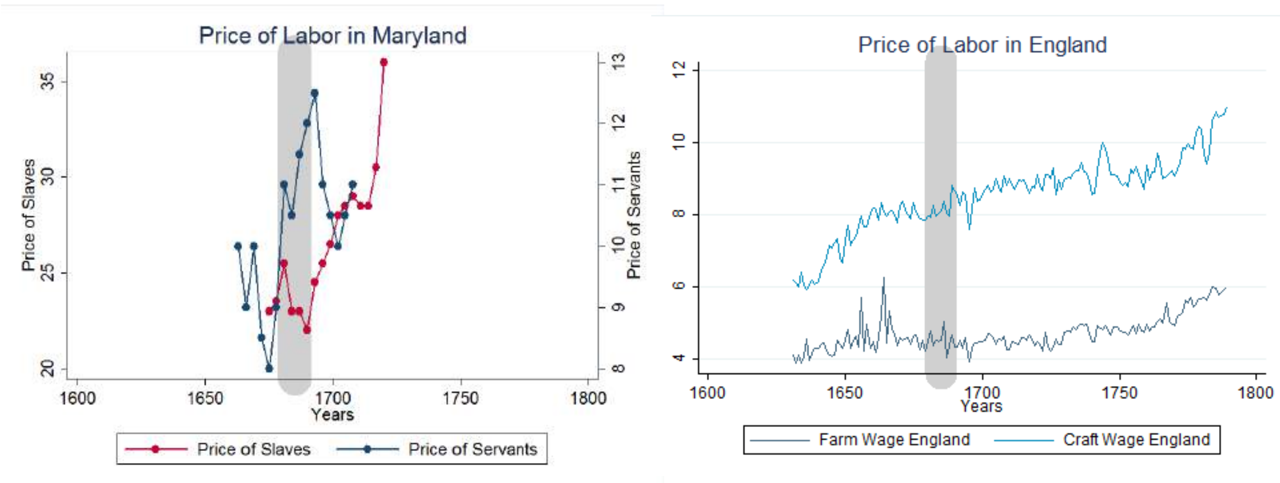
Table 27: Common Polymorphisms That Affect Resistance to Malaria

	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 to 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 7 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 7.

Figure 7: 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 8: Map of Africa 1808

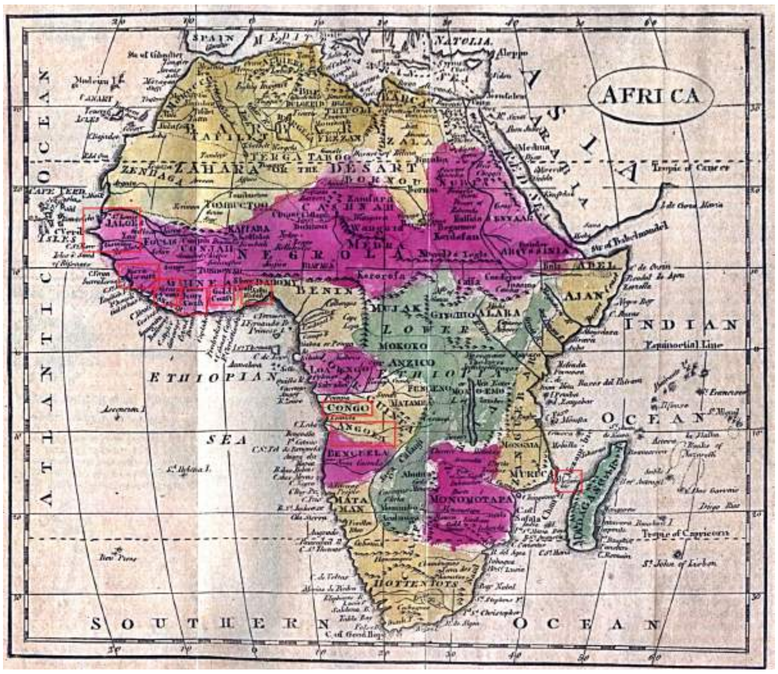


Figure 9: Map of Africa 1829

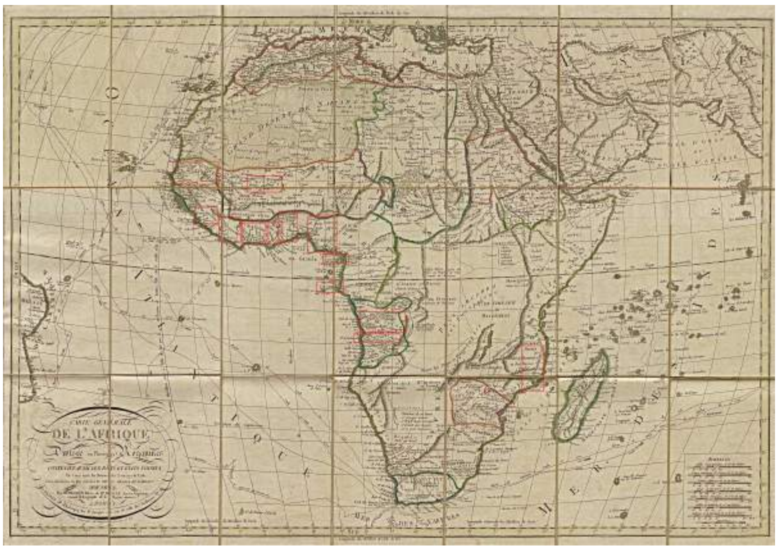


Figure 10: Advertisements