The Side Effects of Immunity: Malaria and African Slavery in the United States

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Abstract

This paper documents the role of malaria in the diffusion of African slavery in the United States. The novel empirical evidence reveals that the introduction of malaria triggered a demand for malaria-resistant labor, which led to a massive expansion of African enslaved workers in the more malaria-infested areas. Further results document that, among African slaves, more malaria-resistant individuals, i.e. those born in the most malaria-ridden regions of Africa, commanded significantly higher prices.

Keywords: Slavery, Malaria, African Slave Trade, Colonial Institutions.
JEL Codes: I12, N31, N37, N57, J15, J47.

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1 Introduction

Why did African slavery spread in the Southern United States? Given the relevance of African slavery to some of the most persistent and extreme cleavages in the country, a great effort has been dedicated to identifying its drivers. The most influential explanations point to the role of the crops grown in the South, with their increasing returns to scale in production and their harsh labor practices (see Fogel and Engerman, 1974; and Sokoloff and Engerman, 2000; among others). However, Southern US did not just grow different crops, it was also burdened by a very specific disease environment. Due to the complexity of teasing apart the geography of crops from the geography of diseases, in addition to the many other concurring cultural and institutional drivers, the role of diseases on the spread of African slavery remains a debate.

The present paper explores the diffusion of African slavery in the United States and singles out the role that malaria played in its spread. Wherever malaria flourished in the United States, a high demand for malaria-resistant labor emerged. In such areas, malaria-resistance of Sub-Saharan Africans increased the profitability of African labor. The new empirical evidence we provide reveals that African slavery was predominantly widespread in more malaria-infested areas of the United States. Going back in time to the period surrounding the introduction of malaria to the United States, we show that the presence of African workers soared after the introduction of a deadly malaria species. By looking at the historical prices of African slaves in the United States, we find evidence of a malaria premium granted by resistance to the disease. In fact, we show that on Louisiana plantations, more malaria-resistant individuals - those born in regions of Africa with a higher prevalence of malaria - commanded significantly higher prices.

Malaria was absent from North America before European settlement and was introduced into the United States during colonization.[1] Importantly, malaria – which requires specific bio-climatic conditions for transmission – did not spread throughout North America but became endemic only in regions warm and humid enough for it to thrive. In areas where malaria began to spread, European workers' health and productivity were greatly affected by the disease, which rendered them "useless for almost one half of the year" (Wood, 2007) and pushed planters to search for more malaria-resistant workers.[2] Land-owners made several attempts to enslave Native Americans, but finally gave up due to their high susceptibility to malaria and other diseases.[3] As a consequence of longer historical exposure

[1] Two major strains of the disease were introduced. *Vivax* malaria – a milder form of the disease – was introduced as early as the first decades of the seventeenth century. On the contrary, the introduction of *falciparum* malaria – the most virulent species – took place in the 1680s. Sections 2.2 and 3.2 details the main features of the diffusion of the disease in the colonies.

[2] Another planter noted that "... a white servant cost three times what he could produce" (Kenzer, 1998).

[3] Native Americans were not only highly susceptible to malaria but also to the full set of Old World diseases, such as

to malaria, certain African populations had developed innate resistances to malaria, granting them protection from the disease.[4]

Malaria incidence influenced settlers in malaria-infested areas to opt for African labor, and in particular for labor of individuals from more malarial regions of Africa. Malaria geography, therefore, helps to explain why African slavery spread more in certain areas of the United States than in others, and why Africans - and particularly Africans from certain regions - were employed in these lands in such large numbers. Note, however, that the higher demand for malaria-resistant labor did not necessarily have to be met through a system of labor coercion, and its moral and political viability lies outside the scope of this work.

The first piece of empirical evidence we provide 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, we employ indexes that predict malaria transmission intensity *only* on the basis of bio-climatic characteristics.[5] The results show a strong positive cross-county correlation between malaria incidence and the share of African slaves.[6] The size of the coefficient indicates that a one standard deviation increase in malaria suitability was related to almost a doubling of the mean county share. Since malaria represented a constraint to agricultural productivity, the increase in the share of African slaves was particularly pronounced in fertile areas, in areas producing cotton and other labor-intensive crops, as well as in areas specializing in wheat production.

We further show that more malaria-infested areas did not just have a sizably larger share of the working population composed of African slaves, but were also the ones that developed stronger pro-slavery views. We explore delegate votes at the Constitutional convention of 1878 and find that delegates born in counties with higher risk of malaria were more likely to be pro-slavery. Moreover, counties with higher Malaria Stability were less likely to vote for politicians that were against slavery and supported Black enfranchisement before the Civil War and in the early Reconstruction Era.[7]

To better identify the role played by malaria, we go back in time to the period surrounding the introduction of malaria to the United States. In particular, we look at a *major* and *rapid* intensification

smallpox, measles etc.

[4]Even if innate immunities do not grant full resistance, so that Sub-Saharan Africans also suffered from malaria spells, extensive evidence shows their comparatively better resistance to the most lethal symptoms of the disease. Section 2.1 reviews epidemiological evidence about malaria (symptoms and transmission) and the immunities to the disease.

[5]As baseline measure, we employ the Malaria Stability index devised by Kiszewski et al. (2004), which predicts malaria transmission intensity using information on long-run temperature, precipitation, and mosquitoes' characteristics. We next verify results with all major malaria indexes used in the literature.

[6]Interestingly, we discovered that similar cross-sectional correlations between malaria suitability and African slavery exists when looking across New World regions in the late nineteenth century. This additional evidence suggests that malaria may have played a similar role in the rest of the Americas.

[7]Specifically, we present results looking at votes for Lincoln in 1860 Presidential Election and for Grant in 1868 Presidential Elections.

of malaria incidence that followed the introduction of *falciparum* malaria, the most deadly and virulent of the malaria species.[8] The available evidence indicates that *falciparum* malaria entered North America in the 1680s, supported by weather anomalies that characterized the decade. As the actual timing of the introduction of *falciparum* malaria to each American state could be partly related to endogenous factors, as a baseline strategy we use the decade of the main ascertained *falciparum* epidemics as the threshold for malaria introduction for all states. We then exploit cross-state variation in malaria suitability as a proxy for the likelihood of *falciparum* malaria striking and becoming endemic in each state. This difference-in-difference exercise allows us to examine the increase in the share of African slaves that followed the introduction of *falciparum* malaria, comparing the states that were more suitable for malaria with states that were less suitable.

The main threat to identification lies in the possible existence of other shocks, generating an increase in the share of African slaves, that occurred around the same decades as the introduction of *falciparum* malaria and that could be correlated with our measures of malaria exposure. We address this concern by accounting for several alternative explanations for the switch to African slave labor.[9] To further rule out that the greater inflow of Africans was the driver of the spread of malaria and not vice versa, we exploit exogenous variation generated by weather anomalies and bio-climatic characteristics to predict both *where* and *when* malaria was more likely to take root. We then use this predicted measure in an instrumental variable framework. According to the coefficient size, the effect of the introduction of *falciparum* malaria was sizable, accounting, for instance, for around 40 percent of the difference in the share of African slaves that emerged after 1690 between Pennsylvania and South Carolina, and about a fifth of the difference that emerged between Pennsylvania and Virginia.

Last, we shed light on the underlying economic motives behind the previously discussed macro phenomena. On the one hand, malaria made free labor scarcer, engendering a Domar-type effect (Domar, 1970). On the other hand, the disease made malaria-resistant workers comparatively more productive, tilting the labor demands of slave owners toward African labor, and in particular, toward Africans from more malaria-ridden regions. Epidemiological studies show that malaria resistance is higher in regions historically most exposed to the disease.[10] As long as malaria resistance provides productivity advantages, we expect to see higher prices being paid for slaves born in those regions.

[8] *Vivax* malaria certainly also shifted the slave-owners' preferences towards African labor. Indeed, the county-level results account for the overall effect of all malaria strains. For identification, we focus on the introduction of *falciparum* malaria because its introduction can be dated and because, given its fatality rate, as attested by various sources reviewed below, its introduction engendered a major discontinuity in the threat represented by malaria.

[9] Among the several alternative explanations that we test, results hold when accounting for agricultural productivity shocks, for variations in the wages of English farm workers, for yellow fever epidemics, and for the frequency of white servants' rebellions.

[10] Sections 2.2 and Appendix Section A.1.3 review the epidemiological literature on malaria resistances.

Using historical data from the Louisiana Slave Records 1719-1820 database, which has the unique feature of documenting the *birthplaces* of individuals enslaved across several Louisiana plantations, we assembled a dataset of prices for more than 3000 individuals born in 21 different African countries. We proxy resistance to malaria for each individual in the dataset with the level of Malaria Stability in his/her country of origin.

Results show a positive and precisely estimated effect of the level of resistance to malaria on the selling price of the enslaved individual.[11] Estimates are unaffected when a vast set of control variables – including proxies for health conditions unrelated to malaria susceptibility, body size, slave traders' production costs, and agricultural skills of the enslaved individuals – is taken into account. The existence of a malaria premium in the prices of African slaves suggests that malaria-resistance conferred a productivity advantage to individuals with higher resistance to the disease. At the same time – and most importantly – it further proves the salience of the disease for agricultural labor choices in malaria-infested areas of the United States.

Related Literature This paper enriches the literature on the economics of slavery by contributing an explanation to the long-held debate about why African slavery spread in the Southern United States, and why specifically Africans were employed in these regions. Most influential explanations have centered on the crops (North, 1966; Fogel and Engerman, 1974; Eearle, 1978; Fogel, 1994; Fenoaltea, 1984; Goldin and Sokoloff, 1984; Hanes, 1996 and Sokoloff and Engerman, 2000), the climate (Phillips, 2007), and the marginal productivity of labor in the South (Wright, 2003). While examining a sizable role of malaria in this picture, the present paper's findings suggest that malaria's role interacted with agricultural suitability for profitable crops, as highlighted in the literature. At the same time, accounting for the role of malaria helps simultaneously explain three key aspects of African slavery in the United States: (i) why it occurred in certain places and not others; (ii) why it soared at the end of the seventeenth century; and, crucially, (iii) why it involved Africans, and certain African populations in particular.

The idea that tropical diseases played a role in the diffusion of African slavery in the Southern US is as old as the Southern slave system itself, since slave owners sometimes used it to justify the brutal and inhumane social order. Several historians, such us Curtin (1968) and McNeill (2010), hypothesized a role for tropical disease in the deployment of the Atlantic slave system, while Coelho and McGuire (1997), Kiple and King (2003), Wood (2005), and Mann (2011) focused on the US context.[12] Despite

[11] The size of the estimated effect implies that going from the 25th to the 75th percentile of Malaria Stability in the country of origin leads to a predicted price that is 10% higher, which corresponds to broadly half of the difference between the prices for a male and a female.

[12] Their arguments combined various pieces of qualitative evidence. Curtin (1968), relying on data about British

these important contributions, the difficulty of teasing out the role of disease from other geographical, cultural and institutional features has left the debate so far unsettled.[13] The present study moves the debate forward by providing an empirical framework that singles out the specific role of malaria and of malaria immunities from the web of other geographical, cultural, and institutional features. This study goes beyond a potentially spurious broad spatial correspondence between malaria-infested regions and African slavery by recognizing the introduction of malaria *falciparum* as a turning point and exploiting it for identification. Further, documenting the existence of a malaria *premium* in slave prices, this study singles out the specific role of malaria immunities.

A rich literature documented how African slavery left ruinous legacies for both the enslaved and the enslavers. On the one hand, it stunted the long-term growth prospects of African populations who were subject to slave raids and enslavement, fostering fractionalization and distrust (Nunn, 2008a; Nunn and Wantchekon, 2011).[14] On the other hand, in societies that historically relied on African slavery as a source of labor, it planted the seeds for deep political and economic inequalities that persist to this day (Engerman and Sokoloff, 2000).[15]

More broadly, a large literature pointed to colonial histories as the key for countries' growth and developmental trajectories, and has thus prompted investigating their determinants. In particular, researchers have asked: did settlers bring their home institutions to the colonies or did colonial institutions emerge as a response to the local geographic environment of the newly settled lands? On the one hand, evidence suggests that cultural attitudes, institutional set-ups, and the consequent economic fortunes followed from persistent, ingrained traits that settlers brought over with them (La Porta et al., 1997, 1998; Grosjean, 2014).[16] Other researchers have argued that settlers adapted to the local geographic environment of the newly settled lands (Engerman and Sokoloff, 1997; Acemoglu,

soldiers serving in the Caribbean, provides statistics documenting a substantial difference in mortality rates between Africans and Europeans in the American tropics. For the United States, Kiple and King (2003) present historical studies comparing Europeans and African susceptibility to malaria, while Coelho and McGuire (2006) propose evidence of a differential susceptibility to hookworms. Moreover, Coelho and McGuire (1997), Wood (2005), and Mann (2011) present maps that suggest an association between the geography of African slavery and the geography of tropical diseases.

[13]Other scholars, such as the celebrated historian of slavery Kenneth Stampp, have 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).

[14]See also Nunn and Puga (2012), Whatleay and Gillezeau (2011), and Fenske and Kala (2015).

[15]See, among others, Nunn (2008b), Bertocchi et al. (2014), and Acharya et al. (2016). There is a rich literature on the legacy of slavery also outside of the United States. See Dell (2010), Acemoglu, García-Jimeno, and Robinson (2012), Bobonis and Morrow (2014) and Caicedo, Fujiwara, Laudares (2017) for the long-term effects of slavery in Central and South America. Markevich and Zhuravskaya (2017) and Buggle and Nafziger (2015) examine the medium- and long-term consequences of Russian serfdom.

[16]La Porta et al. (1997, 1998), for instance, argue that the identities of the colonizers - with the related historically determined legal tradition - determined what type of legal system was transplanted to the colonies, fundamentally affecting the consequent economic success of the country. Focusing on the prevailing culture of violence, Grosjean (2014) shows that different social norms emerged in areas of the US South where Scots-Irish, traditionally shepherds, migrated in large numbers.

Johnson, and Robinson, 2002). By showing that malaria generated a demand for African workers and thus concurred to the spread of African slavery in the United States, the present paper documents how colonial history responded to the geographic environment. Compared to these last studies, however, the present paper has the advantage of examining a major change in disease geography. Moreover, to our knowledge, this study presents the first micro-evidence unveiling the direct impact of geography on the economic motives at the root of different colonization histories. Finally, by documenting the existence of a link between disease geography and African slavery, the findings enrich the economic literature that explored the relationship between health and infectious diseases, institutions, and long-term growth (see, among others, Gallup, Sachs, and Mellinger, 1999; Weil, 2007; Bleakley, 2007 and 2010; and Alsan, 2015).

The remainder of the paper is organized as follows. In Section 2, we provide an epidemiological and 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. We turn to slave prices in Section 4, while the final section concludes.

2 Background

We start by summarizing relevant information about malaria and immunities to malaria in Section 2.1, and describing how malaria was introduced into the US colonies in Section 2.2. Section 2.3 analyzes the consequences of malaria introduction on labor choices in the United States.

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: the specific parasite, the mosquitoes, and the weather.[17] The single-cell parasite, the *Plasmodium*, exists in different strains and, among the most widespread strains, *vivax* malaria is a milder form of the disease, whereas *falciparum* malaria is the most virulent and lethal form. 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). While after repeated infections malaria virulence and mortality risk are reduced, continual infections deteriorate an individual's general health conditions, decreasing the ability to resist other diseases.[18] Precisely because it tends to weaken the immune system and drain a person's

[17] Appendix Section A.1 reviews in greater detail the role of the parasites, the mosquitoes, and the weather for malaria transmission.

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

energy, malaria has been nick-named "the great debilitator" (Dobson, 1989).

The weather is a fundamental factor for malaria transmission. Higher temperatures reduce the duration of the development of the parasite within the mosquito, aiding malaria transmission. At the same time, mosquitoes require enough water and hot enough temperatures to reproduce, develop, and survive.[19] For this reason, stable malaria transmission is mostly confined to tropical and semi-tropical areas.

The best proof of the health burden that malaria represents is written in the genetic code of a share of the world's population: during the last millennia a vast range of innate adaptations 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).[20] Importantly, a recent stream of research has pointed out that innate immunities are likely to interact with acquired ones, i.e. infections can trigger innate responses that might facilitate the acquisition of acquired immunities. 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. Importantly, even across Sub-Saharan African populations substantial heterogeneity is present in the degree of resistance to malaria (Kwiatkowski, 2005).[21]

2.2 Malaria Reaches the US Colonies

For the *Plasmodium* of malaria to be introduced into the US colonies an individual infected with malaria had to embark on a ship and the destination region had to host some variety of *Anopheles* mosquitoes and have a climate warm and humid enough for the mosquitoes and the pathogen to thrive.

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 to the colonies was higher than for *falciparum* malaria.[22] In effect, historical evidence shows that already at the beginning of the seventeenth century US settlers suffered from the type of relapsing fevers that characterize *vivax* malaria infections. On the other hand, *falciparum* malaria struck later. It

[19] On top of this, the two major strains of malaria require different climatic conditions, with *falciparum* malaria needing higher temperatures than *vivax* malaria to become infectious.

[20] Blood cell abnormalities are the most well-known and studied genetic resistances to malaria. 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).

[21] See Appendix Section A.1.3 for a background review on the role of immunities for malaria protection.

[22] Moreover, at destination, the weather conditions compatible with *falciparum* transmission existed only during the warmest seasons and only in the warmer states, whereas *vivax* malaria was often transmitted as far north as the state of New York.

was during the 1680s that an unusually virulent and deadly epidemics of *falciparum* malaria started to ravage the colonies (Childs, 1940; Wood, 1974; Rutman and Rutman, 1976), in conjunction with weather anomalies related to the El Niño events of 1681 and 1683-84.[23]

There is no way to know with certainty *who* carried *falciparum* malaria into the US colonies and *from where* the individual was traveling.[24] At that time, *falciparum* had already been introduced in South America and in the Caribbean (Curtin, 1993; Yalcindag et al., 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.[25] What is certain is that in the US colonies where it took root and flourished malaria started to take a "dreadful toll" among settlers.[26] A factor that increased the effective burden of malaria was its rural nature, so that it took its largest toll from farmers. Often hitting during harvest time, malaria caused serious losses in terms of worker time and efficiency.

This paper emphasizes the health consequences of *vivax* and *falciparum* malarias 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.[27] For this reason, we also investigate the role of yellow fever suitability and yellow fever epidemics, and find yellow fever's role of second-order importance compared to malaria.[28] The reason why in the United States, malaria explained the spread of African slavery much more than yellow fever is strictly related to the epidemiology of the two diseases. Yellow fever requires abundant, closely packed human population.[29] Moreover, immunity for yellow fever is largely acquired, so that the disease

[23]Section 3.2 and Appendix Section C.1 review existing evidence on the introduction of *falciparum* malaria in the United States.

[24]Interestingly, Packard (2007) points out that because "... 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 than were West Africans."

[25]It is important to note that malaria was already present in the British West Indies by the early 16th century. It is, however, impossible to precisely date its introduction there from available sources, and to distinguish *vivax* malaria, *falciparum* malaria and yellow fever epidemics.

[26]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 (Packard, 2007). Unsurprisingly, the great majority of deaths took place in the "ague and fever" months, between August and November (Packard, 2007).

[27]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.

[28]In the empirical section we examine the role of yellow fever and show that, while there is a correlation between yellow fever suitability and the distribution of African slaves, the size of the effect is about a fifth of the one related to malaria suitability. Moreover, yellow fever epidemics do not predict the switch towards African labor that took place at the end of the seventeenth century. Last, the price regressions show no yellow fever premium for individuals born in African countries with higher yellow fever suitability.

[29]This is because *Aedes Aegipty* has limited flying range (333 metres of *Aedes Aegipty* versus 3415 of *Anopheles*

has historically been a burden mostly to never-exposed newcomers (Pritchett and Tunali, 1995). In the United States, these conditions were met only in costal cities, especially in areas/periods where a large part of the population was composed by newcomers. While slavery was primarily a rural phenomenon,[30] and while malaria was to a large extent a rural disease, yellow fever mainly hit in big cities, sea coast cities in particular.[31]

2.3 Malaria and Labor: Europeans, Natives, and African Slaves

For several decades, European workers had been the principal source of labor in the US colonies, where they were mainly employed as *indentured servants* agreeing to work for a designated master for a fixed period of time in return for passage to a specified colony (Galenson, 1981).[32] From the early day of settlement, southern colonies were less healthy than northern ones, and *vivax* malaria was responsible for a large share of this health differential. The introduction of *falciparum* malaria drastically accentuated these health differences and had a big impact on Europeans' workers health and productivity. Georgian planters were lamenting that "Distempers" rendered European workers "useless for almost one half of the year" (Wood, 2007) and that "... a white servant cost three times what he could produce" (Kenzer, 1998).[33] As a consequence, landowners' demand for European laborers declined. At the same time, the supply of European servants willing to serve in malaria-infested low-lands also shrunk.[34]

From the early seventeenth century, and even more so after the introduction of *falciparum* malaria, colonizers had tried to satisfy their labor needs by employing or enslaving Native American tribes. However, Native Americans were considered only partially suitable for employment in plantations because of their high degree of morbidity and mortality. 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. In addition to this was malaria, to which they also had extremely high susceptibility. Humphreys (2001) reports the widespread conviction that Native Americans could

quadrimaculatus, Verdonschot, 2014), and yellow fever has a relatively brief infective period of human sufferers (for an insightful epidemiological discussion on yellow fever transmission, see McNeill, 2010).

[30] Although not solely a rural phenomenon. See Goldin (2001) for a study on the presence of slaves in cities.

[31] In different areas of the New World, however, the relative importance of malaria and yellow fever likely differed. In the Carribeans, for instance - with highly densely populated small islands continuously exposed to the arrival of ships in their active ports, with a large inflow of on-immune newcomers - the yellow fever might have mattered relatively more than in the United States.

[32] It is estimated that between one 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).

[33] Appendix Section A.3 explores the case of Georgia as an insightful case study.

[34] According to Wright (2003), it was not possible to attract free workers in certain locations on commercially viable terms. Menard (2001) notes that while European servants started to avoid unhealthy southern destinations, they continued to flow to the newly established colony of Pennsylvania.

not live in the same areas as 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). Just as telling was the fact that, while the Carolinas were starting to import Africans, they were exporting many more Native Americans out of Charleston (Resendez, 2016).

Until 1680, the presence of Africans in the US colonies remained marginal. They were slightly more numerous in southern colonies, already burdened by *vivax* malaria, but overall they represented only about 6% of the population there. With the introduction of *falciparum* malaria in the suitable US states the reliance on African labor more than doubled.[35]

European settlers seem to have rapidly reached the conviction that Africans were more resistant to malaria than Europeans and Native Americans and that fertile lands in the south could not be profitably cultivated without African labor. 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.[36]" Africans' lower susceptibility to malaria even attracted the inquiry of the scientific community. A study by Dr. Alfred Tebault published in the *American Journal of the Medical Sciences* in 1856 claimed that Blacks suffered from about one-third of the malaria attacks that struck white Americans.[37] Importantly, slave owners' perceptions of this differential susceptibility to diseases went even further, to the point that planters claimed even to be able to discern different health susceptibilities among Africans based on their place of origin.[38]

In malaria-infested areas, African labor became the most economically convenient option. The Atlantic slave trade was then fully established and American planters started to demand African labor in this market, with African workers entering the country as slaves. In the historical context of the Atlantic World of the time, the spread of the disease increased the economic profitability of African slave labor, influencing settlers in malaria-infested areas to opt for African slaves. While their legal status remained initially unregulated,[39] with the large expansion of African laborers in the population, laws and fully-fledged "slave codes" started to be approved in order to rule the status of

[35] According to the Colonial and Pre-Federal Statistics, in 1700 Africans represented 42% of the population in South Carolina and 28% in Virginia. McCusker and Menard (2014) provide estimates on macro-regions that suggest a smaller increase in the Black population between 1680 and 1700: in the Chesapeake region the share of Blacks went from 7% to 13%, while in the Lower South from 6% to 17%. The reason of this difference between aggregate macro estimates and state-measures from the Colonial and Pre-Federal Statistics for the lower South is related to the peculiar trajectory of Georgia (which is detailed as a case study in Section A.3). In any case, these macro-estimates also consistently document a sizable increase in the share of Blacks in the population. In our empirical analysis, we will verify results using data from both sources.

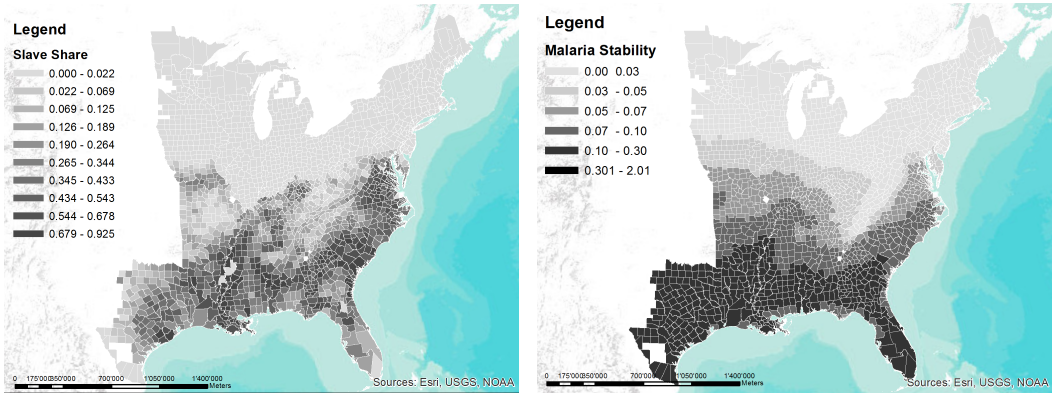
[36] Extract from *Plantation Life Before Emancipation*, Mallard (1892).

[37] Extract from Kiple (2003).

[38] Additional anecdotal evidence on perceptions of differential resistance of Africans to disease of tropical and semi-tropical areas is reported in Appendix Section A.2.2. Additional evidence on differential health susceptibilities among Africans is reported in Section 3.3.

[39] Appendix Section C.1.2 reviews evidence on the legal status of Africans in the US colonies.

Figure 1: Malaria and African Slavery across US Counties in 1860



(a) Share of Slaves (b) Malaria Stability

this growing mass of individuals. The end of the seventeenth century thus represented a turning point, with African slave labor becoming a pillar in certain areas of the country while remaining marginal in others.

3 Empirical Analysis

3.1 Malaria and African Slavery across United States Counties

The first prediction of the hypothesis that we investigate is whether African slavery was more concentrated in malaria-ridden areas. Thus, we start by exploring the cross-county relationship between African slavery and several different measures of malaria incidence.

3.1.1 Data

Malaria Incidence To measure malaria transmission intensity, we would ideally require a historical measure of malaria incidence across United States counties. However, malaria morbidity and mortality are themselves consequences of living standards, agricultural practices, and other features that might be related to broader developmental trajectories. Because malaria transmission can take place only in specific climatic and biological environments, to proxy for effective historical malaria exposure we use an exogenous predicted measure of incidence devised by Kiszewski et al. (2004): the Malaria Stability Index.

The Malaria Stability Index, constructed at a resolution of 0.5 x 0.5 degrees, predicts the risk of being infected with malaria as a function of characteristics of the mosquito vector prevalent in the

region – *i.e.*, the proportion biting people and the daily survival rate – and climate – a combination of long-run temperature and precipitation conditions.[40] The index offers the main advantage of being unaffected by human interventions (such as agricultural practices engendering stagnant water pools or the drainage of swamps and wetlands). In fact, long-term temperature and precipitation are exogenous to human activities, just as much as the characteristics of the dominant mosquito vectors in the area of study, which were indigenous to the New World and were not introduced by colonizers.

While our baseline measure relies on mosquito biology and long-term climate, an index similar in spirit could be constructed ignoring variation arising from different mosquitoes' characteristics. Therefore, as an additional measure of incidence the analysis is fully replicated using an exogenous index of malaria transmission predicted using only long-term temperatures and precipitation.[41] Importantly, we reconstruct this index using only historical data of temperature and precipitation.[42]

While these indices have the advantage of being exogenous to agricultural practices and developmental trajectories more generally, they are constructed to ignore additional - potentially endogenous - information of geographical features, that can explain a lot of the local variation in *actual* malaria intensity. Using historical data of malaria hospitalizations across US forts in the nineteenth century, Hong (2007) predicts malaria transmission risk based on a richer set of variables, including the likely presence of wetlands or swamps. As an additional test of the hypothesis, we verify our findings with this predicted index as well.

African Slavery We first look at the distribution of African slaves across counties using data from the historical censuses of 1790 and 1860.[43] According to our hypothesis, in malaria-prevalent areas the employment of African slaves increased landowners' profits and, as a consequence, in those areas we expect them to represent a higher share of the agricultural labor force. As our baseline measure, therefore, we look at the share of population composed of African slaves.[44] Figure 1 maps the distribution of African slavery in 1860 and the malaria transmission intensity across US counties measured using the Malaria Stability Index.[45]

[40] An in-depth description of all malaria indices used in the paper is provided in Appendix Section B.1.1.

[41] Flückiger and Ludwig (2017) constructed an index very similar in spirit, where mosquitoes' activities are predicted as non-linear functions of temperature, using estimates from the epidemiological literature. We verify our findings with this index as well. Results are available upon request.

[42] Appendix Section B.1.1 demonstrates that long-averages of temperature, precipitation and malaria incidence across sub-periods, 1900-1990, 1900-1950, and 1900-1925, are highly correlated and secular changes in climate do not explain away our findings.

[43] The 1790 census is the first US census ever conducted and there is not, to our knowledge, accurate county-level data for earlier periods. All results would be confirmed if using information from censuses between 1790 and 1860.

[44] This approach also allows us to take into account the large changes in population over time and across regions that took place during colonial times and before the Civil War. As a robustness measure, we also look at absolute number of African slaves in the county population.

[45] The same picture for 1790 is available in Appendix Section B.2.

3.1.2 Estimation and Results

We begin by estimating an Ordinary Least Square (OLS) regression across US counties, where the outcome of interest is the share of slaves in the county and Malaria Stability is the main explanatory variable. Our preferred specifications include state fixed effects, which net out the average differences across states. Looking at within-state variation is especially important because several states were in the process of banning or had already banned slavery. Even if state legislation sanctioning slavery might itself have been a response to economic motives shaped by malaria exposure, we are now interested in highlighting the effect of higher malaria incidence even when comparing counties sharing the same legal and institutional features.[46]

The county-level controls include a full set of soil suitability indices taken from FAO GAEZ, namely soil suitability for cotton, indigo, sugar, rice, tea, and tobacco. Using data from actual agricultural production across US counties in 1860, we show that these suitability indices are good predictors of actual historical production (see Appendix Table B10). We add two possibly relevant measures of distance, to the closest river and to the closest sea, and geographic controls of average historical temperature, precipitation, elevation, latitude, longitude and the interaction between latitude and longitude.[47] Summary statistics of all main variables of interest are summarized in Appendix Table B2, whereas Appendix Figure B4 portrays the binned scatterplot between Malaria Stability and African slavery. Turning to statistical inference, to account for spatial correlation in the errors we compute Conley standard errors adjusted for two-dimensional spatial dependence. Appendix Figure B7 plots confidence intervals for Conley standard errors obtained with all cutoff threshold ranging from 10 to 500 km and shows that standard errors are largest when the cutoff threshold is between 100 and 250 km, and then tend to decline. In the main text, therefore, we report estimates with cutoff values at 100 km and 250 km. Additionally, we verify baseline results with standard errors clustered at the state level.

Malaria and African Slavery Table 1 reports the main results. In Columns 1 to 3 and Column 6 the dependent variable is the share of African slaves in 1790, and in Columns 4, 5, and 7 the dependent variable is the share of African slaves in 1860. In Columns 6 and 7 we restrict the analysis to slave states. The results are consistent throughout the specifications and show that malaria exposure is positively and strongly correlated with the share of African slaves in the county. The estimated coefficient of our favorite specification shown in Column 5 suggests that a one standard deviation increase in Malaria Stability implies an increase in the share of African slaves of around 20 percentage

[46] Specifications without state fixed effects provide quantitatively and qualitatively identical results.
[47] Temperature and precipitation are long-averages from 1900 to 1950.

Table 1: Malaria and African Slavery across US Counties

Dependent Variable:	Share of Slaves (%)						
Sample:	**All States**			**All States**		**Slave States**	
Year:	1790			1860		1790	1860
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.164***	0.154***	0.065**	0.172***	0.192***	0.057*	0.125**
Conley s.e. 100km	(0.040)	(0.038)	(0.030)	(0.031)	(0.042)	(0.032)	(0.049)
Conley s.e. 250km	[0.054]	[0.044]	[0.030]	[0.048]	[0.050]	[0.031]	[0.053]
Conley s.e. 500km	(0.044)	(0.044)	(0.007)	(0.058)	(0.011)	(0.011)	(0.015)
State Cluster	{0.050}	{0.032}	{0.043}	{0.050}	{0.050}	{0.044}	{0.051}
Crop Suitability	No	Yes	Yes	No	Yes	Yes	Yes
Distances & Geography	No	No	Yes	No	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	285	285	285	1,956	1,956	244	1,081
R-squared	0.601	0.718	0.826	0.661	0.709	0.794	0.603
Mean Dep. Var.	0.224	0.224	0.224	0.164	0.164	0.261	0.285

Notes: The table reports Ordinary Least Square (OLS) estimates. The unit of observation is the US county in 1790 (Columns 1, 2, 3, and 6) and in 1860 (Columns 4, 5, and 7). Columns 6, and 7 include only "slave states." The dependent variable is the county share of slaves. 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 "Crop Suitability" controls include soil suitability for cotton, indigo, sugar, rice, tea, and tobacco. The "Distance" controls include distances to the sea, to the closest river, latitude, longitude and latitude x longitude. The "Geography" controls include average temperature, precipitation, and elevation. Conley standard errors, with cutoff thresholds for latitude and longitude at 100, 250, and 500 km, are reported in brackets. Standard errors clustered at the state level in curly brackets. ***, **, * indicate significance at 1%, 5%, and 10% levels respectively, computed with 100 km Conley standard errors.

points, which represents almost a doubling of the mean county share. 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 County in Massachusetts – to a county with the maximum Malaria Stability in the sample – like another Washington County, this time in Georgia – leads to an increase in the ratio of slaves to the total population of about 30 percentage points.[48] The same relationship estimated for 1790 gives a coefficient of a smaller magnitude, since the smaller sample does not permit us to fully disentangle the role of Malaria Stability from the role of climate.[49]

In standard deviation terms, Malaria Stability is the main predictor of African slavery out of all included controls in both 1790 and 1860. Of all the other explanatory variables included, the other other predictors of the African slave share are the average temperature of the county, county elevation, and the distance to the sea. Regarding crop suitability, results summarized in Appendix Table B11 show that cotton, indigo, rice, and sugar suitability are positively correlated with slavery (and cotton

[48]Interestingly, a regression including Malaria Stability alone would give an R-square as high as 0.37, 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 0.24.

[49]The introduction of average temperature, precipitation and elevation decrease the coefficient in Column 3 because, given the reduced number of states in 1790 (14 states), there is limited residual variation in Malaria Stability (the correlation between temperature and precipitation in this sample is 0.92). The same regression in 1860, with 37 states in the sample, allows us to better disentangle the role of temperature from Malaria Stability (the correlation here is 0.44).

- as expected - matters only in 1860).

Table 2: Interaction with Crops and in the New World

	Panel A				Panel B	
Sample:	United States, Counties 1860				US, Brazil, Cuba	
	All Counties				States/Provinces	
Dependent Variable:	**% Slaves**				**% Blacks 1872**	
Malaria Stability	0.168***	0.166***	0.157***	0.204***	0.147***	0.100**
	(0.041)	(0.042)	(0.044)	(0.042)	(0.026)	(0.041)
	[0.050]	[0.048]	[0.054]	[0.050]		
Type of Crop:	**Cotton**	**Any Cotton**	**Labor-Intensive**	**Wheat**		
Crops	0.036	0.028*	0.073***	0.045**		
	(0.029)	(0.015)	(0.016)	(0.022)		
Crops × Malaria Stability	0.022	0.061***	0.055***	0.076***		
	(0.023)	(0.023)	(0.013)	(0.027)		
Crop Suitability	No	No	No	No	No	Yes
Distances & Geography	Yes	Yes	Yes	Yes	No	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	No	Yes
Observations	1,956	1,956	1,956	1,956	73	73
R-squared	0.750	0.704	0.721	0.698	0.352	0.886
Mean Dep. Var.	0.164	0.164	0.164	0.164	0.300	0.300

Notes: The table reports Ordinary Least Square (OLS) estimates. The unit of observation is the US county in 1860 in Panel A. In Panel B, the unit of observation is the region/province (20 states in 1872 Brazil, 13 provinces in 1872 Cuba and 40 states in 1860 United States). Malaria Stability is an index measuring the force and stability of malaria transmission, standardized in order to have 0 mean and unit standard deviation. "Cotton" is a standardized measure of cotton pound bales produced in 1860. "Any Cotton" takes value 1 if the county produces any cotton in 1860. "Labor-Intensive" indicates counties that specialize (top 90 percentile) in the production of at least one labor-intensive crop (cotton, rice, tobacco, and sugar); "Wheat" indicates counties that specialize (top 90 percentile) in the production of wheat. In Panel A, Conley standard errors are reported in brackets, with cutoff thresholds for latitude and longitude at 100 km (round brackets) and 250 km (square brackets). In Panel B robust standard errors are in brackets.

Robustness Checks The results are confirmed: with all alternative malaria indices (Tables B3 and B4 in the Appendix), using alternative ways of measuring African slavery (Appendix Tables B5 and B6), as well as when controlling for actual production of crops traditionally associated with slavery, instead of suitability (Appendix Table B12).

Controlling for Geography Baseline estimates controlled for the linear effects of temperature, precipitation, and altitude; however, it might be desirable to control for the role of geographical features in a more flexible way. Since we do not know the precise functional forms that might capture the specific role of geography, we rely on the high-dimensional LASSO methods presented by Belloni, Chernozhukov, and Hansen (2014). We thus account for the effects of first, second, and third order geographical factors (temperature, precipitation, altitude, longitude, and latitude) and a full set of interactions between various order terms. Results, summarized in Appendix Table B13, show that the role of malaria transmission intensity remains unaffected and precisely estimated even when accounting for the effect of geography in a more flexible way.

Appendix Table B15 explores the role of two other tropical/semi-tropical diseases: yellow fever and hookworm. While results highlight a positive correlation with African slavery, the coefficient size

has significantly lower orders of magnitude. Appendix Figure B6 presents a "thought experiment" that allows us to visualize more concretely the difference in magnitude of the effect of yellow fever and of malaria suitability. We randomly assign to US counties in 1860 malaria and yellow fever suitability values taken from a normal distribution mimicking the disease suitability of counties in Rhode Island (mean and standard deviation). The graphs show that the decrease in the share of slaves if all counties had the distribution of yellow fever suitability of Rhode Island would be marginal, whereas with the Malaria Stability of Rhode Island the share of enslaved individuals in the population would be more than halved. Last, binned scatterplots in Appendix Figure B10 show that there is no relation between malaria suitability and cholera/smallpox mortality.[50]

Interaction with Labor-Intensive Crops Malaria thrived in the countryside and represented a scourge primarily for the agricultural sector. Since the malaria season coincided with the peak of the agricultural season, the disease exposed agricultural workers to suffering, days of absence, and low productivity. In fertile lands suitable for lucrative crops - such as cotton, rice, sugar, and tobacco - the profitability of African labor was thus even larger.[51] In Panel A of Table 2, we document the extra effect of Malaria Stability in counties producing cotton (Columns 1 and 2) and in counties that were major producers of cotton, rice, sugar and tobacco (Column 3). While the main effect of Malaria Stability remains sizable and precisely estimated, results document that indeed in these areas the effect of malaria on labor choices is stronger than in other areas.[52] Interestingly, the same (extra) effect is present in counties that specialized in wheat production (Column 4) and it is not specific of labor-intensive crops. In other words, wheat was cultivated by African slaves in counties with malaria, and by free laborers in counties with no malaria.

Relatedly, Appendix Table B14 also shows that wherever the land was not fertile/unsuitable for agriculture, we see no link between malaria prevalence and the share of African slaves in the population. Wherever the land was very fertile, instead, there were strong incentives to employ African labor to deal with the scourge of malaria.

[50] Appendix Table B9 shows that results would remain unaffected if we were to control for - potentially endogenous - characteristics of the county such as total population or average farm-land value.

[51] According to several scholars, these crops could be profitably cultivated by slave labor. Fenoaltea (1984) claimed that slavery was profitable for "effort intensive crops" such as sugar. Goldin and Sokoloff (1984) suggested that slavery was well adapted to tobacco and cotton, as children and females were relatively more productive for their cultivation. Fogel and Engerman (1974) and Fogel (1994) argued that free workers preferred to avoid crops, such as rice and sugar, cultivated through gang labor, a set of particularly unpleasant labor routines. Earle (1978) pointed out that these crops had lengthier growing season while Hanes (1996) shed light on the high turnover costs related to the cultivation of certain crops.

[52] For instance, in the case of cotton, a one standard deviation increase in Malaria Stability in counties producing cotton (any amount) was correlated to a to a 40% increase in the share of African slaves in the population (with respect to counties with the same level of malaria suitability that were not producing cotton).

Pro-Slavery Attitudes and the Progressive Eradication of Slavery Malaria-ridden areas also developed the strongest pro-slavery attitudes and support. In these places, political preferences for slavery took stronger roots, and when a large part of the country, on ethical and political grounds, started to question the institution, malaria-ridden places remained the last advocates for the persistence of slavery. We explore pro-slavery attitudes at three fundamental crossroads in the history of the country. First, we look at pro-slavery votes of delegates at the Constitutional Convention of 1787.[53] We assign to each of the 55 delegates the Malaria Stability of their county of birth (or of death, whenever the politician was not born in the United States).[54]

Table 3: Malaria, Pro-Slavery Attitudes and Presidential Elections

	Constitutional Convention		**Presidential Elections**			
	1787		1860		1868	
	Pro-Slavery Votes		Lincoln	Breckinridge	Grant	Seymour
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	0.319**	0.319**	-10.274***	12.391**	-14.736***	14.738***
Conley s.e. 100 km	(0.129)	(0.129)	(3.226)	(5.051)	(4.131)	(4.130)
Conley s.e. 250 km	[0.129]	[0.129]	[3.616]	[3.767]	[3.084]	[3.083]
State Cluster	{0.296}	{0.299}	{5.431}	{6.703}	{5.400}	{5.400}
Crop Suitablitiy	Yes	Yes	Yes	Yes	Yes	Yes
Distances and Geography	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
Vote Fixed Effects	No	Yes				
Observations	495	495	1,091	1,096	1,539	1,539
R-squared	0.238	0.296	0.883	0.712	0.463	0.463
Mean Dep. Var	0.275	0.275	37.684	4.704	51.495	48.503

Notes: The table reports Ordinary Least Square (OLS) estimates. In columns 1 and 2, the unit of observation is the vote of the delegate on slavery issues. The dependent variable takes on value 1 when the vote is pro-slavery. Malaria Stability is an index measuring the force and stability of malaria transmission in the delegate county of birth. In columns 3 to 6, the dependent variables is the share of county votes for Lincoln (1860), Breckinridge (1860), Grant (1868) and Seymour (1868). Malaria Stability is an index measuring the force and stability of malaria transmission in the county, standardized in order to have 0 mean and unit standard deviation. The "Crop Suitability" controls include soil suitability for cotton, indigo, sugar, rice, tea, and tobacco. The "Distance" controls include distances to the sea, to the closest river, latitude, longitude and latitude x longitude. The "Geography" controls include average temperature, precipitation, and elevation. Conley standard errors, with cutoff thresholds for latitude and longitude at 100 and 250 km, are reported in brackets. Standard errors clustered at the state level in curly brackets. ***, **, * indicate significance at 1%, 5%, and 10% levels respectively, computed with 100 km Conley standard errors.

As an additional exercise, we explore the distribution of electoral votes for the Republican and Democratic presidential candidates during the 1860 and 1868 Presidential elections.[55] This is interesting because of the opposition of Lincoln to slavery and also because, until the middle of the twentieth century, the Democratic party was the racially conservative party, while Republicans had

[53] In the definition of pro-slavery votes we followed Dougherty and Heckelman (2008), which examined voting behavior of delegates on convention votes 39, 132, 136, 145, 147, 253, 367, 368, and 999.

[54] Each delegate represented a state. Note that the state where the delegate was born (or died) does not always belong to the state the delegate represented at the convention. We decided to focus on the delegate county of birth as this might very likely affect deep-rooted views regarding slavery.

[55] More precisely, we focus on the county share of votes for Lincoln and Breckinridge, in the Presidential election of 1860, and for Grant (Republican) and Seymour (Democrat), for the Presidential elections of 1868. Importantly, data for 1860 elections miss most Southern states, but still includes several former slaves states such as Arkansas and Missouri. To get a more complete picture, therefore, we also look at 1868 elections.

maintained moderate to extreme anti-slavery positions before the Civil War and acted as defendant of Black rights afterwards.[56] Results summarizing a sizable effect of malaria transmission intensity on pro-slavery attitudes are reported in Table 3.

In Table 3, Columns 1 and 2 report estimates on the likelihood of casting a pro-slavery vote at the constitutional convention, comparing delegates from counties with different levels of malaria. A one standard deviation increase in Malaria Stability implies to a 30 percentage points rise in the likelihood of a pro-slavery vote, thus doubling the mean likelihood. Columns 3 to 6 show the votes for the main candidates of the 1860 and 1868 Presidential elections. Similarly, counties with high Malaria Stability voted significantly less Republican Presidents: a one standard deviation increase in malaria risk is related to a 10 percentage points reduction in votes for Lincoln in 1860 and a 14 percentage points reduction in votes for Seymour in 1986, which corresponds to a percent reduction of about one-third of the mean county votes for Lincoln and Seymour.

Equally as important, the more malaria-ridden areas remained anchored to slavery until the very end. Results reported in Appendix Table B16 show that more malaria-ridden counties experienced lower reduction if the share of African slaves in the population between 1790 and 1860 than less malaria-ridden counties.

Malaria and African Slavery in the New World Finally, we show that the same relationship between malaria suitability and African slavery is present across regions/states in the New World (the US, Cuba, and Brazil) in Panel B of Table 2.[57] These results suggest that similar dynamics are likely to have played a role in all these different colonial settings. Importantly, focusing on the United States allows us to go beyond a purely cross-sectional empirical framework, as for the United States we can explore the effect of the rapid deterioration in the malaria environment brought about by the introduction of *falciparum* malaria.

3.2 The Introduction of Falciparum Malaria into the Colonies

The cross-sectional results may be flawed if the areas with higher malaria transmission were different from other areas along dimensions that we do not observe, which could be the actual reasons for greater exploitation of African slaves. As a way out, since malaria was not present in the US before

[56] For related approaches, see Masera and Rosenberg (2018), and Acharya, Blackwell, and Sen (2016).

[57] Note that, in the specification looking at regions/states in the New World (the US, Cuba, and Brazil), we can control for country fixed effects as the unit of observation is at the sub-national level. The sample includes 20 states in 1872 Brazil, 13 provinces in 1872 Cuba, and 40 states in 1860 United States. Outside of the New World, Reilly (2015) suggests that within Arabian Peninsula oases, which were extremely unhealthy locations for people with no acquired and genetic resistance to the disease, the prevalence of malaria was the reason for the large-scale employment as slaves of people of African ancestry.

European colonization, we can go back in time to investigate the diffusion of African slavery before and after the arrival of malaria in the colonies. In particular, we will be exploring the changes triggered by the introduction of the last and most virulent malaria species, *falciparum* malaria, responsible for a drastic deterioration in the health environment of the colonies.

3.2.1 Empirical Strategy

Timing of Introduction The first challenge is to identify the exact timing of the introduction of *falciparum* malaria into the US colonies. Indeed, thanks to historical evidence, the introduction of the disease can be dated with a sufficient degree of accuracy. In fact, epidemiology would suggest that when a *falciparum* malaria infection hits a population never previously exposed to the parasite, violent epidemics must follow. Epidemics are expected to hit until a new equilibrium is reached, when *falciparum* malaria starts to be endemic to the region. In effect, a series of epidemics started to hit the southernmost US colonies during the 1680s. 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.

Exploring anecdotal evidence with these epidemiological considerations in mind leads Wood (1974) and Rutman and Rutman (1976) and several other historians – whose contributions are summarized in Appendix Table C1 – to date the introduction of *falciparum* malaria around the mid-1680s. The deterioration of the health environment was major and rapid, as vast anecdotal evidence documents. Table 4, for instance, reports extracts for South Carolina before and after the *falciparum* epidemic that hit Charleston in 1684. Weather anomalies that characterized the decade facilitated the introduction of *falciparum* malaria in the colonies. As Figure 2 suggests, based on data that climate historians have pieced together, starting in the 1680s we observe an increase in extreme weather events, whose variation we will use to predict when and where malaria introduction was more likely to materialize.[58]

Differences in Differences Ideally, the analysis would require information on the specific timing of the introduction of *falciparum* malaria into each US state. However, while historical analyses of the health environment of the major colonial states are vast and informative, smaller and more peripheral states have received less investigation. Moreover, the actual timing of the introduction of the disease

[58]El Niño 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 Niño event (1671) in the 1670s, and one event in the 1660s (1661). It is well-established that extreme weather events increase malaria risk and transmission intensity 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.

Table 4: 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.*

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 endemic.

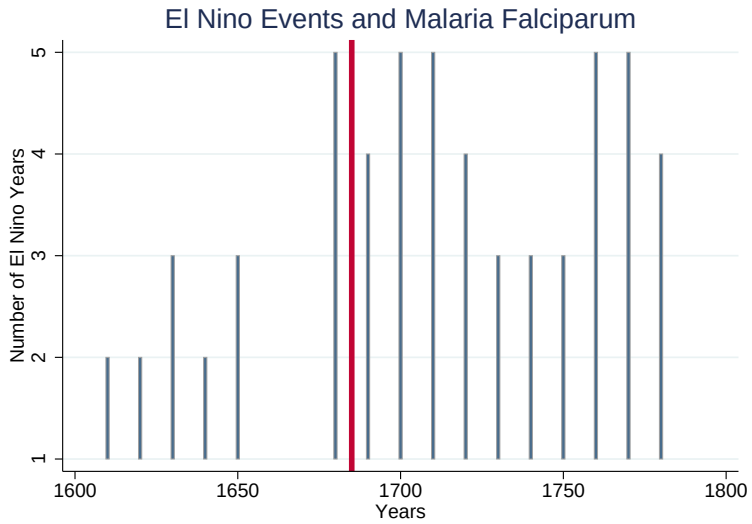
To overcome data limitations and the potential source of endogeneity that might drive the actual timing of the introduction in different states, in our baseline analysis we use the same date of *falciparum* malaria arrival for all states. Based on the work of historians, we consider the decades up to 1680 (included) as prior to introduction, and the subsequent decades as post-introduction. Moreover, we exploit the differential geographic suitability for malaria across states to predict where malaria was more likely to hit and then become endemic. In a difference-in-differences exercise, we 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 was more likely to thrive with the other states.

The main threat to this strategy is posed by shocks that differentially affected states more or less suitable for malaria and that 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, we account for several competing hypotheses, including the potential increase in the production of important colonial crops,[59] changes in English farm wages,[60] yellow fever epidemics, and the frequency of servants' rebellions.

[59] 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. 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 is that the bushels of rice were exported to England from the US colonies. The figures are available starting in 1698. The amount of rice exported from the producing areas was still very little in 1698, with 10,407 pounds of rice exported. However, exports increased quickly, so that in 1700 the colonies exported 394,130 pounds of rice. In 1750, the amount of rice exported was more than 27 million pounds. Source: Colonial and Prefederal Statistics, Chapter Z.

[60] An alternative explanation behind the rise in African labor in the US colonies centers around the role played by English wages. The cost of hiring 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).

Figure 2: Weather Anomalies and Malaria Introduction - El Niño Events



†The graph plots the time series of El Niño events in the seventeenth and eighteenth centuries. The gray bars measure the numbers of El Niño years registered in the decade. The red line plots the year 1684, year of the first major *falciparum* malaria epidemics in the country.

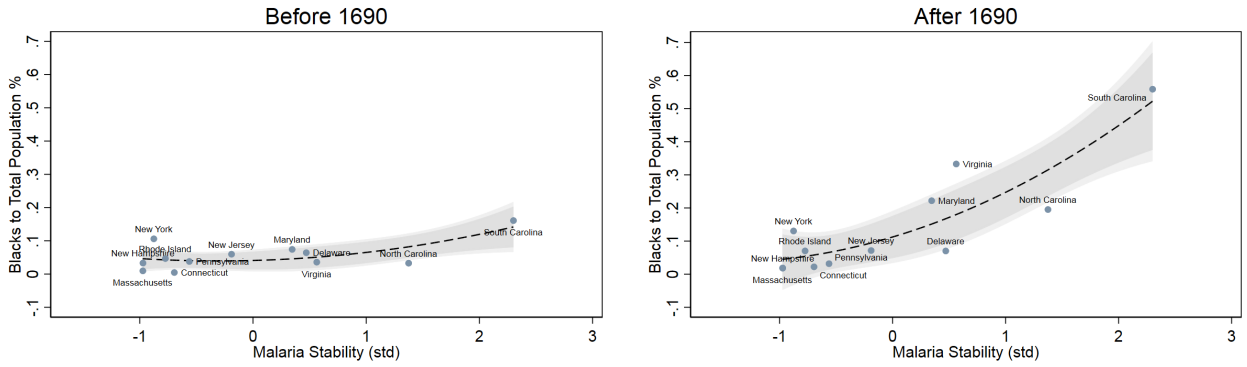
Instrumental Variables A concern that may arise is the possibility that the *falciparum* malaria epidemics observed were a consequence of the greater inflow of Africans to high malaria states, and not vice versa.[61] As an alternative strategy, in order to get rid of the endogeneity that might drive the timing and location of *falciparum* malaria introduction, we 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 we exploit the interaction between a time-series of weather anomalies (common for all states) and the cross-sectional variation in malaria suitability across states.[62] Out of sample evidence, exploiting data on malaria mortality in the same states of our sample but for later decades (1850 to 1930) confirms the validity of the intuition.[63] Thus, we collect all available information on the appearance of *falciparum* malaria for each state and instrument it with this predicted index.

[61]Note that the differences-in-differences exercise attempted to tackle this concern by mimicking a reduced form specification. In fact, the inflow of workers from malaria-infested areas certainly increased the likelihood of epidemics, but the 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.

[62]More formally, let our exogenous predictor be the term $El\ Nino_t * MS_c$, the interaction term between the number of El Niño events in the decade, which varies by decade, and the Malaria Stability index in the state, which varies across states.

[63]We can provide only out of sample evidence, as data on malaria mortality are missing for the colonial era.

Figure 3: Malaria and Share of Blacks Before and After 1690



†The left graph plots the correlation between Malaria Stability and average share of Blacks in the population before 1690. The right graph plots the same correlation after 1690.

3.2.2 Data

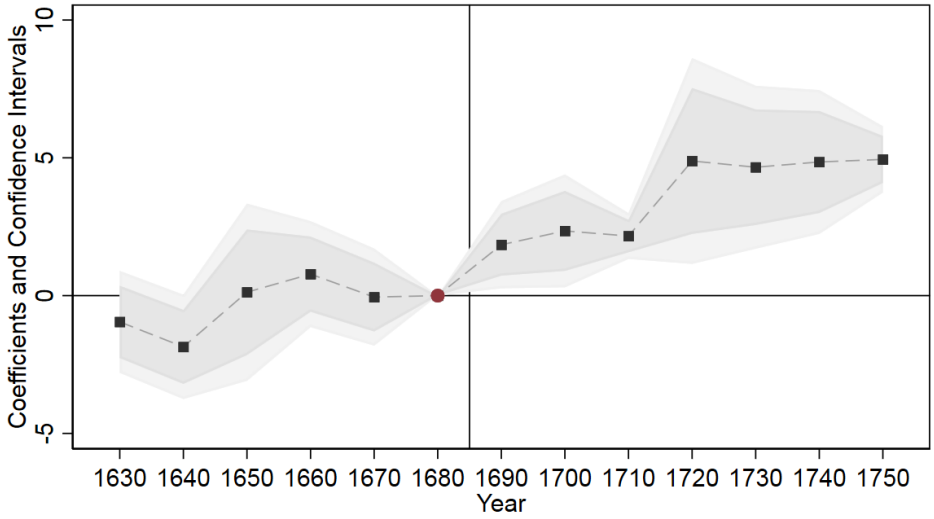
Share of African Slaves The colonial and pre-federal statistics of the US Census provide figures on the numbers of "Whites" and "Negroes" in each state over the decades from the early days of settlement. This is the most disaggregated panel information documenting the presence of Whites and Blacks, going back before 1680.[64] In the final sample, where the unit of observation is the state in each decade, we assemble an *unbalanced* panel of twelve states. To keep comparable pre- and post-periods, in our baseline specifications we restrict attention to decades ranging from 1630 to 1750, and provide several robustness analysis with shorter/longer post-treatment periods.[65]

We use the Malaria Stability index of Kiszewski et al. (2004) to proxy for the average geographical suitability for malaria in the state, and a similar index predicting malaria intensity based on long-run temperature and precipitation - as previously discussed. Figure 3 shows that, before 1690, there is no correlation between the average share of Blacks and the Malaria Stability of the state. The correlation emerges, instead, when looking at the period starting in 1690. This is indicative of the fact that malaria suitability did not matter before the malaria *plasmodium* reached the United States, but started to matter after its introduction. Data sources and summary statistics for all main variables

[64]Greene and Harrington (1993) provide the most comprehensive attempt to map the local distribution of population before 1790 at the sub-state level. Unfortunately, however, for almost no state is it possible to retrieve local information on Blacks and Whites both before and after 1680. As a robustness check, we replicate results using revised data from McCusker and Menard (2014).

[65]As they would not contribute to the empirical analysis, we exclude states that are observed only after 1690 (Georgia). Moreover, to be able to compare data over time we consider Maine, Plymouth, and Massachusetts as a single state. The panel is unbalanced because certain colonies were founded later and have information on population only after 1640, such as for South Carolina (1670). Note that results hold when excluding South Carolina and controlling for the size of the population in a number of ways (see Section C.3.4).

Figure 4: Malaria and Share of Blacks - Event Study



†The graph plots coefficients in an event study type of framework, comparing states with different level of Malaria Stability over time. The red dot indicates the omitted decade, 1680. Black squares mark regression coefficients. Gray areas depict confidence intervals, at 99% (dark gray) and 95% (light gray) significance.

are reported in Appendix Tables C2 and C3.

3.2.3 Estimation

Turning to a more formal analysis, we propose a set of estimates based on the specification below:

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

The main interest lies in β, the coefficient of the interaction term between $Post$-1690_t, an indicator taking value 1 for the decades following 1690 (with 1690 included), and the variable MS_s, which is the 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. We start by looking at $\%Black_{s,t}$, the share of the Black population in the state in the decade, as the variable of interest. $\mathbf{I_{s,t}}$ is a vector of time-varying controls, which we will detail below.

The difference-in-difference framework identification rests on the assumption of identical counterfactual trends at different treatment intensities. Before turning to the results, we provide more formal evidence in favor of the identifying assumptions in an event study framework, where we observe the evolution over time comparing states with different levels of Malaria Stability. Figure 4 shows that all leads (the main coefficients in the periods prior to the treatment) are very close to zero. Part of the effect materializes immediatedly in 1690, and then grows bigger after 1720.

3.2.4 Results

Main results are summarized in Tables 5 and 6. The estimated coefficients consistently show that after 1690 more malaria-suitable states have a significantly larger share of African laborers in the population compared to less malaria-suitable states. In terms of magnitude, according to the baseline specification in Column (1) of Table 5, a standard deviation increase in Malaria Stability brings about a 0.096 increase in the share of Blacks, which correspond to a doubling of the mean state share.

Table 5: Malaria Introduction and African Slavery – US States

Dependent Variable:	Share of Blacks				**Δ Sh. Blacks**	**Δ Blacks**	**Δ Whites**
	All Sample		No South	No	All Sample	All Sample	All Sample
	1630-1750	1650-1720	Carolina	Virginia	1630-1750	1630-1750	1630-1750
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability × Post-1690	0.096***	0.072**	0.074**	0.083**	0.017**	2,836.533*	-935.761
	(0.024)	(0.026)	(0.031)	(0.026)	(0.005)	(1,822.657)	(719.751)
	[0.024]	[0.025]	[0.032]	[0.027]	[0.006]	[1,376.326]	[756.733]
Bootstrap s.e. p-value	*0.006*	*0.038*	*0.013*	*0.020*	*0.043*	*0.007*	*0.261*
Decade Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	133	88	124	120	121	121	121
R-squared	0.868	0.851	0.745	0.882	0.236	0.486	0.700
Number of States	12	12	11	11	12	12	12
Mean Dep. Var.	0.104	0.089	0.078	0.096	0.013	1947.273	7593.669

Notes: The table reports OLS estimates. The unit of observation is the US state in the decade. The panel includes decades from 1640 to 1780 in Columns 1, 3, 4, 5, 6, and 7, and decades from 1650 to 1720 in Column 2. The dependent variable is the share of Black people in the state in Columns 1, 2, 3, and 4; the first difference of the share of Black people in the state $\Delta Sh.Blacks_t = Sh.Blacks_t - Sh.Blacks_{t-1}$ in Column 5; the first difference in the absolute number of Black people in the state $\Delta Blacks_t = Blacks_t - Blacks_{t-1}$ in Column 6; the first difference in the absolute number of White people in the state $\Delta Whites_t = Whites_t - Whites_{t-1}$ in Column 7. 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 double-clustered at the decade and state level are reported in round brackets, standard errors clustered at the state level are reported in squared brackets. P-values for the null hypothesis (Malaria Stability x Post1690 = 0) computed with wild bootstrap standard errors are reported in italics. ***, **, * indicate significance at the 1%, 5%, and 10% levels respectively (related to standard errors clustered at the state level).

The results are confirmed when restricting the analysis to the decades between 1650 and 1720, as per Column 2 of Table 5. Moreover, results are not driven by the trajectory of any specific state, or by the particular histories of South Carolina and Virginia. Estimates in Columns 3 and 4 of Table 5 exclude each of these states and a binned scatter plot in Appendix Figure C1 confirms that the effect is not driven by outliers.[66] To account for potential auto-correlation in the errors, Columns 5, and 6 look at the first difference in the share of Blacks and in the absolute number of Black people. The negative coefficient in Column 7 suggests that the shock might have also triggered a reduction in the white population, but the coefficient is not precisely estimated.[67]

[66] Appendix Table C10 reports results excluding one of the states in the sample one by one.

[67] Binned scatter plots in Appendix Figure C2 tend to confirm a negative, and noisy, relationship. There is anecdotal evidence, for instance from Wood (1974), mentioning that several European immigrants left Charleston, South Carolina, as a consequence of the deterioration in the health environment. Certainly, white settlers adopted strategies to cope with

In Table 6 we account for an extensive set of time-varying controls. In Panel A, we first account for the potential concurring effect of agricultural productivity shocks. As documented in Appendix Table C4, the two major colonial crops were rice and tobacco. To take into account the possible effect of agricultural productivity shocks on the share of African labor, we control for a structural change in the role of average suitability for several crops in 1690 - shown in Columns 1, 2, and 3, which mirrors our main specification where Malaria Stability is the variable of interest. In addition, we control for a time-varying effect of average crop suitabilities on the share of Blacks, interacting average state suitabilities with decade fixed effects - as seen in Columns 4, 5 and 6. As a way to control for possible higher profitability of certain crops, we account for the interactions between the time series of crop prices and state fixed effects in Column 7.[68] The coefficient of interest decreases but only mildly.

In Panel B of Table 6 - Columns 1, 2, 3, and 4 - we account for the role of other potential health shocks. Column 1 controls for the number of yellow fever epidemics in the decade, whereas Column 3 controls for the interaction term of yellow fever suitability with the Post-1690 variable. The inclusion of these controls does not reduce the magnitude of the coefficient of interest and the number of yellow fever epidemics has no positive and significant effect on our outcome.[69] Moreover, it is reassuring that the inclusion of interaction tertm between temperature and decade fixed effects does not reduce, but actually increases, the size of β, indicating that the estimated effect is not driven by time-varying variables acting along a climatic gradient (see Column 3). It is also possible that the number of African laborers previously imported might increase the likelihood of malaria epidemics, or other epidemics, to occur. For this reason, in Column 4, we account for a time-varying effect of the black population in 1680, as a way to account for a path dependence determined by the total number of African laborers present in the colonies in 1680.

In Panel B of Table 6 - Columns 5, 6, and 7 - we also account for the effect of changes in the wages and availability of European workers. To exclude the possibility that the results are driven by the lower availability of European servants, which might have affected certain states more than others, we account for state-specific effects of the English wage time series.[70] While the inclusion of these controls decreases the coefficient somewhat, the effect of malaria introduction remains sizable and precisely estimated. As servants' revolts might have discouraged land-owners from relying on European servants' labor, in Column 7 we include the total number of revolts enacted by white servants in the decade,

the disease. Narratives document a change in the habits of the white inhabitants, with a large share of the population that could afford it leaving the areas during the worst malaria months in the summer and fall.

[68] More formally, we control for a full set of interaction variables between the time series of crop prices and state fixed effects, $price_t * \mu_c$.

[69] In the same way, when we control for the time-varying effect of the suitability to *Aedes Aegipty*, results do not change.

[70] Note that if the effect of higher English farm wages affected all the states in the same way, accounting for aggregate shocks hitting all the colonies at once would already eliminate this potential bias.

Table 6: Malaria and African Slavery – Accounting for Alternative Explanations

Dependent Variable:	Share of Blacks						
	PANEL A: CROPS						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability × Post-1690	0.084***	0.091**	0.075**	0.084***	0.092**	0.073*	0.091***
	(0.023)	(0.030)	(0.028)	(0.025)	(0.032)	(0.033)	(0.026)
	[0.023]	[0.032]	[0.031]	[0.025]	[0.034]	[0.034]	[0.023]
Bootstrap s.e. p-value	*0.052*	*0.066*	*0.148*	*0.050*	*0.059*	*0.173*	*0.005*
Controls:	Rice Suit × Post-1690	Tobacco Suit × Post-1690	Rice & Tob. Suit × Post-1690	Rice Suit × Decade FE	Tobacco Suit × Decade FE	Rice & Tob. Suit × Decade FE	Rice & Tob. Prices × State FE
Observations	133	133	133	133	133	133	116
R-squared	0.872	0.868	0.872	0.906	0.882	0.916	0.918
Mean Dep. Var.	0.104	0.104	0.104	0.104	0.104	0.104	0.114
	PANEL B: OTHER CONTROLS						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability × Post-1690	0.093***	0.233***	0.147**	0.083***	0.089***	0.089***	0.105***
	(0.022)	(0.051)	(0.048)	(0.024)	(0.026)	(0.024)	(0.027)
	[0.022]	[0.054]	[0.050]	[0.024]	[0.023]	[0.025]	[0.028]
Bootstrap s.e. p-value	*0.001*	*0.056*	*0.073*	*0.025*	*0.007*	*0.009*	*0.043*
Controls:	Yellow Fever	Yellow Fever × Post-1690	Temp × Decade FE	Sh. Black 1680 × Post-1690	English Farm Wage × State FE	Servants Revolts	South Nantucket × Decade FE
Observations	133	133	133	133	133	133	133
R-squared	0.872	0.888	0.909	0.875	0.900	0.875	0.883
Mean Dep. Var.	0.104	0.104	0.104	0.104	0.104	0.104	0.104
	PANEL C: ALL CONTROLS - LASSO AND PCA						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability × Post-1690	0.092***	0.046***	0.059**	0.115***	0.095***	0.054**	0.089***
	(0.024)	(0.012)	(0.025)	(0.033)	(0.025)	(0.018)	(0.023)
	[0.025]	[0.009]	[0.027]	[0.036]	[0.025]	[0.018]	[0.023]
Bootstrap s.e. p-value	*0.005*	*0.001*	*0.199*	*0.002*	*0.000*	*0.011*	*0.005*
LASSO & PCA Controls:	Baseline	LASSO Crops	PCA Crops	LASSO Geo	PCA Geo	LASSO All	PCA All
Linear & Quadratic Trend		Yes	Yes	Yes	Yes	Yes	Yes
Observations	116	116	116	116	116	116	116
R-squared	0.876	0.935	0.900	0.927	0.876	0.909	0.878
Mean Dep. Var.	0.114	0.114	0.114	0.114	0.114	0.114	0.114
Number of States	12	12	12	12	12	12	12
Decade Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: The table reports OLS estimates. The unit of observation is the US state in the decade. The panel includes 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. In Panel A we account for crops and in Panel B for a host of other potential confounders. In Panel C we account for all confounders jointly (all crops controls, all geographical controls, and all controls in the last two columns), using high-dimensional LASSO method and principal component analysis. Regression in panel C also add linear and quadratic state-specific time trend to the set of LASSO and PCA controls. All the regressions include decade fixed effects and state fixed effects. Robust standard errors double clustered at the decade and state level are reported in round brackets, standard errors clustered at the state level are reported in squared brackets. P-values for the null hypothesis (Malaria Stability x Post1690 = 0) computed with wild bootstrap standard errors are reported in italics. ***, **, * indicate significance at the 1%, 5%, and 10% levels respectively (related to standard errors clustered at the state level).

which has no positive effect on the outcome. Baseline results are confirmed. Finally, major economic and political events taking place in the United Kingdom around 1690 might affect different states differently if the sailing distance from UK varied significantly across the US colonies. Being located north or south of Nantucket, Massachusetts, represented the biggest factor determining the sailing distance from the UK.[71] For this reason, in Column 1 of Panel C we account for a time-varying effect of being positioned south of Nantucket.

Ideally, we would like to jointly account for all these other potential drivers. However, given the large number of fixed effects and the limited amount of observations, a similar regression cannot be estimated. The high-dimensional LASSO method presented by Belloni, Chernozhukov, and Hansen (2014) permits us to account contemporaneously for all confounders presented in previous tables, allowing for a principled search of meaningful controls. As an alternative approach, using principal component analysis, we can extract the first component of our very rich set of time-varying controls. We apply the two strategies on two sets of controls: one containing only crop-related controls from Panel A (Columns 2 and 3), the second set containing only geographic controls (Columns 4 and 5), and the third set containing all controls from Panels A, and B (Columns 6 and 7). In addition to these sets of controls, we also add state-specific linear and quadratic time trends to the set of controls fed to LASSO and PCA. Results reported in Panel C - Column 1 - show the baseline estimate on the subsample of interest (Columns 1), next the estimates including selected controls from the LASSO estimation (Columns 2, 4, and 6) and the first component of the principal component analysis (Columns 3, 5 and 7). The estimated coefficients for specification including all controls range between 0.054 (LASSO) and 0.089 (PCA). Although not always as precisely estimated, estimates confirm results throughout different samples, control sets, and methods. Taking at face value coefficient estimates of Column 6 Panel C (LASSO), malaria accounts for about 40% of the difference in the higher share of African laborers, which emerged after 1690 between South Carolina and Pennsylvania, and about one fifth of the difference that emerged between Virginia and Pennsylvania.

Robustness Checks In the Appendix we show that results are confirmed when: accounting for crops that had limited or no importance during colonial times (see C5), using alternative malaria indices (Table C6), and accounting for colonies with scarce population or population concentrated on the coast (Sections C.3.4 and C.3.5). Appendix Section C.3.3 discusses the role of potential measurement errors in the data, replicating results with revised data from McCusker and Menard (2014). Appendix

[71] Historians pointed out that the Gulf Stream dictated sailing distance from the motherland: on the one hand were New England and nearby regions and on the other hand were North Carolina and the Chesapeake Bay region. Nantucket represented the dividing line of travel, to the point that a difference in destination of a degree in latitude necessitated a completely different and longer route.

Section C.3.7 presents a spatial randomization analysis, where we randomly assign a Malaria Stability value to each state 1,000 times and compare results to our baseline effect.

Instrumental Variable Estimates The instrumental variable results are summarized in Table 7.[72] Our predicted measure of malaria incidence is weather anomalies in malaria-suitable states; our proxy measure for weather anomalies (common to all states) is the number of El Niño events registered in the decade, and our predicted measure is the interaction term between El Niño events $\#El\ Niño_t$ and the Malaria Stability in the state MS_c. As a proxy for actual malaria incidence, we construct a variable – *Falciparum Malaria* – indicating for each state when historical evidence documents *falciparum* malaria's appearance.[73] Because the variable *Falciparum Malaria* might be measured with error and might be endogenously driven by spurious factors, we instrument it with our predicted measure. The results show that weather anomalies in more malaria-suitable states significantly predict malaria deaths, when looking at later decades for which malaria mortality data are available (Panel A, Out of Sample Evidence).[74] As expected, weather anomalies in the highly malaria-suitable states also predict the variable *Falciparum Malaria* (Panel B, First Stage). Exploiting our predicted measure of malaria incidence, we show that weather anomalies in the more malaria-suitable states are associated with a sizable increase in the share of Blacks (Panel C, Reduced Form). Finally, instrumenting the variable *Falciparum Malaria* with weather anomalies in the more malaria-suitable states, we find instrumental variable estimates in line with the OLS ones (Panels C, OLS, and IV).

Malaria Introduction and Slave Codes With the expansion in the number of Africans in the colonies, states started to approve legislation aimed at regulating the legal status of Black laborers, generally bringing about a reduction in the liberties of African laborers. The process culminated in the approval of "slave codes," comprehensive sets of laws that attempted to define slave status and sanction once and for all its elementary characteristics.[75] Given that slaveholders often served as colonial legislators, the approval of slave codes in the US colonies can be used as a proxy of a greater demand for African labor. We thus look at the effect of malaria *falciparum* introduction on the likelihood of approval of a "slave code." Table C11 reports the linear probability model (LPM) estimations exploring the likelihood of approval of slave codes as the dependent variable. The results show that *falciparum* malaria increased by 22 pecentage points the likelihood of approving a slave

[72]Given that the instrumental variable analysis relies on variation arising from multiple weather fluctuations, we present results using the full sample until 1780.

[73]See Appendix Table C2 for details on data construction and sources.

[74]Note that higher soil suitability for rice, as expected, does not predict higher malaria deaths.

[75]Note that property rights in man were well defined in the West Indies before the codes were approved in the US colonies. Appendix Section C.1.2 presents additional background material on the legal status of African workers in the US colonies and details the sources used to construct the variable.

Table 7: Malaria and the Share of Blacks – Instrumental Variable Estimates

	PANEL A **OUT OF SAMPLE EVIDENCE**		PANEL B **FIRST STAGE**	
Dependent Variable:	**Malaria Deaths (100s)**		**Falciparum Malaria**	
Malaria Stability × # El Niño Events	3.266***	2.443**	0.068***	0.078**
	(0.952)	(0.898)	(0.019)	(0.026)
Rice Suit. × # El Niño Events		0.691		-0.016
		(0.430)		(0.025)
Bootstrap s.e. p-value	*0.095*	*0.098*	*0.002*	*0.061*
State Fixed Effects	Yes	Yes	Yes	Yes
Year Fixed Effects	Yes	Yes	Yes	Yes
Observations	84	84	169	169
R-squared	0.553	0.569	0.802	0.804
Mean Dep. Var.	2.455	2.455	0.237	0.237

	PANEL C			
Dependent Variable:	**% Blacks**		**% Blacks**	
	REDUCED FORM		**OLS**	**IV**
Falciparum Malaria			0.229***	0.223***
			(0.047)	(0.032)
Malaria Stability × # El Niño Events	0.015***	0.016**		
	(0.005)	(0.005)		
Rice Suit. × # El Niño Events		-0.001		
		(0.004)		
Bootstrap s.e. p-value	*0.003*	*0.063*	*0.000*	*0.002*
State Fixed Effects	Yes	Yes	Yes	Yes
Year Fixed Effects	Yes	Yes	Yes	Yes
Observations	169	169	169	169
R-squared	0.803	0.803	0.888	0.888
Mean Dep. Var.	0.118	0.118	0.118	0.118

Notes: The table reports OLS and IV estimates. In Panel A, the dependent variable is the number of malaria deaths (in the hundreds) in the state/decade for which data are available (1850-1860-1870-1900-1910-1920-1930). In Panel B, Falciparum Malaria is an indicator variable taking value 1 if *falciparum* malaria is endemic in the state/decade. In Panel C, 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. # El Niño Events is a variable counting the number of El Niño episodes registered in the decade. All regressions include decade fixed effects and state fixed effects. Robust standard errors clustered at the state level are reported in round brackets. ***, **, * indicate significance at the 1%, 5%, and 10% levels respectively (related to standard errors clustered at the state level).

codes, about a 50% increase in probability with respect to the mean.

4 Malaria and African Slavery: Uncovering the Micro-Motives

After having looked at the macro patterns, this section attempts to bring to the surface such underlying economic motives that malaria shaped. According to our main hypothesis: malaria concurred to the spread of African slavery because it affected the relative profitability of the available sources of labor: European workers, African slaves and Native American slaves. On the one hand, malaria decreased the availability of free (European) workers, on the other hand, the disease engendered a demand for malaria-resistant labor.

4.1 Malaria, Whites and Native Americans

Working in malaria-ridden environments exposed workers to sickness and death. This made free laborers harder to recruit and more expensive to employ, engendering a Domar-type effect (Domar, 1970). In addition to this, malaria did not expose all workers to sickness and death in the same way. Because of a history of exposure to the disease, certain African populations had developed resistances to the disease (see Section 2.3), which European and Native Americans completely lacked.

As addressed in Section 2.3, Native Americans lacked immunities to malaria. It is not surprising therefore to observe that, even when accounting for state fixed effects, in 1860 we observe a negative correlation between Native American population and Malaria Stability (left panel Figure 5, extended specifications available in Appendix Table B8). Whites also suffered in malaria-infested areas. After the Civil War and the ban on slavery, Whites left more malaria suitable counties.[76] The right panel of Figure 5 plots the association between the change (between 1870 and 1860) in population of Whites and Blacks, and Malaria Stability across counties: while Whites fled, Blacks stayed (extended specifications available in Appendix Section B.4.4).[77]

4.2 Malaria Resistance and the Prices of African Slaves

This section shows that malaria also made certain workers comparatively more productive, documenting slave owner preferences for malaria-resistant labor. Finding a malaria premium in slave prices suggests that malaria resistance conferred a productivity advantage to individuals with higher resistance to the disease. Indirectly, and most importantly, it proves the salience of the disease for agricultural labor choices in the United States.

4.2.1 Historical Background

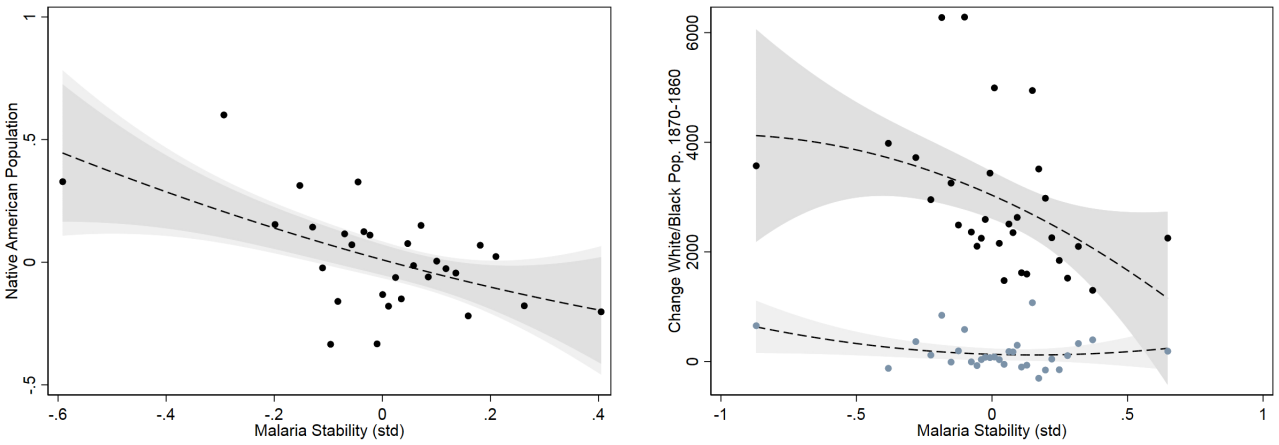
This section tests whether resistance to malaria granted higher productivity in malaria-infested areas. Given that, among the African workers shipped to North America, individuals born in African regions with more malaria had a higher stock of immunities to the disease, we examine whether they were sold for higher prices in the transactions occurring in the Louisiana slave market.[78]

[76] Before the ban on slavery, wealthy whites avoided malaria-ridden areas during the malaria season. As already discussed in Section 2.3, since pre-colonial times and throughout the nineteennneth century, slave-owners left the plantation during the malaria months. As McCandless suggestively documents: "By the early nineteenth century, the gentry's seasonal abandonment of their plantations had become so prevalent that the Episcopal Church ceased to provide Sunday services in some parishes for at least five months".

[77] Appendix Table B7 shows that there is no correlation between the free Black population and malaria, neither in 1790 nor in 1860.

[78] 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

Figure 5: Malaria, Native Americans, Blacks and Whites



(a) Native American Population 1860

(b) Change in Black/White Pop. 1870-1860

Note: Left Figure. Binned scatterplot mapping the association between malaria suitability and Native Americans Population (hyperbolic sine transformation) in 1860. Right Figure. Binned scatter plot mapping the association between malaria suitability and the change in white (black dots) and black (blue dots) population between 1860 and 1870, i.e. $Delta_c = Whites_{c,1870} - Whites_{c,1860}$ and $Delta_c = Blacks_{c,1870} - Blacks_{c,1860}$.

A large body of anecdotal evidence documents the fact that colonists preferred certain African groups over others, and that health played an important role in the shaping of these preferences. 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 (1983) 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." 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.[79] 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

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 differentials resulting from characteristics such as age, sex, and skills. 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).

[79] Certain diseases, such as smallpox, had clearly identifiable symptoms while the symptoms of malaria were more difficult to identify.

we know that malaria was in fact the major disease of the lowlands. On top of this, malaria-resistance conferred general health advantages, since malaria tends to weaken the immune system and individuals resistant to malaria infection are much less vulnerable to several other diseases.

4.2.2 Data

We 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 (2005). This 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 the analysis.[81] We aggregate all the available information at the African *country* level. This allows us to maximize sample size as for almost half of the individuals we only know the country of birth.[82] Most importantly, 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] 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). We exclude individuals in the sample with ethnicity not clearly traceable to a single modern country.[84] Moreover, we perform a vast set of robustness checks, excluding individuals whose place of origin can be only imperfectly matched to a modern country.

Further information about data construction, variables, and sample selection are detailed in Appendix Section D.1.1. For our baseline specifications, we assemble a final sample composed of 3,186 individuals sold in the Louisiana slave market between 1741 and 1820, born in 21 different African countries.[85]

[80] Individuals born in Africa represent more than half of the individuals for whom we know the place of birth.

[81] Details for the mapping are summarized in Section D.1.1 of the Online Appendix.

[82] For more than 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. For about one-third of the records, the ethnicity of the slave is provided, whereas city or geographical location for the place of birth is indicated for about 10% of individuals. For the remaining records, we 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 Sierra Leon, Windward Coast, and Rice Coast. Narratives in (Wax, 1973), who studies US slave owners' preferences by place of origin, show that slave owners referred to the origins of their slaves with terms such as Calabari, Gold Coast, Wydah, Gambia, Angola, and Congo. Hall (2005) claims that, wherever the ethnic origin of the slave was specified, it was information provided by the slave him- or herself.

[84] In particular, we 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.

[85] The average selling price in the sample is equal to 570 US dollars. The majority of the transactions involved male individuals (69%), and the average age was 29. Note that baseline controls are available for 3,175 individuals, from 20 countries. And this represents the baseline sample of interest.

4.2.3 Estimation and Results

Turning to our empirical exercise, we propose the following baseline specification:

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

where the dependent variable is the natural logarithm of the price for the individual i sold in year t, born in African country c, located in region r. The main variable of interest is the level of Malaria Stability in the country of origin of the enslaved individual $MS_{c,r}$.

Individual controls $\mathbf{X_{i,c,r,t}}$ include age, age squared, a dummy variable taking value 1 if the slave is a male, type of sale transaction, and the language of the document that registered the transaction. Document type refers to the kind of transaction – i.e. sale, estate sale, seizure for debt – and allows to compare the same type of transaction, while document language fixed effects allows us to compare transactions executed under the same regime jurisdiction (French, Spanish, and US).

We add an extensive set of country-level controls $\mathbf{Z_{c,r}}$, to account for other factors that might influence productivity and be correlated with malaria. All the specifications include three macro African regions fixed effects, to avoid comparing slaves born in overly distant locations.[86] Moreover, since the health of individuals enslaved from Africa was also influenced by the hardship of the long voyage, we control for the length of the journey from the African country of origin. Moreover, slave origin might have mattered for physical and cultural characteristics beyond health, such as body size and agricultural skills.[87] We therefore control for several proxies for (i) body height and size; (ii) country suitability for agriculture, and in particular for rice cultivation; and (iii) agricultural skills or type of social organizations prevalent in the African country of origin of the enslaved individual. Finally, we control for various factors that might have impacted slave traders' production costs.[88]

Results Table 8 summarizes the main results. 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). To get a sense of the magnitude, this also implies that going from the 25th to the 75th percentile of Malaria Stability in the country of origin leads to a predicted price that is 10%

[86] The three macro-regions are Upper Guinea, Bight of Benin, and Western and Southeastern Africa.

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

[88] For instance, following Nunn and Puga (2012) and Fenske (2015), we control for ruggedness and average temperature.

higher, which is a very large effect, broadly about half of the difference between the prices for a male and a female. 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.[89] Furthermore, the relationship is not driven by outliers, see Appendix Figure D2.[90] The coefficient of interest is little affected by the inclusion of controls which, if anything, tend to increase the size of the estimated coefficient.

Table 8: Malaria in the Country of Birth and Slave Price

	Ln(Slave Price)					
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	0.063***	0.056***	0.076***	0.070***	0.075***	0.085***
	(0.015)	(0.013)	(0.020)	(0.015)	(0.019)	(0.022)
Bootstrap s.e. p-value	*0.005*	*0.014*	*0.013*	*0.002*	*0.004*	*0.019*
Male Slave	0.194***	0.194***	0.195***	0.194***	0.194***	0.193***
	(0.021)	(0.021)	(0.022)	(0.022)	(0.022)	(0.022)
Slave Age	0.048***	0.048***	0.048***	0.048***	0.049***	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		0.026***				0.030***
		(0.009)				(0.007)
Distance Atlantic Markets			0.032**			0.035***
			(0.012)			(0.008)
Land Suitability				-0.011		-0.017
				(0.009)		(0.014)
Average Rice Suitability					-0.011	-0.006
					(0.011)	(0.020)
Region & Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Document Language & Type FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	3175	3175	3175	3175	3175	3175
R-squared	0.446	0.446	0.446	0.446	0.446	0.447
Mean Dep. Var.	6.177	6.177	6.177	6.177	6.177	6.177

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 enslaved individual. 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 enslaved individual, the square of the age and whether the enslaved individual is a male. "Region" fixed effects are indicator variables that define three African macro-regions. "Year" fixed effects are indicator variables for the year of the contract. "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. Standard errors are clustered at the country level (21 clusters). Given the number of clusters, we 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.

In Table 9 Panel A, we show that country of birth higher exposure to trypanosomiasis and yellow fever, measured as suitability to the tsetse fly and Aedes Aegypti respectively, is not associated with any premium in price. These results suggest that the effect is related to malaria, and not more generally to health conditions in the country of origin. In Panel B we show that results hold when controlling

[89] Both controls have a positive effect on the slave price, possibly reflecting a selection effect in the subset of slaves who survived as far as the Louisiana plantations.

[90] In Appendix Figure D3 we show that there is no clear relationship between temperature and precipitation in the country of origin and slave prices.

for possible determinants of differences in production costs, such as temperature and ruggedness. In Panel C of the same table, we control for proxies of historical agricultural skills, while in Panel D we account for historical population density and state antiquity. The inclusion of these controls has little effect on our coefficient.

Table 9: Accounting for Production Cost Human Capital

Dependent Variable:	Ln(Slave Price)								
	PANEL A **Other Deseases**		PANEL B **Production Costs**		PANEL C **Agricultural Skills**		PANEL D **Human Capital**		PANEL E **All**
Malaria Stability	0.069***	0.089***	0.064***	0.066***	0.060***	0.081***	0.056**	0.054***	0.144***
Cluster (s.e.)	(0.016)	(0.021)	(0.013)	(0.013)	(0.018)	(0.028)	(0.024)	(0.019)	(0.037)
Bootstrap s.e. p-value	*0.007*	*0.008*	*0.002*	*0.002*	*0.002*	*0.064*	*0.015*	*0.045*	*0.051*
Yellow Fever Suit.	-0.006								-0.016
	(0.008)								(0.012)
TseTse Fly Suitability		-0.021*							-0.073
		(0.011)							(0.070)
Ruggedness			-0.011						0.065
			(0.008)						(0.043)
Avg. Hist. Temperature				0.017*					0.087
				(0.009)					(0.090)
Historical Croplands Cover					0.011				-0.021*
					(0.009)				(0.011)
Transition to Agriculture						-0.014			0.003
						(0.008)			(0.033)
Ln(Population in 1400)							-0.007		0.018
							(0.016)		(0.025)
State Antiquity								-0.010	-0.053**
								(0.011)	(0.021)
Observations	3175	3175	3175	3175	3175	3175	3175	3169	3169
R-squared	0.446	0.446	0.446	0.446	0.446	0.446	0.446	0.446	0.448
Mean Dep. Var.	6.177	6.177	6.177	6.177	6.177	6.177	6.177	6.177	6.177

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 enslaved individual. 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, the square of the age, the sex, "Region" fixed effects, "Year" fixed effects, "Document Language" fixed effects, "Document Type" fixed effects. Standard errors are clustered at the country level (21 clusters). P-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors are reported in italics. ***, **, * indicate significance at the 1%, 5%, and 10% levels respectively.

We control for differences in body size in two ways. First, we assemble the available data on historical body heights for different African countries/ethnic groups. As a second way, we construct a variable indicating whether a famine/drought took place during the first two years of the life of the individual slave in his/her African country of birth. Our results, reported in Table 10, show that (i) being born in a county during a drought indeed reduces the price of the individuals enslaved years later in the Louisiana slave market, suggesting that our price measure captures some underlying productivity; and (ii) the inclusion of these controls leaves the coefficient of interest unaffected.

Robustness Checks and Additional Results Appendix Table D5 reports specifications including a vast list of geo-climatic controls, that could be correlated with Malaria Stability. In Appendix

Table 10: Accounting for Height and Body Size

Dependent Variable:	**Ln(Slave Price)**				
	PANEL A		PANEL B		
	Height		**Body Size**		
Malaria stability	0.054**	0.074**	0.066***	0.063***	0.062***
Cluster (s.e.)	(0.018)	(0.025)	(0.017)	(0.017)	(0.016)
Bootstrap s.e. p-value	*0.015*	*0.037*	*0.011*	*0.013*	*0.013*
Slave Height (Country/Ethnic Group)		0.028			
		(0.026)			
Famine in Childhood (2 Years)				-0.035	
				(0.020)	
Drought in Childhood (2 Years)					-0.039***
					(0.012)
Observations	2564	2564	3159	3159	3159
R-squared	0.453	0.453	0.448	0.448	0.448
Mean Dep. Var.	6.177	6.177	6.177	6.177	6.177

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 enslaved individual. 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, the square of the age, the sex, "Region" fixed effects, "Year" fixed effects, "Document Language" fixed effects, "Document Type" fixed effects. Standard errors are clustered at the country level. P-values for the null hypothesis, i.e. Malaria Stability = 0, computed with wild bootstrap standard errors are reported in italics. ***, **, * indicate significance at the 1%, 5%, and 10% levels respectively.

Table D6 we use a reconstructed version of the Malaria Stability index, that uses only historical climatic variables and ignores variation arising from different *Anopheles* vectors. Table D7 explores the correlation between prices and modern measures of genetic malaria resistance, namely sickle-cell trait, G6PD, and Duffy antigen. Furthermore, results summarized in Appendix Tables D8 and D9 show that baseline findings do not depend on any specific choice in the mapping of individual origins to countries. Results are confirmed when excluding all major indicated places of origin and, one by one, each country in the sample.

We further explore whether the malaria *premium* changed before and after the ban on the importation of individuals from Africa (1808). Table D10 reveals that the effect is much stronger in the years after the ban, consistent with the idea that until 1808 slave traders could partially adjust to meet the demands of planters. Finally, we show that a malaria *premium* existed for transactions of French, Spanish, and English-speaking slaveowners (Table D.4.2).

Slave Quantity 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 individuals raided. We look at the total number of individuals enslaved in the New World by African country and show that countries with higher Malaria Stability were more massively exposed to the trade (Table D12).

5 Conclusion

The novel empirical evidence of this paper documents the contribution of diseases, and notably malaria, to the diffusion of African slavery in the Southern United States. The spread of the disease, and the demand for malaria-resistant labor that it engendered, made African labor more economically attractive in malaria-infested areas and concurred to its massive expansion. We first showed that African slavery was predominantly concentrated in American counties that were more suitable for malaria transmission. Second, looking at the period surrounding the introduction of *falciparum* malaria into the United States in a difference-in-differences exercise, we showed that the introduction of the particularly virulent strain of malaria caused the number of African workers to soar. Third, looking at the historical prices of African slaves sold in the Louisiana slave market, we show that individuals from certain African populations were preferred because of their higher malaria resistance.

This paper brings to the surface some of the underlying economic motives that the geography of malaria shaped. Clearly, for the establishment of African slavery in the American South, underlying economic motives interacted with the cultural and political contexts of the Atlantic World at the time, where the forced enslavement of individuals was a politically and morally viable option. In this sense, this paper is silent about the cultural and political context that made it possible for slavery to prevail as a system of labor and social organization. At the same time, it shows that the geography of malaria, by affecting the relative economic profitability of African slave labor, interacted with that given context, and to a certain extent shaped it.

While the victory of the North in the American Civil War meant that the enslavement of individuals was at last banned, malaria transmission continued to be a burden in the US South. How did the labor market and the southern economies adjust to the end of slavery and the continued burden of the disease? Did enfranchised Blacks attempt to flee malaria-infested lands? Did they start to receive higher wages, or did widespread intimidation and violence by land-owners prevent this from happening? Future research will have to address these important questions connected to this historical phase.

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