Abstract: This dissertation endeavours to examine long-lasting sequels of malaria on economic development in sub-Saharan Africa. Despite the general consensus that countries where malaria is endemic are not only poorer but also grow economically at a slower pace than non-malarial ones, the channels through which malaria incidence translates into lower subsequent development are still under-examined. The aim of this thesis is assessing one of such channels, namely cognitive impairment. According to our hypothesis, malaria sufferers during childhood who survive see their cognition impaired because of the disease, which is reflected into lower human capital as adults and, consequently, affects economic outputs, not only at the individual level but at the aggregate one as well. By using Pearson’s Correlation Coefficients and bivariate and multivariate regressions, this study has found initial support for the hypothesized channel in sub-Saharan Africa. According to our preferred estimates, a one standard deviation increase in malaria ecology is associated to a 1.18 points reduction in IQ and a one-point increase in IQ would be associated on average, to a $268 increment in GDP per capita, when controlling for education. Moreover, results suggest a potential indirect effect of malaria on education via cognition as well. While remaining cautious about our results, we suggest that improvements in human capital need not be in the form of more education but rather in the form of better health outcomes that would allow for better-quality education by improving, for instance, cognition.

Keywords: malaria, health, cognition, IQ, economic performance, income, GDP per capita
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1 Introduction

«Where malaria prospers most, human societies have prospered least» (Sachs & Malaney, 2002).

For a long time, one of the greatest public health achievements has been the response to malaria at the global level (World Health Organization, 2017). However, in the decade of the 1990’s, despite the efforts by the World Health Organization, an estimated 300-500 million people were infected with malaria annually, with around 1.5 to 2.7 million deaths yearly, a million of which happening among African children under five. By then, malaria was indigenous in around a hundred countries with more than 40% of global population exposed to some degree of risk (Novelli, 1993; World Health Organization, 1997). Deemed sometimes as a “disease of place”, malaria transmission relies heavily on geographical and ecological conditions and happens when a female Anopheles mosquito takes a blood meal from a person infected with the parasite and then after some time takes another meal from a different individual, transmitting it to the latter. Because of the role played by the vector mosquitoes, while a century ago tropical, subtropical and temperate climates favoured its spread, nowadays it is mainly concentrated in the tropics, where warmth and humidity likely sustain the endemicity of malaria. Indeed, 90% of the burden is accounted for by sub-Saharan countries, home to three of the most effective vector mosquitos, and where the predominant strain, Plasmodium falciparum, is one of the most fatal compared to the ones prevailing in other continents (World Health Organization, 1997; Gallup & Sachs, 2001; Sachs & Chambers, 2009; Bleakley, 2010b).

Malaria has been shown to be highly related to poverty conditions. As Sachs and Malaney (2002) make clear in their statement, those areas showing high poverty levels happen to be almost the same as those framed by malaria transmission. Countries where malaria is endemic are not only poorer but also grow economically at a slower pace than non-malarial ones, as discussed by existing literature (McCarthy et al, 2000; Gallup & Sachs, 2001). Over the last decades, health economics research has primarily focused on the distribution of disease burden, as well as on the economic aftermath of the HIV epidemics. Although to a somewhat lesser extent, it has also examined economic consequences of other tropical infectious diseases such as malaria, especially in sub-Saharan Africa. Even though there is general consensus on the
deleterious effect of malaria on the economy, particularly through its detrimental impact on human capital, the underlying mechanisms to which researchers have pointed are wide-ranging and disentangling them is at least challenging. Despite these mechanisms have been identified at the theoretical level, the channels through which malaria morbidity\(^1\) translates into lower subsequent development are still under-examined. This lack of attention to the precise channels might be hampering the implementation of efficient public policies to fight off malaria or even to foster economic development, which could be better targeted with the improvement of the understanding of linkages between malaria and development.

Although some of the symptoms, namely cough, nausea, diarrhoea, fever or vomits, may be confused with those of a virus, malaria can lead to far more severe complications than those of a simple virus. One of the most arduous is cerebral malaria which occurs mainly among children aged under five, and leads to permanent neurological damages (Novelli, 1993). This suggests important consequences for cognitive development that can translate into long-lasting effects on the economy at the aggregate level. Thereby, this thesis hypothesizes that one of the channels whereby malaria has an impact on sub-Saharan underdevelopment at the aggregate level is cognition. Malaria sufferers during childhood who survive see their cognition impaired because of the disease, which translates into lower human capital as adults and, consequently, affects economic outputs, not only at the individual level but at the aggregate one as well. We use the exogenous variation in malaria as proxied by the malaria ecology index (MEI) in the 1990’s and intelligence quotients (hereon, IQs) as of late 2000’s or early 2010’s in each of the countries under assessment to analyse the potential effect of the former on the latter. Then the correlation of IQs with economic performance as measured by GDP per capita and average annual growth in the period spanning 2010-2016 is assessed. According to our hypothesis, malaria levels to which sub-Saharan children (or unborn babies) in the 1990’s were exposed should negatively correlate with their cognitive abilities as adolescents in the late 2000’s or early 2010’s. Consequently, these should positively correlate with economic development as of 2016, once they are supposed to have joined the labour market as young labour force. Even though literature on the economic consequences of malaria is more abundant and well-founded, so far, the evidence supporting a link between the disease and cognitive abilities is at best suggestive. Moreover, to the extent of our knowledge, despite a crude exercise by Venkataramani (2012)

\(^1\) Morbidity in this context stands for incidence of a disease.
with Mexican data, no such literature linking both the effect of malaria on cognition to subsequent development exists. Thus, this work contributes to a growing body of knowledge providing evidence of the long-lasting effects of malaria on the economy, relating early-life exposure to the disease to adulthood outcomes through an often-disregarded channel.

Our core findings suggest a negative correlation between malaria exposure during childhood and adolescent cognition. They also suggest a positive correlation between IQ and later economic performance. Nonetheless, when considering education as a further measure of human capital, results suggest that the effect from cognition to per capita GDP might go through enrolment as well. Better cognitive abilities derived from lower malaria exposure might turn into higher enrolment rates. This can yield significant policy implications. For instance, aiming at increasing school attainment by means of higher-quality teaching in schools might be useless if afflicted children are unable to attend school or if their cognitive abilities prevent proper learning. A health and nutrition programme might however yield better results if it improves children’s ability to concentrate. Nonetheless, we call for caution when deriving conclusions from these results since, although, a priori, they seem to be in line with the proposed channel, we cannot interpret them as causal but rather as mere correlations. Further research should deepen in this question controlling for additional channels to isolate the real impact. It would also be appropriate to use alternative cognitive skills measures, not available so far at the country level.

Therefore, this thesis will research the intertwined relation between malaria and economic development via human capital. Concretely, we will try to provide evidence of the correlation between malaria during childhood and cognition and the subsequent economic development, given that this is precisely one of the infectious diseases that hits the youngest population segment the most. Untwisting this channel is utterly important provided that young survivors can carry along malarial pernicious effects to the economy even decades after the disease affliction, being this the reason for it to be our focal point.

The rest of the paper is structured as follows. Section 2 provides theoretical background on the topic. It also surveys an extensive review of existing literature about health outcomes and economic development, malaria and economic development, and, finally, about IQ, health and economic development. Section 3 discusses the data. Section 4 describes the research strategy and the empirical foundations on which it is grounded. Section 5 presents the results and section 6 discusses them and concludes.
2 Theory

This section will assess existing literature from both a theoretical and an empirical standpoint. First, theory dealing with the relation between health and economic outcomes will be reviewed, assessing some of the possible channels whereby health affects income. The scope will be narrowed down to the focus of this thesis, the potential effects of malaria on income through cognitive impairment, and our theoretical approach will be examined. Finally, a literature review of previous studies dealing with similar research questions will be provided.

2.1 Theoretical Approach

Seminal works by Lucas (1988) and Romer (1990) contributed to the Endogenous Growth Theory by acknowledging the crucial role of human capital as a major determinant of economic growth. Even though most of growth studies incorporating human capital focus on education, human capital is a much broader concept that includes training, health or migration, among others (Gyimah-Brempong & Wilson, 2004). Indeed, the role of health as a form of human capital has long been recognized. For instance, the Ben-Porath (1967) mechanism stands as a popular channel through which improvements in health spur economic development. This postulate proposes that when life expectancy, a general measure of health, increases, one would expect education investment to increase as well. However, despite the general agreement on the importance of health for human capital and subsequent economic performance, the relation between health and economic outputs is difficult to unravel and many mechanisms have been discussed. We will provide some of the most recent theory examining such relation and will focus on malaria specificities afterwards.
2.1.1 Economic Theory and Health

Health is both a measure of a country’s stage of development and a contributor to income. It is a sort of human capital itself and an input for producing further types of human capital. The relation between health and income is complex and difficult to detangle. To begin with, as we already stated, health is a measure of human capital which is by nature a multidimensional concept and must be treated as such (Bleakley, 2010a; Weil, 2014). Different measures have been used in literature to assess the relation of health with economic outcomes. Life expectancy at birth, Years Lost to Disability, Disability Adjusted Life Years, and individual health indicators such as fraction of women who are not anaemic, birth weight, age of menarche or height, among others, are some of the measures commonly used to portrait health. An additional measure of health that has been proposed in the literature is intelligence, an aspect determined by both biological features and education. The part related to biological health has been on the rise over time in developed nations. Such phenomenon is usually referred to as Flynn Effect and is often regarded as a consequence of improvements in nutrition and health over time. More concretely, this effect is reflected by the IQ, a measure of intelligence which, by construction of the tests used to assess it, should only reflect skills not acquired in schooling (Weil, 2014).

Secondly, the nature of the statistical correlation between health and income broadly evidenced in the literature depends on the setting. To begin with, health and income can be thought of as being determined simultaneously, health is a function of income while income is as well a function of health. According to Weil (2014), three kinds of forces would induce the positive correlation between these two variables. First, factors other than health, such as availability of natural resources or non-health aspects of human capital, that have an impact in income lead to an increase in health because of the raising income. Second, factors other than income, namely changes in the disease environment, induce a positive impact on income because of its positive effect on health. Third, external factors that affect both health and income, for instance institutional quality, technology or education, would yield a positive correlation between both variables even if there was no causality running from one to the other. Moreover, the author emphasizes the multiplier effects that derive from this two-way causality. An exogenous factor raising income directly would lead to a further health-induced increase in income because of the positive relation running from health to income. Overall, the whole debate about the role of health in economic growth can be boiled down to how sensitive health is to changes in income and how sensitive income is to changes in health. However, there is good reason to think that the relation between health and income varies depending on the different causal channels that
shape it in the different contexts. Thus, the response of income to health improvements need not be the same across countries or even within each country at different points of time.

Another important aspect to account for in health economics theory is the dynamics in the relation between health and income, that is, the timing of each of them. Health improvements many times do not happen immediately as a response to a better health environment. For instance, health conditions during childhood or even prior to birth strongly affect adult health and, therefore, have an impact on labour input. An example is given by height which is crucially determined by in-utero and up to four years old conditions. Moreover, according to the Barker Hypothesis, ill health during adulthood, particularly chronic diseases after reproductive age, is associated with foetal malnutrition (Almond & Currie, 2011). This is also the case of in-utero insults like congenital rubeola syndrome, maternal smoking, foetal alcohol syndrome or childhood diseases like cerebral malaria that can leave life-long scars. Another example of the importance of gestation period for adult health outcomes is intelligence as pointed by Bleichrodt and Born (1994), who suggest that iodine deficiencies in such period lead to reductions in adult IQ. Furthermore, many studies have highlighted the positive effects on educational attainment, intelligence, wages and height derived from better childhood nutrition. However, the dynamics of health economics are sensitive to the extent to which adult health is a combination of the health environment during childhood and during adulthood. The speed at which health improvements in the environment are translated into adult health is determined by such combination. The relevance of this lies on the fact that adult health is the one that matters the most for output (Weil, 2014).

On the other hand, improvements in health as a result of improvements in income can also happen with delays. The improvement of public health infrastructure is an important contributor to health increases. The rise in national income leads to an increase in public health infrastructure expenditure but often with lags. What is more, often, economic growth can have deleterious effects on health in the short term. For instance, international or even inter-regional migration usually brought along by economic growth can impact health negatively. As an instance, the spread of the old-world diseases after the discovery of the Americas translated into millions of deaths. In like manner, urbanization brings people into contact with infectious agents new for them and favours the spread of diseases because of the collection of food and waste in cities.
A third consideration regarding timing in the dynamics between health and economic growth is population growth. When mortality is high, parents decide to have more children to compensate for many new-borns not reaching adulthood, thus increasing fertility. According to the demographic transition theory, when there is a decline in mortality the positive response of fertility is not immediate. The delay in the fertility response leads to a spurt of population growth. This is precisely behind the claim by Acemoglu and Johnson (2007) that per capita income declines as a result of reduced mortality, the negative economic impact of population growth overweighs in their study the benefits form better improved health. This is also in line with Ashraf et al (2008) as shown in the following section.

But the dynamics in the effects of health on income depend on the mechanism of transmission, meaning the channels whereby health (disease) conditions might have an impact on economic outputs. Such channels are well documented in literature. A review of these possible channels is provided by Weil (2014). Based on several empirical studies, the author identifies several channels of which productivity stands as the simplest one. Healthier workers are both physically and mentally more effective and adults who used to be healthy children will presumably have acquired more human capital via more education. In this regard, Bleakley (2010b) also acknowledges two ways by means of which health affects adult productivity. Firstly, being sick now reduces one’s ability to work now. The second way pertains to the fact that people are more willing to invest in human capital during childhood if this is not going to be idled because of sickness during adulthood. Another channel identified by Weil (2014) is longevity: living longer raises the return to investments in human capital, and, therefore, schooling. However, the author, based on existing empirical evidence, acknowledges that it is not the main determinant of raising schooling. Additionally, decreasing mortality, both adult and infant, may lead to parents to have less offspring, given that the precautionary childbearing can be reduced. Because of this they might even decide to invest more in their children’s human capital. Also, poor health can impact economic outcomes because of the short-sighted behaviour of the afflicted. If the perception is that the probability of using one’s savings is low due to high mortality, physical capital accumulation will be negatively affected. This is the reason for claiming that higher life expectancy can lead to higher national savings.

Nevertheless, despite the important role given to investment in human capital this is not the only view. Bleakley (2010b) casts doubts on those studies that focus on schooling as the only outcome of childhood health. Following him, childhood health affects both returns to schooling
and schooling opportunity costs, wages. Healthier children might directly earn more in the market simply because of the fact that they have a better health, which would raise the opportunity cost of schooling. Parents might, therefore, send children to work if they are healthier because they are already more productive. However, better health can improve the quality of education received as children can learn better. Thus, parents might decide to provide them with more education. The final impact on years of schooling is, thereby, ambiguous. What is more, the author approaches the issue by means of Envelope Theorem. Applied to this issue, the theorem assesses the effect of a small change in health on optimal lifetime income. The result would imply that health improvements would affect income by making human capital more productive rather than by increasing investment. Therefore, lifetime income would increase because being healthier during childhood permits to grow up stronger and learn faster. Changes in schooling time, this means in quantity of education, would not be of first-order importance. The main implication derived from this theorem is that those studies measuring the impact of health on years of schooling are measuring impacts that are not utterly important to income. Furthermore, the author goes on asseverating that, according to the Envelope Theorem, changes in health lead to changes in the quality of education received which, in turn, have first-order importance impacts on income. This is line with Barro (1998) who supports the view of those claiming that education quality matters more than years of schooling for subsequent economic growth.

In this regard, Bleakley (2010b) points to the usefulness of measuring health impacts on lifetime income via other outcomes such as literacy or IQ tests. The exposure to disease environments at young ages may influence cognitive abilities since some infections pose an impediment to brain development, which may lead to cognitive impairments throughout life. Early exposure to such diseases may even result in speeding cognitive decline at older ages (Case and Paxon, 2009). Children’s cognitive abilities are influenced by both environmental and health-related conditions. Risk factors during infancy are particularly important given that the first years of life are a crucial period of rapid development (Berkmant et al, 2002). In the concrete case of disease environments, one might think of these as environmental conditions that, if afflicted by the disease, might turn into health conditions. In this regard, the parasite-stress hypothesis recently proposed by Eppig et al (2010) states that human beings at their early stages of development will find impediments to brain growth if they have to fight off infectious diseases. From a metabolic standpoint, the brain is very energy-demanding, especially in new-borns and young children. When affected by infectious diseases, children’s brains’ energetic demands will
not be met and thus, cognitive development will suffer. Moreover, when exposed to disease environments, human beings may develop a permanent immune function that constantly requires more energy even when they are healthy. This is in line with the theory by Fogel and Costa (1997) who argue that reducing the incidence of infectious diseases renders more energy available for work since less energy is devoted to mobilizing the immune system and the gut can absorb nutrients better. This would favour the efficiency with which energy input is converted to work output. Applied to the case of children morbidity and following Eppig et al (2010), this would mean that fighting off infectious diseases comes at the expense of other energy-costly traits such as intelligence.

On the other hand, and in line with the hypothesized channel of malaria effects on income through cognition, there is a well-established body of theory, data and literature providing support for IQ as a determinant of income among individuals. Since childhood IQs are strong predictors of adolescence IQs and these, in turn, determine adulthood earnings, it follows that national IQs determine national incomes, being this so well-founded that could be deemed as almost a law (Lynn & Vanhanen, 2009). People with high IQs may acquire complex skills that are remunerated with high earnings. Thus, those countries with high IQs have efficient economies, with skilled and semi-skilled workers that produce high-value goods and services with strong international demand that cannot be produced by low IQ nations. Those countries also have efficient and intelligent public personnel such as teachers or scientists, who strengthen their economies, or even politicians who effectively run such economies. Accordingly, skilled management of the economy is paramount for economic growth and development (Lynn & Vanhanen, 2009). Moreover, Jones (2011) proposes an even larger effect of IQ on income at the national level than at the individual one given the human capital spill-overs. Four are the channels that support his hypothesis. To begin with, intelligent individuals are said to be patient, which translates into higher saving rates at the national level. Secondly, they are deemed to be risk-tolerant, which makes them more cooperative and propitious to get closer to the Pareto optimum in the economy. Thirdly, high group intelligence is complementary to sophisticated

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2 In their Theory of Technophysio Evolution, Fogel and Costa (1997) identify thermodynamic and physiological factors that influence economic growth. The latter relates to how human beings work as engines that transform energy input into work output.
and high-value technologies for production. Finally, intelligent individuals are associated with supporting better economic policies.

As depicted in Figure 2.1 and according to the previous statements, disease exposure during childhood would have a direct effect on income during adulthood through its effect on cognition. However, the potential effect on adult income through its impact on schooling would be indirect. Indeed, the effect through schooling of second-order importance given that, as suggested by Bleakley (2010b), it might well be the case that schooling decisions are based on cognition, which is affected by the exposure to the disease. As we can see, aside from affecting adult health, early-life exposure to illness affects human capital accumulation. The truthfulness of this stylized fact lies on two generally accepted statements. Firstly, much of an individual’s development, both physiological and cognitive, takes place during childhood (direct effect). Secondly, economic theory proves that investments in human capital should be made during one’s early life (indirect effect). These two statements are represented as the direct and indirect effects in the figure. The figure also makes clear that timing stands as an essential aspect to take into account when assessing the correlation between disease exposure or health improvements and economic development through their effects on intelligence.

Despite the mechanism of which Figure 2.1 is well-grounded, the literature on which it is based is quite recent and other strategies have been traditionally used to deal with the issue at hand, usually disregarding the mechanism of transmission and assessing simply the impact of the health (disease) indicator on the economic outcome. For instance, a common macroeconomic approach has focused on simulation models which allow for comparisons of disease-free scenarios versus actual disease scenarios, calculating this way the impact of diseases (Cuddington & Hancock, 1993; Cuddington, 1993; Jefferis et al, 2008). Arndt & Lewis (2000) provide a similar approach by using a general equilibrium model while some others make use of Solow model style regressions augmented with human capital measures (Gyimah-Brempong & Wilson, 2004; Grimard & Harling, 2004) or production function models (Bloom et al, 2004). Some additional pieces of work simply perform regression analyses were the economic indicator, usually GDP per capita or GDP growth, is regressed on an indicator of the prevalence of the disease under assessment, while controlling for other covariates that may also have an impact on economic development (see Dixon et al., 2002 for a brief review).
Nonetheless, a major concern usually arises from the analysis of the impact of the disease environment on economic performance, namely endogeneity or reverse causality. As we have pointed, better health has a positive impact on income through several channels, but it is also the case that the higher the income, the higher the health given that more resources are devoted to the prevention and cure of diseases, providing the population with higher quality health services and goods. Additionally, there might be factors affecting both diseases and economic performance or any other dependent variable, such as irresponsible governments or geographical factors, posing a threat to the causal effect of the former on the latter (Ahuja, 2010; Bleakley, 2010a). Because most of the studies disregard the complexity of the channels and mechanisms of transmission, endogeneity arises. Several approaches have been traditionally used in the health economics literature to deal with this issue. Bloom et al (2004) try to overcome the problem by making use of lagged values of regressors and regressands to measure the effect of life expectancy on economic growth. Likewise, Gyimah-Brempong and Wilson (2003) use lagged values of endogenous regressors to ensure that they can be treated as predetermined and avoid serial correlation in error terms. Also using IV, Ahuja (2010) estimates the impact of HIV prevalence on economic measures by instrumenting the disease with male circumcision rate. As an alternative although somehow similar approach, Lorentzen et al (2008) propose a structural model identifying the causal links between economic growth, the mechanisms linking it to mortality, and the mortality indicators. By using a large set of instruments, including malaria ecology indicators, they estimate a simultaneous equations system through three-stage least squares.
2.1.2 Epidemiological Literature: Malaria

The theory exposed above broadly applies to different measures of health and different disease environments. However, the impact of health changes on income depends on the kind of change and when it takes place, being the study of different diseases subject to particularities. Therefore, assessing the particular channels through which malaria can affect adult income deserves some attention.

Some of the channels through which malaria can yield long-lasting consequences, are, for instance, the burden of medical costs, since resources devoted to children that do not survive are considerable, or foregone adult income because of lost workdays. In addition, changes in household behaviour such as schooling, fertility, savings and migration decisions might also happen. High infant mortality leads parents to have more children and probably to invest less in their education. This can even change dependency ratio since there will be more children at any time than those expected to survive into adulthood. Furthermore, women employment opportunities are also reduced because they have to be at their children’s disposal which can translate into sizable consequences for productivity and economic growth. Moreover, if the disease has an epidemic character it can bring about trade, foreign direct investment or even tourism consequences since economic linkages are suppressed and malarial countries are avoided for the fear of contracting the disease. The most discussed channel is, nonetheless, as generally done in health economics literature, school attainment since malaria favours school absenteeism because of high children morbidity. A final channel, acknowledged in literature but not given much attention, is cognitive impairment in afflicted children (Sachs & Malaney, 2002).

Several are the channels through which a child’s cognitive development can be affected because of malaria. To begin with, unborn children from afflicted pregnant women might suffer from their mothers’ anaemia and interruption of nutritional transmission, which, in turn, affect their cognitive abilities, just as the physical ones. Secondly, suffering severe malaria during childhood might lead to cognitive impairment. Indeed, one of the most arduous complications derived from the disease is cerebral malaria, which back in the 1990’s occurred in 1% of the cases, mainly among children aged under five, and leads to permanent neurological damages (Novelli, 1993). Thirdly, malaria sufferers in childhood may also see their posterior development affected given that advanced cognitive development relies on previous development. Thus, the full damage is not perceived until higher-order functions are required.
at later stages of development at older ages. Finally, and in a more indirect way, children not
affected themselves by the disease but who have a sick relative are as well exposed to its
consequences, given that household income allocated to nutrition or schooling can be reduced,
with the subsequent effect (Lucas, 2010). The importance of childhood exposure to malaria lies,
therefore, in the fact that most of one’s human capital development happens in the early-life
stage, being the damage often hard to reverse (Bleakley, 2010b). Despite all the theoretical
foundations about these channels, the evidence supporting the existence of a link between early-
life exposure to malaria and subsequent cognitive abilities is sparse.

2.2 Previous Research

Based on the theory we have examined, this section will review empirical literature dealing
with the effect of different diseases on economic development and will narrow down the scope
to focus then on malaria studies. Then, literature assessing the effects of diseases on cognition
and how cognition relates to economic development will be surveyed.

2.2.1 Diseases and Economic Development

Literature assessing the association between economic development and health is neither new
nor scarce. There is a broad stream of literature trying to give an answer to how much disease
hampers economic development in the aggregate. However, there is broad heterogeneity in the
effects found in literature and so far, no consensus has been achieved on the implications of
better health on income (Currie & Vogl, 2013). Research studies in this field have been carried
out at the macro and micro level and at country, regional and cross-country level. While micro
empirical studies provide compelling evidence, they move away from the initial question. On
the other hand, while macro studies are more tightly connected to such question, they provide
less compelling evidence (Bleakley, 2010b). Before focusing on macro approaches to the issue
more interesting for the study at hand, a brief micro literature review is presented.

By surveying and following up South African HIV/AIDS-affected and unaffected households,
Bachmann and Booysen (2003) find that the income of households affected by the disease was
found to be 12% lower at baseline than that of non-affected households, and so were
expenditures. Employment rate among households with at least one member affected was also
found to be lower than for those where no member is affected. On the other hand, Bleakley (2007) assesses the impact of a deworming campaign in South America in the 1910’s. By means of micro data, the author finds a considerable gain in income coinciding with the campaign exposure, as well as an increase in the return to schooling. Likewise, the deworming programme among Kenyan schools analysed by Miguel and Kremer (2004) not only increased school attendance among participants but also created positive health and schooling externalities for untreated pupils. Their results lay bare positive externalities or benefit spill-overs from health, something that usually is not given much attention.

The macro literature is, however, much broader, both from a theoretical and an empirical approach. Weil (2007) estimates the wage returns to health at the micro-level to then derive the effect of macro-level health outcomes on GDP per worker. According to the author’s preferred estimates, getting rid of health differences would close the income per worker gap between poor and rich countries by 9.9%. In like manner, estimating a production function model, Bloom et al (2004) find out that improvements in health have a positive impact on economic outputs not only through productivity but also via a positive influence on savings and capital accumulation, as well as returns to schooling, which is consistent with Weil (2014). Likewise, Zhang and Zhang (2005) construct a growth model building on an overlapping generations approach. They theoretically show how increased life expectancy has a negative impact on fertility, and a positive on saving and schooling decisions. On the other hand, Gyimah-Brempong and Wilson (2004) estimate an augmented neoclassical growth model considering health human capital separately from other kinds of human capital, namely education. The stock of health is proxied as the inverse of child mortality as well as with life expectancy, while investment in health is assumed to be a proportion of income. Results show that both the investment in and stock of health human capital have important positive effects on per capita income growth. Also, by means of an augmented Solow style growth model, Grimard and Harling (2004) find that countries bearing a higher burden of tuberculosis grew at a slower pace than those less afflicted. Every 10% additional incidence is said to be associated with between 0.2 and 0.4% lower growth. By adopting a different approach, Lorentzen et al (2008) try to answer their research question with a structural model using 3SLS and malaria ecology as an instrument. They find that fertility and investment are stronger channels through which mortality can impact economic growth than human capital accumulation.
Quite an important amount of studies, on the other hand, deals with the HIV/AIDS epidemic economic consequences. All in all, regardless the channel through which it takes place, most of studies in this field find a negative impact of HIV/AIDS on economic growth in African countries (Over, 1992; Cuddington, 1993; Cuddington, 1994; Arndt and Lewis, 2000; Bonnel, 2000; Barings, 2000; Sackey & Raparla, 2000; Dixon et al, 2001; James et al, 2001; Sackey & Raparla, 2001; Dixon et al, 2002; Jefferis et al, 2008). Additionally, there is a broad branch of literature assessing the total economic costs derived from both productivity losses and treatment costs to the society. Some of these studies concern neglected tropical diseases and compute the final economic burden considering a cost-effectiveness approach (Torgerson, 2003; Carabin et al, 2005; Garg et al, 2008; Budke et al, 2005; Waters et al, 2004; Blas et al, 2006; Frick et al, 2003).

Nonetheless, the dominant view of the positive (negative) effects of health (diseases) on economic outcomes has been challenged by a line of thought led by Acemoglu and Johnson (2007), who claim that the final effect on GDP per capita can be the opposite. Using data about different diseases prior and posterior to the epidemiological transition, these authors suggest that an increase in life expectancy due to health improvements might not be sufficiently compensated by a decrease in fertility leading to a rise in population and a decline in GDP per capita. From a different approach, by building on microeconomic estimates to run a simulation model, Ashraf et al (2008) provide further support to this standpoint. The effects of changes in health measured as improved life expectancy or disease eradication are only visible quite long after they take place and they are surprisingly small, according to these authors’ results. The former case would yield a reduction in income of about 5% over the following 30 to 40 years, only raising per capita income around 15% in the long term. The latter case, represented by eradicating either malaria or tuberculosis, would lead to a small 2% increase in GDP per capita in sub-Saharan Africa in the long run. In the case of malaria, in the short term, GDP per capita would even shrink. Similarly, Young (2005) predicts a hopeful future scenario for South Africa in which the economy is endowed with more resources because of the HIV epidemic. Two opposite effects counteract according to his findings. On the one hand, the accumulation of human capital suffers a detrimental effect. On the other hand, fertility decreases. The latter is found to predominate and, according to the author, the positive economic effects derived from lower population outweigh even the worst forecasts on human capital loss. Thus, in his own words the epidemic “is a humanitarian disaster of millennial proportions […] It is not, however,
an economic disaster”. This view would fit within the line of thought headed by Acemoglu and Johnson (2007).

As we have seen, studies assessing the impact of health (disease) on the economy are wide-ranging and results mixed.

2.2.2 Malaria and Economic Development

At the micro level, the effects of malaria on economic development have primarily focused on impacts on educational attainment. Using micro data from malaria eradication campaigns in Sri Lanka and Paraguay, Lucas (2010) exploits the differences in school attainment between cohorts born prior to, during or after the epidemic period to see the effect of malaria on female school attainment. The differences-in-differences approach yields a significant and negative effect of malaria on completed years of education and literacy. Moreover, those regions with initial less exposure to malaria experienced smaller gains in terms of education than those more infected. However, the author does not explore posterior effects on income. Similarly, Bleakley (2010a) assesses the extent to which childhood exposure to malaria reduces labour productivity by using data of eradication campaigns in the US (circa 1920) and Mexico, Brazil and Colombia (circa 1955). The author provides evidence that children born after the implementation of the eradication campaign had higher income and literacy as adults than previous cohorts exposed to higher malaria. However, the effect on schooling is mixed, which is consistent with the fact that childhood health possibly affects both the return to schooling and its opportunity cost, being the final effect of better health on years of education unclear. Likewise, Cutler et al (2010) assess the impact of malaria eradication exposure during childhood on economic status and human capital accumulation in India. Male exposed to the campaign as children are found to have better household economic status than those not exposed. However, no effect is found on educational attainment, which might be due to the trade-off between labour and schooling as pointed by Bleakley (2010a; 2010b).

In addition, several have been the studies that have estimated the production loss due to malaria morbidity using the marginal product of labour (Gazin et al, 1988; Ettling et al, 1991; Sauerborn et al, 1991; Leighton & Foster, 1993; Ettling et al, 1994; Asenso-Okyere & Dzator, 1997; Guigemde et al, 1997). Other studies have focused on the economic burden that dealing with malaria poses at the household level (Mills, 1994; Onwujekwe et al, 2000).
On the other hand, malaria research carried out at the macro level is scantier. McCarthy et al (2000) find that malaria enters standard growth regressions with negative sign and significantly, once controlled other factors. Pronounced growth reductions due to malaria are robust to adding several controls as well as sub-Saharan and Latin American region dummies. Gallup and Sachs (2001) use a malaria intensity index to see whether malaria causes poverty and curbs economic growth and find that malarial countries only have 30% of the income of those not afflicted by the disease. Moreover, after controlling for initial income and human capital stock, life expectancy and policy and geographical factors, they find that malaria leads to a 1.3% reduction in growth. Accordingly, those countries affected in the totality of their territories by malaria could raise their annual growth by 2.6% if they completely eradicated the disease. Also, from a macro approach, McCord et al (2017) use the malaria ecology index to instrument for child mortality and study the positive effect of this on fertility. The main caveat of their results is the fact that malaria impacts on fertility through several channels, not only through mortality. One of such channels is said to be effects on children’s cognition, which might then lead parents to provide them with more or less education and change the price of children’s quality.

Regarding precisely the potential effect that malaria exposure might have on cognitive abilities, despite the acknowledgement of this hypothetical channel, most of studies focus on something else but cognition, being few the pieces of research that examine it. Holding et al (1999) try to reveal the long-lasting effects of malaria on cognitive abilities in Kenyan children given that in the 1990’s cerebral malaria was one of the main causes of acute encephalopathy in African children. Results reveal that children who had suffered severe malaria present difficulties in language development, behaviour and attention. The authors also point out the possibility of other cognitive damages not evident at the age the individuals were surveyed and followed up. Moreover, they suggest that if such damages happen to be irreversible, differences between treatment and control groups could be expected to be even greater at older ages and further stages of development. Nonetheless, they do not examine subsequent income consequences, therefore exploring only the first stage of the mechanism we propose. From a different perspective, Venkataramani (2012) exploits the implementation on a malaria eradication campaign in Mexico to test the hypothetical effects of early-life exposure to malaria on later cognition. The author finds that birth year exposure to the campaign led to better results in the cognition tests in adulthood, although, similarly to Bleakley (2010b), he finds no significant impact on schooling. Additionally, in a crude exercise, the author shows that the improvement in cognition might account for up to 20% of the improvement in consumption. This study is, to
the extent of our knowledge, pioneer in attempting to link both the effect on cognition and the effect on economic outcomes.

2.2.3 Cognitive abilities, Diseases and Economic Development

The theory previously exposed is fostering two branches of literature dealing with the effect of diseases on cognitive abilities, on the one hand, and relating IQ as a further measure of human capital to economic outcomes, on the other hand. To the extent of our knowledge, there is no such literature that quantitatively links both to see how diseases impact cognitive abilities and how this translates into effects on economic outcomes, even more so if we concretely focus on malaria.

Regarding the first branch of literature, A paramount study to our research is the one by Eppig et al (2010). They find that their measure of disease burden, which accounts for the years of life lost and spent disabled as a result of a total of 28 infectious diseases, correlates negatively with cognitive abilities. Not only it is statistically significant but also remains the main predictor of IQ when other variables, namely temperature, distance from sub-Saharan Africa, average years of education or GDP are controlled for.

Nevertheless, studies of the effect of different infections on cognitive abilities are anything but recent. Nokes et al (1992) already studied the impact that parasitic worm infection had on Jamaican children in the late 1980’s. Results showed a significant improvement in cognition after the removal of the infection. Sakti et al (1999) also provide similar evidence that helminth infection in children can translate into negative sequelae for reading comprehension and reasoning ability with data from Indonesian school children. Berkmant et al (2002) surveyed and followed up a cohort of Peruvian children in the early 1990’s and found that malnutrition during infancy is related with cognitive function in later stages of childhood. Furthermore, Smith et al (2006) find evidence that children who experienced AIDS at early ages have significant cognitive impairment relative to HIV free children or even to HIV positive who did not developed AIDS. Building on the work by Miguel and Kremer (2004), Ozier (2014) deepens in the externalities of the Kenyan case of deworming. The cognitive impact of the campaign on children who did not receive the treatment but whose community did equals that of increasing schooling between 0.5 and 0.8 years. This effect almost doubles when considering children whose siblings did directly receive the treatment. Because infection was reduced, those surrounding the treated benefited nearly as much as the dewormed. By exploring the disease
exposure of children in the first half of the 21st century in the US, Case and Paxton (2009) provide further grounds for believing that early-life exposure to disease environments is correlated with cognitive performance at older ages. Particularly, malaria is found to be associated to the inability to count backward and is also negatively correlated with educational attainment. In like manner, by means of the first national IQs estimates (Lynn & Vanhanen, 2002) for 1976, Barber (2005) assesses the correlation of these with education attainment and health as proxied by infant mortality rates and low birth weight. The sign of the preliminary correlations points to a negative effect, as expected, providing support to the hypothesis we are proposing.

Concerning the second and more recent branch of literature, Jones and Schneider (2006) provide evidence that IQ can be considered a useful human capital measure which collects a crucial portion of human capital stock that presents an economically large and statistically robust positive correlation with economic performance. Similarly, running an augmented Solow’s growth model, Ram (2007) provides evidence that IQ is a strong measure of human capital whose effect on output growth is statistically significant. Moreover, IQ seems to be a stronger predictor of economic growth than health or education. Just like the previous authors, Meisenberg (2012) finds evidence supporting IQ as the strongest predictor of economic growth, being also a better measure of human capital than exposure to education. 23% of differences in economic growth are, according to Meisenbergs’s (2012) results, attributable to IQ differences. Such results are robust and not likely to be caused by the chance of coincident IQ and economic performance. Also, by means of Lynn and Vanhanen’s (2012) National IQs dataset, Hafer (2017) provides further support for the view that IQ plays a major role in economic growth, adding to this evidence the effect that it also has on welfare, which considers additional quality of life factors. IQ effects on growth and welfare stand as economically significantly meaningful. The author insinuates that, while the gains from higher IQs may have already translated into higher welfare and GDP in developed countries, there is still some room for improvement in developing nations. Hanushek and Woessmann (2008) provide further evidence of the effect of cognitive skills rather than school attainment on individual earnings and economic growth, even when other factors come into play. In addition to the previous, Lynn and Vanhanen (2012) review a myriad of studies in which their IQ measure correlates with numerous economic and social phenomena. A bunch of studies positive and significantly correlate IQ with various measures of per capita GDP. The channels through which this translates into higher growth,
namely effective economic institutions, market economies or higher saving rates, support what Jones (2011) claimed about human capital spill-overs.

That IQ has a positive and significant impact on economic growth has been, as proved in previous paragraphs, highly discussed in literature, being such relation taken as a law for some. However, it is more seldom to find studies assessing the different impact of different IQ classes. This is precisely what Burhan et al (2014) do. In accordance with their division of population according to IQ, the intellectual class, those gifted with higher intelligence, seems to contribute more to economic growth than the average-ability population or the non-intellectual class. They also find that such result applies to technological progress.

As we can see, literature so far explores either the first stage of the hypothesized mechanism or the second one. In other words, existing studies research either the effects of diseases on cognition, or simply on schooling, or the impact of cognition on economic outcomes. Even though we find support for both stages, we have not find any attempt, except for the crude exercise by Venkataramani (2012), to link both, being this our reason to try to fill this gap in the literature.
3 Data

This chapter will critically discuss the data used for this dissertation. In addition to show the reliability of data sources, the validity of each of the three main variables used is assessed and the representativity of the sample is discussed.

3.1 Source Material

Information on economic performance, demographic situation and disease environment has been gathered for sub-Saharan countries. Even though the region is made up of a total of 47 nations according to the United Nations, data are not available for all of them, an issue which will be discussed in the following paragraphs. Most of the information regarding economic outcomes is only updated to 2016, and rarely 2017, which is the reason for using the former in our analysis when we refer to current performance. The main data source used for this research has been the World Development Indicators downloadable from the World Bank’s Databank. This compiles indicators from a variety of sources. The ones we will be using are GDP per capita in 2016 in 2011 PPP constant US$, gross enrolment rate in secondary education in 2011, and mortality rate for children aged under 5 per 1,000 live births in the 1990’s. All of them but the first one will be used as additional controls to the main analyses. Enrolment rate is defined by the UNESCO, the source that compiles such information, as the “ratio of total enrolment, regardless of age, to the population of the age group that officially corresponds to the level of education shown”. On the other hand, mortality rate, under 5 is defined as the “probability per 1,000 that a new-born baby will die before reaching age five” according to the UN Inter-agency Group for Child Mortality Estimation. Average economic growth for the period 2010-2016 has been computed as the difference of logarithms using GDP per capita in 2011 PPP constant US$, which is the gross domestic product converted to US dollars keeping the purchasing power parity. Additionally, those variables not compiled in this dataset, namely country-level cognitive abilities and malaria ecology, were looked up in alternative sources.
Regarding IQ there is no such an indicator that provides the IQ for each country and year. Lynn and Vanhanen’s (2006) estimates represent the main attempt to gather all available information on cognitive abilities worldwide to create a dataset of global intelligence distribution. These authors provided measured IQ’s for 113 countries and estimated 79 more using information on IQ in neighbouring nations. Such data corresponded to several surveys that sampled most often teenagers and were primarily based on the non-verbal Raven’s Progressive Matrices (Meisenberg, 2012). Such values were then normalised to British IQ standards of 100 by 1976, and, later on, to those of 1992 (Lynn & Vanhanen, 2009). Current values could be deemed as representative of the actual country level, given the revision performed in 2012 on the original data by Lynn and Vohannen (2012) themselves, and that corrected for the upward secular trend predicted by the Flynn Effect (Lynn & Meisenberg, 2010). Thus, these values could be deemed as representative of national cognitive abilities in the late 2000’s or early 2010’s. Even though it is an approximate measure, we would not expect national IQ to vary significantly in a short time span. This measure is not free of criticisms and alternative values have been proposed. In our case we will be using the NIQ dataset (Becker, 2017) which contains two final measures. The first measure is simply the latest version of Lynn and Vanhanen’s IQ calculations, updated in 2012. The second one is an updated version of the original Lynn and Vanhanen (2006) estimates carried out by Becker (2017), who uses the largest additional information available to revisit the calculus for some countries. We will be using this as an alternative measure, but the focus is on the original updated estimates. As already mentioned, data were compiled and updated using a variety of sources, different for the different countries. However, the validity of the data has been proven in a wide range of studies showing a high correlation between IQs and test scores in maths, science or reading. Moreover, Lynn and Meisenberg (2010) compare this measure of intelligence quotient with educational attainment and find that the correlation is quite high. Thus, the measure seems to present a high degree of validity. The authors suggest that both IQ and education attainment are equally good measures of human capital and can be interchanged since they predict each other, although as previously mentioned, some literature even considers IQ as a better measure of human capital. In addition, environmental conditions seem to affect IQ scores, which brings support to the analysis we are to perform (Lynn & Vanhanen, 2012).

On the other hand, Malaria Ecology Index data come from the dataset developed in both McCord (2016) and McCord et al (2016). The data are publicly available in the form of the average ecology for the period 1960–2010. However, the country-level year-by-year data are
Models of malaria dynamics based on how the distribution of the dominant vector mosquito, Anopheles, interacts with climate are used to construct a spatial index of malaria transmission stability. The index is an updated version of the original index in Kiszewski et al (2004) that considers a 0.5-degree spatial grid to determine the monthly temperature and precipitation to construct a yearly value for each of the countries assessed for the period spanning 1900-2010. Such climatic conditions determine whether the parasite can develop in the mosquito vectors, and how long the incubation period takes, this means, whether the habitat is propitious for the parasite to become infectious and lead to malaria transmission. The conditions for mosquitos to exist are exogenous to public health interventions given that are solely based on the ecology. While interventions can break the transmission cycle, they cannot get rid of the vector. In addition to considering climatic factors, the Malaria Ecology Index combines indicators of different vectors’ presence and their corresponding human biting rate. The index we will be using is population weighted so that it accounts for potential higher variance in smaller countries (McCord et al, 2016).

3.2 Descriptive Statistics

Summary statistics for the main variables used in the analysis are provided in Table 1. The standard deviation seems particularly high for economic outcomes which might suggest the existence of some outliers that might be pulling the mean up or down. This is not quite clear so far since the minimum and maximum values are considerably different, taking into account the fact that GDP per capita is converted to constant prices and considers PPP. Similarly, we could tell about mortality rate under five and enrolment measures. The main shortcoming is that not all variables are available for all countries, being this especially noticeable in the case of

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3 We thank Professor Gordon McCord for making the data available to us.
4 The formula used in McCord (2016) and McCord et al (2016) is the following:

\[
Ecology\ Index = \sum_{m=1}^{12} \frac{a_i \cdot p_{i,m}^F}{-\ln p_{i,m}}
\]

Where \(i =\) grid cell; \(y =\) year; \(m =\) month

\(a =\) proportion biting humans of dominant vector in grid \(i\) and month \(m\) (0 – 1);

\(p =\) daily survival rate of mosquito (0 – 1);

\[E = \frac{\text{degree days for parasite development}}{T - T_{min}} = 11 \frac{1}{T - 16}\] for Plasmodium falciparum

\(T =\) temperature
secondary enrolment rate. If we add to this that those missing values are not always the same for all the variables, in some cases we might face a restricted sample, making it utterly important to remain cautious when assessing the results.

Table 3.1. Summary Statistics

<table>
<thead>
<tr>
<th>Variables</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>Min.</th>
<th>Max.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GDP per capita (2011 PPP constant US$, 2016)</td>
<td>44</td>
<td>4,945.53</td>
<td>6,284.83</td>
<td>647.88</td>
<td>26,319.16</td>
</tr>
<tr>
<td>Average Growth (2010-2016)</td>
<td>43</td>
<td>0.03</td>
<td>0.17</td>
<td>-0.33</td>
<td>0.58</td>
</tr>
<tr>
<td>IQ as calculated by L&amp;V</td>
<td>46</td>
<td>71.25</td>
<td>5.33</td>
<td>60.10</td>
<td>88.00</td>
</tr>
<tr>
<td>IQ as calculated by David Becker</td>
<td>47</td>
<td>70.56</td>
<td>5.68</td>
<td>58.66</td>
<td>89.83</td>
</tr>
<tr>
<td>Average Malaria Ecology Index (1990-1995)</td>
<td>44</td>
<td>6.92</td>
<td>4.66</td>
<td>0.00</td>
<td>15.89</td>
</tr>
<tr>
<td>Mortality rate, under 5 (per 1,000 live births, 1990)</td>
<td>47</td>
<td>156.82</td>
<td>70.18</td>
<td>16.70</td>
<td>328.90</td>
</tr>
<tr>
<td>School enrolment, secondary (% gross, 2011)</td>
<td>33</td>
<td>43.03</td>
<td>20.63</td>
<td>9.07</td>
<td>93.30</td>
</tr>
</tbody>
</table>

Note: N stands for number of observations, SD for standard deviation, and Min. and Max. for minimum and maximum values within the sample, respectively.

To check whether the sample we will be using when performing the analyses differs much from the complete sample, Table 3.2 provides summary statistics restricting the sample only to those observations for which economic outcomes, IQ and malaria data are available at the same time. The mean values do not seem to vary much, although, for most of the variables, standard deviations decreased slightly. We have got rid of several observations, one of which seemed to be driving average GDP per capita up, since the maximum is now somewhat smaller. Another observation with quite low mortality was also put aside. The final sample is made up of 41 countries, although information about school enrolment is only available for 27 out of the 41. From now on, results will be given for this sample, except for when we also include the latter variable, in which case we will use only those observations for which data are available. We do not expect our results to be far from those that an analysis of the 47 countries that make up the full sub-Saharan region would yield. Therefore, we expect our sample to be representative of the full region, but we have to remain cautious since this cannot yet be proved as we make use of all data available so far.
Table 3.2. Summary Statistics with Restricted Sample

<table>
<thead>
<tr>
<th>Variables</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>Min.</th>
<th>Max.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GDP per capita (2011 PPP constant US$, 2016)</td>
<td>41</td>
<td>4,613.95</td>
<td>5,492.43</td>
<td>647.88</td>
<td>24,162.53</td>
</tr>
<tr>
<td>Average Growth (2010-2016)</td>
<td>41</td>
<td>0.03</td>
<td>0.16</td>
<td>-0.33</td>
<td>0.58</td>
</tr>
<tr>
<td>IQ as calculated by L&amp;V</td>
<td>41</td>
<td>70.99</td>
<td>5.02</td>
<td>60.10</td>
<td>88.00</td>
</tr>
<tr>
<td>IQ as calculated by David Becker</td>
<td>41</td>
<td>70.60</td>
<td>5.44</td>
<td>58.66</td>
<td>89.83</td>
</tr>
<tr>
<td>Average Malaria Ecology Index (1990-1995)</td>
<td>41</td>
<td>6.89</td>
<td>4.70</td>
<td>0.00</td>
<td>15.89</td>
</tr>
<tr>
<td>Mortality rate, under 5 (per 1,000 live births, 1990)</td>
<td>41</td>
<td>155.69</td>
<td>67.75</td>
<td>23.00</td>
<td>328.90</td>
</tr>
<tr>
<td>School enrolment, secondary (% gross, 2011)</td>
<td>27</td>
<td>43.63</td>
<td>20.97</td>
<td>14.22</td>
<td>93.30</td>
</tr>
</tbody>
</table>

*Note: N stands for number of observations, SD for standard deviation, and Min. and Max. for minimum and maximum values within the sample, respectively*
4 Methods

This chapter briefly exposes the quantitative framework used to assess the hypothesized channel whereby malaria affects economic outcomes in the long-run.

4.1 Empirical Strategy

The strategy we propose is theoretically grounded on the ideas described in chapter 2. As depicted in Figure 2.1, our strategy establishes that malaria levels in the 1990’s might have affected the IQs of those that recently joined the labour market and whose cognitive abilities were measured when they were adolescents around the late 2000’s or early 2010’s. We will not presume here as Lynn and Vanhanen (2009) that the relation between IQ and per capita incomes is causal and runs from the former to the latter. There are reasons to believe that income might also affect IQ. However, we will assume that, since individual IQ when adolescent determines one’s earnings when being an adult, national IQs will determine national incomes. It follows from the previous that malaria exposure more than two decades ago is likely to still have an impact on present day economic outcomes through its long-lasting effects on human capital by means of permanent cognitive damage.

The empirical approach is, however, different to those reviewed. As already mentioned, the purpose of this research is not finding the causal relationship but rather assessing whether there exists some significant correlation that links past incidence of the disease to current economic performance, giving support to our hypothesised channel through cognition. To begin with, exploratory correlations will be assessed by calculating the Pearson’s $r$ correlation coefficient for the variables of interest. Then, bivariate regressions will be run to complement the previous results. Additional covariates will be added then to check how robust the significance is by means of multiple regressions. More concretely, the models to run are the following:
\[ Human \ Capital_{j,t_1}^i = \alpha_0^i + \alpha_1^i \cdot Malaria \ Ecology_{j,t_0} + \alpha_n^i \cdot X_n \] (1)

\[ GDPpc_{j,t_2} = \beta_0^i + \beta_1^i \cdot Human \ Capital_{j,t_1} \] (2)

\[ Average \ Growth_{j,T} = \gamma_0^i + \gamma_1^i \cdot Human \ Capital_{j,t_1} \] (3)

\[ GDPpc_{j,t_2} = \rho_0 + \rho_1 \cdot Malaria \ Ecology_{j,t_1} \] (4)

\[ Average \ Growth_{j,T} = \sigma_0 + \sigma_1 \cdot Malaria \ Ecology_{j,t_1} \] (5)

\[ GDPpc_{j,t_2} = \pi_0 + \sum \pi_1^i \cdot Human \ Capital_{j,t_1}^i + \pi_2 \cdot Malaria \ Ecology_{j,t_0} \] (6)

\[ Average \ Growth_{j,T} = \phi_0 + \sum \phi_1^i \cdot Human \ Capital_{j,t_1}^i + \phi_2 \cdot Malaria \ Ecology_{j,t_0} \] (7)

Where \( i \) refers to either IQ or enrolment rate as measures of human capital, \( t_0 \) represents childhood (during de 1990’s), \( t_1 \) represents adolescence (late 2000’s or early 2010’s), \( t_2 \) represents adulthood (in 2016) and \( T \) stands for the period spanning 2010-2016. \( X_n \) is a vector of covariates that might be included in the analysis. As equation (1) shows, firstly, we will assess the impact of malaria on both measures of human capital. Even though our focus is on cognition, the entangled relation with education makes it useful to see analyse a potential effect of malaria on this, as well. Later on, the robustness of malaria effects when a different measure of the disease environment is included will be checked. Then, we will assess the effect of the both measures of human capital and malaria, independently, on economic output. Finally, we will see whether the effect of cognition on economic performance is robust when we consider, simultaneously, a potential direct effect of malaria and the effect of education.

To show the hypothetical channel proposed and avoid the aforementioned endogeneity, we will be using the Malaria Ecology Index as a proxy for malaria levels given that by construction it is exogenous to health interventions or economic factors. Built by McCord (2016) and based on temperature and rainfall measurement, its functional form is said to be capturing the part of the dynamics relatively uncorrelated to other processes also determined by temperature and precipitation and that might affect income, as well, for instance agricultural production. This way, it is suitable for regressions of economic performance on this disease’s risk (McCord, 2016). In our case, the channel linking GDP to malaria levels in the 1990’s is the IQ of the current working class, whose development of cognitive abilities might have been affected by
fighting off the disease as children. Given the timing, it seems reasonable to consider that the current IQ levels correspond to those of the generation exposed to malaria ecology back in the 1990’s.

Overall, we will be assessing the two stages that make up our mechanism. First, we will explore the effects of malaria on IQ and schooling. Then we will examine the effects of IQ on economic outcomes, as well as those of schooling as measured by secondary enrolment. Finally, we will see whether the effects of cognition hold when the impact of malaria on economic outcomes is assessed directly, rather than through this channel.
5 Empirical Analysis

Once the theoretical background has been provided and the hypothesized channel has been framed within a particular empirical strategy, results are provided in this chapter. First, a graphical analysis will provide some intuition of the potential correlations between malaria and cognition, and this and economic performance. Then, the quantitative results yielded by the Pearson’s correlation and regression analysis are assessed.

5.1 Graphical Analysis

To try to answer our research question, whether malaria has long lasting impacts on economic development through its effect on IQs, we hypothesised that malaria ecology in the 1990’s had an impact on current levels of IQ, which subsequently affects economic performance. To give support to this potential channel through which malaria detrimental effects stretch on over time, past malaria ecology should be negatively correlated with current IQs. For it to also affect economic development via this measure, IQ should show positive correlation with output measures, per capita GDP and growth over the period spanning 2010-2016, in this case.

A prior analysis consisting in visually displaying IQ and average malaria ecology levels in the period spanning 1990 to 1995 has been carried out. The map in Figure 5.1 provides some intuition that those countries in which cognitive abilities are not so developed, represented by darker green hues, were also those more favourable to malaria occurrence given their ecology some decades ago, represented with bigger spots. Additionally, such potential correlation can be seen in Figure 5.2 which depicts a negative linear trend, being higher levels of malaria ecology correlated with lower IQs. It can also be seen that Mauritius presents an extraordinarily high IQ given its malaria ecology, which might be due to the fact that malaria in Mauritius in the 1990’s was mainly caused by the Plasmodium Vivax parasite rather than Plasmodium Falciparum whose transmission is captured by the malaria ecology index (World Health Organization, 1997). The opposite holds for both Malawi and The Gambia, whose IQ levels are very low and so are their malaria ecologies. This fact makes them three potential outliers.
Figure 5.1. Geographical Distribution of IQ and Malaria Ecology in the 1990’s.
Source: Author’s development. Data from McCord (2016) and Becker (2017).

Figure 5.2. Scatterplot of IQ and Average Malaria Ecology.
Source: Author’s development. Data from McCord (2016) and Becker (2017)
Given the entangled relation between disease environment, cognition and education as described in the theory section, it is useful to evaluate, as well, the association between malaria ecology in the 1990’s and enrolment rates during adolescence in the 2010’s. Recalling from the theoretical framework, parents of children whose cognition was affected by malaria might decide to send them to work if they think that their returns to schooling will be lower. Otherwise, they might send them to school if the opportunity costs of schooling, namely wages at the labour market, are even lower. Figure 5.3 shows, indeed, that the former case might hold since there seems to be a strong negative correlation between malaria and enrolment rates. This would a priori provide support to the potential effect of malaria on schooling through cognition.

Figure 5.4 also shows some potential outliers since Equatorial Guinea seems to have a relatively high per capita GDP given its IQ compared to other countries. The opposite holds for Madagascar, one of the countries with higher IQ but with very low per capita GDP. It can also be seen that those with higher per capita GDP have an IQ superior to 70, being the correlation between these two variables positive and relatively strong, a priori. This, however, is not the case for GDP growth in the period spanning from 2010 to 2015, as Figure 5.5 depicts very scattered observations. The pattern of correlation between growth and IQ is beforehand not clear-cut. Nonetheless, it might be the case that since we are assessing the effects of malaria on those that joined the labour market relatively recently, only the most recent figures of GDP per capita capture such effect. Those whose IQ we are considering have not yet contributed long enough to the economy so as to perceive an effect on growth. It might also be the case that IQ only affects GDP levels, as proposed by Bleakley (2010b).
Figure 5.3. Scatterplot of Enrolment Rate and Average Malaria Ecology.
Source: Author’s development. Data from McCord (2016) and World Bank Databank (2017)

Figure 5.4. Scatterplot of GDP per capita and IQ.
Source: Author’s development. Data from Becker (2017) and World Bank Databank (2017)
5.2 Regression Analysis

Taking into account that the IQs we have represent cognitive abilities of adolescents between 2000 and 2010, we focus on the potential effects of malaria between the years 1990 and 1995. An exploratory study of correlations shown in Table 5.1 suggests a negative correlation between malaria and IQ (see first row). Even though we do not intend to establish causality, it seems reasonable to assume that changes in malaria are associated with changes in cognition, rather than the other way around, given the timing at which the variables were measured. Thus, we can say that countries with a malaria ecology above the mean, have IQ levels below the mean. However, results seem to be non-significant. Only average levels of malaria ecology for the aforementioned period are displayed, provided the high correlation between malaria ecology at different years makes results for each year quite alike. Figures in Table A.1 in Appendix 1, however, show that correlation decreases slightly as we approach 1995, which could mean that malaria levels that affected the most those individuals whose cognitive abilities are represented in our IQ figures are those closer to 1990 rather than 1995. Moreover, an additional inference
can be drawn: the strong, almost perfect, correlation between malaria ecology in different years is nothing but a sign of the endemicity that characterizes such infectious disease.

Table 5.1. Pearson’s Correlations (r)

<table>
<thead>
<tr>
<th></th>
<th>IQ as calculated by L&amp;V</th>
<th>Average MEI (1990-1995)</th>
<th>GDP per capita (2016)</th>
<th>Average Growth (2010-2016)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-0.231</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>GDP per capita (2016)</td>
<td>0.342*</td>
<td>-0.151</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Average Growth (2010-2016)</td>
<td>-0.060</td>
<td>-0.040</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Enrolment Rate (2011)</td>
<td>0.433*</td>
<td>-0.273</td>
<td>0.751***</td>
<td>0.201</td>
</tr>
</tbody>
</table>

Note: * p<0.05, ** p<0.01, *** p<0.001; Number of observations are in parentheses

Similarly, the first column (second and third rows) in the same table contain correlations between IQ and economic performance measured as GDP per capita in levels and average growth in the period spanning 2010-2016, respectively. As one might expect, they correlate positively, meaning that values of GDP above the average are also associated with values of IQ over the mean, although, in the case of growth, these values are not statistically significant. On the other hand, the second column shows that past levels of malaria seem to correlate with the expected negative sign with current economic performance, even though such correlations lack statistical significance, being moreover especially low for growth figures. The correlation between education outputs as measured by enrolment rates in secondary education in 2011 has also been assessed in the last row. Despite having only few observations, enrolment rate correlates positively and significantly at the 5% level with IQ. Recalling from the theory, this might imply that children with higher cognition are provided with more education, pointing to a potential higher effect of disease on returns to schooling than on wages. Indeed, the effect of malaria on education seems to be similar to that on cognition. On the other hand, it seems that enrolment rate is highly and significantly correlated with GDP per capita, although not that much with economic growth.

Nonetheless, as pointed out in previous paragraphs, some observations had quite atypical values, and including such outliers in the analysis could derive in misleading conclusions. Several combinations were attempted, dropping each of the outliers separately at first. The results led to the conclusion that correlations were meaningfully different when all of them, namely Mauritius, Malawi, the Gambia, Equatorial Guinea and Madagascar, were excluded.
from the analysis on the basis of the mentioned above reasons for each of them. The new correlations between malaria ecology in the first half of the 1990’s and IQ are shown in first row in Table 5.2. As in Table 5.1, they correlate negatively although now the value is somewhat higher. Regarding the correlation between IQ and economic performance, the second row in column one shows a positive association between the former and per capita GDP in 2016 in constant 2011 prices, being this slightly smaller than before and non-significant. The correlation with growth seems to be higher now in absolute terms, although still having an unexpected negative sign. On the other hand, the association between malaria ecology and per capita 2016 GDP, displayed in the second column and second row, is still not significant, although the magnitude of the coefficients has almost doubled when removing the effect of the outliers. The effect on growth seems almost negligible. Finally, enrolment rate displays smaller coefficients although similar as before, being the correlation with cognition being now much lower and non-significant.

Table 5.2. Pearson’s Correlations (r) (without outliers)

<table>
<thead>
<tr>
<th></th>
<th>IQ as calculated by L&amp;V</th>
<th>Average MEI (1990-1995)</th>
<th>GDP per capita (2016)</th>
<th>Average Growth (2010-2016)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-0.307</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(37)</td>
<td></td>
<td>(37)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GDP per capita (2016)</td>
<td>0.288</td>
<td>-0.275</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(37)</td>
<td></td>
<td>(37)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Growth (2010-2016)</td>
<td>-0.121</td>
<td>-0.066</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(37)</td>
<td></td>
<td>(37)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enrolment Rate (2011)</td>
<td>0.291</td>
<td>-0.313</td>
<td>0.709***</td>
<td>0.193</td>
</tr>
<tr>
<td>(24)</td>
<td></td>
<td>(24)</td>
<td>(24)</td>
<td>(24)</td>
</tr>
</tbody>
</table>

Note: * p<0.05, ** p<0.01, *** p<0.001; Number of observations are in parentheses

As we have seen, coefficients many times lack statistical significance, especially when we get rid of potential outliers. Tables A.2 and A.3 in Appendix A provide alternative results when using pairwise correlations instead. In that case, the maximum number of observations available from the whole sample is used for calculating each correlation, being the coefficients higher in absolute terms and in terms of significance. All in all, the correlation coefficients have pointed to the direction we expected according to our hypothesized channel. This way, childhood exposure to malaria seems to have a negative effect on cognition and schooling. These, in turn, seem to be positively correlated between them which could be a sign of parents investing more in the education of those whose cognition has not been affected by the disease, and who might have a higher return from schooling, as already pointed by Bleakley (2010b). In addition, both
variables seem to be positively associated with per capita GDP in adulthood. To shed some more light in the issue, regression analyses have been carried out.

To begin with, Table 5.3 provides results of running IQ on average malaria ecology. Column (1) shows, as expected, a negative impact of the diseased environment on cognitive abilities, significant at the 5% level. With respect to the size of the effect, a one standard deviation increase in malaria ecology (4.7 points, see Table 3.2) might lead to a 1.18 points reduction in IQ. Removing the outliers yields quite similar results in this case, with the coefficient now slightly smaller in absolute terms and $R^2$ almost doubling the previous (see column (3)). Column (5) contains the same analyses for enrolment rate as an alternative measure of human capital on which malaria might have had an impact. Contrarily to the previous case, it seems that malaria did not have a significant effect on enrolment rate. This is in line with Venkataramani (2012) who finds no significant impact of malaria on schooling.

Table 5.3. Malaria Effects on IQ and Enrolment Rate, OLS Regressions

<table>
<thead>
<tr>
<th></th>
<th>Panel A</th>
<th>Panel B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IQ as calculated by L&amp;V</td>
<td>Enrolment Rate (2011)</td>
</tr>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-0.25** (0.11)</td>
<td>-0.01 (-0.17)</td>
</tr>
<tr>
<td>Mortality Rate, Under 5 (1990)</td>
<td>-0.03*** (-0.02)</td>
<td>-0.02* (-0.01)</td>
</tr>
<tr>
<td>Constant</td>
<td>72.69*** (-1.28)</td>
<td>76.37*** (-2.15)</td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.05 0.22 0.09 0.15 0.07 0.57</td>
<td></td>
</tr>
<tr>
<td>Outliers Included</td>
<td>YES YES NO NO YES YES</td>
<td></td>
</tr>
<tr>
<td>Number of observations</td>
<td>41 41 37 37 27 27</td>
<td></td>
</tr>
</tbody>
</table>

Note: * p<0.10, ** p<0.05, *** p<0.01; Robust standard errors are in parentheses

On the other hand, the main intelligence measure on which we base our study has a positive impact on GDP per capita, significant at the 10% level, as displayed in column (1) of Table 5.4. A one-point increase in the national IQ might raise per capita GDP in 2016 by almost $374. As one might expect, the effect of malaria is negative, with a one standard deviation increase in the malaria index potentially leading to a GDP per capita reduction of $827.25, although the effect is not significant. If we additionally get rid of potential outliers, the effect of a one-point increase in national intelligence would lead to a $313.72 increase in per capita GDP. The effect of a typical positive deviation from malaria ecology would shrink national income by approximately $1,034 per capita, being this time significant at the 5% level. Nonetheless, these
results should be interpreted as mere correlations given that, probably, adding other covariates more significant for explaining per capita GDP variance would render malaria ecology insignificant. The results for economic growth regressions on IQ and malaria are displayed in columns (6) and (7), respectively. Neither cognition nor disease environment seem to have a significant effect on growth. Indeed, both coefficients are negligible.

Table 5.4. GDP per capita and Economic Growth, OLS regressions

<table>
<thead>
<tr>
<th></th>
<th>Panel A</th>
<th>Panel B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GDP per capita (2016)</td>
<td>Average Growth (2010-2016)</td>
</tr>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>IQ as calculated by L &amp; V</td>
<td>373.89*</td>
<td>267.63*</td>
</tr>
<tr>
<td></td>
<td>(-191.27)</td>
<td>(-150.18)</td>
</tr>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-176.01</td>
<td>-219.92**</td>
</tr>
<tr>
<td></td>
<td>(-110.89)</td>
<td>(-102.77)</td>
</tr>
<tr>
<td>Enrolment Rate (2011)</td>
<td>116.76***</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td>(-31.61)</td>
<td>(0.00)</td>
</tr>
<tr>
<td>Constant</td>
<td>-21,928.39</td>
<td>5,826.05***</td>
</tr>
<tr>
<td></td>
<td>(-13,407.58)</td>
<td>(-1,229.93)</td>
</tr>
<tr>
<td>R²</td>
<td>0.12</td>
<td>0.02</td>
</tr>
<tr>
<td>Outliers Included</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>N. of observations</td>
<td>41</td>
<td>41</td>
</tr>
</tbody>
</table>

Note: * p<0.10, ** p<0.05, *** p<0.01; Robust standard errors are in parentheses

Even though both the correlation analyses and the bivariate regressions have reinforced our hypothesis of a detrimental long-lasting effect of malaria on GDP per capita, we should check the robustness of such correlations to the inclusion of other relevant covariates.

Regarding malaria ecology, Table 5.3 column (2) shows that when adding a measure of mortality rate of children aged under five, its effect on IQ can be considered as null given that the previous significance vanishes. This is a broader measure among which children mortality due to malaria is included and can be deemed as a proxy of the unfavourable and diseased environments in which children in sub-Saharan Africa have to grow up. Life expectancy could alternatively have been chosen. Nonetheless, mortality rate for children under five is more representative of the part of the population we talk about, and life expectancy is much broader, capturing also the effect of those diseases affecting older population. Hence, malaria ecology is rendered non-significant when adding mortality rate given that malaria incidence is somehow already captured by this more comprehensive measure. The effect on IQ when mortality increases by one death per 1,000 live births is a 0.03-point decrease. If we consider instead a
one standard deviation increase (67.75 deaths per 1,000 live births), the decrease in IQ is around 2 points. Using both of them is enough to collect 22% of the variance of national cognitive abilities. Excluding outliers yields very similar results although the drop in the malaria ecology coefficient is not that acute. When switching IQ for secondary enrolment, malaria ecology is again non-significant although the coefficient enters the equation with negative sign. However, mortality rate for children under five enters negatively, significant at the 1% level and with a decline of 16.26 percentage points for each standard deviation increase in mortality. This provides support for the theory claiming that higher mortality raises fertility and decreases the investment in education in each child.

With respect to the effect of cognition on GDP, it does not vanish as we consider a further measure of human capital, namely enrolment rate in secondary education. We should consider these results with caution since scarce data are available and only few observations remain when we consider both covariates. Even though IQ refers to the early 2010’s, we do not think enrolment rates could have changed meaningfully from 2010 to 2011, and since more observations are available for the latter, we present results for this as another measure of human capital. Thus, column (3) in Table 5.4 provides further evidence of the robustness of what column (1) tells us. Both IQ and school attainment seem to capture the effect of human capital on GDP, being both significant even when the other is included in the equation. These could be deemed as the most important results. The effect of a one-point increase in IQ on GDP is now slightly smaller, around $268 per capita, ceteris paribus. The fact that the coefficient declines might be a sign that IQ alone captures part of the effect that should be captured by schooling. These results render IQ as a robust measure of human capital, important for economic performance, and provide further support to the hypothesis of IQ being an important channel through which malaria could have permanent deleterious effects, although, again, we call for caution when interpreting them. Moreover, the fact that the coefficient for IQ is still significant and its size is still considerable might be additional support for its effect to be of first-order importance while that of enrolment might be only of second-order importance as pointed by Bleakley (2010b). The same model is run with economic growth as the dependent variable as displayed in column (8). However, once more, none of the variables are significant and the coefficients could be deemed as negligible. This might be pointing again to an effect not yet visible given that the part of the population whose cognition is represented by our IQs recently joined the labour market. What is more, according to Bleakley (2010b) the usual interpretation in growth regressions is that health has an effect on levels, on the target level of output needed
to achieve such growth, but not growth itself. Therefore, it might be the case that cognition only affects per capita GDP in levels.

A final consideration relates to how the effects of both malaria ecology and IQ on per capita GDP change as we consider them simultaneously. The first column in Table 5.5 shows that the coefficients of both variables are smaller in absolute terms than when we were considering simple linear regressions for each of them. IQ, nevertheless, remains significant at the 10% level, being a one-point increase associated with almost a $355 increment in per capita GDP, ceteris paribus. The effect of malaria in the 1990’s on current economic performance can be deemed as void since we do not reject the null hypothesis that the coefficient is zero at any level of significance. If we add secondary enrolment rate in 2011 to the equation (column (2)), a variable that could be correlated with both of them, IQ remains significant but malaria ecology, not only is again non-significant but its coefficient also changes sign. Again, enrolment rate is highly significant, further supporting the robustness of both this variable and IQ. In addition, the $R^2$ seems to be almost 70%, unfolding again the importance of human capital for explaining GDP per capita variance. In this case, a one-point increase in IQ would be associated, on average, to a $268 increment in GDP per capita, while a 1% increase in enrolment in secondary education would be associated to a $117 increase, ceteris paribus. Even though, these results must be interpreted with caution given the scant available observations, they are basically the same as obtained in column (3) in Table 5.4, where malaria was not included. Considering that schooling decisions might be dependent on children’s cognitive abilities, one might expect the effect of cognition to vanish as we introduce enrolment in the equation. However, not only the effect remains significant, but the magnitude of the coefficient is still considerable. Once more, this might be a sign of the fact that the effect of cognition is of first-order importance while that of schooling, although important, might be of second-order. Regarding growth, however, once again, neither IQ nor malaria seem to have a long-lasting impact on this economic outcome a priori. Neither does enrolment rate in secondary education in 2011. Again, this supports Bleakley’s (2010b) claim about health human capital measures having an effect on levels rather than growth.

If we perform the same analysis but excluding the outliers, results are surprisingly quite different. Column (3) in Table 5.5 shows that when regressing per capita GDP on IQ and malaria ecology, cognitive abilities are not significant any longer while malaria’s coefficient presents the right negative sign, higher in absolute terms and significant at the 10% level. If we
add enrolment rate to the equation (column (4)), both variables of interest are rendered non-significant, presenting the enrolment rate the only significant coefficient. Notwithstanding, only few observations are used for running such model, which makes us call for caution when interpreting these results.

**Table 5.5. Joint Effects: Malaria Ecology Index, IQ and Enrolment**

<table>
<thead>
<tr>
<th>Panel A</th>
<th>Panel B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GDP per capita (2016)</td>
</tr>
<tr>
<td></td>
<td>(1) (2) (3) (4) (5) (6)</td>
</tr>
<tr>
<td>IQ as calculated by L&amp;V</td>
<td>354.71*</td>
</tr>
<tr>
<td></td>
<td>(-201.14)</td>
</tr>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-88.67</td>
</tr>
<tr>
<td></td>
<td>(-110.74)</td>
</tr>
<tr>
<td>Enrolment Rate, Secondary (2011)</td>
<td>117.01***</td>
</tr>
<tr>
<td></td>
<td>(-33.49)</td>
</tr>
<tr>
<td>Constant</td>
<td>-19,956.48</td>
</tr>
<tr>
<td></td>
<td>(-14,274.07)</td>
</tr>
<tr>
<td>R²</td>
<td>0.12</td>
</tr>
<tr>
<td>Outliers Included</td>
<td>YES</td>
</tr>
<tr>
<td>Number of observations</td>
<td>41</td>
</tr>
</tbody>
</table>

*Note: * p<0.10, ** p<0.05, *** p<0.01; Robust standard errors are in parentheses

According to our results, both IQ and schooling matter to explain GDP per capita variance. In fact, their effects are robust to the introduction of malaria to the equation. This would support the idea of malaria having long-lasting effects on economic performance two decades later only through the channels that have been permanently damaged, namely cognition. Malaria ecology in the 1990’s might have influenced cognitive abilities of survivor children afflicted by the disease as shown in previous analyses. Furthermore, parents might then have decided about human capital investment by sending them to school or to the labour market. Considering that the correlation of IQ and the economic outcome at hand diminishes but remains quite important as enrolment rate is added to the equation, we might think that the effect of schooling is not of first-order importance as it could be that of cognition, as pointed by Bleakley (2010b). It might be the case, simply, that enrolment is an outcome itself of cognition. Thus, it would not be a big leap to think that parents of those children not affected might have decided to provide them with more education. Thereby we might expect countries with lower malaria ecologies to present higher enrolment rates. The graphical analysis performed in earlier in this chapter provided further evidence for this. Figure 5.5 clearly showed a negative effect of malarial environments on enrolment rates about a decade later. These results would therefore be in line
with Bleakley’s (2010b) standpoint and Venkataramani’s (2012) results at the micro level in Mexico.
6 Conclusions and Discussion

The aim of this thesis was trying to give an answer to the question of whether malaria has long-lasting impacts on sub-Saharan economic development through its effect on cognitive abilities of survivors affected during their childhoods. It is important to bear in our minds that the effect we have assessed is not immediate, since we allow for some time to pass by between the measurement of the three variables. While malaria ecology is that of the 1990’s, IQs represent the cognitive abilities of adolescents in the late 2000’s or early 2010’s, who were exposed to malaria back then as children. The evidence we have provided with the available data so far supports the hypothesized mechanism: malaria affected cognition of children exposed to the disease environment, which in turns determined schooling decisions and affects economic performance during their adulthood. Although further research is required, both the Pearson’s correlations and the regression analyses here presented point in this direction.

Overall, our results suggest that where climatic conditions were favourable to malaria transmission, IQs were lower. According to our preferred model, this negative correlation is significant at the 5% level and would imply that one standard deviation from a country’s malaria ecology would be associated to a 1.18 points reduction in IQ. However, no significant effect of malaria is found to affect enrolment rates. Similarly, both cognition and schooling are significant to explain the variation in GDP per capita. Indeed, a one-point increase in IQ would be associated to a raise in income of around $268 per capita, ceteris paribus, and a 1% increase in enrolment in secondary education would raise it by approximately $117 per capita, ceteris paribus, when considering both at the same time. These results remain almost unchanged as we introduce malaria ecology simultaneously into the equation. As expected, malaria ecology does not present a significant correlation with current economic performance, being in line with the assumption that, decades later, the only effect prevailing is the one through channels that have been permanently damaged. It seems reasonable to believe that direct impacts of malaria on economic development happen primarily when the outbreaks take place. By that time, the relationship might be bidirectional, implying that economic development can also affect the incidence of the disease via more resources devoted to prevention and treatment of the affected.
But once the outbreak is over, only the effects related to permanent damage on victims’ abilities might extend over the years.

On the other hand, results for GDP per capita growth were not significant in any case, which, as explained by Bleakley (2010b), could be due to the fact that the usual interpretation in growth regressions is that health has an effect on levels, on the target level of output needed to achieve such growth, but not on growth itself. Our interpretation is, however, that it is too soon to notice any effect on growth since the individuals exposed to malaria in the 1990’s must have recently joined the labour market, affecting only the economic performance in the very last years.

Although we call for caution when interpreting these results since the sample is not as large as would be desirable, they seem to point in the direction of our hypothesis. We could consider that IQ comprises all the effect that malaria ecology in the 1990’s could still have on economic outcomes. Despite obtaining some initial hints that the proposed mechanism could be right, adding education to the equation complicates the interpretation. As previously discussed, parents decide on children’s human capital investments according to children’s cognitive abilities. A cognition impairment due to malaria might affect not only schooling returns, since children cannot make the most of education, but also labour wages, since productivity is also affected. Depending on which effect predominates, parents might decide not to invest in their children’s education and send them directly to the labour market, or the other way around, send them to school by investing more in education. Therefore, decisions on schooling might be related to cognition, which is affected by malaria.

As already stated, several authors have identified both IQ and educational attainment as equally good measures of human capital to include in income and growth regressions. We do not argue against this statement. However, we do think that, as held by Bleakley (2010b), the effect of cognition might be of first-order importance while that of schooling might be of second-order importance. With the results at hand, one might interpret that since IQ coefficient becomes smaller as enrolment is introduced in the equation, the effect of malaria on IQ and subsequent economic outcomes might happen via education as well. Notwithstanding, given that the magnitude of the IQ coefficient is still considerable, and its significance remains, we lean towards Bleakleys’ (2010b) claim. Moreover, the robust and strong positive coefficient of enrolment rate to explain GDP per capita variance but the non-significant effect of malaria on enrolment rate could mean that lower malaria translates into higher cognition and this into higher educational attainment. Education would be picking some of the effect of malaria on
cognition but would not be showing an effect of malaria on education itself. Put in other words, our results suggest that cognitive abilities might be driving adulthood incomes, and, thus, aggregate output, through the effect in returns to schooling. Nonetheless, once more, we should interpret this with caution because of the scant data at our disposal. These results are correlations which we have given economic sense to, but which need from deeper assessment to be considered definite and robust.

This correlational study contributes to the growing literature assessing the long-lasting effects of infectious diseases via hard-to-undo impacts on human capital by analysing an often-overlooked mechanism. Although further research is required, both at the micro and macro level, these initial conclusions point to some policy implications. As we have discussed, education decisions might be driven by exposure to disease environments which affect children’s cognition. Improvements in human capital, therefore, need not be in the form of more resources allotted to education but rather in the form of more resources devoted to obtaining better health outcomes, which in turn allow to learn better and make the most of education. Health policies targeted at repairing cognition damages during childhood, when possible, or simply improving nutrition to fight off malaria when afflicted, might translate into higher cognition at later stages of life. Higher cognition might imply higher returns to schooling and higher educational attainment. Even if the increase in returns to schooling is smaller than the increase of wages, and educational attainment is not brought up, human capital will be greater simply because of the importance of health itself as a determinant of human capital. This would be in line with Hanushek and Woessmann (2008) who suggest a shift in the policy focus from years of schooling to cognitive abilities. This implies a shift from quantity of education to quality of education, from time spent at school to knowledge acquired. John and Schneider (2006) also propose optimizing the environmental factors children are exposed to, among which parasites causing childhood diseases are included, aiming at reducing cross-country differences in IQs in order to close living standards gaps.

Overall, our results are only suggestive given the data constraints. Future research could overcome this and allow for the establishment of causality when more data become available. Indeed, one of the main limitations relates to the fact that we only make use of a subsample of sub-Saharan countries which initially was 41 out of the 47 sub-Saharan nations but which is substantially reduced when using data on education. Using the whole sample would probably allow to check whether our results hold all over the region and infer consistent conclusions.
Moreover, further assessment of the relation between malaria and cognition, and this and economic output, including the role played by education, would provide useful insights on the topic. Particularly, research at the micro level would reveal how decisions at the household level are made which could help depicting a broader picture of the economy at the aggregate level. It could also unfold other potential channels through which malaria might affect the economy on the long term. Finally, more accurate measures of cognition would help addressing the issue of endogeneity. Given that IQ is, by construction of the Raven’s Progressive Matrices used to assess it, a reflection only of skills not acquired in schooling (Weil, 2014), we might expect correlation running primarily from IQ to economic performance. However, the fact that the measure we use is a revision of previous measures may cast doubts on the exogeneity in the last stage of the mechanism proposed. The measurement of current cognition at the national level would be useful to approach these issues, and so would be additional covariates to control for education.
References


Appendix A

Alternative correlations to those in the main results are here presented.

Table A.1. Malaria (1990’s) Correlations

<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>MEI (1990)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MEI (1991)</td>
<td>0.997***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MEI (1992)</td>
<td>0.989***</td>
<td>0.991***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MEI (1993)</td>
<td>0.997***</td>
<td>0.998***</td>
<td>0.990***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MEI (1994)</td>
<td>0.993***</td>
<td>0.996***</td>
<td>0.987***</td>
<td>0.995***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MEI (1995)</td>
<td>0.994***</td>
<td>0.994***</td>
<td>0.992***</td>
<td>0.993***</td>
<td>0.991***</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MEI (1990-1995)</td>
<td>0.998***</td>
<td>0.999***</td>
<td>0.994***</td>
<td>0.998***</td>
<td>0.997***</td>
<td>0.997***</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>IQ (L&amp;V)</td>
<td>-0.249</td>
<td>-0.212</td>
<td>-0.237</td>
<td>-0.227</td>
<td>-0.221</td>
<td>-0.234</td>
<td>-0.231</td>
<td>-</td>
</tr>
<tr>
<td>IQ (DB)</td>
<td>-0.186</td>
<td>-0.153</td>
<td>-0.181</td>
<td>-0.167</td>
<td>-0.163</td>
<td>-0.172</td>
<td>-0.171</td>
<td>0.951***</td>
</tr>
</tbody>
</table>

Number of observations: 41

Note: * p<0.05, ** p<0.01, *** p<0.001

Table A.2. Pearson’s Pairwise Correlations (r)

<table>
<thead>
<tr>
<th></th>
<th>GDP per capita (2016)</th>
<th>Average Growth (2010-2016)</th>
<th>IQ as calculated by L&amp;V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Growth (2010-2016)</td>
<td>0.647***</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>(43)</td>
<td></td>
<td>(43)</td>
<td></td>
</tr>
<tr>
<td>IQ as calculated by L&amp;V</td>
<td>0.482***</td>
<td>0.113</td>
<td></td>
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<tr>
<td>(44)</td>
<td>(43)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-0.151</td>
<td>-0.040</td>
<td>-0.273</td>
</tr>
<tr>
<td>(42)</td>
<td>(41)</td>
<td>(44)</td>
<td></td>
</tr>
</tbody>
</table>

Note: * p<0.05, ** p<0.01, *** p<0.001; Number of observations are in parentheses

Table A.3. Pearson’s Pairwise Correlations (r) (without outliers)

<table>
<thead>
<tr>
<th></th>
<th>GDP per capita (2016)</th>
<th>Average Growth (2010-2016)</th>
<th>IQ as calculated by L&amp;V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Growth (2010-2016)</td>
<td>0.631***</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>(39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IQ as calculated by L&amp;V</td>
<td>0.551***</td>
<td>0.155</td>
<td>-</td>
</tr>
<tr>
<td>(40)</td>
<td>(39)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average MEI (1990-1995)</td>
<td>-0.287</td>
<td>-0.066</td>
<td>-0.356*</td>
</tr>
<tr>
<td>(38)</td>
<td>(37)</td>
<td>(40)</td>
<td></td>
</tr>
</tbody>
</table>

Note: * p<0.05, ** p<0.01, *** p<0.001; Number of observations are in parentheses