Malaria and Education: Evidence from Mali.*

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Abstract

This article examines the influence of malaria on human capital accumulation in the village of Diankabou in Mali. To account for malaria endogeneity and its interaction with unobservable risk factors, we exploit natural variations in malaria immunity across individuals of several sympatric ethnic groups - the Fulani and the non-Fulani - that differ in their susceptibility to malaria. The Fulani are known to be less susceptible to malaria infections, despite living with a similar malaria transmission intensity compared with other ethnic groups. We also use natural variation of malaria intensity in the area (during and after the malaria transmission season) and we use this seasonal change as a treatment. We find that malaria has a causal impact on cognitive and educational outcomes in this village. We discuss the implications of this result for human capital investments and fertility decisions with the help of a quantity-quality model.

Keywords: Malaria, Immunity, Education, Cognition, Fertility

JEL: O12, I15, I25

1 Introduction

Malaria is thought to be among the oldest of human diseases with profound impact on human evolution. Indeed, the "malaria hypothesis" posits that certain human genetic polymorphisms[1] have been naturally selected in high frequencies because they have protected against the effects of malaria infections. Such protection involves the immune system, i.e. the ability of an organism to resist disease[2]. Moreover, the employment of such defense mechanisms against malaria is recognized as a costly life-history trait (Williams, 2006). For instance it is well known that inherited conditions such as sickle cell anaemia and beta-thalassaemia, which cause deformities in red blood cells and are common in people from malaria regions, make it more difficult for malaria parasites to infect red blood cells.

Though tentative, our investigation exploits immunity to malaria in an attempt to explain the causal effects of malaria on human capital accumulation. One of the possible approaches in the study of human variation in the susceptibility to malaria consists in comparing malariological indicators between populations differing in their genetic background but living in the same epidemiological context, i.e., exposed to the same transmission level and to the same parasite strains. The possible observation of inter-ethnic differences of susceptibility in such conditions provides opportunities to detect factors associated with protection (Modiano et al., 1995, 2001). In Sub-Saharan Africa and in Mali specifically, the Fulani ethnic group has proved to be less susceptible to malaria[3] as reflected by lower parasite rate (the proportion of the population found to carry asexual blood-stage parasites), lower parasitemia (the quantitative content of parasites in the blood) and fewer clinical symptoms than other sympatric ethnic groups (Dolo et al., 2005; Farouk et al., 2005). Sympatric in this context refers to the fact that they inhabit the same overlapping geographical area but the gene

[1] The recurrence within a population of two or more discontinuous genetic variants of a specific trait.

[2] The immune system is the collection of cells, tissues and molecules that protects the body from numerous pathogenic microbes and toxins in our environment. Such defense can be divided into two general types of reactions: reactions of innate immunity and reactions of adaptive immunity. The innate system is the first line of defense and comprises mechanisms that defend the host from infection by other organisms in a non-specific manner. The adaptive immune system, or acquired immune system, is specialized and eliminates or prevents pathogen growth. In acquired immunity, pathogen-specific receptors are "acquired" during the lifetime of the organism.

[3] Four species of these protozoan parasites account for almost all infections seen in humans: *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale* and *Plasmodium malariae*. *Plasmodium falciparum* is the most aggressive of all and accounts for the majority of infections in Africa. Malaria stands for *Plasmodium falciparum* malaria in this article.

pools are not mixed. Therefore, we use this fact to identify the possible impacts of malaria on cognition and education at the pupil level and we use it to understand the complex links between infection, educational investment and fertility at the household level. Our analysis is divided into two main parts.

First, we study the direct impact of malaria on educational outcomes. In the area we consider, there are two ethnic groups (Fulanis and non-Fulanis) and two seasons (high and low malaria transmission). The Fulani have some immunity to malaria so the infection rates are much lower among the Fulani than the non-Fulani in the high season. They are comparable in the low season. Given this, by comparing the change in cognitive and educational scores across seasons between the Fulani and the non-Fulani, and rescaling it based on the difference (across ethnic groups) in the change in infection rates, one can assess the impact of malaria on cognitive and educational scores. This strategy is similar to the difference-in-difference estimation strategy proposed by Bleakley (2010*b*), though we use data collected at a much finer scale. Bleakley (2010*b*) exploits the variation in malaria prevalence at baseline across US states, whereas we exploit the variation across ethnic groups. Bleakley (2010*b*) exploits the reduction in transmission due to an eradication campaign, whereas we exploit the seasonal change. The approach suggested by Bleakley is improved in two ways: (i) malaria measurement is improved, using a polymerase chain reaction (PCR)[4], and (ii) we assess a "natural" treatment effect (which presents the advantage to be independent of human activities). Cognitive outcomes are Raven Progressive Matrices tests scores (Raven, 1984). Educational achievement outcomes are measured with a set of educational variables taken from previous studies (Jukes et al., 2006; Thuilliez et al., 2010) and French models for primary educational tests (BatelemR and Boehm3). We find that malaria and notably asymptomatic malaria (i.e., the infection without any symptoms) have a causal impact on cognitive and educational outcomes in this village. These results are in keeping with

[4]PCR (polymerase chain reaction) is a technique in molecular genetics that permits the analysis of any short sequence of DNA (or RNA), even in samples containing only minute quantities of DNA or RNA. PCR is used to reproduce (amplify) selected sections of DNA or RNA for analysis. PCR is now extensively used for malaria diagnosis as PCR can distinguish between the different species of human malaria parasites. When used in the best possible conditions, the PCR technique has been reported capable of detecting parasitaemia of less than 0.00002%, which corresponds to one parasite per mm3, or 5 parasites per 5 l sample of blood. By comparison, microscopy has been reported capable of detecting parasitaemia of 0.0001%. PCR is therefore much more sensitive and specific than microscopy, generally used in other microeconomic studies. Put differently, PCR is more specific and sensitive than previous techniques used such as microscopy (Anchinmane and Shedge, 2011)

Venkataramani (2012) for cognition and Bleakley (2010*b*) for literacy rates, among others. Moreover, although we focus on short-run impacts, we improve the understanding of potential competing mechanisms.

Second, we discuss the economic implications of this result by considering a quantity-quality model where parents choose their fertility and their investment in the education of their surviving children. The framework is similar to Ehrlich and Lui (1991), Kalemli-Ozcan (2003), Lagerlöf (2003), Cervellati and Sunde (2007), Estevan and Baland (2007), Bell and Gersbach (2009), and Baudin (2012) except that we focus on early child mortality that occurs before the schooling period and distinguish it from child morbidity. The main novelty is to consider a differential exposure to malaria. The model explores the potential cost of immunity for populations at a lower risk of disease, when the disease reduces the returns to education. It shows that, if malaria increases the child mortality rate and reduces educational outcomes, the unprotected group (the group with a higher probability of being infected) will invest more in education and will have more children. The predictions of the model are illustrated using some results of the literature and stylized facts collected at the family level at baseline.

The paper is organized as follows. Section 2 describes the historical difference between sympatric ethnic groups in Mali and gives previous evidence on the links between malaria, cognition and education. We analyse the causal effect of malaria on education in section 3. We discuss the economic implications of this result in section 4. Section 5 summarizes and discusses implications of the results.

2 Background evidence

2.1 Differential malaria prevalence amongst sympatric ethnic groups in Mali

Malaria is one of the most serious public health problems in Mali. It accounts for 37.5% of health clinic consultations and 72% of deaths amongst children under 5 years old, and is the main cause of anaemia amongst pregnant women (Système Local d'Information Sanitaire or SLIS, 2007). Immunity to malaria is dependent on both the innate and the adaptive arms

(both cell- and antibody mediated) of the immune system, which are required for adequate protection. Previous studies have shown that Fulani children have a stronger inflammatory and antibody response against malaria parasites compared to the Dogon, and that these differences are evident already at an early age, thus probably depending on the innate arm.

The Fulani traditionally were nomadic pastoral people and have now settled in various parts of the African continent. One such site is northern Mali where they have lived for at least 200 years. The Dogon are farmers that migrated to the study area of northern Mali, about 500 years ago. The inhabitants of the Fulani and the Dogon villages in this area do not inter-marry and are therefore referred to as sympatric ethnic groups. The Dogon are in a numerical majority in this area, but other marginal ethnic groups (Malinke, Bambara, Bobo, Mossi) also live in the study area. We thus refer to the Dogon ethnic group and other minority groups as the "Non-Fulani" ethnic group in the tables. However, we also perform specific analysis using the Dogon group alone (referred as the "Dogon only" ethnic group).

When assessing malaria parasite rate, parasite density, malaria specific antibodies of several sympatric ethnic groups in West Africa, interethnic differences were found (Modiano et al., 1995). The Fulani were less infected and had lower parasitemia than other ethnic groups. Findings from this study suggest that the Fulani were more resistant to malaria. Further studies from Sudan showed consistent inter-ethnic differences in malaria infection rates, malaria morbidity, prevalence and levels of anti-malarial antibodies (Nasr et al., 2009). The Fulani were again less infected by parasites, less affected by the infection and more responsive to the antigens tested. These differences could not be explained by the use of malarial protective measures such as bednets, other protective measures, or exposure to infective bites, nor did socio-cultural or environmental factors seem to play a role in the relative better protection against malaria seen in the Fulani. In addition, the Fulani did not show higher frequencies of *known* genetic factors of malaria resistance (Modiano et al., 1995, 2001).

When studying differences in susceptibility to malaria between two sympatric ethnic groups in Mali, it was shown that the Fulani had higher spleen enlargement rates[5], lower

[5]Splenomegaly is an enlargement of the spleen resulting from an abnormal immune response to repeated attacks of malaria. Many of the mechanisms leading to an enlarged spleen are exaggerated forms of normal spleen function, and a wide variety of diseases (schistosomiasis, post-necrotic cirrhosis, thalassemia, leukemia, lymphoma, myelofibrosi) are associated with enlargement of the spleen. The role of the spleen during malaria in humans remains unclear.

parasite density and were less affected by malaria than the Dogon group. The Fulani also had higher titers of antimalarial antibodies than the Dogon group (Dolo et al., 2005) which means that they had stronger immune responses.

In further studies, inter-ethnic differences were observed in some cytokines. Cytokines and chemokines are essential mediators during malaria infection, and the balance between pro- and anti-inflammatory cytokines may be important for the clinical outcome of malaria responses in the Dogon[6]. Attempts to link this relative better protection during malaria infection seen in the Fulani to different polymorphisms in candidate genes have so far not given a satisfying explanation of the underlying cause of the protection, and the more pro-inflammatory response seen in the Fulani but not in other sympatric ethnic groups: they could not provide a convincing explanation of the relative better protection against malaria seen in the Fulani (Driss et al., 2011). Put differently, genetic factors - usually associated with malaria protection and common to different populations - do not seem to be the key explanatory variables of this protection. Epigenetics studies[7] could help understanding what regulates these differences, but research has not reached this stage so far.

2.2 Previous evidence on malaria, cognition and education

Malaria can impact children's educational achievement through a number of ways. First, malaria during pregnancy can lead to foetal growth retardation, which translates into cognitive and physical impairments among children. Barreca (2010) analyzes the long-term impact of *in utero* and post-natal exposure to malaria. He finds such exposure leads to considerably lower levels of educational attainment, and higher rates of poverty later in life.

Second, during early childhood (under the age of five) complicated forms of malaria may

[6]When comparing plasma levels of pro-inflammatory cytokines between the Fulani and the Dogon, it was found that regardless of the infectious status, the Fulani had higher levels of IL-6, IL-8, IL-12, IFN-γ and IFN-γ compared to the Dogon (Giusti, 2009). When children were further subdivided and the uninfected children from both ethnic groups were compared, uninfected Fulani had higher levels of IFN-γ, IL-6, IL-8 and IL-12 compared to the uninfected Dogon. This indicates that Fulani children might be more prone to combat malaria infections, possibly due to higher baseline levels of these cytokines. When comparing uninfected individuals, plasma levels of IL-6, IL-8, IL-12 and IFN-γ were significantly higher in the Fulani as compared to the Dogon. In infected individuals, higher plasma levels of IFN-γ were observed in the Fulani, compared to the Dogon (Arama et al., 2011). Boström et al. (2012) found that Fulani children had higher levels of all tested cytokines compared to the Dogon, in particular IFN-γ, a cytokine known to be involved in parasite clearance. Furthermore, the Fulani also had higher titers of malaria-specific antibodies (IgG and IgM as well as IgG1-3 subclasses) compared to the Dogon.

[7]Epigenetics is the study of inherited changes in phenotype or gene expression caused by mechanisms other than changes in the underlying DNA sequence (Bird, 2007).

develop rapidly. Indeed, acquired immunity in children does not play an efficient protective role until the ages of 5 to 6, even in highly endemic areas, which highlights why malaria is a major threat to child survival. The effects of severe malaria, better known as cerebral malaria, have been quantified by numerous studies [8]. For instance, Ngoungou et al. (2007) provide a quantification of the burden in West Africa. In this study, 101 subjects (mean age of 5.6 $\pm$ 3.6 years) who had had cerebral malaria in Mali were followed from 1999 to 2001. The authors find that 28 children exhibited persistent neurological sequelae (26.7 %). Among them, eight (7.9 %) children had developed these sequelae just after cerebral malaria, and 20 (19.8 %) a few months later. These included headaches, mental retardation, speech delay, bucco-facial dyspraxia, diplegia and frontal syndrome (one case each), dystonia (two cases), epilepsy (five cases), as well as behaviour and attention disorders (15 cases).

Third, even during late childhood, which usually extends from 6 to 16 years of age, the protection conferred by acquired immunity is only partial. If cerebral malaria is rare at this stage, "simpler" cases of clinical malaria (called "uncomplicated malaria"), repeated illness, or chronic malaria infections are not. They can have a non-cognitive impact on educational achievement through school absenteeism, general health conditions, and investment in curative strategies (coping strategies against the disease detrimental to educational investments). For instance in a Kenyan case study, Brooker et al. (2000) attribute 13 % to 50% of medically related school absences to malaria. In Kenya, primary school students were found to miss 11% of the school year (20 school days missed per child-year). In Nigeria, school days missed varied between 2% to 6% of the school year (3 to 12 days per year per student). In Mali, malaria was the primary cause of absenteeism during the full school year (Thuilliez et al., 2010). Moreover, although the age distribution of uncomplicated malaria and asymptomatic malaria depends on transmission intensity, the total burden of the disease may be similar or even higher in settings of low transmission due to patterns of acquired immunity. Malaria morbidity among school-age children increases as transmission intensity decreases, but asymptomatic infections are more frequent in high transmission settings (Clarke et al., 2004). Fernando et al. (2003) show a significant negative correlation between the total number of malarial attacks experienced by children and test scores during a six year follow-up. Fernando et al. (2006) and Jukes et al. (2006) also show a substantial effect of preventive

[8] See Mung'ala-Odera, Snow and Newton (2004) for a literature review.

treatment in two randomized studies. Yet, *asymptomatic malaria* has proven to have detrimental effects on children's cognitive and educational skills in three studies, one being a cluster-randomized control trial which suggests either a direct causal effect of the disease or an antimalarial treatment effect (Clarke et al., 2008; Thuilliez et al., 2010; Nankabirwa et al., 2013). The mechanism could include a "*toxicity effect, leading to biochemical changes in the central nervous system (CNS); excitation of the immune system, leading to changes in behaviours related to appetite and reaction time; and physiological effects such as discomfort and disturbed sleep,leading to reductions in activity levels or causing behavioural change*" (Holding and Snow, 2001).

3 Short-run impacts of malaria on cognitive test scores and educational performance

3.1 Study area and survey methods

The study took place from early October to early December 2010 in the village of Diankabou in the Mopti area, 850 km northeast of Bamako, the capital of Mali. Malaria is mesoendemic[9] in this area with *P. falciparum* being the main parasite species. The entomological inoculation rate[10] was previously shown to be similar in both ethnic groups. Transmission is seasonal from July to October (Farouk et al., 2005; Dolo et al., 2005; Bereczky et al., 2006; Vafa et al., 2007, 2009).

The sample design was for an exhaustive survey of all children living in Diankabou, enrolled in the school of Diankabou who accepted to participate in the study. 300 children were enrolled in the village school in September 2010. Our study includes 296 children at baseline and 291 at endline (5 children were lost at the endline mostly due to absences at the endline)[11]. Data was collected at the beginning of the school year during two cross-sectional surveys in October 2010 and December 2010. As illustrated by New et al. (2002)

[9]Mesoendemic malaria endemicity is defined by a parasite rate (PR) between 0·11 and 0·5

[10]The "entomological inoculation rate" is the commonly-used measure of the intensity of malaria transmission. EIR is a commonly used metric that estimates the number of bites by infectious mosquitoes per person per unit time (Smith et al., 2007).

[11]Note that we also have a limited number of missing values for some of the variables under study here due to the difficulties inherent to biomedical data collection and storage in remote areas, refusals to conform to particular procedures or to perform some tests.

October still corresponds to the malaria season whereas December is not considered anymore as a transmission season in this area[12]. Once informed consent had been obtained, all participating pupils received clinical and laboratory examinations during two "active" follow-ups. Tests were administered the same day directly in the school[13].

The data collected included the children's malaria infection status, measured with high precision through PCR, anthropometrics, cognitive outcomes, and the ethnic group. Definitions and details on the variables are provided in Appendix A.

3.2 Natural experiment and empirical equation

In order to provide evidence on the causal effects of malaria on educational outcomes, we use a quasi-experimental approach in which we exploit ethnic variations across individuals of the two sympatric ethnic groups, and the natural variation of malaria intensity in the village (at the end of the malaria transmission season which fixes our before/after framework). Two factors combine to determine the identification strategy of this first part: (i) the exogenous origin of change in malaria exposure (pre- and post-seasonal change); (ii) the use of the Fulani ethnic group for comparison (the Fulani are less susceptible and provide a good counterfactual). Figure I provides a synthetic overview of our natural experiment.

Figure I about here.

To estimate the average treatment effect (ATE), a natural approach is to estimate equation (1):

$$E_{it} = \alpha_2 + \gamma_1[Post_t \times NonFulani_i] + \gamma_2 Post_t + \omega X_{it} + FE_i + \epsilon_{it}, \quad (1)$$

where E are the outcomes of interest here (cognitive or educational outcomes)[14], $Post$ is the period of observation (taking a value of 1 for early December (just after the seasonal

[12]Malaria transmission in this area increases from July to October. It then declines during the other months of year. New et al. (2002) show similar seasonal trends from 1960 to 1990, and epidemiological studies in this area confirm the timing of this seasonal shift (Farouk et al., 2005; Dicko et al., 2005; Dolo et al., 2005; Bereczky et al., 2006; Vafa et al., 2007, 2009). Notably, this seasonal change leads to a differential decline in malaria infection in both groups. As it is naturally more susceptible, the non-Fulani group is more affected by this decline.

[13]The study protocol was approved by the institutional review board (IRB) ethics committee of the National School of Medicine and Pharmacy of Mali. Community and individual written informed consent was obtained before starting the study. Panel data were collected directly in the field by the authors.

[14]We define cognition as the mental process involved in knowing, learning, and understanding things. The

change), and 0 for early October (just before the seasonal change)), $NonFulani$ is a dummy variable for non-Fulani ethnic groups, X, a set of covariates at individual level that vary across time, including other health covariates (malaria symptoms, splenomegaly, BMI for age z-score, haemoglobin concentration), FE are child fixed effect intended to capture time invariant effects (including ethnicity, practice effects, other baseline parasite infections and socioeconomic factors at baseline), and ϵ is an individual-specific error term.

However, the natural immunity amongst the Fulani (the control group) is not fully protective and the reduction in malaria infection after the exogenous seasonal change amongst non-Fulani is not fully efficient to eliminate all infections. It is useful for our approach to think about the infectious status with respect to seasonal change. We have four groups of pupils: (i) those who were infected at baseline and sane at endline, (ii) those who were sane at baseline and endline, (iii) those sane at baseline and infected at endline, (iv) those infected at baseline and endline[15]. Indeed, our control (Fulani) and treated group (non-Fulani) are a mixture of these four categories and we want to estimate the average treatment for those individuals whose malaria infection and parasite density were affected negatively by seasonal change, knowing that the non-Fulani have a higher probability to be affected by this change over the three months of study. Consequently, there is no subpopulation available for whom the probability of treatment is zero. In this case, instead of a simple difference-in-difference analysis, Imbens and Angrist (1994) recommend to use the Local Average Treatment Effect (LATE) approach, based on natural experiments. LATE is the average treatment effect for individuals whose treatment status is influenced by changing an exogenous regressor that satisfies an exclusion restriction.

Therefore, we rely on an instrumental variable strategy, where we instrument for malaria status using the interaction term between *Ethnicity* and *Post* with main effects for *Ethnicity* and *Post* to rescale the simple difference-in-difference, provided in Table 1, in a within child fixed-effects model, following equations (2) and (3), where equation (3) corresponds to a difference-in-difference design applied to malaria infections. We identify the treatment effect

cognitive outcome is given by Raven Progressive Matrices test, a non-verbal assessment comprised of pattern matching exercises that progressively increase in difficulty (Raven, 1984). Educational achievement refers to the ability of succeeding in education, as measured here by a synthetic primary educational test score.

[15]These four categories can be defined on the basis of a binary infection status or on the basis of the continuous parasite density. In the last case, the reasoning is made in terms of density increase or decrease instead of binary infectious status.

for "compliers", i.e. the average treatment for those individuals whose malaria infection and parasite density were affected negatively by the seasonal change (a local average treatment effect, LATE).

$$E_{it} = \alpha_1 + \beta Mal_{it} + \omega X_{it} + FE_i + \epsilon_{it}, \tag{2}$$

where Mal is a measure of malaria infection, and β is the coefficient of interest. We estimate equation 2 by two-stage least squares (2SLS), using the following first-stage equation:

$$Mal_{it} = \alpha_2 + \gamma_1[Post_t \times NonFulani_i] + \gamma_2 Post_t + FE_i + \mu_{it}, \tag{3}$$

where $Post$ is the period of observation (taking a value of 1 for early December (just after the seasonal change), and 0 for early October (just before the seasonal change)), $NonFulani$ is a dummy variable for non-Fulani ethnic groups, FE is a child fixed effect intended to capture time invariant effects (including ethnicity).

The identifying assumption is that there are no unobserved factors directly affecting the outcomes that (i) are correlated with seasonal change and (ii) are correlated with ethnic specific malaria infectious status. There is absolutely no evidence that ethnic differences, a marker of risk for malaria infection, plays any role in the education production function. There is also no reason to believe that ethnic differences *per se* should play any role in this function. In addition, there is no reason that the difference in performance between infected and uninfected individuals should be smaller during the low malaria season than during the high malaria season. However a number of threats to inference in the LATE design can still threaten our results. We discuss theses issues in greater detail in section 3.5.

3.3 Descriptive statistics and simple difference-in-difference

The results from the simple difference-in-difference approach are provided in Table I and A1. Table I provides the results of the descriptive analysis for selected variables but does not rescale the difference-in-difference based on the differential change in infection rates across ethnic groups. Our results are similar to previous findings from other villages located in the same area. Comparing the two groups, the Fulani were less affected by malaria than the Non-Fulani, as expected. For instance, in November 1998 and November 1999 (corresponding

approximately to our baseline survey made during the rainy season), in the same area and with a similar sample size, Dolo et al. (2005) find a parasite prevalence difference of 15.6% and 13% respectively (*P-values*<0.001). We find a 19.2% (*P-values*<0.001) difference in October 2010. At the very end of the dry season in July 2004, they find a parasite prevalence difference of -2.2% (*P-value*=0.220) between the Non-Fulani and the Fulani. We find a non-significant difference of -0.015% (*P-value*=0.825) in December 2010, at the beginning of the dry season.The same was observed for malaria symptoms: the Fulani ethnic group has fewer clinical symptoms. By contrast spleen enlargements, lower nutritional status and anaemia were more prevalent within Fulani children, still in accordance with the literature on this topic (Dolo et al., 2012*a*). However, all educational and cognitive test results were not statistically different between the two groups and no other factors (except age at the baseline, *P-values*<0.100) differed across groups. The prevalence of other parasite infections (urinary *schistosoma* and intestinal parasites) was similar in the two groups at the baseline.

Amongst the non-Fulani ethnic group, the parasite rate fell from 87.1% to 25% (i.e. a 62% decline) between October and December 2010. Amongst the Fulani ethnic group, the parasite rate fell from 67.9% to 23.5% (i.e. a 44.4% decline). The decline was thus large. The last column presents the OLS regression results (standard errors are clustered at the family level) from a simple difference-in-difference analysis. The decline in malaria exhibited significant variation across ethnic groups (17.8% differential decline between the non-Fulani and the Fulani).[16] The only other differential changes observed across time are malaria related symptoms (cephalgia and vomiting decreasing more amongst the non-Fulani ethnic group) whereas haemoglobin concentration increased significantly more amongst the non-Fulani. Notably, the last column of Table 1 shows no significant effect of the interaction term ($Post_t \times NonFulani_i$) on cognition and education. Table A1 provides additional results and shows no significant effect of the interaction term ($Post_t \times NonFulani_i$) on cognition and education.

Table I about here.

[16]Note that similar results are found with the Dogon "alone" ethnic group instead of the non-Fulani category.

3.4 Results

Table II presents the regression results on the effects of malaria on cognitive and educational outcomes, using the LATE approach. Column (1) presents the coefficients from the OLS estimate of equation 2. All other columns provide the coefficients from the 2SLS estimates. Robust standard errors are clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results (results available upon demand). The within-child fixed-effects results are given in columns (5) and (6)[17]. The low p-values from the Durbin-Wu-Hausman test indicate that malaria infections should be treated as endogenous. Note that PCR is detecting a very low parasite loads, which is useful to detect asymptomatic malaria. However the PCR technique used here only provides us a dummy variable for cases tested positive for malaria. As an alternative measure of malaria infection we also use malaria positive cases detected through microscopy and the parasite loads measured by microscopy. Microscopy-based parasite density, though less sensible and sensitive than PCR, enable us to rely on a continuous variable, and thus much more variation. The results are provided in Table A2. Column (1) from this table shows that the microscopy measures suffer from measurement errors.

Table II about here.

The results show significant negative effects of malaria infection on cognitive and educational outcomes. The regressions with controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain), health status characteristics (splenomegaly, BMI-for-age, haemoglobin concentration). Notably, the coefficients of interest do not change significantly as fixed effects and control variables are added to the specifications, thus providing some validation to the identification strategy. More particularly, adding symptom controls does not change the size of the coefficients, suggesting an effect of the presence of the parasite *per se*. When reducing the sample size to the Fulani and "Dogon alone" ethnic group the sample size is reduced by 28 to 30 observations, but the results remain unchanged.

To put this in context, the estimate implies that malaria increase resulted in a decrease in cognition of 0.887, a decrease in PCA score of 1.157 and a decrease in educational av-

[17]Note that the F-statistics mitigate "weak instrument" concerns.

erage score of 0.424 (Table II, column 5). The range of these variables being respectively $[-2.217; 3.832]$, $[-3.780; 4.631]$ and $[-1.884; 1.978]$, the magnitudes of the coefficients imply that being infected by malaria decreases the cognition score by 0.89 standard deviation, decreases the PCA educational score by 0.84 standard deviation and decreases the average educational score by 0.648 standard deviation. The latter suggests that results are also robust across educational outcomes. Table III provides the first stage estimates. We show the results of a linear probability model where we regress a dummy variable equal to one if the child was infected on ethnicity, the period of observation and the interaction term.

Table III about here.

To further understand the mechanisms through which malaria might affect cognition and education, table IV reports estimates for competing channels (haemoglobin levels and having at least one malaria symptom). One hypothesis suggested in the medical literature on the effects of malaria on cognition and education is through decreases in haemoglobin concentrations. Clinical longitudinal follow-up data showed that Fulani children have lower hemoglobin levels than Dogon children (Dolo et al., 2012*b*). Put differently, the Fulani suffer more from anaemia than the Dogon, despite their lower susceptibility to malaria[18]. This can be interpreted as a health cost related to protection. We could also expect a greater effect of malaria symptoms on cognition and education compared to parasite infections. Malaria infection could thus have both a direct effect on education and an indirect effect through symptoms. The direct mechanism could include a "toxicity effect," leading to biochemical changes in the central nervous system (Holding and Snow, 2001). Moreover, if malaria infection had a direct impact on symptoms but no direct impact on cognition and education, the PCR measure could just be a proxy for symptomatic infections. This does not seem to be the case.

The coefficients of interest do not change significantly when new channels are added and instrumented. In addition, none of the variables tested in these tables and instrumented with the same procedure (haemoglobin levels and having at least one malaria symptom) seem to have an impact on education.

However, there could be a two-fold penalty here, as the presence of malaria parasites had

[18]Malaria is a major cause of anaemia (Kurtzhals et al., 1999).

a direct effect on the educational achievement and cognitive performance, but symptoms also have a negative impact on education (Fernando et al., 2003). Note that the number of symptomatic cases was rather limited during the two "active follow-ups" (as opposed to continuous clinical case detection or clinical consultations), in accordance with previous studies (Dicko et al., 2005). This fact might explain why we do not find any effects for symptoms here.

Acute cases of illness (of any type) were treated immediately and monitored by the medical team directly in the field. This could bias our estimates of the treatment effect. Nonetheless, for the specific cases of malaria, it is important to highlight that even after receiving an antimalarial treatment, patients can still be at risk in short-term (14-day) treatment failure, late recrudescence (28 days) and re-infection with new parasite strains. Indeed, after a treatment, a period of 14 days is generally used for considering a second malaria infection as a new case (Dorsey et al., 2002). Consequently, as there were more than 14 days between the baseline (October) and the endline (December), infections at the endline can be considered as new cases. However, we test the robustness of our results to the inclusion of treatments in our regression models. The results are provided in Table IV and do not change our main conclusions. The following section discusses systematically the potential threats to the estimation strategy and provides a series of robustness tests.

Table IV about here.

3.5 Potential threats to validity

The first is related to ethnic-specific elasticities of cognition and education with respect to health. One concern is that the responsiveness of education to health improvements might differ by ethnic group. As suggested by Jayachandran and Lleras-Muney (2009) for gender, if a given improvement in health has a larger effect on the education of the Fulani than the non-Fulani, then our results could be driven, not by the differentially larger improvement in health for the non-Fulani due to seasonal change, but by their differential sensitivity to health improvements. This different elasticity by ethnic group could operate through morbidity affecting absences or improvements in family members' health. We tested for ethnic-specific cognitive or educational effects of other health improvements that were common to the Fulani

and the non-Fulani (such as BMI-for-age z-scores) in Table IV. There is no strong evidence for a greater elasticity of the non-Fulani or the "Dogon alone" as the results do not change our main conclusions.

Second, pre-existing endowment differences could also drive the results. For instance if non-Fulani are drawn from the sicker part of the distribution, then one might retrieve similar results. This could be particularly true for anemia and splenomegaly, that could reflect differences in other investments made in Fulani children that have a seasonal pattern. Indeed, anemia is a marker of malaria severity but is also driven by other factors (nutritional intake for instance). Appendix Figures A1 to A4, provide densities of hemoglobin density, splenomegaly, cognition and education variables. First the anemia level is relatively high in average for both ethnic groups and there is no evidence that non-Fulani are drawn from the sicker part of the distribution. We recall that the prevalence of anemia among school-age children is generally defined as a hemoglobin concentration of less than 11.0g/dL. Similarly the distribution of splenomegaly, cognitive or educational outcomes does not show major differences across ethnic groups. In order to confirm that differences in baseline health does not affect our results, Table A3 provides estimates interacting baseline anemia - as defined by the previous threshold -, baseline splenomegaly and malaria on cognitive and educational outcomes. Table A3 also analyzes the question of cross-group spillovers (e.g., malaria among non-Fulani children could impact learning among Fulani either positively or negatively due to classroom effects) by interacting classrooms with malaria. Therefore our results do not seem to be influenced by potential heterogeneity.

The third concern is related to cross seasonal behavioral reinforcing or compensating investments that differ by ethnic group or differential access to health care across groups across seasons. For instance different seasonal economic activities could be a concern. Though we have limited information about ethnic specific activities across seasons in Diankabou, as the time frame of the survey was limited, we did check in the Mali Demographic and Health Survey (DHS) collected from 2001, 2006 and 2012-2013 in rural areas of the Mopti region across different months (couple recode). A regression of the DHS wealth index on the interaction term between the month of interview and ethnicity shows no particular cross seasonal ethnic specific differences (results available upon demand). In addition, we also provide a robustness test of our results in another context involving the Fulani ethnic group. We use

data from another village in Mali. This village (Donéguébougou) is a small village located in a malaria-endemic area near Bamako (17 km, North of Bamako) providing free access to health care to all villagers. This village is not a place where Fulanis settled in the past and Fulani representation is very low, which is the reason why we did not use it in the main analysis. Details about this second village and the survey procedure - that is similar to the one describe above - can be found in (Thuilliez et al., 2010). 12 fulanis were surveyed across 8 follow-ups as compared with 213 non-Fulanis across 8 follow-ups from November 2007 to June 2008 (hence with much more seasonal variation). Still, this different place provides a useful test of our LATE approach because the village presents distinct cultural characteristics. First, other ethnic groups are different. The non-Fulani ethnic group is represented by Sarakole and Bambara. Second, economic and agricultural activities are different in this area as the climate is slightly different. In addition, there was no attrition in this village among the Fulani ethnic group leading to a total of 96 Fulani observations. The results, provided in Table A4, are similar to the ones found in Diankabou.

Another concern is that Diankabou is a remote village (65 km from the nearest city and 850 km northeast of Bamako, the capital of Mali) with difficult field work conditions. No research programs were undertaken previously in this village to our knowledge. Therefore we face a number of missing values for some of the variables under study here due to the difficulties inherent to biomedical data collection and storage in remote areas, and refusals to conform to particular procedures or to perform some tests. For instance, the data on cognition comprises 550 observations instead of 587 (data are missing for 6% of observations) and there are 583 observations for educational average score (data are missing for 0.6% of observations) and 503 for PCA score (data are missing for 14% of observations). Although we cannot directly rule out this concern, sample size variation across variables does not change the conclusions. In addition, restricting the sample to the Dogon ethnic group alone (and thus changing the sample size) does not affect our results. However, in an additional robustness check, all the missing educational outcomes for the Fulani were coded at the maximum value of the outcome in the post period and all the missing educational outcomes for the Non-Fulani were coded at the minimum value of the outcome. The results provided in Table V are robust to this check.

Finally, considering the very short time period of the study and the limited size of the vil-

lage, it is highly improbable that the village of Diankabou underwent major changes during the study period, such as unobserved education-related changes. The identification will be threatened if changes had ethnic-specific effects that varied by individuals in a manner correlated with malaria seasonal change. We are not aware of any major ethnic-specific changes between October and December, in addition to the ones discussed above. Consequently the discussion section provides other arguments on long term potential compensating mechanisms that differ by ethnic group and could explain the observed difference between Table 1 and Table 2.

Table V about here.

4 Discussion

In the previous Section, we have shown that malaria decreases the return to educational investment. It has a causal impact on cognitive test scores and educational performance. Nevertheless, Table 1 shows that the non-Fulanis do not outperform the Fulanis at the baseline or the endline separately. Moreover, the simple difference-in-difference estimates provided in Table 1 and A1 show no significant difference between both groups. This suggest that malaria has a short-run impact and that catch-up effects may take place. Indeed malaria is endemic in the village, i.e. transmitted all over the year, though the intensity of transmission varies with seasons.

In order to provide a discussion on the potential implications of short-run impacts of malaria on educational performance on the economic decisions inside the household, we built a model whose theoretical predictions are discussed using stylized facts collected at the family level at baseline, the Mali Demographic and Health Surveys (from 2001 to 2013) and the literature on the topic. An extensive test of our model is beyond the scope of this paper but constitutes avenues for future research.

We consider a quantity-quality model, in which parents choose their fertility and their investment in the education of their surviving children, and applied it to the case of a differential susceptibility to malaria.The probability to be infected (i.e. the infection rate), denoted p with $p \in (0, 1)$, is exogenous and varies across ethnic groups.

Malaria has two impacts. First, it increases the child mortality rate, which is well established (Murray et al., 2012). We define a survival function, which measures the probability to reach schooling age, and assume it decreases with the infection rate of the considered ethnic group. The survival function is denoted $s(p)$ and satisfies $0 < s(p) < 1$ and $s'(p) < 0$. We denote by n the fertility rate and therefore consider that $s(p)n$ is the number of children that reach schooling age in the considered household. We notice that according to the model, malaria has no impact on adult longevity. The second impact of malaria is to reduce educational outcomes (which is the result obtained in section). We denote by e the monetary cost of education per child made by the parents. We then assume that returns to education are equal to Re provided that the child has been ill and to δRe otherwise. We consider in the model that $\delta \geq 1$, which permit to distinguish two cases: $\delta = 1$ means that malaria has no impact on educational outcomes whereas $\delta > 1$ implies that children who were ill exhibit lower outcomes (which is the result of Section 3).

Parents share their income, denoted w, between consumption, c, and the costs of their surviving children:

$$w = c + (e + \lambda) s(p) n, \tag{4}$$

where $\lambda \geq 0$ accounts for fixed costs per child. The expected utility of the parents depends on consumption, net fertility and returns to education. It can be written as:

$$u(c) + s(p) n \left[pv(Re) + (1-p) v(\delta Re) \right], \tag{5}$$

where functions u and v are increasing and strictly concave and $v(0) = 0$. We notice that considering the utility of expected returns rather than (5) would not affect the results presented below. The optimization problem of the parents is to choose (c, n, e) that maximize (5) subject to (4). We assume that solutions are interior and denote them by (c^*, n^*, e^*). They satisfy two first-order conditions that say that e^* is such that the marginal utility of consumption is equal to the expected marginal utility of education and that n^* is is such that the marginal utility of consumption is equal to the "return" of one additional surviving child; a "return" that is given by the ratio of the expected utility of education over the cost $(e + \lambda)$. Using those conditions, we derive below some theoretical relationship between exogenous parameters and the optimal pair (e^*, n^*). Let us first analyze the predictions of

the model concerning the education choices.

Proposition 1. *The optimal investment in education* e^*,

i) does not depend on parental income w,

ii) increases with the infection rate p *provided that the relative risk aversion is constant or increasing and that* $\delta > 1$.

Proof. See Appendix B.

By combining the first-order conditions of the problem, we obtained that the expected marginal utility of education should equal the "return" of one surviving child. This relation gives the optimal investment in education, which therefore does not depend on parental income. The main result of Proposition 1 is to exhibit a condition such that the relationship between infection rates and investment in education is positive. The relationship is *a priori* ambiguous as the "return" of a child decreases with p while the expected marginal utility of education is increasing with p. If the relative risk aversion is increasing or constant (which is empirically relevant: Holt and Laury (2002) and Chiappori and Paiella (2011)), the second effect is found to dominate the first. Hence, parents compensate a lower expected return by a larger investment. We notice that our result still hold if the relative risk aversion is decreasing, provided that the fixed cost of each child is not too large, which is also generally considered to be the case in rural Africa, and notably by considering the high opportunity cost of schooling (Estevan and Baland, 2007; Bargain, Donni and Kwenda, 2011; Dunbar, Lewbel and Pendakur, 2013).

Theoretical results contained in Proposition 1 might be surprising as one would expect that a decrease in the return to education should lower the investment in education (see, most notably, the nice presentation by Bleakley (2010*a*)). Nevertheless, as argued by Soares (2005) and Hazan and Zoabi (2006), the quantity-quality tradeoff challenges this intuition as changes in morbidity also influence the return on the quantity of children.

To discuss the empirical relevance of Proposition 1, we established some stylized facts that illustrate the sign of the theoretical relationships we obtained. Table VI uses data collected at the family level in October 2010 during the baseline survey. In particular, in Column (1) in Table VI, it is shown that educational investment in education does not depend on parental income but increases with the probability to be infected.

To discuss the empirical relevance of Proposition 1 we recall that $\delta > 1$ is the main result we presented in section 3. Let us now establish some stylized facts to illustrate the sign of the theoretical relationships. Table VI uses data collected at the family level in October 2010 during the baseline survey to illustrate this proposition. Column (1) in Table VI shows that educational investment in education does not depend on parental income but increases with the probability to be infected.

We now turn to the predictions of the model concerning the fertility choices.

Proposition 2. *The optimal fertility, n^*,*

i) increases with parental income w,

ii) increases with the probability to be infected, p, if δ is not too large.

Proof. See Appendix B

The relation between fertility and the infection rate depends on two factors. First, as malaria negatively impacts the survival rate of infants, the infection rate tends to increase fertility trough a compensation effect. Second, as we have seen in Proposition 1, the infection rate may increase the investment in education, which turns -through a quantity-quality trade-off- to decrease fertility. Proposition 2 says that if the second effect is not too strong (i.e. if the relative advantage of healthy pupils measured by $\delta - 1$ is not too large), the first effect dominates.

This condition fits well with the estimates we presented in Section 3 where we showed that δ is significant but not too large (less than one standard deviation for all outcomes). Before illustrating Proposition 2, we remind that evidence on the relationship between malaria and fertility are mixed. Using data from the 1961 Italian census when malaria was endemic in Sardinia, Zei, Lisa and Astolfi (1990), Lisa et al. (1994) and Astolfi et al. (1999) observed differential fertility between women living in areas with differing degrees of malaria. A positive relationship was found between fertility and malaria transmission. The average number of live-born children was invariably higher in the areas with the highest malaria risk, after controlling for socio-cultural factors. for socio-cultural factors. Conversely, Lucas (2013) showed that the national malaria eradication campaign in Sri Lanka rose fertility. Concerning Africa, there is no direct evidence on that issue. Several studies reported a lower fertility of the Fulani. Several studies in Africa also reported a lower fertility of the

Fulani ethnic group. Furthermore, the observed differential fertility among the Fulani has been observed in different cultural contexts (David and Voas, 1981; Hill and Thiam, 1987; Hampshire and Randall, 2000). However, as the discovery of a lower susceptibility of the Fulani group to malaria infections is relatively recent, the "malaria hypothesis" has not been deeply explored yet.

Column (2) from Table VI shows that the total number of children per family in one considered household increases with parental income and increases with the probability to be infected (as measured by ethnicity). Column (3) from table VI illustrates Proposition 2 with the Mali Demographic and Health Survey (DHS) collected in 2001, 2006 and 2012-2013 in rural areas of the Mopti region. Recall that our study takes place in this area and that the different ethnic group live in sympatry in this area, which is not the case elsewhere in Mali. We use the couple data. This dataset has one record for every couple. It contains data for married or living together men and woman who both declared to be living together to each other. The unit of analysis (case) in this file is the couple in which both partners were interviewed. We use the DHS wealth index as a measure of long-term income constructed from household assets. Regression (3) shows similar results. The total number of children ever born per family increases with parental income and increases with the probability to be infected (as measured by the non-Fulani variable). Unfortunately, DHS does not provide educational investment to test Proposition 1 on a larger dataset. Testing external validity of these results in a larger area and analyzing catch-up effects of the non-Fulani ethnic group in the long-run, constitute important avenues for further research.

Table VI about here.

5 Conclusions

Our empirical results suggest that malaria infection (as measured by PCR) has a direct causal impact on cognitive and educational outcomes. These results are also robust to including a number of control variables and a series of tests. Notably, we use a natural experiment which differs from a randomized control trial.

Although an extensive biomedical literature analyzes the costs of immunity, the social

costs of immunity have not been explored yet. The findings of the model proposed in the discussion section suggest that, for the ethnic groups under study here, a higher probability to be infected by malaria might increase fertility and human capital investments. On the contrary, the survival of those groups that are partially immunized against malaria, might be threatened. Indeed and interestingly, despite having lower malaria-specific mortality rates, the survival of the Fulani ethnic group will depend on fertility decision when they live in sympatry with other ethnic groups. This is certainly not a negligible consequence of immunity, from an economic evolution perspective. Notably, the model converges with observed unexplained fertility behaviors of the Fulani in different contexts and with our aggregate data. Therefore though stylized, this discussion could be highly relevant to explore these issues in the future.

Our article also emphasizes coping mechanisms and catch-up effects that may be at play in the long-run across ethnic groups. Indeed the seasonal shock under study here is only a temporary shock that does not affect the probability to be infected by ethnic group in the long run or the average probability to be infected over a full year of schooling. Moreover, this seasonal change is an imperfect treatment to eliminate malaria amongst all ethnic groups. The originality of our results lies in that they go beyond the findings of the above-mentioned previous studies on income or human capital accumulation and we emphasize potential coping mechanisms that can lead the estimation of that effect to be biased or difficult to assess with a simple difference-in-difference framework. We emphasize the potential changing behavior of those households that have a lower probability of infection because of natural protection against the disease. This is particularly important, considering malaria selective pressure on the human genome.

The findings of this paper also suggest that the increase in human capital accumulation that results from malaria decrease is an important component of malaria control policies and cost-benefit analyses of malaria control programs. First protective factors against malaria might be taken into account in defining the target population of malaria control programs to avoid disadvantaging specific groups. This could be particularly relevant for Glucose-6-phosphate dehydrogenase deficiency (G6PD deficiency) in Africa. Carriers of the G6PD allele appear to be protected to some extent against malaria and highest median prevalence of G6PD deficiency (peaking at 32.5%) was predicted across sub-Saharan Africa (Howes et al.,

2012). Second, testing the relative impacts of malaria, nutritional status and hemoglobin levels on education is necessary in defining health policy priorities. Our analysis suggests that for school-age children, malaria might be a priority compared to other health problems. However, proper nutrition, especially from conception to age two, and early childhood stimulation play a critical role in the process of brain formation and development. Low levels of child development are associated with worse school participation and performance, and increased reliance on health care, potentially perpetuating inter-generational poverty cycles. Therefore, the timing of interventions is probably crucial here.

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Figure I: Simple DiD framework

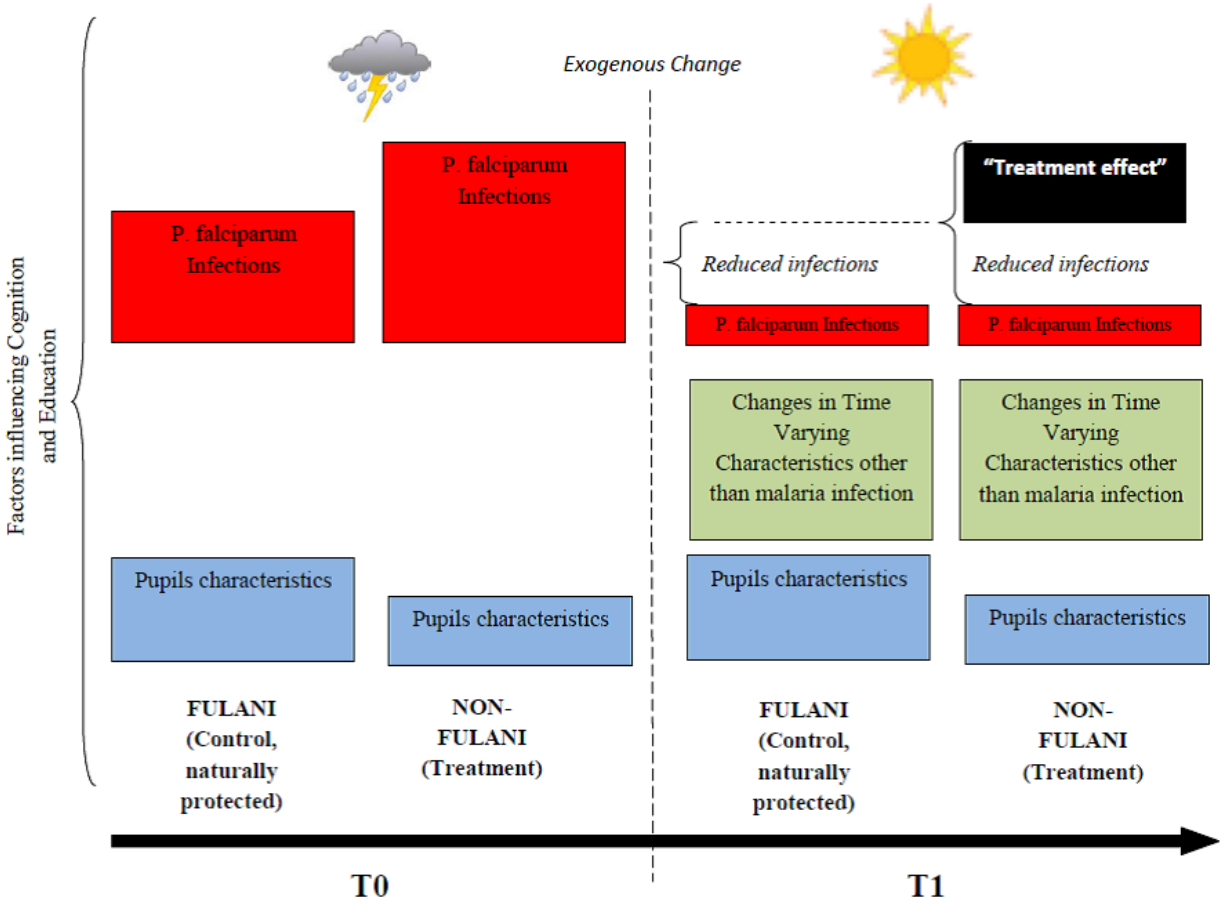

Table 1: Descriptive Statistics

	Rainy (Malarious) season							Dry (non-Malarious) season							Diff-in-Diff	
	Fulani (56)			Non Fulani (240)			Diff	Fulani (51)			Non Fulani (240)			Diff		
	N	Mean	Sd	N	Mean	Sd	Mean	N	Mean	Sd	N	Mean	Sd	Mean	N	Mean
Malaria																
PCR positive	56	0.679	0.471	240	0.871	0.336	-0.192***	51	0.235	0.428	240	0.25	0.43	-0.015	587	-0.178*
Microscopy Positive	56	0.250	0.436	240	0.661	0.474	-0.411***	51	0.040	0.197	240	0.162	0.37	-0.122**	587	-0.288***
Parasite Density (Microscopy)	56	5.500	12.933	240	89.419	329.984	-83.919***	51	0.140	0.756	240	12.76	75.1	-12.621	587	-71.297***
Malaria Symptoms																
Fever (Temp>37.5C)	56	0.143	0.353	240	0.188	0.391	-0.045	51	0.039	0.196	236	0.03	0.170	0.010	583	-0.054
Cephalgia	56	0.054	0.227	240	0.146	0.354	-0.092	51	0.196	0.401	236	0.093	0.290	0.103*	583	-0.195***
Vomiting	56	0.000	0.000	240	0.013	0.111	-0.013	51	0.059	0.238	236	0.000	0.000	0.000	583	-0.013*
Diarrhea	56	0.018	0.134	240	0.004	0.065	0.014	51	0.157	0.464	236	0.004	0.070	-0.004	583	0.018
Abdominal pain	56	0.107	0.312	240	0.083	0.277	0.024	51	-1.924	1.496	236	0.055	0.230	0.004	583	0.020
Health																
Splenomegaly (Hacket classification)	56	0.339	0.745	240	0.100	0.375	0.239***	51	0.157	0.464	236	0.059	0.290	0.098	583	0.142
BMI for age z-score	56	-1.696	1.432	240	-0.473	1.032	-1.224***	51	-1.924	1.496	236	-0.461	1.020	-1.463***	583	0.239
Haemoglobin concentration	56	11.498	1.682	240	11.884	1.193	-0.386*	47	11.172	1.646	232	11.99	1.300	-0.821***	575	0.436***
Presence of intestinal parasites	36	0.361	0.487	211	0.455	0.499	-0.094	-	-	-	-	-	-	-	-	
Presence of eggs of Bilharzia in urine	42	0.690	0.468	197	0.604	0.490	0.086	-	-	-	-	-	-	-	-	
Cognitive tests																
Raven's Progressive Matrices	45	-0.416	0.911	226	-0.223	0.979	-0.194	50	0.231	1.022	229	0.236	0.953	-0.005	550	-0.189
Academic school outcomes																
Educational PCA score	45	-0.306	1.358	201	-0.417	1.314	0.111	44	0.661	1.197	214	0.315	1.317	0.346	504	-0.235
Educational average score	56	-0.074	0.732	240	-0.140	0.626	0.066	51	0.194	0.628	236	0.130	0.638	0.064	583	0.002
Others																
Age	56	10.179	2.629	240	11.000	2.729	-0.821*	51	10.157	2.501	239	10.946	2.747	-0.789	586	-0.196
Gender (Female)	56	0.554	0.502	240	0.496	0.501	0.058	51	0.588	0.497	239	0.502	0.501	0.086	586	-0.028

Notes: This table provides the average or proportion of selected variables before and after the seasonal change for the different groups. The simple difference

columns use simple Student's t-tests. The equation estimated in the last column (Diff-in-Diff) is similar to equation (1) without controls. Put differently the last colum provides γ_1 from an OLS estimation of this equation: $Y_{it} = \alpha_2 + \gamma_1[Post_t \times NonFulani_i] + \gamma_2 Post_t + \gamma_3 NonFulani_i + \epsilon_{it}$, where standard errors are clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table 2: Effect Malaria on Cognitive and Educational Outcomes: Local Average Treatment Effect.

	Whole Sample (Fulani & Non Fulani)					Fulani & Dogon only
	(1)	(2)	(3)	(4)	(5)	(6)
Cognition score (Raven)	-0.370***	-0.777***	-0.692***	-0.712***	-0.877***	-0.880***
	(0.075)	(0.115)	(0.105)	(0.106)	(0.124)	(0.132)
N	550	550	550	542	504	474
R-squared	0.034	-	-	-	-	-
Durbin-Wu-Hausman chi-sq test	-	54.47***	55.861***	54.061***	45.747***	42.574***
Educational score (PCA score)	-0.388***	-1.275***	-1.163***	-1.193***	-1.157***	-1.173***
	(0.111)	(0.149)	(0.150)	(0.154)	(0.164)	(0.171)
N	504	504	503	498	430	402
R-squared	0.020	-	-	-	-	-
Durbin-Wu-Hausman chi-sq test	-	51.309***	52.558***	50.507***	42.853***	39.652***
Educational score (Av. score)	-0.142***	-0.452***	-0.404***	-0.423***	-0.424***	-0.426***
	(0.046)	(0.065)	(0.057)	(0.057)	(0.068)	(0.071)
N	583	583	582	574	556	526
R-squared	0.012	-	-	-	-	-
Durbin-Wu-Hausman chi-sq test	-	53.281***	54.172***	52.934***	42.316***	39.403***
OLS	Yes	No	No	No	No	No
2SLS	No	Yes	Yes	Yes	Yes	Yes
Malaria symptoms	No	No	Yes	Yes	Yes	Yes
Health controls	No	No	No	Yes	Yes	Yes
Wihtin Child fixed effects	No	No	No	No	Yes	Yes

Notes: This table provides the Local Average Treatment Effect. Each cell reports the coefficient from a separate regression corresponding to equation (2) where the first stage is equation (3), provided in Table IV. The measure used for malaria is the PCR measure. We provide similar tables for the other malaria (microscopy either binary or continuous) in Table A2. The last column reduces the sample to the Dogon ethnic

group for the non-Fulani group whereas all other columns include all non-Fulani groups. Malaria controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain. Health controls include splenomegaly, BMI-for-age, haemoglobin concentration. Standard errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table 3: First stage estimates (Linear Probability Model)

	Whole Sample (Fulani & Non Fulani)				Fulani & Dogon only			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Non-Fulani	0.192***	0.197***	0.244***	-	0.188***	0.193***	0.240***	-
	-0.061	-0.061	-0.064		-0.061	-0.06	-0.065	
Post	-0.443***	-0.441***	-0.444***	-0.402***	-0.443***	-0.438***	-0.440***	-0.399***
	-0.095	-0.094	-0.096	-0.107	-0.095	-0.094	-0.096	-0.108
Non-Fulani x Post	-0.178*	-0.185*	-0.177*	-0.206*	-0.181*	-0.192*	-0.183*	-0.211*
	-0.100	-0.100	-0.102	-0.113	-0.101	-0.101	-0.103	-0.113
N	587	583	575	575	554	551	544	544
R-squared	0.359	0.361	0.37	0.511	0.359	0.362	0.368	0.513
Malaria symptoms	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Health controls	No	No	Yes	Yes	No	No	Yes	Yes
Wihtin Child fixed effects	No	No	No	Yes	No	No	No	Yes

Notes: This table provides the first stage of Table 2 and column (4) and (8) corresponds exactly to equation (3). The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani groups. Malaria controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain. Health controls include splenomegaly, BMI-for-age, haemoglobin concentration. Standard errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table 4: Testing other mechanisms

	Whole Sample (Fulani & Non-Fulani)			Fulani & Dogon only		
	Cognition score (Raven)	Educational score (PCA score)	Educational score (Av. score)	Cognition score (Raven)	Educational score (PCA score)	Educational score (Av. score)
PCR postive	-0.933***	-1.564***	-0.497***	-0.901***	-1.457***	-0.475***
	(0.185)	(0.480)	(0.089)	(0.188)	(0.457)	(0.094)
Hgb Level	-0.832	-3.227	-0.511	-0.876	-3.435	-0.548
	(0.780)	(2.735)	(0.316)	(0.821)	(3.027)	(0.334)
N	504	430	556	474	402	526
PCR postive	-1.081***	-1.379***	-0.545***	-1.049***	-1.346***	-0.527***
	(0.266)	(0.269)	(0.094)	(0.256)	(0.268)	(0.095)
At least one symptom	2.251	3.078	0.877	2.35	3.078	0.908
	(1.920)	(1.945)	(0.568)	(2.000)	(1.959)	(0.588)
N	504	430	556	474	402	526
PCR postive	-1.427***	-1.472***	-0.553***	-1.347***	-1.403***	-0.534***
	(0.394)	(0.313)	(0.147)	(0.385)	(0.296)	(0.145)
Treatment	0.502*	0.315	0.132	0.436	0.243	0.116
	(0.292)	(0.222)	(0.103)	(0.286)	(0.210)	(0.100)
N	502	430	552	472	402	522
2SLS	Yes	Yes	Yes	Yes	Yes	Yes
Malaria symptoms	Yes	Yes	Yes	Yes	Yes	Yes
Health controls	Yes	Yes	Yes	Yes	Yes	Yes
Wihtin Child Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes

Notes: This table provides the Local Average Treatment Effect. Each cell reports the coefficient from a separate regression corresponding to equation (2) where we instrument both malaria and the additional health variables simultaneously following equation (3) where two health factors are considered as endogenous. The measure used for malaria is the PCR measure. The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani groups. Standard errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table 5: Effect of Malaria on Cognitive and Educational Outcomes (replacing missing values)

	Whole Sample (Fulani & Non Fulani)					Fulani & Dogon only
	(1)	(2)	(3)	(4)	(5)	(6)
Cognition score (Raven)	-0.433***	-0.967***	-0.864***	-0.886***	-1.019***	-1.029***
	(0.078)	(0.127)	(0.117)	(0.117)	(0.129)	(0.136)
N	565	565	565	557	532	502
Educational score (PCA score)	-0.735***	-2.290***	-2.231***	-2.224***	-2.071***	-2.101***
	(0.122)	(0.215)	(0.224)	(0.216)	(0.219)	(0.226)
N	550	550	549	543	502	472
Educational score (Av. score)	-0.142***	-0.452***	-0.404***	-0.423***	-0.424***	-0.426***
	(0.046)	(0.065)	(0.057)	(0.057)	(0.068)	(0.071)
N	583	583	582	574	556	526
OLS	Yes	No	No	No	No	No
2SLS	No	Yes	Yes	Yes	Yes	Yes
Malaria symptoms	No	Yes	Yes	Yes	Yes	Yes
Health controls	No	No	Yes	Yes	Yes	Yes
Wihtin Child fixed effects	No	No	No	No	Yes	Yes

Notes: This table provides is similar to Table 2 where all the missing educational outcomes for the Fulani were coded at the maximum value of the outcome in the post period and all the missing educational outcomes for the Non-Fulani were coded at the minimum value of the outcome. Each cell reports the coefficient from a separate regression corresponding to equation (2) where the first stage is equation (3), provided in Table IV. The measure used for malaria is the PCR measure. We provide similar tables for the other malaria (microscopy either binary or continuous) in Table A2. The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani groups. Malaria controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain. Health controls include splenomegaly, BMI-for-age, haemoglobin concentration. Standard

errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table 6: Stylized facts at the family level (baseline survey and Mali Demographic and Health Surveys)

	(1)	(2)	(3)
	Educational Investment per children per year (in XOF)	Total number of children per household	Total children ever born per woman (DHS)
Non-Fulani	2,847**	0.949***	0.313*
	(501.400)	(0.037)	(0.189)
Wealth Index	-199.4	0.188***	0.135**
	(99.700)	(0.016)	(0.055)
Constant	7,166***	6.176***	-3.297***
	(570.200)	(0.055)	(0.321)
N households	106	106	989
R-squared	0.03	0.023	0.457

Notes: Cluster-robust Standard errors (at the ethnic level) are in parentheses. *, ** and *** indicate significance at the 10, 5 and 1% levels.

6 Appendix

6.1 Appendix A: Data

6.1.1 Cognitive tests and educational outcomes

We focused on a set of outcomes that are intended to capture a child's human capital accumulation. These include specific tests which have already been used before in Mali. Tests were grade-specific, but not time-specific for two main reasons. First, children get used to the procedure quickly, and second, it is possible to account for learning progression and achievement.

Cognitive outcomes are Raven's Progressive Matrices test scores (Raven, 1984). Raven matrices are available in three different forms for participants of different ability. Only two of them were used for the purpose of this study: Raven's Standard Progressive Matrices and Raven's Coloured Progressive Matrices. Raven tests measure general mental ability and offer information about individual capacity for analyzing and solving problems, abstract reasoning, and the ability to learn. Colored Progressive Matrices were used for children aged 5 to 11 years old and Standard Progressive Matrices were used for older children, following Raven's recommendation. Results from these tests were then standardized by grade across the two follow-ups.

Specific educational tests were developed with the team of Diankabou teachers. They were derived from validated questions already used in other studies (Jukes et al., 2006; Thuilliez et al., 2010) and French models for primary educational tests: BatelemR (Savigny et al., 2001) and Boehm3 (Boehm, 2001). They were next adapted to the village context. This was a multi-step process: first, new questions or statements were developed, based on the format of the original test questions. Next, the content of the assessment was modified, but the basic format remained the same. The teachers assessed the suitability of the content to the age and grade-level programs and defined five categories: mathematics, vocabulary, writing, reading, and visual memory. All tests were administered by the teachers in one session lasting around 45 minutes in the school. All tests were standardized by class, but not by cross-sectional follow-up to take into account the evolution of scores over time. A principal component analysis was then performed on the five variables to calculate an educational

function score (Bartlett, 1937). Condensing the information contained in the original variables into one dimension is justified as the aim is not to assess the impact of malaria only on particular skills, but on the common underlying process involved in knowing, learning, and understanding. In addition, the validity of such an educational function score as a measure of educational achievement in Mali has been tested in Thuilliez et al. (2010), where a high significant correlation between the annual average standardized academic school marks and the annual educational function score was found. However, although the educational function score seems to be a good measure of educational achievement, we also use an arithmetic average as a robustness test[19].

6.1.2 Health status

In order to detect an ongoing *P. falciparum* malaria infection, finger prick blood was collected on filter papers for PCR analyses. Malaria infection is defined as a PCR positive sample (binary variable). This technique is much more sensitive and specific than miscroscopy, as previously noted by Anchinmane and Shedge (2011). In addition, each participant underwent a full clinical examination. Clinical symptoms compatible with malaria are body temperature higher than 37.5 Celsius degrees, cephalgia, vomiting, diarrhea or abdominal pain. Note that most malaria cases were asymptomatic cases (i.e., without any symptoms; Table I). This is consistent with epidemiological studies (Clarke et al., 2004; Dicko et al., 2005): school-age children are the age group most commonly infected with malaria parasites. These infections are usually asymptomatic (without clinical symptoms), so go undetected and thus never get treated. In this study, only acute cases of clinical illness (of any type) were treated immediately and monitored by the medical team directly in the field[20]. Splenomegaly, an enlargement of the spleen resulting from an abnormal immune response to repeated attacks of malaria, was assessed via spleen measurement using the Hackett classification (Hackett, 1944).

The weight and height of each child was measured using an electronic scale and stadiometer respectively. The age of each child was calculated in years and months using the date of birth given by the birth certificate, according to school records and confirmed, if

[19]Note that the numbers of observations for the two academic school outcomes differ slightly because the PCA score excludes all missing values whereas the arithmetic average ignores missing values.

[20]Treatment against asymptomatic forms was provided to all pupils at the end of the study.

necessary, by the village census. In order to compare children of different ages by sex, the anthropometric measurements were converted into Body Mass Index (BMI)-for-age z-score, using the 2000 centre for disease control and prevention growth charts. Blood was obtained by a finger prick under aseptic conditions and the haemoglobin level was determined using a field haemoglobin analyzer. Intestinal parasites such as hookworms, roundworms or microscopic parasites (*Hymenolepis nana*, *Giardia intestinalis* and *Ascaris lumbricoïdes* were detected) and bilharzia (urinary schistosomiasis due to *Schistosoma haematobium*) were detected using WHO recommended methods[21]. As these procedures (collecting feces or urine) are demanding for the children and families, these variables were only collected at the baseline. However, no significant differences were found between the Fulani and the non-Fulani at the baseline and all children were treated preventively for urinary and intestinal parasite diseases at the end of the baseline survey[22]. Note that including other parasite baseline infections in the regression analysis does not affect our results but decreases the size of the sample for the reasons mentioned above.

[21]To detect a maximum number of intestinal parasite cases, three methods were used for the analysis: (i) direct analysis for detecting vegetative forms of protozoan, (ii) Kato-Katz technique (used for the detection of helminthes eggs such as *Ascaris lumbricoïdes*). (iii) Ritchie method, a quantitative method that allows low density of parasite eggs and cysts to be detected. For urinary helminthes, the test consists of detecting the presence of *Schistosoma haematobium* eggs in urine. The filtration method with a Whatman filter was used to quantify egg densities per milliliter of urine.

[22]At the baseline, we could not perform these tests for 49 and 57 pupils among the 296 participants (for intestinal and urinary tests respectively) due to refusals

6.2 Appendix B: Proof of propositions

Proof of Proposition 1. The first order conditions of the optimization problem can be written as:

$$\begin{aligned} -u'\left(w-\left[\left(e+\lambda\right)s\left(p\right)\right]n\right)+R\left[pv'\left(Re\right)+\left(1-p\right)\delta v'\left(\delta Re\right)\right] &= 0, \quad (6)\\ -\left(e+\lambda\right)u'\left(w-\left[\left(e+\lambda\right)s\left(p\right)\right]n\right)+\left[pv\left(Re\right)+\left(1-p\right)v\left(\delta Re\right)\right] &= 0. \quad (7)\end{aligned}$$

By replacing (6) in (7), we obtain that e^* is the solution of:

$$-\left(e+\lambda\right)R\left[pv'\left(Re\right)+\left(1-p\right)\delta v'\left(\delta Re\right)\right]+\left[pv\left(Re\right)+\left(1-p\right)v\left(\delta Re\right)\right]=0. \quad (8)$$

We immediately conclude that

$$\left.\frac{de}{dw}\right|_{e=e^*}=0, \quad (9)$$

which proves the first claim of the proposition. By applying the implicit function theorem to (8), we can compute the derivative of e^* with respect to λ and p and obtain:

$$\begin{aligned} \left.\frac{de}{d\lambda}\right|_{e=e^*} &= \frac{-R\left[pv'\left(Re\right)+\left(1-p\right)\delta v'\left(\delta Re\right)\right]}{\left(e^*+\lambda\right)R^2\left[pv''\left(Re^*\right)+\left(1-p\right)\delta^2 v''\left(\delta Re^*\right)\right]}, \quad (10)\\ \left.\frac{de}{dp}\right|_{e=e^*} &= \frac{-\left(e^*+\lambda\right)R\left[v'\left(Re^*\right)-\delta v'\left(\delta Re^*\right)\right]+\left[v\left(Re^*\right)-v\left(\delta Re^*\right)\right]}{\left(e^*+\lambda\right)R^2\left[pv''\left(Re^*\right)+\left(1-p\right)\delta^2 v''\left(\delta Re^*\right)\right]}. \quad (11)\end{aligned}$$

For $\delta=1$, we immediately see that $de^*/dp=0$. For $\delta>1$, we replace (6) and (7) in (11) to obtain:

$$\left.\frac{de}{dp}\right|_{e=e^*}=\frac{\left(e+\lambda\right)R\delta v'\left(\delta Re\right)-v\left(\delta Re\right)}{\left(e+\lambda\right)pR^2\left[pv''\left(Re\right)+\left(1-p\right)\delta^2 v''\left(\delta Re\right)\right]}. \quad (12)$$

The sign of de^*/dp is thus the opposite of the one of

$$f\left(\lambda\right)=\left(e\left(\lambda\right)+\lambda\right)R\delta v'\left(\delta Re\left(\lambda\right)\right)-v\left(\delta Re\left(\lambda\right)\right) \quad (13)$$

where $e\left(\lambda\right)$ denote the relationship between e^* and λ, which is given by the implicit function (8). We first notice that the strict concavity of v implies $xv'\left(x\right)<v\left(x\right)$ and, thus $f\left(0\right)<0$.

Second, using (10), we obtain:

$$f'(\lambda) = pR\delta \frac{v''(Re)\, v'(\delta Re) - v'(Re)\, \delta v''(\delta Re)}{[pv''(Re) + (1-p)\, \delta^2 v''(\delta Re)]}. \tag{14}$$

Thus, we have for all $x > 0$:

$$f'(\lambda) \geq 0 \Leftrightarrow \frac{d\left(-\frac{xv''(x)}{v'(x)}\right)}{dx} \leq 0. \tag{15}$$

We conclude that if the relative risk aversion is constant or increasing $f(\lambda) < 0$ for all $\lambda \geq 0$. If the relative risk aversion is decreasing, we use a continuity argument to state that $f(\lambda) < 0$ for λ sufficiently small.

Proof of Proposition 2. Let e^*, which is the solution of (8), be expressed as a function of the infection rate $e^* := e(p)$ where $e'(p)$ is given by (12). Using (7), the optimal fertility is given by:

$$-(e(p) + \lambda)\, u'(w - [(e(p) + \lambda)\, s(p)]\, n) + [pv(Re(p)) + (1-p)\, v(\delta Re(p))] = 0. \tag{16}$$

By applying the implicit function theorem, we compute:

$$\frac{dn^*}{dw} = \frac{1}{(e(p) + \lambda)\, s(p)} > 0, \tag{17}$$

which proves the first claim of the proposition. Similarly, we have:

$$\frac{dn^*}{dp} = \frac{v(\delta Re(p)) - v(Re(p))}{(e(p) + \lambda)^2\, s(p)\, u''(.)} - e'(p) \frac{n}{(e(p) + \lambda)} - s'(p) \frac{n}{s(p)}. \tag{18}$$

Using the fact that $de^*/dp = 0$ if $\delta = 1$ (that has been proved in the proof of Proposition 1), we obtain that

$$\left.\frac{dn^*}{dp}\right|_{\delta=1} = -s'(p) \frac{n}{s(p)} > 0, \tag{19}$$

and we conclude by continuity.

6.3 Appendix C: Appendix Figures and Tables

Figure A1: Density of Hemoglobin levels by Ethnic group

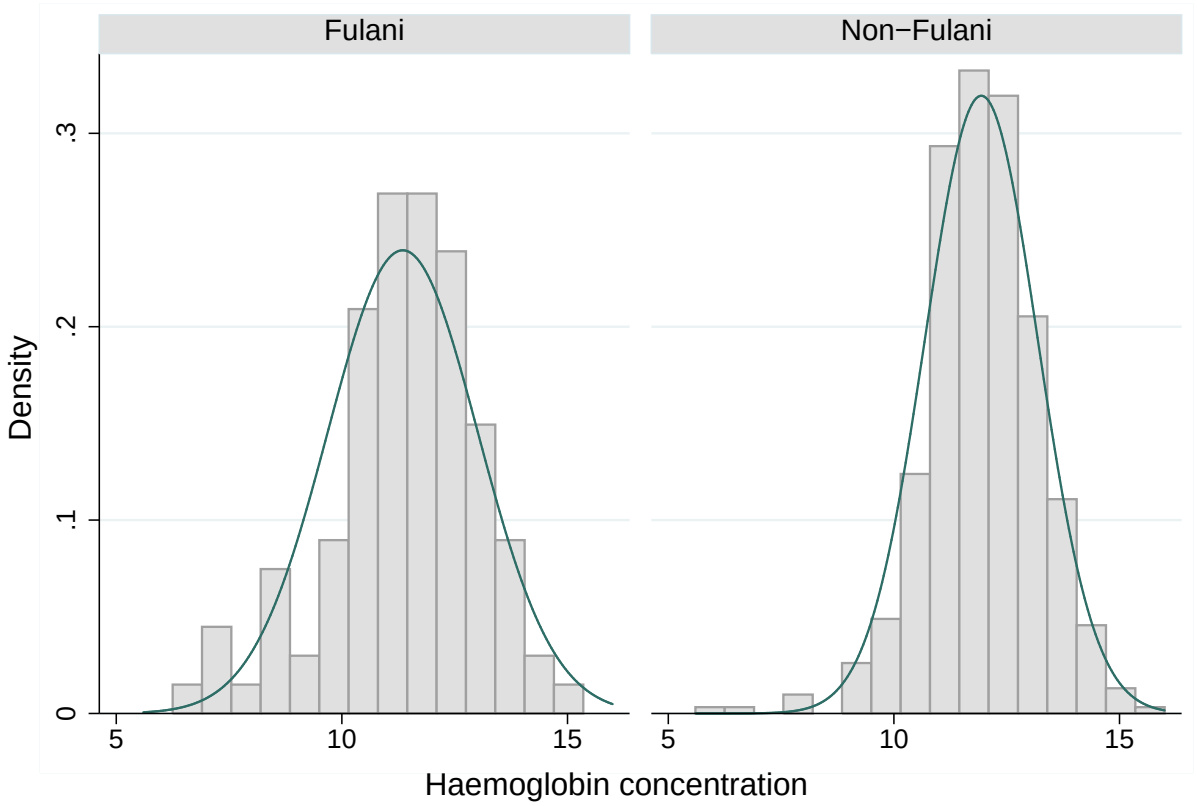

Figure A2: Density of Splenomegaly by Ethnic group

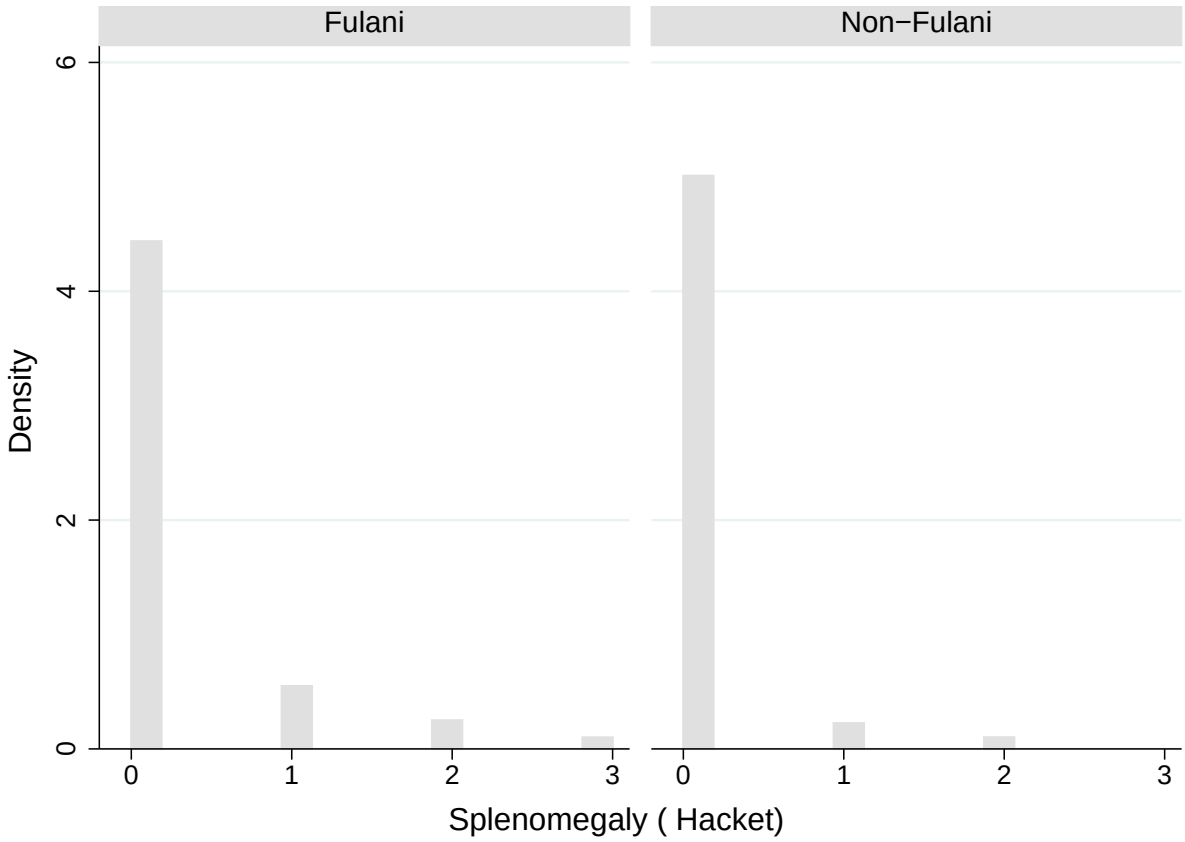

Figure A3: Density of Cognitive outcome by Ethnic group

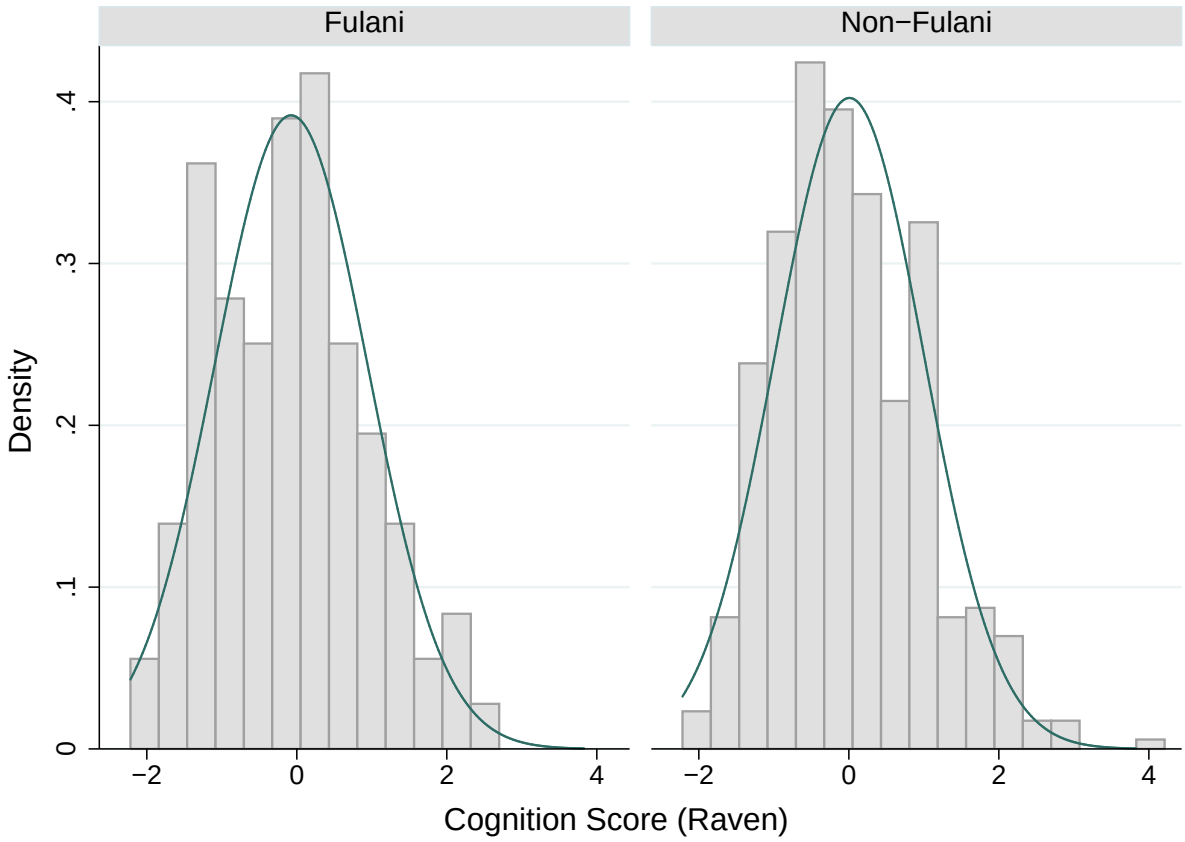

Figure A4: Density of Educational outcome by Ethnic group

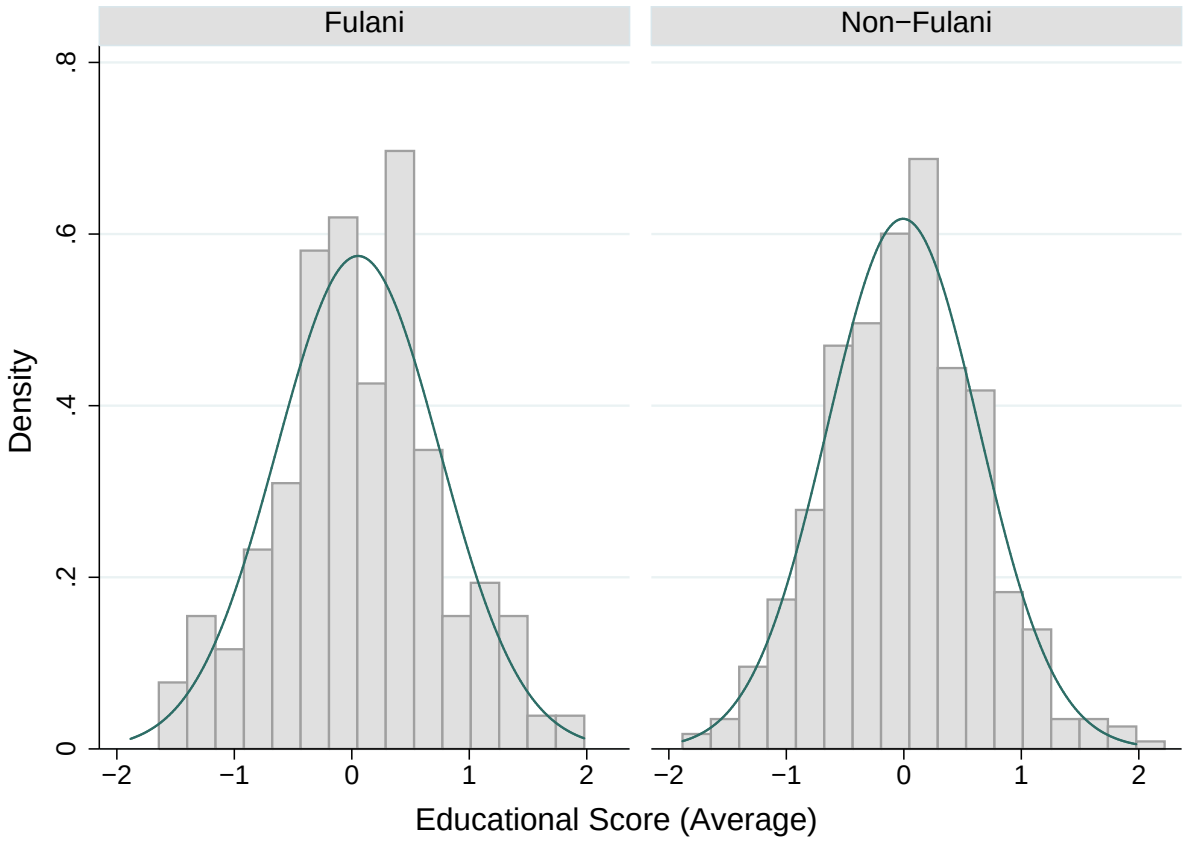

Table A1: Simple DiD with all controls

	Whole Sample (Fulani & Non Fulani)					Fulani & Dogon only
	(1)	(2)	(3)	(4)	(5)	(6)
Cognition score (Raven)	-0.189	-0.165	-0.173	-0.177	-0.118	-0.116
	(0.227)	(0.221)	(0.228)	(0.228)	(0.282)	(0.291)
N	550	550	550	542	504	474
R2	0.063	0.077	0.094	0.097	0.292	0.284
Educational score (PCA score)	-0.235	-0.252	-0.316	-0.298	-0.326	-0.309
	(0.239)	(0.243)	(0.250)	(0.264)	(0.298)	(0.299)
N	504	504	503	498	430	402
R2	0.085	0.092	0.100	0.119	0.397	0.392
Educational score (Av. score)	0.002	-0.006	-0.068	-0.079	-0.051	-0.057
	(0.109)	(0.111)	(0.114)	(0.112)	(0.117)	(0.116)
N	583	583	582	574	556	526
R2	0.044	0.049	0.059	0.074	0.340	0.339
Malaria symptoms	No	Yes	Yes	Yes	Yes	Yes
Health controls	No	No	Yes	Yes	Yes	Yes
Wihtin Child fixed effects	No	No	No	No	Yes	Yes

Notes: This table provides the Average Treatment Effect using the simple DiD presented in equation (1) estimated with OLS and including all controls progressively (malaria controls, health controls, within child fixed effects). Each cell reports the coefficient from a separate regression where γ_1 is reported (the coefficient of the interaction term between $Post \times NonFulani$. The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani groups. Malaria controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain. Health controls include splenomegaly, BMI-for-age, haemoglobin concentration. Standard errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table A2: Effect Malaria on Cognitive and Educational Outcomes: Local Average Treatment Effect (using Microscopy based malaria infection instead of PCR).

	Whole Sample (Fulani & Non Fulani)					Fulani & Dogon only
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Malaria Microscopy (binary)						
Cognition score (Raven)	-0.143	-0.790***	-0.705***	-0.747***	-0.961***	-0.952***
	(0.090)	(0.155)	(0.146)	(0.150)	(0.173)	(0.182)
N	544	544	544	536	494	464
R2	0.005	-	-	-	-	-
Educational score (PCA score)	-0.481***	-1.429***	-1.352***	-1.391***	-1.384***	-1.418***
	(0.113)	(0.210)	(0.213)	(0.211)	(0.200)	(0.212)
N	498	498	498	493	422	394
R2	0.029	-	-	-	-	-
Educational score (Av. score)	-0.165***	-0.518***	-0.478***	-0.513***	-0.503***	-0.501***
	(0.051)	(0.087)	(0.083)	(0.080)	(0.079)	(0.083)
N	575	575	575	567	544	514
R2	0.015	-	-	-	-	-
Panel B: Malaria Microscopy (continuous parasitaemia)						
Cognition score (Raven)	0.000	-0.005***	-0.004**	-0.003*	-0.009***	-0.009***
	(0.000)	(0.002)	(0.002)	(0.001)	(0.003)	(0.003)
N	544	544	544	536	494	464
R2	0.001	-	-	-	-	-
Educational score (PCA score)	0.000	-0.008***	-0.008***	-0.006***	-0.011***	-0.011***
	(0.000)	(0.003)	(0.003)	(0.002)	(0.004)	(0.004)
N	499	499	498	493	422	394
R2	0.001	-	-	-	-	-
Educational score (Av. score)	0.000	-0.003***	-0.003***	-0.002***	-0.004***	-0.004***
	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
N	576	576	575	567	544	514
R2	0.001	-	-	-	-	-
OLS	Yes	No	No	No	No	No
2SLS	No	Yes	Yes	Yes	Yes	Yes
Malaria symptoms	No	Yes	Yes	Yes	Yes	Yes
Health controls	No	No	Yes	Yes	Yes	Yes
Wihtin Child fixed effects	No	No	No	No	Yes	Yes

Notes: This table reproduces Table 2 but use microscopy-based malaria measures. Each cell reports the coefficient from a separate regression corresponding to equation (2) where the first stage is equation (3), provided in Table 4. The measure used for malaria is the PCR measure. We provide similar tables for the other malaria (microscopy either binary or continuous) in Table A1 and A2. The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani

groups. Malaria controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain. Health controls include splenomegaly, BMI-for-age, haemoglobin concentration. Standard errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table A3: Estimating interactions of pre-seasonal differences and malaria on cognitive and educational outcomes.

	Whole Sample (Fulani & Non-Fulani)			Fulani & Dogon only		
	Cognition score (Raven)	Educational score (PCA score)	Educational score (Av. score)	Cognition score (Raven)	Educational score (PCA score)	Educational score (Av. score)
PCR postive*Baseline Anemia	-4.356***	-6.959***	-2.323***	-4.436***	-7.349***	-2.383***
	(0.871)	(2.764)	(1.804)	(0.961)	(1.655)	(0.595)
N	504	430	556	474	402	526
PCR postive*Baseline Splenomegaly	-3.482***	-4.782***	-1.987***	-3.383***	-4.761***	-1.968***
	(1.245)	(1.428)	(0.613)	(1.241)	(1.440)	(0.617)
N	504	430	556	474	402	526
PCR postive*Grade	-0.190***	-0.268***	-0.091***	-0.192***	-0.274***	-0.091***
	(0.029)	(0.040)	(0.016)	(0.031)	(0.043)	(0.018)
N	504	430	556	474	402	526
2SLS	Yes	Yes	Yes	Yes	Yes	Yes
Malaria symptoms	Yes	Yes	Yes	Yes	Yes	Yes
Health controls	Yes	Yes	Yes	Yes	Yes	Yes
Familly fixed effect	Yes	Yes	Yes	Yes	Yes	Yes

Notes: This table provides the Local Average Treatment Effect. Each cell reports the coefficient from a separate regression corresponding to equation (2) where we instrument both malaria and the additional health variables simultaneously following equation (3) where two health factors are considered as endogenous. The measure used for malaria is the PCR measure. The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani groups. Standard errors are reported in parenthesis and clustered at the family level. Note that using

other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.

Table A4: Local Average Treatment effect in another village in Mali.

	Whole Sample (Fulani & Non Fulani)				
	(1)	(2)	(3)	(4)	(5)
Panel A: Malaria Microscopy (binary variable)					
Educational score (see Thuilliez et al., 2010)	-0.084	-4.826***	-5.127***	-5.483***	-7.435***
	(0.069)	(0.646)	(0.725)	(0.824)	(0.946)
N	1794	1794	1794	1794	1794
R2	0.001	-	-	-	-
Panel B: Malaria Microscopy (continuous parasitaemia)					
Educational score (see Thuilliez et al., 2010)	-0.000***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
N	1794	1794	1794	1794	1794
R2	0.001	-	-		-
OLS	Yes	No	No	No	No
2SLS	No	Yes	Yes	Yes	Yes
Malaria symptoms	No	Yes	Yes	Yes	Yes
Health controls	No	No	Yes	Yes	Yes
Wihtin Child fixed effects	No	No	No	No	Yes

Notes: This table provides the Local Average Treatment Effect in another village in Mali. The Table reproduces table II. Each cell reports the coefficient from a separate regression corresponding to equation (2) where the first stage is equation (3), provided in Table IV. The measure used for malaria is the PCR measure. We provide similar tables for the other malaria (microscopy either binary or continuous) in Table A2. The last column reduces the sample to the Dogon ethnic group for the non-Fulani group whereas all other columns include all non-Fulani groups. Malaria controls include all malaria symptoms provided in Table I (five dummies for fever, cephalgia, vomiting, diarrhea, and abdominal pain. Health controls include splenomegaly, BMI-for-age, haemoglobin concentration. Standard errors are reported in parenthesis and clustered at the family level. Note that using other clustering options, either at the individual level or ethnic level, does not change the results. *, ** and *** indicate significance at the 10, 5 and 1% levels.