DISCUSSION PAPER SERIES

DP14705

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DEVELOPMENT ECONOMICS

ISSN 0265-8003

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Discussion Paper DP14705
Published 04 May 2020
Submitted 23 April 2020

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www.cepr.org

This Discussion Paper is issued under the auspices of the Centre's research programmes:

- Development Economics

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JEL Classification: D1, H51, I1, J13, O15

Keywords: Health expenses, Malaria, Africa, Human Capital, Competing Risks

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Private Health Investments under Competing Risks: Evidence from Malaria Control in Senegal

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March, 2020

Abstract

This study exploits the introduction of high subsidies for anti-malaria products in Senegal in 2009 to investigate whether malaria prevents parents from investing in child health. A simple model of health investments under competing mortality risks predicts that private expenses to fight malaria and other diseases should increase in response to anti-malaria public interventions. We test and validate this prediction using original panel data from a household expenditure survey combined with geographical information on malaria prevalence. We find that health expenditures in malarious regions catch up with non-malarious regions. The same result holds for parental health-seeking behavior against other diseases like diarrhea. These patterns cannot be explained by differential trends between regions. Our results suggest that behavioral responses to anti-malaria campaigns magnify their impact on all-cause mortality for children.

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‡The authors are grateful to two anonymous referees, Manisha Shah, Laurent Gobillon, Sylvie Lambert, Pascaline Dupas, Erica Field and Adriana Lleras-Muney for their careful reading of the paper and their numerous suggestions, and to Kenneth Houngbedji, Rozenn Hotte, and Karine Marazyan for their invaluable help with data matching. The paper was inspired by discussions with Denis Cogneau and Pascaline Dupas. The authors would like to thank William Dow, Rachid Laajaj, Karen Macours, Paola Manzini, Hessel Oosterbeek, Yao Pan, Erik Plug, and Liam Wren-Lewis for helpful comments, as well as participants of seminars at PSE, UvA, Lille, UNamur, UNU-Wider, Laval and Wageningen, and several conferences: CSAE, JMA, LAGV, DIAL, AFSE, EEA, Oxdev, ESPE, EALE, and NEUDC. Pauline Rossi gratefully acknowledges financial support from the Netherlands Organisation for Scientific Research (NWO) through the individual starting grant Veni 451-17-012. Declarations of interest for both authors: none.

1 Introduction

Malaria has long been the leading cause of child death in Sub-Saharan Africa, and is expensive to treat and difficult to prevent. In this context, does malaria depress parental investment in child health? To answer this question, we exploit recent interventions that have made anti-malaria products suddenly affordable to most households.

At the beginning of the 21st century, there was a series of initiatives coordinated by the international community under the Roll Back Malaria partnership to galvanize the fight against malaria in Africa. Very large-scale interventions were implemented to distribute anti-malaria products for free or at highly subsidized prices. On the preventive side, 900 million insecticide-treated nets (henceforth ITNs) have been distributed since the early 2000s. Nowadays, an estimated two-thirds of children sleep under an ITN compared to virtually none before the distribution started.[1] On the curative side, free access to treatments called artemisinin-based combination therapies (henceforth ACT) has been promoted. The scope of this intervention is more modest with an estimated 16% of children treated in 2015, but the coverage is increasing rapidly. Over the past 15 years, malaria prevalence and mortality have been halved (World Health Organization, 2015).

In this paper, we examine how health-seeking behavior has changed in response to these interventions. We argue that, before Roll Back Malaria, poor households had few incentives to invest in child health because fighting a major cause of death was unaffordable. Substantially decreasing the price of preventive and curative anti-malaria treatments made it profitable to invest in health, not only to avoid malaria but also other causes of death. Dow et al. (1999) were the first to claim that subsidizing treatments against one disease might boost households' expenses to prevent other diseases, because people allocate efforts to equalize lifetimes from all causes of death. We adapt their framework to show that complementarities also arise in a stochastic setting at the extensive margin. Our model predicts that households should start spending on child health in response to anti-malaria public interventions.

To test this prediction, we exploit original panel data from a Senegalese household survey providing detailed information on health expenses. Malaria control efforts in Senegal took off between the two waves of the panel (2006 and 2011), providing a perfect setting to analyze households' responses. Our empirical strategy is to compare the evolution of child health expenditures between malarious and non-malarious regions of Senegal. We find that child health expenditures were initially lower and increased more, principally at the extensive margin, in malarious regions. The catch-up was not only in levels but also in composition, with parents spending more on preventative care. Triple differences exploiting intra-household comparisons and variation in intensity of campaigns across health districts support the idea that anti-malaria campaigns caused the change in spending behavior: the catch-up (i) does not happen for health spending on

[1] Another preventive intervention is to have public agents spray the inside of dwellings with an insecticide (indoor residual spraying, henceforth IRS), but it covers less than 5% of the population at risk in Africa.

adults, who are less at risk; and (ii) is stronger in districts where more bednets were distributed. Finally, we exploit Demographic and Health Surveys conducted roughly at the same time (2005 and 2010) to show that health-seeking behavior against other diseases, like diarrhea, increased more in malarious regions.

Our results could potentially be driven by differential pre-trends or changes in other determinants of expenditures. We provide evidence that this is not the case. When we account for total income, distance to health facilities, other large-scale public health campaigns, rainfall patterns and region-specific linear trends, our estimates are even larger and more significant. Last, our results are qualitatively unchanged once we account for selective migration, attrition, changes in family structure, and use alternative definitions of high prevalence. We finally discuss the relative roles of the subsidy component and the information component of anti-malaria interventions.

Broadly speaking, our paper fits in the literature on private investments in human capital in developing countries. We examine how health investments respond to changes in a specific mortality risk, while most existing studies look at educational investments. For instance, Jayachandran and Lleras-Muney (2009) show that reductions in maternal mortality risk lead to an increase in girls' educational attainment in Sri Lanka. Similar results have been established by the HIV literature. Areas with the largest increase in HIV prevalence experience the largest decline in schooling in Sub-Saharan Africa (Fortson, 2011). This is later mitigated by access to therapies, which reduces perceived mortality risks and prompts parents to spend more on child education (Baranov and Kohler, 2018; Lucas et al., 2019). As for health outcomes, Oster (2012) finds that reductions in risky sexual behavior in response to the HIV epidemic in Africa are larger in areas with lower non-HIV mortality. A related strand of the literature is primarily interested in whether parents reinforce or attenuate differences in endowments across children. These studies use public interventions as an exogenous change in endowments and typically find that parental investments are higher for children who benefit from the intervention (Almond and Mazumder, 2013). Of particular interest for us are the articles by Adhvaryu and Nyshadham (2014) and Ravindran (2018). The first one shows that children are breastfed longer and vaccinated more often if they previously received iodine in Tanzania; and the second one argues that parental investments reinforce the impact of an early-childhood development program in India. Our focus is different: we aim to explain why complementarities arise, rather than exploring the consequences for intra-household allocations.

Specifically, we are the first to use data on private health expenditures to validate a model with complementarities between disease-specific investments. In their paper, Dow et al. (1999) provide empirical support by showing that birth outcomes improve after child vaccination campaigns in Sub-Saharan Africa. The evidence is only suggestive, because we do not know what additional interventions were embedded in the campaigns, and they might have directly influenced maternal health. We argue that data on health outcomes is not enough and that data on health expenses is necessary to implement a proper, direct test of the model. Providing empirical support for such a model is useful to explain why poor people in insalubrious environments invest little in

their children's health. Treatments to avoid a given disease might be affordable, but once we recognize that there are many diseases, the total cost of fighting against all of them may be prohibitive.

Another contribution is to provide evidence of behavioral responses to health subsidies in Africa. Whether these responses undermine or magnify the intended impact of programs is a long-lasting discussion. A large literature in economics studies whether public interventions crowd out private investments. In the case of health in poor countries, the debate is not over. On the one hand, Bennett (2012) argues that the public provision of health products might generate moral hazard issues. He documents the case of the Philippines, where the introduction of piped water worsened household sanitary behavior. On the other hand, Dupas (2014) argues that subsidies might foster long-run adoption through positive learning effects. Using experimental data from Kenya, she shows that subsidizing ITNs has a positive impact on households' willingness to pay in the future. She finds no evidence of negative behavioral responses such as anchoring effects or cross-product entitlement effects. Armand et al. (2017) do not find empirical support for crowding-out effects, either. They examine the relationship between a private (ITN) and a public (IRS) investment to fight malaria in Eritrea. In line with our results, they find that households were *more* likely to buy a bednet when public health agents had sprayed their own dwellings with insecticide. In our context, we argue that behavioral responses magnify the impact of Roll Back Malaria because there is a complementarity between public and private health expenditures.

Our paper has strong implications for health policies in Africa. It is often argued that horizontal (health system-wide) interventions should be preferred over vertical (disease-specific) interventions, because targeting one disease may lead to negative spillover effects in the health system by drawing resources away from other conditions. Our results suggest that, in the case of anti-malaria campaigns, spillovers can also be positive: people reallocate their resources to fight other diseases. This mechanism helps explain "one of the surprising results to emerge from large-scale trials of insecticide treated bednets – that the reduction in all-cause mortality with the use of bednets is considerably greater than the reduction in malaria-attributed mortality" (Sachs and Malaney, 2002, p.684).

The paper is organized as follows. Section 2 reviews existing evidence on malaria control and child health. Section 3 presents a simple model of health investments. Section 4 describes the data and section 5 explains our empirical strategy. Section 6 provides the main empirical results. Sections 7 and 8 deal with alternative explanations and robustness checks. Section 9 discusses the price and information channels and concludes.

2 Malaria control and child health in Sub-Saharan Africa

Since 2000, the evolution of malaria in Africa has been closely monitored by medical studies.[2] The general consensus is that Roll Back Malaria substantially contributed to reducing malaria

[2]See reviews by World Health Organization (2015), Kesteman et al. (2017) and Appendix 10.1 for more details.

prevalence and mortality across the continent (Bhatt et al., 2015). In Senegal, three articles provide evidence of a causal relationship between malaria control interventions and progress in child health using different, complementary methods: a cross-sectional matching model (Lim et al., 2011), a longitudinal study of a surveillance site (Trape et al., 2014) and a plausibility framework designed by the American Journal of Tropical Medicine and Hygiene (Thwing et al., 2017). In particular, the last study documents that (i) child mortality decreased by 40% between 2005 and 2010, with greater declines among age groups and regions initially most at risk for malaria; and (ii) only 3% of consultations and 4% of deaths were due to malaria after the interventions, against 34% and 18%, respectively, in 2006.

An open question in the literature evaluating the effectiveness of recent malaria control interventions is whether effects are heterogeneous (Kesteman et al., 2017; Yazoume et al., 2017). We shed light on this question in Table 1. Using Demographic and Health Surveys conducted in 19 African countries, we estimate the trends in child mortality before and after the start of Roll Back Malaria, for regions with low and high initial malaria prevalence, distinguishing between rich and poor households. Before the intervention, over the period 1995–2001, child mortality was decreasing for both rich and poor households in regions with low initial prevalence. Whereas in regions with high initial prevalence, only the rich households display a decreasing trend; there was no progress for poor households. After the intervention, mortality also started to decline also for poor households in highly malarious regions; they are the only ones who exhibit a significant break in trends.

The different pre-trends cannot be explained by different health interventions between regions because rich households in malarious regions were able to progress as much as rich households in non-malarious regions. They cannot be explained either by a poverty trap, because in non-malarious regions poor households were able to progress as much as rich households. There seems to be some obstacles specific to being poor *and* living in malarious environments. The explanation we put forward in this paper is that malaria makes health investments unprofitable for poor households and prevents them from benefiting from improvements in other causes of death. For richer households, anti-malaria products were affordable prior to 2002, making health expenses on other diseases worthwhile.[3]

3 Conceptual framework

The main insight from the theoretical literature on health investments decisions is that competing mortality risks generate complementarities between disease-specific investments. In a seminal paper, Dow et al. (1999) argue that the production function of overall lifetime under competing risks is Leontief. In a deterministic setting, this implies that the optimal allocation of investments

[3] An alternative explanation would be that malaria depresses adult health that is, either maternal health or the breadwinner's health. This would limit poor households' ability to care and pay for their children's health. However, the adult health channel fails to account for the patterns in total expenditures that we document in section 7.

equalizes the times of death across all causes. Therefore, a public subsidy related to a specific disease raises private incentives to fight *all* causes of deaths. Chang (2005) further introduces uncertainty about lifetimes in the model and specifies the conditions under which a disease-specific reduction in price is predicted to increase other investments.

These models only discuss interior solutions, whereas corner solutions play a crucial role in our context. Indeed, the majority of parents do not spend anything on child health. In our Senegalese baseline data, less than 40% report having some child health expenditures during the previous year, and seeking some medical care when a child is sick. Child health expenditures account for 3% of total expenditures.[4] In Appendix 10.2, we propose a simple model explaining how competing mortality risks influence the extensive margin i.e., the decision to invest or not in child health. The main idea is that the expected benefit of preventing one cause of death depends on the overall survival function, which is itself a product of cause-specific survival functions. This generates complementarities because some health investments are only worth it when the mortality risk from competing causes has been sufficiently reduced. If the risk of dying from one disease is high, and little can be done because treatments are unaffordable, this makes other investments unprofitable.

Our model predicts that private health expenses should increase in response to Roll Back Malaria. Before the intervention, malaria was a major cause of death. The disease accounted for 17% of deaths among children aged under five in Sub-Saharan Africa in 2000 (World Health Organization, 2015). If we exclude neonatal deaths, resulting primarily from prematurity, birth asphyxia and birth trauma, roughly one death in four was caused by malaria.[5] Anti-malaria products were very expensive, representing between 20% and 40% of total out-of-pocket health expenditures (Mugisha et al., 2002; Onwujekwe et al., 2000). In a competing risk framework, this implies that those households that could not afford to fight malaria also gave up on some other health investments. Roll Back Malaria radically changed the situation by reducing the cost of preventing and curing the disease. Once anti-malaria treatments became affordable, other health investments became worth it. So the dramatic decrease in the price of anti-malaria products is predicted to increase not only investments against malaria but also investments against other diseases.[6] Besides the change in prices, Roll Back Malaria may also have changed beliefs about the mortality risk of malaria. The disease is clearly perceived by parents as the main threat to their children – e.g., Ndiaye et al. (1994) in Senegal, Tarimo et al. (2000) in Tanzania and Deressa and Ali (2009) in Ethiopia. Information campaigns are likely to make the risk of dying from malaria even more salient and to increase confidence in treatments (Armand et al., 2017). In our framework, this would accentuate the cross-disease effect. In the last section of this article we discuss the relative contribution of prices and information in explaining the

[4] To give some perspective, out-of-pocket health expenditures – including adult health – represent about 8–10% of total household expenditures in rural Kenya and urban India (Dupas, 2011).

[5] Other major causes were upper and lower respiratory infections (influenza, diphtheria, pneumonia, bronchiolitis, ear infections), diarrheal diseases, and measles, accounting together for one half of post-neonatal deaths.

[6] Note that a standard model of investment in human capital, without complementarities, would fail to explain why some parents start spending on other diseases when the price of a disease-specific treatment decreases.

changes we observe.

Empirically, as described in the next section, we observe total expenditures on child health and health-seeking behavior against other diseases. To formalize, denote q treatments (curative and preventative) bought by households at price p, and Q treatments provided for free; malaria is subscripted by m and other diseases by o. We have data on $(p_m.q_m+p_o.q_o)$ and $Pr((q_o+Q_o) > 0)$, whereas our theoretical predictions are for q_m and q_o. Changes in total expenditures capture both changes in quantities and changes in prices. Changes in health-seeking behavior are driven by changes in both costly and free treatments. To single out the variation in private investments, we combine three predictions:

P1 Total expenditures on child health should increase.

P2 The proportion of households with no child health expenditures should decrease.

P3 Health-seeking behavior against other diseases should increase.

After Roll Back Malaria, p_m is close to zero so child health expenditures mainly consist of $p_o.q_o$. A rise in p_o would be consistent with prediction 1, but not with predictions 2 and 3. Free distribution of treatments against other diseases would be consistent with prediction 3, but not with predictions 1 and 2. Taken together, the three predictions imply an increase in q_o.

4 Data

We test these predictions in the Senegalese context, where malaria control endeavors started in 2009. We combine three datasets providing information before and after 2009.

Panel data on household expenditures on child health. Our main dataset is the Poverty and Family Structure[7] (*Pauvreté et Structure Familiale*, PSF by its French acronym) panel of individuals. The PSF dataset is a unique panel of individuals, with the first wave in 2006–2007 and the second one in 2011 (DeVreyer et al. (2008)). The first wave (PSF1) is representative of the national population and was conducted on 1,800 households. All individuals from this sample were tracked down during the second wave (PSF2) and interviewed along with all the members of the household they were found to belong to at that point. The number of household splits is sizeable and the second wave covers about 3,200 households.

One original feature of this dataset is that households were divided into sub-units according to their budgetary arrangements. The survey provides information on non-health expenditures made during the last 12 months at the sub-unit level. This is particularly relevant when the household head lives with his brother(s) or has multiple wives. In that case, each mother and

[7]Momar Sylla and Matar Gueye of the Agence Nationale de la Statistique et de la Démographie of Senegal (ANSD), and Philippe De Vreyer (University of Paris-Dauphine and IRD-DIAL), Sylvie Lambert (Paris School of Economics-INRA) and Abla Safir (now with the World Bank) designed the survey. The data collection was conducted by the ANSD.

her dependent children[8] form one sub-unit, since the mother is usually the main caregiver and responsible for her children needs and well-being. In the rest of the paper, we use to the term "household" to refer to the sub-unit. Importantly for our purpose, the survey registers information on private health expenditures paid during the last 12 months before the interview.[9] Roughly two-thirds of health expenditures are devoted to medication purchase, followed by consultation, hospitalization, and commuting to health facilities. These expenses are recorded at the individual level so we have two potential units of observation: either the child or the sibship (all dependent children of the same mother). In the child-level analysis, we have more observations and we follow the exact same individuals so it might seem the relevant unit. However, this approach has several drawbacks. Children in PSF2 are by construction five to six years older than in PSF1. As a consequence, when comparing both waves, we cannot disentangle changes in health-seeking behavior and life-cycle effects. What we want to measure is parental health investment in children, especially in young children who are the most vulnerable. Moreover, some health expenditures might be hard to assign to a given child if they benefit many of them. That is why our preferred unit of analysis is the sibship; we discuss regressions at the child level in robustness tests presented in section 8.

The first column of Table 2 shows some descriptive characteristics. Our sample of interest is made up by 1,594 households that we observe in both waves. In addition, 789 households are only observed in the second wave: these mothers did not have any dependent children in the first wave. In addition, 573 households are only observed in the first wave: 368 mothers no longer had a dependent child in the second wave, and 205 mothers could not be found. Implications of this attrition are discussed in section 8. The main advantage of PSF is to provide a panel so we can estimate regressions with mother fixed effects. The main disadvantage of this dataset is that only expenses are registered, and not health-seeking behavior broken down by disease.[10]

Repeated cross-sections on child health status. To get information by disease, we exploit the Demographic and Health Surveys (DHS hereafter) conducted in Senegal in 2005 and 2010–11. They measure trends in child morbidity and health-seeking behavior. DHS report cases of children aged five and under suffering from diarrhea, fever, and coughing. Parents were asked if they sought treatment when the child was sick. DHS also collects data on vaccination but we do not consider vaccines as an outcome of interest for two reasons. First, they reflect changes in public rather than private health investment. As part of the Expanded Program on Immunization, children are vaccinated for free in public health care facilities or during outreach activities like mobile vaccination teams or annual national vaccination campaigns. Second, coverage was already high in 2005: depending on the vaccine, between three quarters and 90% of children had been vaccinated (Ndiaye and Ayad, 2006). The main drawback is that the survey is a repeated cross-section, so changes over time may capture both changes in behavior

[8] A dependent child is a child under 18 or an unmarried child living with the mother. In both waves, about 17% of children do not live in the same household as their mother.

[9] Only 1% of these expenditures are reimbursed by health insurance schemes.

[10] PSF contains questions about the health status of children and health-seeking behavior, but unfortunately they are not comparable between the two waves of the panel.

and changes in population.

Geographical data on malaria prevalence Our identification strategy exploits the spatial variation in the initial exposure to malaria. We use the Malaria Atlas, a map constructed by epidemiologists, to get a measure of the prevalence before anti-malaria interventions (Bhatt et al., 2015). We chose 2000 as our year of reference because of measurement issues in later years.[11] Both PSF and DHS contain GPS information about the village or city block (that we call "cluster") where the respondent lives, making it possible to merge them with the Malaria Atlas.

5 Empirical strategy

Our model predicts how private investments in child health should respond to anti-malaria campaigns in regions where malaria is endemic. The first source of variation that we exploit is temporal, comparing household expenditures on child health before and after the campaign. However, expenditures may change over time for many reasons unrelated to the intervention of interest. To account for the time-varying determinants of expenditures, we exploit another source of variation, comparing malarious and non-malarious regions of Senegal. Under the assumption that trends in these determinants are the same in all regions, our difference-in-differences strategy identifies responses to anti-malaria campaigns.

In 2008, the Programme National de Lutte contre le Paludisme (PNLP; National Malaria Control Program) initiated a four-year-plan of massive anti-malaria interventions. The PNLP actions were coordinated to achieve the goals of the Roll Back Malaria partnership and involved nearly all national and international partners engaged with malaria prevention and control in the country. As shown by Figure 1, the funds allocated to fight the disease jumped in 2009, once USAID joined the team of donors. The US aid agency coordinates the President's Malaria Initiative, which started in Angola, Tanzania, and Uganda, and was gradually extended to other African countries. Senegal was part of the second wave, along with Malawi, Mozambique, and Rwanda. We therefore argue that large-scale interventions were the result of an exogenous push by foreign aid donors. Beforehand, in the period 2002–2008, only very targeted and small distributions of bednets and other malaria-related goods and services took place (President's Malaria Initiative, 2008). The first nation-wide ITNs distribution campaign took place in June 2009 and targeted specifically children under five and pregnant women. More than 6 million ITNs were distributed between 2008 and 2010 throughout the country, and no specific area was singled out (Plan National de Lutte Contre le Paludisme au Sénégal, 2015). For pregnant women and mothers of under-five children, ITNs could be obtained either for free or at a very subsidized price:

[11] For the year 2000, estimates of prevalence are based on a map of climatic suitability for malaria transmission. For later years, estimates are derived from an epidemiological model combining information on initial conditions as well as coverage and impact of anti-malaria interventions. This model relies on strong assumptions in terms of external validity, linearity and exogeneity, that we are not willing to make. Therefore, we prefer to use the estimates based on initial climatic conditions only. As a robustness test, we consider estimates of prevalence in 2006 and find comparable results (cf. Tables A.1 and A.2 in the Appendix).

maximum of 1 euro, instead of the 10-12 euros market price (President's Malaria Initiative (2008)). The main coverage scheme involved a door-to-door approach to deliver a voucher for an ITN to be redeemed later at a distribution point. The campaign also communicated the importance of using ITNs. As a result, the ITN coverage measured in the DHS-MICS doubled from 20% in 2006 to 40% in 2010. Moreover, in 2010 curative treatments (ACT) were made free for all ages in public health facilities. To sum up, in 2009–2010, the price of preventive and curative treatments against malaria decreased substantially to become virtually zero.

Before anti-malaria campaigns started, there was considerable variation in malaria prevalence across regions of Senegal. The map in Figure 2 represents the proportion of children infected by the parasite in 2000. The proportion ranges from below 2% in the arid region of Louga to above 60% in the areas bordering Guinea. The national average is 24%. We use this threshold to define areas with a low malaria prevalence (below average, in dark blue on the map) and areas with a high prevalence (above average, in light blue and yellow on the map). In low prevalence areas, the average prevalence rate is below 10%, which is considered by epidemiologists as hypoendemic (Bhatt et al., 2015). At the bottom of Figure 2, a map shows the location of the 150 clusters surveyed in PSF, either in high prevalence areas (black triangles) or in low prevalence areas (gray circles). We use the High-Low categories in the descriptive statistics, for clarity of exposition, whereas in the econometric specification, we exploit the continuous variation in prevalence.

We proceed to a difference-in-differences analysis with individual fixed effects:

$$Y_{i,t} = \alpha_0 + \alpha_1 \, Prevalence_i \; + \alpha_2 \, Post_t + \alpha_3 \, Post_t \times Prevalence_i + u_i + \epsilon_{i,t} \tag{1}$$

$Y_{i,t}$ is the outcome of interest: the annual level of child health expenditures per capita in the household (prediction 1), a dummy variable equal to one if the household has no health expenditure (prediction 2), a dummy indicating whether a child was sick and left untreated (prediction 3). $Prevalence_i$ indicates the initial level of prevalence in the city block or village (the "cluster") where household i lives. $Post_t$ equals one if the survey took place after 2009. Standard errors are clustered at the cluster level because malaria prevalence is measured at that level and we have enough clusters. We test predictions 1 and 2 using panel data, in which case we include a mother fixed effect u_i (note that α_1 cannot be estimated). We test prediction 3 using repeated cross-sections, in which case we are not able to include a mother fixed effect. In sections 7 and 8, we introduce time-varying controls to account for potential confounders.

The ideal experiment would be to randomly allocate free anti-malaria treatments in endemic areas and to examine the impact on households' health-seeking behavior.[12] In our setting, anti-malaria campaigns were implemented in the whole country at the same time. There is no area

[12]This experiment is run in Dupas (2014) with another objective: check whether subsidizing ITN decreases the willingness to pay for another health product, water chlorine. She finds no significant impact and therefore rules out cross-product entitlement effects. But the sample size is small and the coefficient is positive and large, suggesting that subsidizing ITN might have fostered the adoption of water chlorine.

excluded or targeted at a later stage that can be used as a control group. Instead, we use areas where the campaign could not make a difference because malaria was already under control. Our counterfactual is not what would have happened in malarious regions in the absence of the intervention, but what would have happened if there was no malaria in these regions. Such a strategy is used by Bleakley (2010), Cutler et al. (2010), and Lucas (2010) to assess the impact of childhood exposure to malaria on socio-economic outcomes. They exploit malaria eradication campaigns in several American and Asian countries. In the same vein, we exploit Roll Back Malaria to test the hypothesis that, when anti-malaria treatments are not heavily subsidized, the disease prevents poor households from investing in child health.

This strategy was recently formalized and named "difference-in-differences in reverse" in an econometric article by Kim and Lee (2018). In a standard difference-in-differences (DD), the treatment and control groups are similar in the pre-period and different in the post-period. In a difference-in-differences in reverse (DDR), they are different at the start, and become similar. This changes the interpretation of the coefficient on the interaction term. DD identifies post-period effects, while DDR identifies pre-period effects. In our context, we can define the treatment as "being affected by malaria." In the pre-period, malaria prevalence is higher in some areas: they form our treatment group. In the post-period, malaria prevalence is low everywhere, as documented in section 2. The interaction term therefore captures the impact of malaria in the pre-period, provided other drivers of health expenditures evolved in the same way for all households.

Is this assumption likely to hold? Columns 2 and 3 of Table 2 provide some descriptive statistics of our sample, by initial malaria prevalence. Household structure is roughly the same everywhere: there are 2.6 children on average, the mother is around 35 years old and the average age of children is seven. But rural and poorer areas tend to be more affected by the disease. We argue that, despite these differences, comparing areas with high and low initial prevalence can be informative for four reasons. First, we use previous DHS waves to look at pre-trends in health-seeking behavior and show that the catch-up had not started beforehand. Second, we exploit the panel structure of PSF to account for composition effects in health expenditures. Indeed, mother fixed effects allow us to disentangle the effect of public subsidies from that of changes in population characteristics. What remains to be discussed are changes in the environment that could have affected low and high prevalence areas differently during our period of interest. This is our third test: we examine differential trends in other determinants of child health expenses: total income, health infrastructure, other health campaigns, rainfall patterns, and geographical dynamics. We show in section 7 that they are unlikely to explain our results. Fourth, we use two triple differences to circumvent the concern that unobserved time-varying heterogeneity between high and low prevalence areas is driving the catch-up.

6 Results

Health expenditures

Figure 3a shows descriptive statistics on health expenditures in the two waves of PSF, comparing areas with a high and low prevalence of malaria. In the first wave, households in high prevalence areas spent much less: on average, 1,720 CFA francs per child per year against 7,335 in low prevalence areas. Between the two waves, they tripled their consumption of health commodities, up to 5,215, while there was no significant change in low prevalence areas. Figure 3b displays the extensive margin. Households in high prevalence areas were 15.7 p.p more likely to not pay expenses toward health in 2006 than the others. In 2011, they had almost caught up. Between the two PSF waves, the proportion of households with zero expenditure decreased by 17.7 p.p whereas a more moderate downturn of 4.7 p.p happened in low prevalence areas.[13]

In Table 3, we include mother fixed effects and exploit the continuous variation in prevalence. Regarding the average level of expenditures, in column 1, results are less strong, suggesting that part of the catch-up is driven by composition effects. But there is also evidence of a change in behavior: the coefficient on the interaction term is no longer significant at conventional levels, but the magnitude remains large. Given the distribution of initial prevalence, it corresponds to a catch-up of 3,835 (resp. 1,726) CFA francs between areas with the highest and the lowest initial prevalence (resp. above and below the average initial prevalence). To put these numbers in perspective, Lépine and Le Nestour (2012) report that, in rural Senegal in 2009, health facilities charged an average of 200 and 100 CFA francs for adult and child outpatient care, respectively. So households increased their expenses on child health by an amount which is not negligible. But this amount is lower than the price of a bednet before malaria control efforts started – around 6,500–8,000 CFA francs (President's Malaria Initiative, 2008). This is consistent with our claim that, in the first wave, households could not afford preventative malaria measures.

Results at the extensive margin are reported in Table 3, column 5. The coefficient on the interaction term remains of similar magnitude and significance as without fixed effects. The fraction of households that started investing in child health after anti-malaria campaigns was higher by 21 p.p. (resp. 9 p.p.) in areas with the highest initial prevalence, compared to the lowest prevalence (resp. in areas above the average initial prevalence compared to areas below). The extensive margin specification gives more precise results than the average spending per capita. The reason is that health expenditures typically display long right tails. There are several options to deal with this type of distributions: excluding outliers (column 2), using a log transformation (column 3) or an inverse hyperbolic sine transformation (column 4). In all specifications, the difference-in-differences coefficient is statistically significant at conventional levels.

We further inspect the distribution of non-zero health expenditures to understand responses at the intensive margin. Figure 4a plots the kernel density estimates before and after the campaigns, in high and low prevalence areas. As expected, distributions are heavily skewed, featuring low modes and long right tails. In low prevalence areas, the distribution does not change between

[13]Estimation of the difference-in-differences regression without fixed effects can be found in Table A.3 in the Appendix. The coefficients on the interaction terms are significantly different from zero.

the two waves. In high prevalence areas, people switch from very low amounts to slightly higher amounts, converging toward the distribution in low prevalence areas. To formally test whether the change in the distribution of non-zero health expenditures was different between high and low prevalence areas, we estimate a quantile regression. Table A.4 in the Appendix reports the quantile difference-in-differences estimates. Coefficients on *Post* are never significant, confirming the absence of changes in low prevalence areas. Coefficients on the interaction term are all positive, confirming that the distribution shifted to the right in high prevalence areas. Importantly, the quantile difference-in-differences estimate at the median (Q50) is 1,500 CFA francs, significant at 1%, representing an increase by 75% relative to the baseline. As expected, coefficients are precisely estimated for lower quantiles, whereas standard errors are large for upper quantiles. Thus, there is a positive response at the intensive margin as well. But it is difficult to detect due to the large variance in health expenditures, especially at the top.

Last, we discuss changes in the composition of health expenditures illustrated by Figure 4b. In low prevalence areas, medication accounts for 60% of expenses, consultation for 31%, commuting to health facilities for 6% and hospitalization for 3%. The breakdown does not change at all between the two waves. The picture is different in high prevalence areas. Before anti-malaria campaigns, households spent a much larger share on medication (75%) and hospitalization (10%) and only 9% on consultation. The scope for preventative care seemed very limited. The breakdown changes markedly after the introduction of anti-malaria subsidies and converges toward the composition observed in low prevalence areas: less on medication and hospitalization, more on consultation. Parents allocated additional expenses differently, with a smaller share spent on curing episodes of malaria and a larger share spent on medical examination. Another way to distinguish between preventative and curative treatments is to look at sick and non-sick children separately (cf. Figure A.1 in the Appendix). The bulk of health expenditures is made on sick children. Still, we observe an interesting pattern on non-sick children: spending in high prevalence areas increased from virtually zero in the first wave to around 2,000 CFA francs in the second wave, while remaining stable in low prevalence areas. The difference-in-differences is significant at 10%. All in all, we find evidence of a catch-up in health expenditures not only in levels but also in composition.

Health-seeking behavior against other diseases

Table 4 Panel A presents the estimates of Equation 1 for health-seeking behaviors related to diarrhea and a fever/cough.[14]. In columns 1 and 2, we look at the probability of being sick and left untreated among all children under age five. Between the two waves, this probability decreased everywhere, and the decline was stronger in areas with higher prevalence. Is the progress driven by fewer disease episodes or by more remedial care? In a nutshell, we find evidence of changes in health-seeking behavior on the preventive side for fever, and on the curative side for diarrhea. In columns 3 to 6, we look at the probability of being sick. The likelihood of a fever,

[14]These are the only diseases on which we have information. In the data, we can distinguish between fever and coughs for the probability of being sick, but not for the probability of being treated.

a major symptom of malaria, decreased more in more malarious areas, supporting the idea that the distribution of ITNs was effective in preventing malarious episodes.[15] Trends for diarrhea and cough are not statistically different between areas. In columns 7 and 8, we restrict the sample to sick children and look at the probability of being left untreated. In the case of diarrhea, parents were significantly less likely to seek medical advice or treatment in higher prevalence areas in 2005. The correlation entirely disappears between the two waves, suggesting that parents started acting upon diarrhea once they had been relieved from malaria. In the case of fever and coughs, the coefficient on the interaction term is negative, but smaller and not significant. This may be explained by a strong attenuation bias generated by selection into illness, since the pool of children suffering from fever changed in malarious areas after the campaigns. Due to data limitations, we cannot run the regression for only coughs. In Panel B, we switch to logit specifications to account for the binary nature of the outcomes. The difference-in-differences coefficients are more precisely estimated than with linear specifications.

We can assess the external validity of our cross-disease result on diarrhea using DHS in other African countries. We need a similar empirical design: anti-malaria campaigns start between the two waves (time variation) and malaria prevalence is low enough in some regions (spatial variation). Two countries comply with these criteria: Kenya and Rwanda. As shown by Table A.5 in the Appendix, the same pattern holds in both countries: high prevalence areas catch up with low prevalence areas in the second wave. The intensity of the catch-up depends on the initial gap – higher in Rwanda, lower in Kenya.

Triple differences exploiting adults and campaign intensity

According to the model, the perceived change in malaria mortality risk is the key driver of the response. Holding initial prevalence constant, health expenses are therefore predicted to increase more in subpopulations with a higher initial risk or with a stronger change in risk. Children are more likely to die from malaria than adults.[16] We can therefore use the evolution of adult health expenditures to account for confounding changes in the demand and supply of health. So far, we only exploited information on children, but we observe annual health expenditures for each member of the household. We are thus able to construct the average health spending per adult in each household.[17] To identify a causal impact, we need to assume that other drivers of health

[15] In levels, parents are not more likely to report that a child was sick in malarious areas, which is at odds with the medical literature describing malaria's toll on child health. One explanation is that measures of self-reported health are influenced by socio-economic status. For instance, Sen (2002) shows that Kerala, the state with the highest life expectancy in India, consistently displays the highest rates of reported morbidity. This may explain why reported morbidity is not lower in lower prevalence areas, where people are on average better off.

[16] In 2000, 91% of malaria-related deaths affected children aged under five: 694,000 deaths in children out of 764,000 deaths in all ages in Africa (World Health Organization, 2015).

[17] Specifically, we estimate the following equation:

$$\begin{aligned} Y_{i,m,t} =& \beta_0 + \beta_1\ Post_t + \beta_2\ Post_t \times Prevalence_m + \beta_3\ Children_i + \beta_4\ Prevalence_m \times Children_i \\ &+ \beta_5\ Post_t \times Children_i + \beta_6\ Post_t \times Prevalence_m \times Children_i + u_m + \epsilon_{i,m,t} \end{aligned} \quad (2)$$

Where $Y_{i,m,t}$ is the annual health spending per adult if $Children = 0$ and per child if $Children = 1$ in the household of mother m in wave t. We include a mother fixed effect u_m to compare the evolution of adults and

expenditures *specific to children* evolved in the same way in high and low initial prevalence regions. Results are reported in Table A.6 in the Appendix. Between the two waves, adult health expenditures tend to increase, and the change is not correlated with malaria prevalence. The coefficient on $Post \times Prevalence$ is small and never statistically significant. By contrast, child health expenditures do increase proportionally with malaria prevalence. Coefficients on the triple interaction have the same sign and magnitude as the difference-in-differences estimates reported in Table 3. This suggests that the demand and supply of health services in general (not specific to children) did not increase more in areas with higher initial malaria prevalence, and hence do not explain the catch-up in child health expenditures.

Another interesting source of variation is the intensity of the June 2009 national campaign. Some of the 76 health districts distributed more ITNs than others. A monitoring report identifies where the distribution was more massive (PNLP, INFORM and LSHTM, 2015). We interact the variables in Equation 1 with $Intensity$ a dummy equal to 1 if the household is located in a health district with more intense campaigns. Estimates are shown in Table A.7 in the Appendix. The catch-up happens everywhere and seems to be faster in districts where more bednets were distributed. Coefficients on $Post \times Prevalence$ are the same as the difference-in-differences estimates, and coefficients on the triple interaction are large, but not significant. By a preponderance of the evidence, we therefore argue that people indeed responded more strongly when they experienced a stronger change in malaria mortality risk.

7 Alternative Hypotheses

Overall, we find that, after anti-malaria campaigns, private health investments, in total and against other diseases, increased more in highly malarious areas than in low prevalence areas. This is consistent with our framework of investments under competing risks. In this section, we discuss five alternative hypothesis that may generate the same empirical patterns: pre-trends, total income, access to healthcare (infrastructure and campaigns), rainfall, and geographical dynamics. We explain why these explanations do not confound our results, and in fact make them stronger.

Pre-trends. The first concern we need to rule out is that malarious areas may have started to catch up with non-malarious areas long before the campaigns. Using DHS conducted in 1992 and 1997, we can look at the evolution of health-seeking behavior against other diseases. Figure 5 plots the proportion of sick and untreated children in the case of diarrhea. There are some fluctuations, 1997 being an especially good year.[18] But these fluctuations are similar

children in the same household. β_2 captures any change in health expenditures correlated with initial malaria prevalence. These changes may be caused by reasons unrelated to malaria control – these regions are different to start with. Or they may happen because of malaria control, but not through the competing risks channel since malaria mortality is low among adults. The competing risk channel, peculiar to children, is captured by β_6. β_6 identifies an additional change in health expenditures on children, compared to adults, which is correlated with initial prevalence.

[18] We investigated whether this could be driven by seasonality in diarrhoeal diseases but it does not seem to be the case. All waves were conducted in the winter and a methodological report assessing the quality of DHS

everywhere; in particular, the catch-up observed between 2005 and 2010 was not underway in the early 2000s.

Total income. The second alternative hypothesis argues that total income grew faster in highly malarious areas. It might be due to confounding factors, or specifically because of malaria control if health improvements benefited adults, in particular mothers and breadwinners. This could lead to a positive income effect in highly malarious areas, for example, through an enhancement of labor productivity. If health investments are normal goods, an increase in income should translate into an increase in health expenditures. To tackle this issue, we test whether there is a differential rise in *all* expenditures. We measure total consumption at the household level, including adults. Descriptive statistics in Table 2 indicate that households in highly malarious areas were poorer than the others to start with: 185 vs. 384 thousands of CFA francs. Column 1 in Table 5 shows that they did not catch up between the two waves. The coefficient on the interaction term is small, insignificant, and if anything negative. Compared to low prevalence areas, households in high prevalence areas did not become richer; they merely reallocated part of their expenses to child health. We tried to identify which expense items experienced a decrease, but the amounts in question are too small to be detected.[19] We specifically looked at adult health expenditures to see if expenses are reallocated from parents to children. This is not the case: adult health expenditures have increased everywhere between the two waves. This is consistent with the competing risk channel and not with the adult health channel.

Access to healthcare. The third alternative hypothesis is about access to healthcare. We focus on three key elements that changed during our period of interest.[20] First, we exploit information about distance to health facilities recorded in the DHS. Mothers are asked whether distance is a main concern when seeking medical advice or treatment for themselves. As shown by Table 5, column 2, access was a greater problem in areas with more malaria in 2005 and there was no catch-up between the two waves. If anything, access seems to have improved less in higher prevalence areas. Second, there was a concomitant campaign against diarrhea.[21] In 2010, approximately 6 million zinc dispersible tablets were delivered to Senegal by UNICEF, and they were only distributed in a few regions (Derosena, 2011). If these regions were predominantly highly malarious areas, this may explain the change in health-seeking behavior against diarrhea described in Table 4, column 7. In fact, the opposite happened: the intervention was piloted first in the low prevalence region of Thies. Using data published in a technical report from UNICEF (Derosena, 2011), we are able to control for the quantity of tablets distributed in each region when testing prediction 3 in Table 5, column 3. As expected, the coefficient on the interaction term increases in size and significance. Third, we examine the introduction of community health

health data finds no evidence of seasonality of diarrhea in the 1997 Senegalese survey (Pullum, 2008).

[19] Child health expenditures account for 3% of the household total consumption.

[20] Ideally, we would need geo-coded data on health provision before and after 2009 to comprehensively examine this mechanism. Service Provision Assessment surveys provide this type of information but the first wave was collected in 2012–2013, so we cannot measure variations.

[21] Another campaign against measles was implemented between 2005 and 2010. Using DHS data, we checked that the progress in immunization coverage was not correlated with initial malaria prevalence.

workers under the program PECADOM (Prise En Charge A Domicile). Health workers conduct door-to-door visits in their village to improve surveillance, testing, and treatment. The system was introduced in three districts in 2008 and gradually expanded to 37 districts by 2011, covering both high and low prevalence areas (Plan National de Lutte Contre le Paludisme au Sénégal, 2012). During that period, health workers focused exclusively on malaria, so these efforts to reach out to everyone cannot drive our cross-disease result. Still, they lower transaction costs and might explain why people start spending on malaria. Plan National de Lutte Contre le Paludisme au Sénégal (2012) provides detailed information on the change in the number of community health workers in each region between the two waves of our panel. When we include this control in Table 5, columns 4 to 6, our coefficients remain stable. So the increase in spending in high prevalence areas is not driven by a thicker network of health workers.

Rainfall. The fourth alternative hypothesis emphasizes the correlation between rainfall and malaria transmission. The occurrence and intensity of malaria infection is closely related to rainfall patterns. The surge in health expenditures in highly malarious regions could potentially result from variations in the environment. If the year 2006 was particularly dry while 2011 was particularly rainy, people would have spent more on curative treatments in the second wave. We rule out this hypothesis using a geo-coded measure of yearly rainfall provided by the Climate Hazards Group[22] that we were able to merge with the PSF panel, with the exception of one cluster. This allows us to compute positive (flood) and negative (drought) rainfall shocks for each PSF cluster and for both waves.[23] It turns out that the year 2006 was slightly more rainy than usual (6 clusters out of 149 experienced a flood) whereas the year 2011 was slightly more dry (12 clusters experienced a drought). As a consequence, our specification would tend to underestimate the causal impact of anti-malaria subsidies on health expenditures. When we control for the annual rainfall deviation from the historical mean, our coefficients of interest increase in magnitude and in significance (cf. columns 7 to 9 in Table 5).

Geographical dynamics. The last alternative hypothesis is that our estimates capture different dynamics between geographical areas, for instance, between different administrative regions of Senegal or between rural and urban areas. Malaria is more prevalent in rural areas, as shown by Table 2. It may be the case that rural areas caught up with urban areas during the period of interest, for at least two reasons. First, there is more room for improvement. Second, food and fuel prices increased substantially between 2006 and 2011, which might have constrained the growth of health spending in urban areas. To rule out this concern, we first look at urban and rural areas separately. Table A.8 in the Appendix show that the same spending pattern is observed in each subpopulation, although we lose a lot of power when splitting the sample. Estimates are larger in urban areas, which is consistent with the theoretical prediction that switchers

[22]We use the Climate Hazards Group InfraRed Precipitation with Station data (CHIRPS) that combines satellite imagery and rainfall station data to produce annual precipitation measure from 1981 to 2015. For more information on this dataset, see http://chg.ucsb.edu/data/chirps/index.html

[23]We define as positive (resp. negative) rainfall shocks the observations whose annual rainfall measure is one standard deviation above (resp. below) the cluster historical mean of annual rainfall calculated over the 1981-2015 period.

are in the middle of the income distribution. In rural areas, the fraction of people for whom it becomes profitable to invest in child health is likely to be small; other constraints remain binding. More generally, in Table A.9 in the Appendix, we account for potentially diverging regional trends by interacting *Post* with dummies for each administrative region of Senegal (see borders in Figure 2). By comparing clusters with a different prevalence located in the same region, we find stronger results for health expenditures (columns 1 to 3) and health-seeking behaviors (columns 4 and 5). The difference-in-differences coefficients are larger, suggesting that spurious geographical dynamics tend to attenuate the mechanism we want to highlight.

8 Robustness

Finally, this section presents robustness tests using alternative definitions of the treatment, units of observations and standard errors. We also account for migration and attrition.

Alternative definition of treatment. The baseline econometric specification uses a continuous measure of prevalence. Alternatively, we can use a binary variable and check that our results hold up to different cuts. Tables A.1 and A.2 in the Appendix report the estimates of interest using as cutoff points the average prevalence in 2000 and in 2006 as well as the thresholds defining low endemicity (10%) and high endemicity (40%) in the medical literature. Coefficients on the interaction term are often more precisely estimated than with the continuous treatment. In particular, the effect at the extensive margin is stronger and more significant when the cut-off point is lower. This is informative about parental beliefs: in the model, the fraction of switchers is related to the difference between the perceived child survival rate with and without investing in anti-malaria products. This fraction should be larger when the initial malaria mortality risk is larger, but theory does not predict whether the relationship should be linear, concave or convex. Our estimates reveal that even small prevalence levels are perceived as an important mortality risk by parents. Only people living in a malaria-free environment do not respond to the campaigns, while people facing a medium endemicity risk respond almost as much as those facing a high risk.[24]

Alternative unit of observations. In the results presented so far, the unit of observation is the household. Per capita child health expenditures are likely to depend on the household structure, like the number of surviving children and their age.[25] One may therefore worry that our coefficient of interest captures a differential change in household structure. For instance, if mothers in malarious regions are more likely to have another child between the two waves and health expenditures are higher on infants than on older children, then we would observe a relative increase in health expenses in these regions. To address this concern, we change the unit of analysis from the household to the child level.[26] We include all children who were born

[24] We come to the same conclusion if we estimate a regression with *Prevalence* and $Prevalence^2$ (not shown).

[25] Mortality is potentially a concern but the number of households experiencing a child death between the two waves is too small (below 2%) to drive our results.

[26] Another option is to introduce some controls related to the household structure: average age of children, number of children, and share of children under five. Our coefficients remain very stable in magnitude and

and living with the mother in PSF1. We follow them in PSF2, whatever the residence status, and examine the evolution of their health expenditures. In Table A.10, columns 1 to 3, in the Appendix, we confirm that testing predictions at the child level leads to the same conclusion as the household-level test: individual expenses increase much more in high prevalence areas. In columns 4 to 9, we split the sample of children depending on their age at the time of the intervention. Coefficients are larger for children younger than five in 2009. This is consistent with our model: episodes of malaria do greater harm to younger children, in particular the under-fives, therefore parental health-seeking behavior should change more in response to the campaign.

Alternative standard errors. We consider two types of adjustments. First, we use a spatial heteroscedasticity and autocorrelation consistent (HAC) estimator (Conley, 1999, 2008). The difference-in-differences p-values for various distance cutoffs (from 10 to 250 kilometers) range from 0.14 to 0.18 when looking at expenditures in levels, and from 0.01 to 0.03 when looking at the extensive margin. Second, we implement a procedure to account for multiple hypothesis testing (Romano and Wolf, 2005*a*,*b*). Those coefficients that are significantly different from zero without the correction remain significant at 10% with the correction. We also perform a joint test of the three predictions, taking together columns 1 and 5 of Table 3 and columns 1 and 2 of Table 4. The significance level of the joint test is 2%.

Geographical mobility. One potential concern is that we define the area of residence – high or low prevalence – using PSF1, and women might have migrated between the two waves. Migration could explain our results if people migrate from high to low prevalence areas and spend more on health in low prevalence areas. This could be the case if people migrate to cities, for instance. The scope for this concern is limited because 93% of the balanced sample stayed in the same city block or in the same village (cf. Table 2). If we exclude migrants, results are very stable, as shown in columns 4 to 6 in Table A.11 in the Appendix.

Selective attrition Another issue would arise if the attrition observed in the PSF panel were selected differentially between malarious and non-malarious areas. Attrition is limited: only 5% of mothers in malarious areas and 12% of mothers in non-malarious areas were not found in the second wave. Our coefficient of interest could potentially be biased upwards in two cases. First, if attrited mothers in non-malarious areas were precisely the ones with a large increase in health expenditures between the two waves. Second, if attrited mothers in malarious areas were precisely the ones with no change in health expenditures. The first condition is unlikely to hold because attrited mothers in non-malarious areas are richer and spend twice as much as non-attrited ones on child health in PSF1. For them it is reasonable to suppose that they were already investing in the prevention of all diseases. Regarding the second condition, attrited mothers in malarious areas were also richer but they spent relatively little on health commodities for their children. If anything, they seem to be in a situation where switching to positive health

significance, as shown by columns 1 to 3 in Table A.11 in the Appendix.

spending is likely. All in all, our coefficient of interest is more likely to be underestimated rather than overestimated by attrition.

9 Discussion and Conclusions

There are two potential channels through which anti-malaria campaigns may affect behaviors: a strong decrease in price and information.[27] On the one hand, Armand et al. (2017) argue that public interventions raise awareness of the dangers of malaria among the population so that people change their beliefs about the returns to avoiding the disease. On the other hand, Dupas (2009) provides experimental evidence that demand for malaria prevention is very sensitive to price, but is not at all influenced by the framing of marketing messages.[28] Which role does information play in our case? First, note that information alone cannot explain the cross-disease effect. The campaign focused exclusively on malaria: it was called Xeex Sibbiru ("Let's fight malaria" in Wolof) and stressed the importance of sleeping under an ITN every night via various channels: posters, certificates given to families who picked up an ITN, singing competitions, etc. In the absence of changes in prices, it is hard to explain why providing information on malaria would raise private spending on other diseases. Second, we exploit the fact that the information component and the subsidy component of the campaigns did not affect everyone in the same way to disentangle their respective impact. Information was primarily targeted at pregnant women or mothers of young children, while a larger share of the population benefited from subsidies. When we split the sample, the effect is not stronger for women who received more information, but we cannot conclude with certainty due to large standard errors. Last, we provide support for the price channel by showing that households that started investing in health after the campaign are in the middle of the income distribution. This is consistent with our model: rich people had already started to invest before the campaign, and very poor people still could not afford any health expense.[29]

In a nutshell, this paper investigates how private health investments responded to subsidies for anti-malaria products introduced in Senegal in the late 2000s. We combine panel data from

[27] A third potential channel could be that distributing free products impacted health care utilization via increased familiarity with health facilities. However, in our context, the room for improvement seems limited because the vast majority of mothers were already used to visiting local health centers to get free care for themselves and their children. In 2005, 87% of pregnant women had received pre-natal care and over 95% of children had received at least one vaccine (Ndiaye and Ayad, 2006).

[28] Similarly in the HIV literature, Godlonton et al. (2016) find that parents are *not* more likely to circumcise their sons once they are informed that circumcision prevents infection.

[29] We can measure household total expenditures in the first wave, by type of transitions. "Never Invest" are households that make no health expenses in both waves, "Switchers" are households with no health expenses in PSF1 and some expenses in PSF2, and finally "Always Invest" are households with some expenses in both waves. "Switchers" are poorer than "Always Invest" and wealthier than "Never Invest." Another way to investigate heterogeneous responses by income level is estimating an order 2 polynomial of income (measured pre-intervention). We interact $Post$ and $Post \times Prevalence$ with $Income$ and $Income^2$ in the specification with health expenditures per capita as the dependent variable. Coefficients on the triple interaction terms are of expected signs: the response increases up to some point and then decreases. For clarity of exposition, Figure A.2 in the Appendix represents estimates of the difference-in-differences as a function of total expenditures over the support observed in PSF1. We find the same inverted-U shape if we use a wealth index instead of income.

a household expenditures survey and repeated cross-sections on health-seeking behavior with geographical information on malaria prevalence. We find that investments to fight malaria and other diseases increased substantially in malarious areas, while they remained stable in non-malarious ones. Pre-trends and changes in total income and access to healthcare do not explain this pattern. We argue that these private responses to a public intervention are consistent with a model of health investments under competing mortality risks, in which public and private spending are complements. Our study concludes that recent anti-malaria interventions in Africa have not crowded out private spending on child health, quite the opposite. Malaria has long prevented parents from investing in child health and heavy subsidies proved to be necessary to alleviate this constraint.

An interesting lead for further research would be to examine whether changes in spending behavior go hand in hand with changes in perceptions of health agency. Once they can afford some investments in child health, do parents feel more empowered? Are they less likely to believe that child survival is first and foremost a matter of luck? Whether parents consider infant mortality as exogenous or endogenous has strong implications for population dynamics, via the nexus mortality-fertility (Cigno, 1998). When parents believe that there is nothing they can do to improve the survival chances of their offspring, this generates a motive for high fertility, namely diversifying mortality risks. Realizing that those chances improve with the amount of resources spent is a precondition for limiting the number of births and investing more in each of them, catalyzing the accumulation of human capital.

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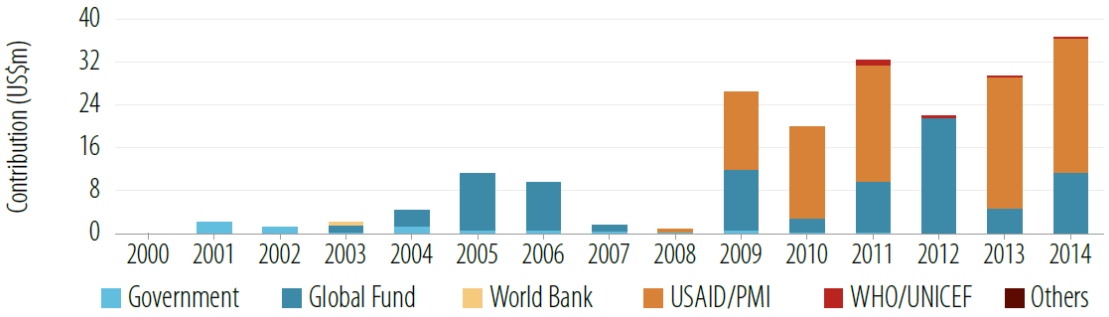
Figures and Tables

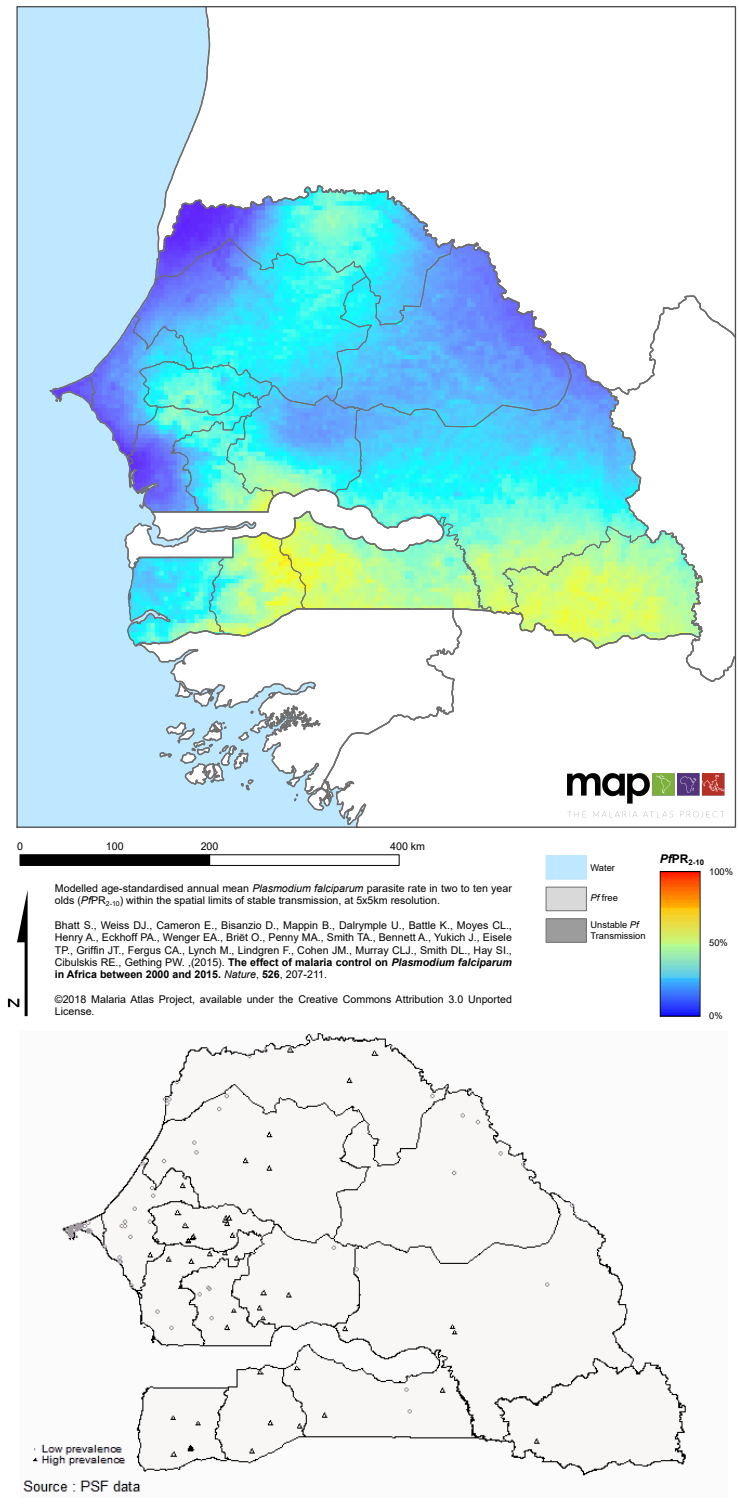
Figure 1: Temporal variation: Funds allocated to anti-malaria interventions



Source: World Health Organization (2015)

The figure shows the amount of funds allocated to anti-malaria interventions in Senegal. There is a jump in 2009, which coincides with the first nationwide distribution of bednets and the free delivery of curative treatments in public health facilities. We have data on private health investments before (in 2005–2006) and after (in 2010–2011) the jump.

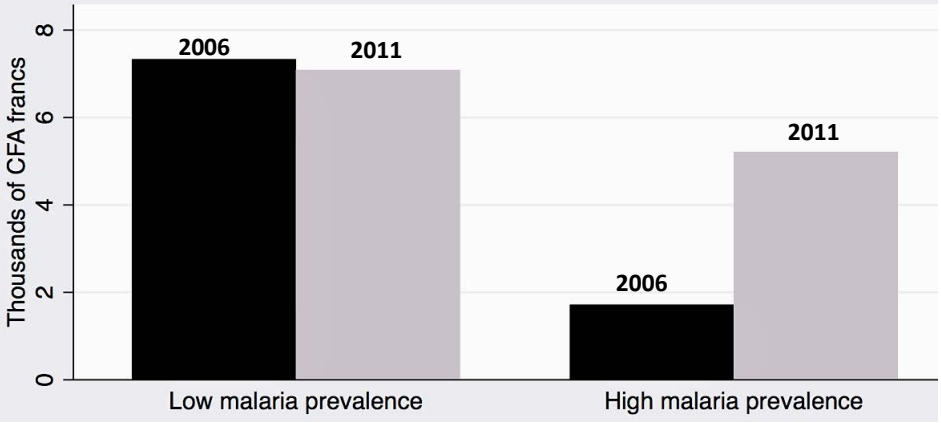
Figure 2: Spatial variation: Initial malaria prevalence



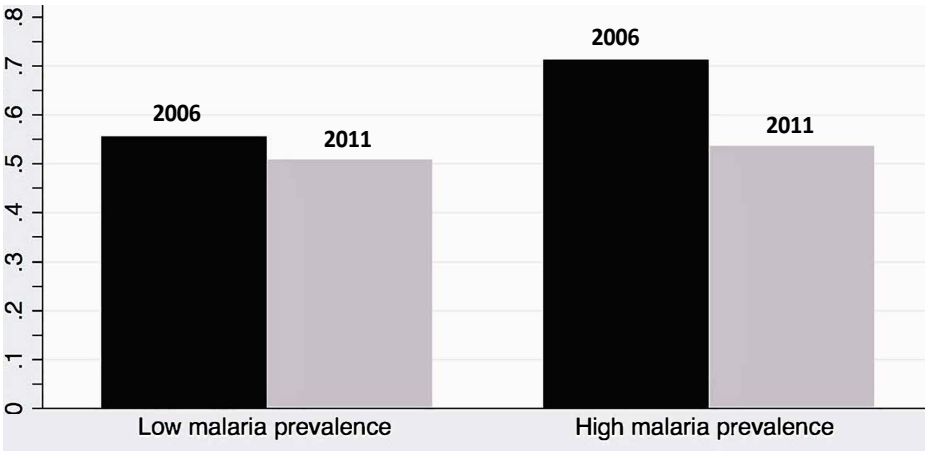
The Malaria Atlas map shows the proportion of children between age 2 and 10 infected by Plasmodium falciparum in 2000. It ranges from below 2% to above 60%. We use the national average, 24%, to define areas with a low malaria prevalence (below average, in dark blue) and areas with a high prevalence (above average, in light blue and yellow). The map on the bottom shows the location of PSF clusters in high (black triangles) and low (gray circles) areas.

Figure 3: Child health expenditures, before and after anti-malaria interventions, by initial prevalence of malaria

(a) Annual expenditures per child



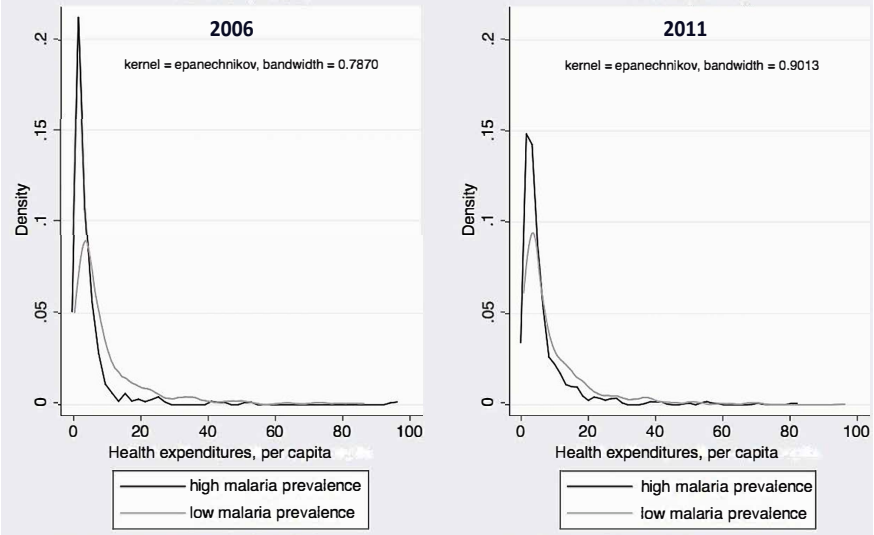
(b) Fraction with zero spending



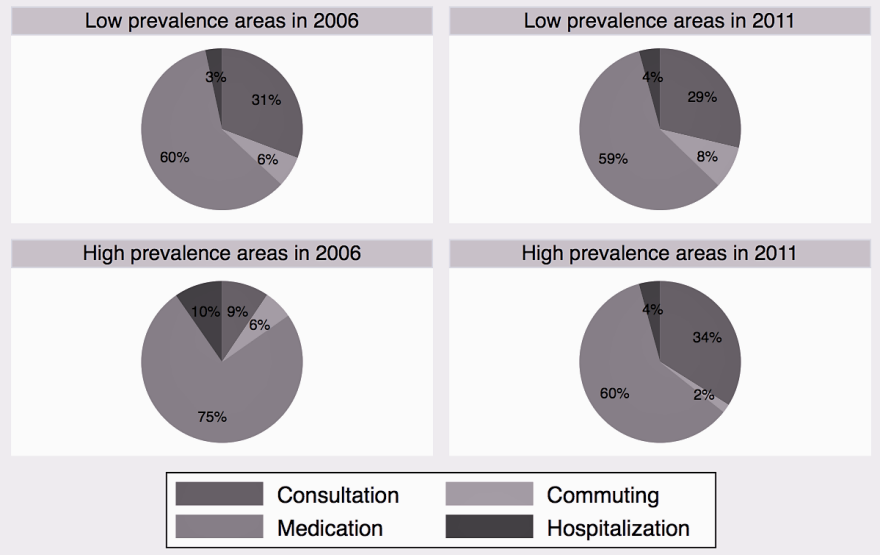
Data: PSF Panel. The figure shows the evolution of (a) annual health expenditures per child, in thousands of CFA francs (1 euro = 656 CFA francs) and (b) the fraction of households with zero spending on child health, in low and high malaria prevalence areas, before (2006 in black) and after (2011 in gray) anti-malaria interventions. Graph (a): the difference-in-differences is equal to 3,739 CFA francs (p-value = 0.051). Graph (b): the difference-in-differences is equal to -13 p.p. (p-value =0.012).

Figure 4: Non-zero child health expenditures, before and after anti-malaria interventions, by initial prevalence of malaria

(a) Distribution

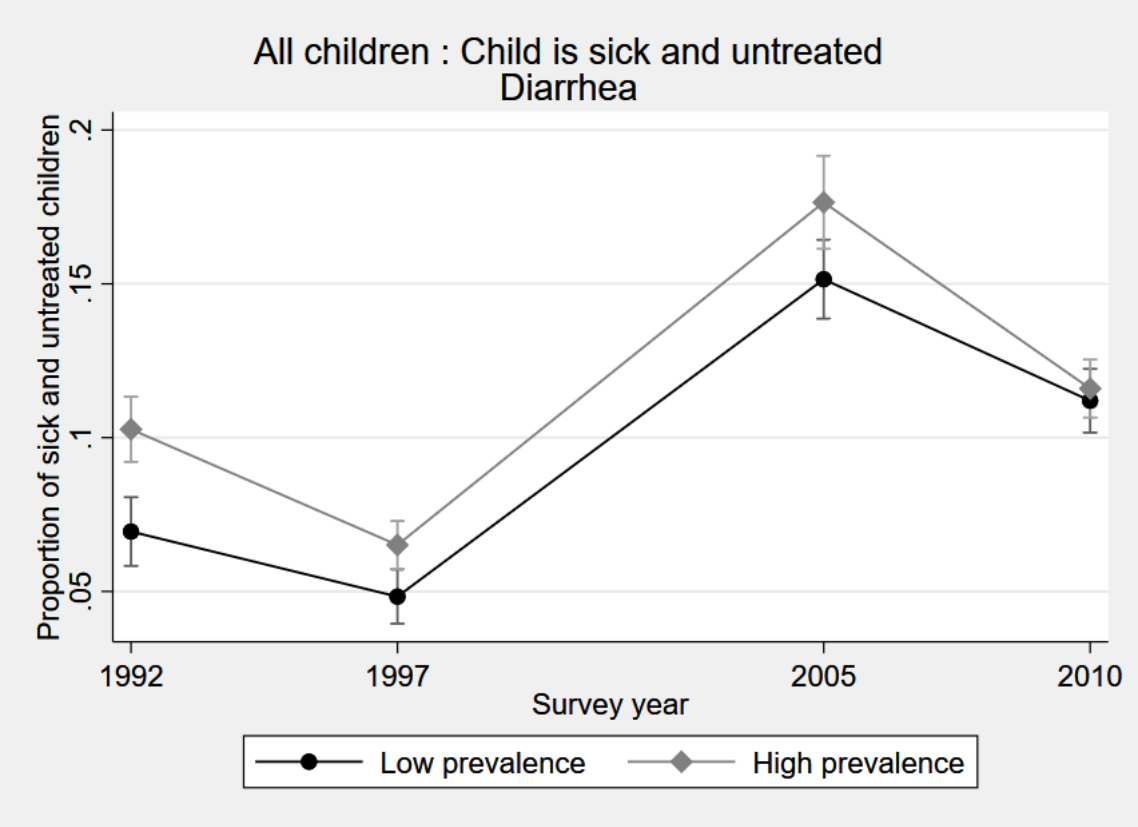


(b) Composition



Data: PSF Panel. Figure (a) plots the kernel density estimates of non-zero expenditures on child health before (graph on the left) and after (graph on the right) anti-malaria campaigns, in low (gray line) and high (black line) malaria prevalence areas.
Figure (b) shows the composition of child health expenditures before (graphs on the left) and after (graphs on the right) anti-malaria interventions, in low (graphs at the top) and high (graphs at the bottom) malaria prevalence areas. Example: in low prevalence areas in 2006, 31% of expenditures were spent on consultation; 6% on commuting to health facilities; 60% on medication; and 3% on hospitalization.

Figure 5: Pre-trends in health-seeking behavior



Data : DHS 1992, DHS 1997, DHS 2005, and DHS 2010. Sample : All children under age 5. The figure shows the proportion of sick and untreated children in the case of diarrhea. Bars represent the 90% confidence intervals.

Table 1: Stylized fact: Trends in child mortality

	High prevalence Poor	*High prevalence Rich*	*Low prevalence Poor*	*Low prevalence Rich*
Linear trend before Roll Back Malaria	−0.0014	−0.0038***	−0.0054***	−0.0044***
	(0.0015)	(0.0013)	(0.0007)	(0.0010)
Linear trend after Roll Back Malaria	−0.0053***	−0.0042***	−0.0057***	−0.0043***
	(0.0007)	(0.0005)	(0.0005)	(0.0005)
Observations	134806	196943	296879	317598
pvalue Before=After	0.033	0.765	0.698	0.950

DHS in: Benin, Burkina Faso, Cameroon, DRC, Ethiopia, Ghana, Guinea, Kenya, Liberia, Malawi, Mali, Namibia, Nigeria, Rwanda, Senegal, Sierra Leone, Uganda, Zambia, Zimbabwe.
The table presents estimates of the linear trend in child mortality before (1995–2001) and after the start (2002–2011) of anti-malaria campaigns for different populations: the richest half and poorest half of households (according to durable goods ownership) in regions with high and low initial malaria prevalence ($\geq 50\%$ or $< 50\%$ of children aged 2 to 10 are infected by the parasite).
We kept only children born at most 10 years before the survey to perform the estimation.
The last line reports the p-value of a test of equality between linear trends before and after 2002.
S.e. in (). * $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table 2: Summary statistics

	Full sample (1)	High prevalence (2)	Low prevalence (3)	pval(diff) (4)
Baseline characteristics in 2006/2007 (PSF1)				
Malaria prevalence in 2000	0.20	0.37	0.09	0.00
Household in Dakar region	0.30	0.00	0.48	0.00
Household in other urban area	0.20	0.17	0.22	0.00
Household in rural area	0.50	0.83	0.30	0.00
Household total consumption (thousands of CFA francs)	310.06	185.40	384.33	0.00
Mother's age	34.99	34.50	35.28	0.11
Panel characteristics				
Number of children in PSF1	2.60	2.70	2.55	0.02
Number of children in PSF2	2.67	2.74	2.62	0.07
Average age of children in PSF1	7.45	7.42	7.47	0.80
Average age of children in PSF2	7.55	7.22	7.75	0.01
Mother in same malaria prevalence cluster btw 2 waves*	0.93	0.93	0.93	0.94
# of clusters	150	48	102	
# of households in PSF1	2167	809	1358	
# of households in PSF2	2383	908	1475	
# of households in both waves	1594	616	978	

Data: PSF Panel. PSF1 is the first wave in 2006–07; PSF2 is the second wave in 2011.
(*): computed only for mothers found in both waves. We define a household as a nuclear family composed of a mother and her dependent children.
The last column shows the p-value of the difference between high and low prevalence areas.

1 euro ≈ 656 CFA francs.

Table 3: Difference-in-differences: Child health expenditures

Specification:	Level (1)	Without top 1% (2)	Log (3)	Ihs (4)	Zero spending (5)
Post × Prevalence	6.392	3.375*	3.092**	0.929**	−0.345**
	(5.800)	(1.885)	(1.348)	(0.369)	(0.168)
Post	0.546	−0.139	0.098	0.014	−0.015
	(1.776)	(0.589)	(0.342)	(0.105)	(0.040)
Mother FE	Yes	Yes	Yes	Yes	Yes
N	4550	4504	4550	4550	4550
Mean of dep. var. in 2006	5.24	3.22	−3.67	0.88	0.61

Data: PSF Panel.
Difference-in-differences regression with mother fixed effects. Linear probability models. The level of observation is a household, composed of a mother and her dependent children.
Dependent variables: health expenditures per capita for children in the household (thousands of CFA francs) in columns (1)-(4); dummy for no health expenditures for any child in the household in column (5).
Column (1): specification in level; column (2): excludes the top 1%; column (3): log transformation - adding 0.001 to deal with zeros; column (4): inverse hyperbolic sine transformation.
Standard errors, in (), are clustered at the PSF cluster level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table 4: Difference-in-differences: Health-seeking behavior

Sample	All children		All children				Sick children	
Dependent variable	*Child is sick and untreated*		*Child is sick*				*Child is untreated*	
	Diarrhea	*Fever and Cough*	*Diarrhea*	*Fever*	*Cough*	*Fever and Cough*	*Diarrhea*	*Fever and Cough*
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*
Panel A: Using a linear probability model								
Post × Prevalence	−0.050	−0.115*	−0.000	−0.137**	−0.021	−0.092	−0.216*	−0.129
	(0.054)	(0.059)	(0.061)	(0.064)	(0.067)	(0.076)	(0.113)	(0.111)
Post	−0.038***	−0.029*	−0.022	−0.047**	−0.053**	−0.059**	−0.128***	0.002
	(0.014)	(0.017)	(0.017)	(0.021)	(0.022)	(0.024)	(0.036)	(0.032)
Prevalence	0.067	0.052	0.019	−0.014	−0.195***	−0.130**	0.240***	0.357***
	(0.045)	(0.051)	(0.048)	(0.050)	(0.054)	(0.061)	(0.081)	(0.081)
Constant	0.148***	0.206***	0.205***	0.293***	0.299***	0.399***	0.726***	0.512***
	(0.011)	(0.014)	(0.012)	(0.015)	(0.017)	(0.018)	(0.026)	(0.023)
Mother FE	No	No	No	No	No	No	No	No
N	21251	21251	21218	21225	21226	21234	4221	6702
Mean of dep. var in 2005	0.17	0.22	0.21	0.29	0.25	0.36	0.79	0.60
Panel B: Using a logit model								
Post × Prevalence	−0.319	−0.774**	0.009	−0.879**	−0.419	−0.566	−1.379**	−0.572
	(0.429)	(0.373)	(0.381)	(0.349)	(0.409)	(0.351)	(0.602)	(0.474)
Post	−0.349***	−0.185*	−0.140	−0.226**	−0.242*	−0.238**	−0.547***	0.016
	(0.122)	(0.111)	(0.109)	(0.111)	(0.124)	(0.109)	(0.180)	(0.132)
Prevalence	0.483	0.303	0.117	−0.068	−1.062***	−0.563**	1.479***	1.515***
	(0.315)	(0.296)	(0.286)	(0.245)	(0.304)	(0.265)	(0.506)	(0.354)
Constant	−1.746***	−1.347***	−1.354***	−0.881***	−0.839***	−0.406***	0.946***	0.036
	(0.083)	(0.083)	(0.075)	(0.073)	(0.087)	(0.077)	(0.148)	(0.096)
Mother FE	No	No	No	No	No	No	No	No
N	21251	21251	21218	21225	21226	21234	4221	6702
Mean of dep. var in 2005	0.17	0.22	0.21	0.29	0.25	0.36	0.79	0.60

Data: DHS 2005 and DHS 2010. Samples: All children under age 5 in columns (1)-(6). Sick children under age 5 in columns (7)-(8).
Difference-in-differences regression without mother fixed effects. Panel A: Linear probability model. Panel B: Logit model.
Dependent variables in:
Columns (1)-(2) and (7)-(8): Dummy equal to 1 if the mother did not seek any medical advice or medical treatment in case of diarrhea (columns (1) and (7)), and fever and/or cough (columns (2) and (8)).
Columns (3)-(6): Dummy equal to 1 if the child suffered from diarrhea (column (3)), fever (column (4)), cough (column (5)), and fever and/or cough (column (6)) in the last two weeks.
Standard errors, in (), are clustered at the DHS cluster level.

Table 5: Tests for alternative explanations

Hypothesis	*Income*	*Health infrastructure*	*Other campaigns*	*Community health workers*			*Rainfall*		
Dependent variable	Household total expenditures	Distance to health facilities is a concern	Child is untreated in case of diarrhea	Expenditures in level	Expenditures in log	Zero spending	Expenditures in level	Expenditures in log	Zero spending
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Post × Prevalence	−84.226	0.064	−0.309**	11.253	3.094*	−0.325*	8.881	3.940***	−0.442**
	(68.264)	(0.144)	(0.133)	(7.316)	(1.582)	(0.194)	(7.096)	(1.447)	(0.177)
Post	23.223	−0.041	−0.125***	0.734	0.098	−0.014	0.588	0.112	−0.017
	(22.569)	(0.044)	(0.036)	(1.770)	(0.341)	(0.040)	(1.772)	(0.343)	(0.041)
Prevalence		0.578***	0.240***						
		(0.109)	(0.081)						
Constant		0.250***	0.726***						
		(0.032)	(0.026)						
Controls	No	No	Zinc distribution	Number of health workers			Rainfall shocks		
Mother FE	Yes	No	No	Yes	Yes	Yes	Yes	Yes	Yes
N	4550	19098	4221	4550	4550	4550	4548	4548	4548
Mean of dep. var. in 2006	310.1	0.40	0.79	5.24	−3.67	0.61	5.24	−3.67	0.61

Data: columns (1) and (4)-(9): PSF Panel. The level of observation is a household, composed of a mother and her dependent children. Columns (2)-(3): DHS 2005 and DHS 2010. Samples: mothers of children under 18 in column (2) and sick children under age 5 in column (3).
Difference-in-differences regression. Linear Probability Model.
Dependent variables: column (1): Total expenditures for the household in the last 12 months in thousands of CFA francs; column (2): Dummy equal to 1 if distance to the nearest facility is a major problem when the respondent is sick and wants to get medical advice or treatment; column (3): Dummy equal to 1 if the mother did not seek any medical advice or medical treatment in case of diarrhea; columns (4) and (7): Health expenditures per capita for children in mother's household (thousands of CFA francs), in level; columns (5) and (8): log transformation - adding 0.001 to deal with zeros; columns (6) and (9): Dummy for no health expenditures for any child in the mother's household.
Controls: Column (3): Quantity of zinc tablets delivered in the region. Columns (4)-(6): number of community health workers in the region. Columns (7)-(9): annual rainfall deviation from the cluster-level mean.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.
Standard errors, in (), are clustered at the DHS or PSF cluster level.

10 Appendix

10.1 Medical literature on the impact of malaria control on child health in Africa

Since the start of Roll Back Malaria in Africa, the evolution of the disease in terms of prevalence and mortality has been closely monitored by the WHO. According to their estimates, the prevalence among children decreased from 33% in 2000 down to 16% in 2015, and the number of deaths caused by malaria among children under five years old decreased from 700K per year down to 300K (World Health Organization, 2015). A growing number of medical studies, recently reviewed by Kesteman et al. (2017), aim at estimating the contribution of malaria control efforts to this progress. The vast majority of articles are either local longitudinal studies, comparing outcomes before and after the campaigns, or cross-sectional studies, comparing areas/individuals with and without anti-malaria treatments. Most studies find a reduction in parasitemia, number of acute cases, and child deaths. An influential article published in Nature concludes that "current malaria interventions have substantially reduced malaria disease incidence across the continent" (Bhatt et al., 2015, abstract).

In Senegal, three articles provide evidence of a causal relationship between malaria control interventions and progress in child health using different, complementary methods. First, Lim et al. (2011) exploit the 2008–09 Malaria Indicators Survey (MIS) to estimate a cross-sectional matching model. They find that malaria prevalence in children aged under five is significantly lower by 33% in households that own an ITN. Second, a longitudinal study in the surveillance site of Dielmo systematically monitored epidemiological data over the period 1990–2012 (Trape et al., 2014). In 1990, malaria was holoendemic in Dielmo: virtually all children were infected, and they experienced five to six malaria attacks per year. Slow progress was made until 2008. The sanitary situation dramatically changed since the deployment of control interventions: the parasite rate fluctuates between 0 and 3% in children, and the incidence of malaria attacks has decreased 98-fold. The authors are able to rule out confounding factors and conclude that "malaria control policies combining ACT and deployment of ITNs can nearly eliminate parasite carriage and greatly reduce the burden of malaria in populations exposed to intense perennial malaria transmission" (Trape et al., 2014, p.1). Third, in a special issue, the American Journal of Tropical Medicine and Hygiene provides a plausibility framework for evaluating the health impact of the scale-up of malaria control interventions on all-cause child mortality in Sub-Saharan Africa. Applying this framework to Senegal, Thwing et al. (2017) argue that "after considering coverage of malaria interventions, trends in malaria morbidity, effects of contextual factors, and trends in child mortality, it is plausible that malaria control interventions contributed to a reduction in malaria mortality and to the impressive gains in child survival in Senegal" (Thwing et al., 2017, p.89). Specifically, they document a decrease in mortality by 40% between 2005 and 2010, with greater declines among age groups and regions initially most at risk for malaria. Using data from routine information systems, they also report that only 3% of consultations and

4% of deaths were due to malaria after the interventions, against 34% and 18%, respectively, in 2006.

10.2 A simple model of private health investment decisions under competing mortality risks

10.2.1 Set up

We model the decision of parents to invest or not in child health, $x = \{0; 1\}$, comparing costs and benefits in a static framework. Consider a setting with two causes of child death: malaria (subscripted by m) and another composite disease (subscripted by o). In a competing risk framework, the overall survival function until date t is given by the product of cause-specific survival functions: $S(t) = S_m(t).S_o(t)$. We denote s_d the probability of surviving cause d until adulthood in the absence of any health investment.

On the benefits side, we assume that parents can eliminate mortality risk from cause d if they choose to invest in the prevention of this disease ($x_d = 1$). We denote v the value of a surviving child, which is assumed to be the same for all households.

On the costs side, we assume that $c(x) = (p_m.x_m + p_o.x_o).\theta$, where p_d is the price of treatments against disease d, and θ is a household-specific parameter reflecting the heterogeneity in costs. The idea is to capture differences in access to healthcare, credit constraints, and proximity to subsistence levels. The same price translates into a higher utility cost if parents have to travel long distances, stand in long lines, go into debt, take on risky jobs, sell valuable assets or forgo satisfying basic needs to get the treatment. We think of θ as an indicator of vulnerability: those households that have a high θ are less able to afford medical care.

The utility depends on (x_o, x_m) as follows:

x_o / x_m	0	1
0	$s_o.s_m.v$	$s_o.v - p_m.\theta$
1	$s_m.v - p_o.\theta$	$v - (p_m + p_o).\theta$

10.2.2 Solution

Let us start by considering cases where there is no trade-off about x_m, either because there is no malaria ($p_m = 0$ so $x_m^* = 1$) or because there is no treatment against malaria ($p_m = +\infty$ so $x_m^* = 0$). Next, we turn to cases where malaria exists and can be prevented.

When malaria does not exist Parents invest in disease o iff the cost is lower than the benefit, i.e., $p_o.\theta \leq (1 - s_o).v$. The distribution of θ in the population gives the fraction of people spending money on child health: $F(\theta_o)$, where $F(.)$ is the c.d.f. of θ, and $\theta_o = \frac{(1-s_o).v}{p_o}$ is

the threshold below which it is profitable to invest in o in the absence of a competing disease. More people invest in preventing a disease if (i) the mortality risk from this disease is higher, and (ii) the price of treatment is lower.

When malaria exists and cannot be prevented Parents invest in disease o iff the utility of investing is greater than the utility of not investing given the mortality risk from malaria, which leads to the condition $\theta \leq s_m.\theta_o$. The presence of malaria reduces the expected benefit from preventing disease o, because it reduces the overall survival probability. The fraction of people investing in child health, $F(s_m.\theta_o)$, is lower than in non-malarious settings.

When malaria exists and can be prevented We derive three segments of interest from the comparison of utilities in the table above:

- when $\theta > \theta_k$, parents never invest in k.
- when $\theta \leq s_j.\theta_k$, parents always invest in k.
- when $s_j.\theta_k < \theta \leq \theta_k$, parents invest in k iff they invest in j.

In the last segment, investing in one disease is profitable iff the other cause of death has been eliminated. This generates a complementarity between disease-specific investments.

The optimal allocation depends on the relative position of the different thresholds. Denote h (resp. l) the disease with the highest (resp. lowest) threshold: $\theta_l < \theta_h$. It is more profitable to invest in h because the mortality risk is higher and/or the price is lower. For clarity of exposition, we assume that $\theta_l < s_l.\theta_h$.[30] There are five segments:

- when $\theta > \theta_h$, parents do not invest in any disease: it is never profitable to invest in l nor in h.
- when $s_l.\theta_h < \theta \leq \theta_h$, parents do not invest in any disease: it is never profitable to invest in l, nor by complementarity in h.
- when $\theta_l < \theta \leq s_l.\theta_h$, parents invest only in h: it is never profitable to invest in l, and always profitable to invest in h.
- when $s_h.\theta_l < \theta \leq \theta_l$, parents invest in both diseases: it is always profitable to invest in h, and by complementarity in l.
- when $\theta \leq \theta_l$, parents invest in both diseases: it is always profitable to invest in h and in l.

[30] If we want to be more general, the two segments of interest are (i) above $max(s_l.\theta_h; \theta_l)$, parents do not invest in any disease, and (ii) below $min(s_l.\theta_h; \theta_l)$, parents invest in both diseases. In-between, many situations can arise, including multiple equilibria, which uselessly complicates the analysis.

10.2.3 Comparative statics

What happens when the price of anti-malaria products drops from very high levels to nearly zero? We assume that the new malaria threshold (θ'_m) moves well above θ_o while the old threshold was well below (cf. Figure A.3). In other words, malaria used to be the binding constraint, depressing investments in (at least some) other diseases, and it is no longer the case once treatments become almost free.

The introduction of subsidies has two effects. First, a direct effect on investments in malaria: when $\theta \in [\theta_m; s_o.\theta'_m]$, parents switch from $x_m = 0$ to $x_m = 1$. Second, an indirect effect on investments in other diseases: when $\theta \in [s_m.\theta_o; \theta_o]$, parents switch from $x_o = 0$ to $x_o = 1$. The fraction of parents who start investing depends on the density of population in those segments. Note that the proportion spending money on o is now the same as in the non-malarious case.

10.2.4 Behavioral insights

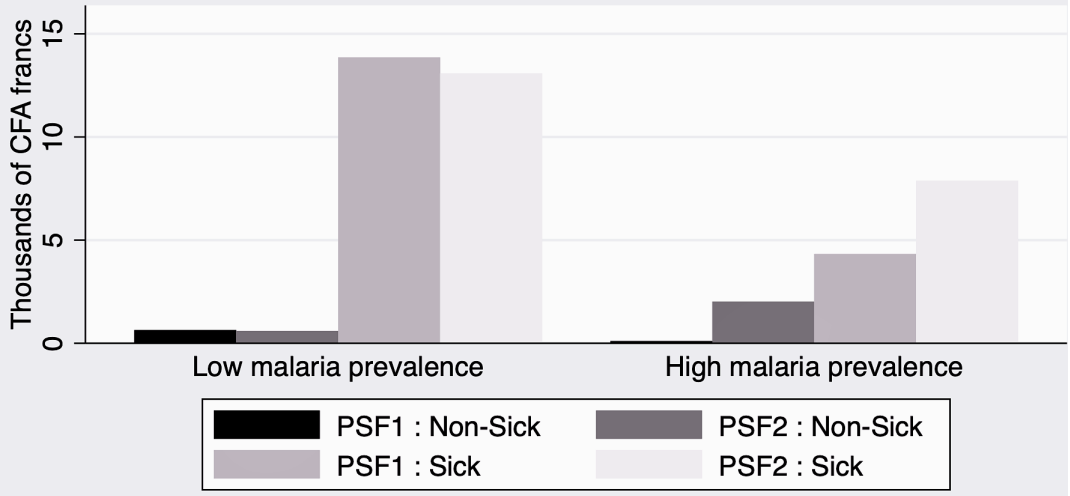
Under the assumption that parents are rational and have perfect information, the model predicts that a fraction of people $F(\theta_o) - F(s_m.\theta_o)$ start investing in the prevention of other diseases when eliminating malaria mortality becomes affordable. One quantity is key in determining the proportion of switchers: the change in malaria mortality risk that can be achieved by investing. This is typically hard to observe for parents. Are there more or fewer switchers if we introduce beliefs?

Let $\widehat{\delta_m}$ be the difference between the perceived survival rate with and without investment. The length of our new segment of interest is $\widehat{\delta_m}.\theta_o$ compared to $(1 - s_m).\theta_o$ in the perfect information setting. There are more switchers when people overestimate the initial malaria mortality risk and believe they can fully eliminate it. But if people underestimate the effectiveness of anti-malaria products, there can be fewer switchers.

The campaign in itself might have an impact on beliefs. It could raise $\widehat{\delta_m}$ by making the risk of dying from malaria more salient and/or increasing confidence in treatments. This would increase the number of switchers.

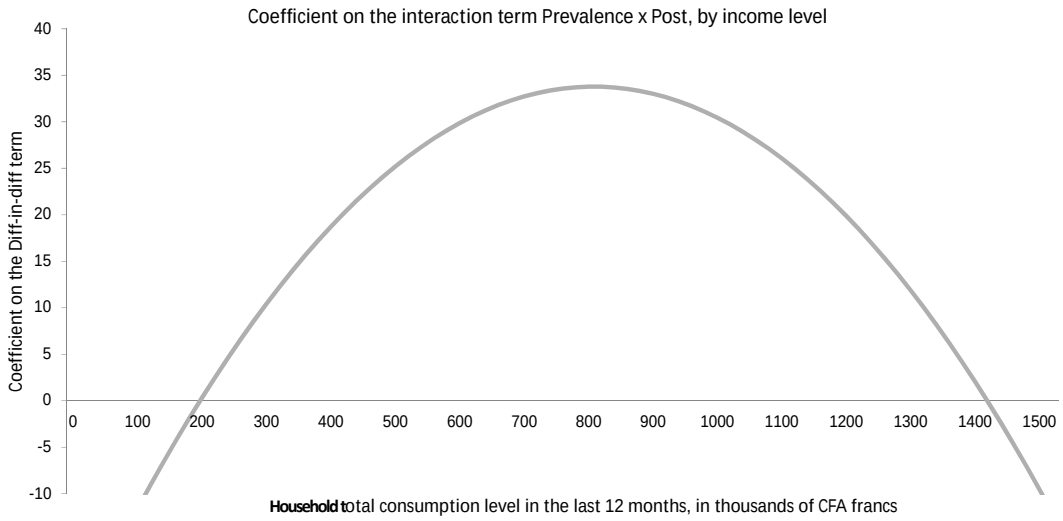
10.3 Figures

Figure A.1: Health expenditures conditional on sickness



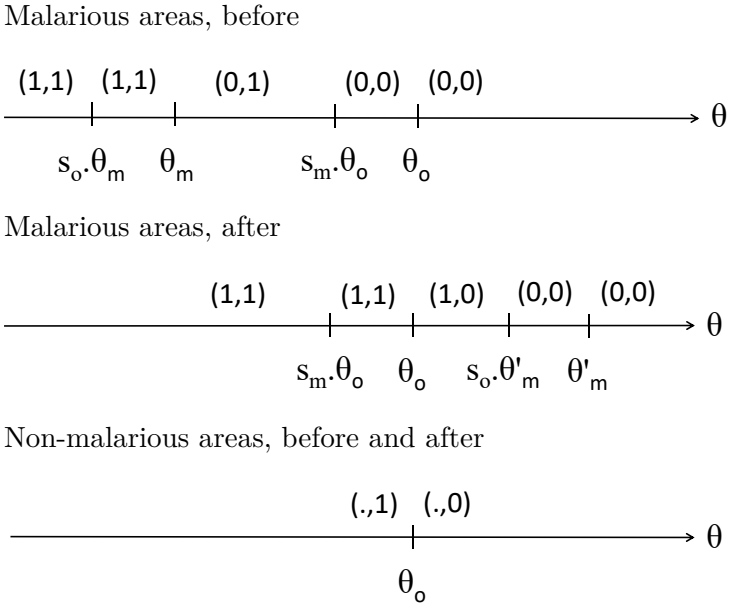
Data: PSF Panel. The figure shows the evolution of annual health expenditures per child, in thousands of CFA francs (1 euro = 656 CFA francs), in low and high malaria prevalence areas, before (PSF1, bars 1 and 3) and after (PSF2, bars 2 and 4) anti-malaria interventions. Households are split into the non-sick sample (those with no sick child during the last 12 months, bars 1 and 2) and the sick sample (those with at least one sick child during the last 12 months, bars 3 and 4). In the non-sick sample, the p-value of the difference-in-differences is 0.10. In the sick sample, the p-value of the difference-in-differences is 0.21.

Figure A.2: Heterogeneity by income



The figure shows the estimated difference-in-differences by income level. Specifically, the graph plots the following equation: $y = -22.4 + 0.1426x - 0.000091x^2$ where x ranges from the minimum to the maximum values of total annual consumption levels observed in PSF1 excluding outliers. The coefficients are obtained by interacting $Post$ and $Post \times Prevalence$ with $Income$ and $Income^2$ in Equation 1.
The coefficient on $Post \times Prevalence$ is -22.4, pvalue $= 0.074$.
The coefficient on $Post \times Prevalence \times Income$ is .1426, pvalue $= 0.049$.
The coefficient on $Post \times Prevalence \times Income^2$ is $-.000091$, pvalue $= 0.123$.

Figure A.3: Health investments (x_m^*, x_0^*) before and after subsidizing anti-malaria products



10.4 Tables

Table A.1: Alternative definitions of prevalence: Child health expenditures

	Level	Log	Zero spending
	(1)	*(2)*	*(3)*
Panel A: average prevalence in 2000			
Post × High Prevalence	2.603	1.036**	−0.115**
	(1.910)	(0.447)	(0.055)
Post	0.842	0.327	−0.041
	(1.465)	(0.277)	(0.033)
Mother FE	Yes	Yes	Yes
N	4550	4550	4550
Mean of dep. var. in low prev. areas in 2006	7.33	−3.06	0.56
Mean of dep. var. in high prev. areas in 2006	1.72	−4.69	0.71
Panel B: high endemicity threshold in 2000			
Post × High Prevalence	3.180	0.773	−0.071
	(3.322)	(0.641)	(0.084)
Post	1.441	0.629***	−0.076***
	(1.071)	(0.238)	(0.028)
Mother FE	Yes	Yes	Yes
N	4550	4550	4550
Mean of dep. var. in low prev. areas in 2006	5.81	−3.57	0.61
Mean of dep. var. in high prev. areas in 2006	1.10	−4.37	0.66
Panel C: low endemicity threshold in 2000			
Post × High Prevalence	0.374	1.481***	−0.179***
	(2.019)	(0.426)	(0.050)
Post	1.607	−0.223	0.030
	(1.503)	(0.320)	(0.036)
Mother FE	Yes	Yes	Yes
N	4550	4550	4550
Mean of dep. var. in low prev. areas in 2006	9.09	−2.40	0.49
Mean of dep. var. in high prev. areas in 2006	2.77	−4.47	0.69
Panel D: average prevalence in 2006			
Post × High Prevalence	2.637	1.712***	−0.204***
	(1.945)	(0.421)	(0.051)
Post	0.657	−0.046	0.007
	(1.636)	(0.270)	(0.032)
Mother FE	Yes	Yes	Yes
N	4550	4550	4550
Mean of dep. var. in low prev. areas in 2006	8.03	−2.73	0.52
Mean of dep. var. in high prev. areas in 2006	1.53	−4.90	0.74

Data: PSF Panel.
Difference-in-differences regression with mother fixed effects. Linear probability model. The level of observation is a household, composed of a mother and her dependent children.
Dependent variable in columns (1) & (2): Health expenditures per capita for children in mother's household (thousands of CFA francs), in level in column (1) and log transformation adding 0.001 to deal with zeros in column (2). Dependent variable in column (3): Dummy for no health expenditures for any child in the mother's household.
Panels A, B and C: we use the malaria prevalence in 2000 to construct the high/low prevalence areas indicators using as cutoff points the mean 24% (Panel A), the high endemicity threshold 40% (Panel B), or the low endemicity threshold 10% (Panel C). Panel D: we use the malaria prevalence in 2006 to construct the high/low prevalence areas indicators (above/below the mean in 2006).
Standard errors, in (), are clustered at the PSF cluster level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.2: Alternative definitions of prevalence: Health-seeking behavior

	Average prevalence in 2000		*High endemicity threshold in 2000*		*Low endemicity threshold in 2000*		*Average prevalence in 2006*	
Sample	All children	Sick children	All children	Sick children	All children	Sick children	All children	Sick children
Dependent variable	*Child is sick*	*Child is untreated*	*Child is sick*	*Child is untreated*	*Child is sick*	*Child is untreated*	*Child is sick*	*Child is untreated*
	Fever	*Diarrhea*	*Fever*	*Diarrhea*	*Fever*	*Diarrhea*	*Fever*	*Diarrhea*
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*
Post × High Prevalence	−0.041**	−0.066*	−0.026	−0.079*	−0.086***	−0.073*	−0.032	−0.077**
	(0.020)	(0.036)	(0.023)	(0.041)	(0.029)	(0.042)	(0.020)	(0.035)
Post	−0.062***	−0.147***	−0.083***	−0.168***	−0.012	−0.126***	−0.074***	−0.154***
	(0.016)	(0.029)	(0.011)	(0.020)	(0.027)	(0.037)	(0.013)	(0.024)
High Prevalence	−0.012	0.076***	0.010	0.067**	−0.021	0.086***	0.008	0.082***
	(0.015)	(0.025)	(0.019)	(0.029)	(0.020)	(0.032)	(0.016)	(0.024)
Constant	0.296***	0.746***	0.287***	0.778***	0.306***	0.721***	0.286***	0.757***
	(0.011)	(0.021)	(0.008)	(0.014)	(0.018)	(0.029)	(0.009)	(0.016)
Mother FE	No	No	No	No	No	No	No	No
N	21225	4221	21225	4221	21225	4221	21225	4221
Mean of dep. var. in low prev. areas in 2006	0.30	0.75	0.29	0.78	0.31	0.72	0.29	0.76
Mean of dep. var. in high prev. areas in 2006	0.28	0.82	0.30	0.84	0.29	0.81	0.29	0.84

Data: DHS 2005 and DHS 2010. Samples: all children under age 5 in columns (1), (3), (5) and (7). Sick children under age 5 in columns (2), (4), (6) and (8).
Difference-in-differences regression without mother fixed effects. Linear probability model.
Dependent variables: we check the robustness of the most important results in Table 4: the child suffered from fever in the last two weeks in columns (1), (3), (5) and (7) and the mother did not seek any medical advice or medical treatment conditionally on the child suffering from diarrhea in columns (2), (4), (6) and (8).
In columns (1) to (6): we use the malaria prevalence in 2000 to construct the high/low prevalence areas indicators using as cutoff points the mean 24% (columns (1) and (2)), the high endemicity threshold 40% (columns (3) and (4)), or the low endemicity threshold 10% (columns (5) and (6)). In columns (7) and (8): we use the malaria prevalence in 2006 to construct the high/low prevalence areas indicators (above/below the mean in 2006).
Standard errors, in (), are clustered at the DHS cluster level.

Table A.3: Regressions corresponding to figure 3

	Level	Zero spending
	(1)	*(2)*
Post × High Prevalence	3.739*	−0.129**
	(1.898)	(0.051)
Post	−0.244	−0.047
	(1.509)	(0.031)
High Prevalence	−5.615***	0.157***
	(1.368)	(0.046)
Constant	7.335***	0.556***
	(1.311)	(0.028)
Mother FE	No	No
N	4550	4550
Mean of dep. var. in low prev. areas in 2006	7.33	0.56
Mean of dep. var. in high prev. areas in 2006	1.72	0.71

Data: PSF Panel.
Difference-in-differences regression without fixed effects. Linear probability model. The level of observation is a household, composed of a mother and her dependent children.
Dependent variables: (1) Health expenditures per capita for children in mother's household (thousands of CFA francs). (2): Dummy for no health expenditures for any child in the mother's household.
Standard errors, in (), are clustered at the PSF cluster level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.4: Quantile Difference in Differences in (non-null) health expenditures

	Health expenditures quantiles								
	Q10	Q20	Q30	Q40	Q50	Q60	Q70	Q80	Q90
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*
Post × High Malaria	0.750***	0.333	0.917**	1.067**	1.500***	1.367*	1.500	4.417**	8.400*
	(0.239)	(0.262)	(0.411)	(0.446)	(0.547)	(0.768)	(1.078)	(1.787)	(4.884)
Post	−0.250	−0.333	−0.333	−0.333	−0.500	−0.200	0.000	−1.000	−1.000
	(0.204)	(0.210)	(0.312)	(0.376)	(0.414)	(0.647)	(0.928)	(1.425)	(4.126)
High Malaria	−0.750***	−1.000***	−1.833***	−2.400***	−3.500***	−4.000***	−6.000***	−10.667***	−19.400***
	(0.216)	(0.167)	(0.264)	(0.302)	(0.408)	(0.515)	(0.697)	(1.205)	(3.749)
Mother FE	No	No	No	No	No	No	No	No	No
N	1981	1981	1981	1981	1981	1981	1981	1981	1981
Mean of dep. var. in low prev. areas in 2006	1.25	2.00	3.00	4.00	5.50	7.00	10.00	16.0	28.0
Mean of dep. var. in high prev. areas in 2006	0.50	1.00	1.17	1.60	2.00	3.00	4.00	5.33	8.60

Data : PSF Panel.
Quantile Differences-in-differences regression. Each column reports the coefficient estimated at the specified quantile. The level of observation is a household, composed by a mother and her dependent children.
Sample: cells with stricly positive expenditures on child health.
Dep var : health expenditures per capita for children in the household (thousands of CFA francs).
Bootstrapped standard errors, in () based on 2,000 replications.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.5: External validity : estimating the cross-disease response in other African countries

	Senegal *(1)*	*Kenya* *(2)*	*Rwanda* *(3)*
Post × High Prevalence	-0.066^{*}	-0.025	-0.167^{***}
	(0.036)	(0.043)	(0.049)
Post	-0.147^{***}	-0.296^{***}	-0.184^{***}
	(0.029)	(0.031)	(0.023)
High Prevalence	0.076^{***}	0.021	0.107^{***}
	(0.025)	(0.038)	(0.027)
Constant	0.746^{***}	0.668^{***}	0.788^{***}
	(0.021)	(0.028)	(0.015)
Mother FE	No	No	No
N	4221	3530	2017
Mean of dep. var. in low prevalence areas in 2006	0.75	0.67	0.79
Mean of dep. var. in high prevalence areas in 2006	0.82	0.69	0.89

Data: Senegal: DHS 2005 and DHS 2010. Kenya: DHS 2003 and DHS 2014. Rwanda: DHS 2005 and DHS 2010.
Sample: sick children under age 5.
In these three countries anti-malaria campaigns start between the two waves (time variation) and malaria prevalence is low enough in some regions (spatial variation). We use the malaria prevalence in 2000 in Senegal to define the high (above the average) and low (below the average) prevalence areas.
Difference-in-differences regression without mother fixed effects. Linear probability model.
Dependent variable: Dummy equal to 1 if the mother did not seek any medical advice or medical treatment conditionally on the child suffering from diarrhea.
Standard errors, in (), are clustered at the DHS cluster level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.6: Triple difference: Intra-household comparisons between adults and children

	Prediction 1		Prediction 2
Specification:	Level	Log	Zero spending
	(1)	(2)	(3)
Post × Prevalence × Children	7.457	2.324*	−0.232
	(9.220)	(1.310)	(0.144)
Post × Prevalence	−0.000	0.764	−0.114
	(9.700)	(1.420)	(0.144)
Post × Children	−2.228	−0.788**	0.078**
	(2.805)	(0.335)	(0.035)
Post	3.181	0.895**	−0.092**
	(2.657)	(0.378)	(0.039)
Prevalence × Children	16.191***	−0.669	0.142
	(6.117)	(1.011)	(0.120)
Children	−13.390***	−2.828***	0.268***
	(2.008)	(0.251)	(0.027)
Mother FE	Yes	Yes	Yes
N	9104	9104	9104
Mean of dep. var. for adults in 2006	15.4	−0.71	0.32

Data: PSF Panel.
Difference-in-differences regression. In both waves, each household provides two observations: the average health spending per adult and the average health spending per child.
Dependent variables: Columns (1)-(2) Health expenditures per adult (Children=0) or per child (Children=1), in thousands of CFA francs. Column (1): specification in level; column (2): log transformation - adding 0.001 to deal with zeros; column (3): Dummy for no health expenditures for any adult (Children=0) or for any child (Children=1).
Standard errors, in (), are clustered at the PSF cluster level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.7: Triple difference: Variation in intensity of the 2009 campaign

	Prediction 1		Prediction 2
Specification:	Level	Log	Zero spending
	(1)	(2)	(3)
Post × Prevalence × Intensity	44.313	3.335	−0.182
	(37.649)	(4.956)	(0.601)
Post × Prevalence	1.830	2.952**	−0.346*
	(4.486)	(1.447)	(0.184)
Post × Intensity	−8.096	−1.031	0.072
	(8.391)	(1.523)	(0.190)
Post	0.840	0.141	−0.018
	(1.817)	(0.351)	(0.041)
Mother FE	Yes	Yes	Yes
N	4550	4550	4550
Mean of dep. var. in 2006 for low intensity districts	5.21	−3.53	0.60

Data: PSF Panel.
Difference-in-differences regression with mother fixed effects. Linear probability model. The level of observation is a household, composed by a mother and her dependent children.
Dependent variables: Columns (1)-(2): Health expenditures per capita for children in the household (thousands of CFA francs). Column (1): specification in level; column (2): log transformation - adding 0.001 to deal with zeros. Column (3): Dummy for no health expenditures for any child in the household.
"Intensity" is a dummy equal to one if the number of ITNs distributed in the household's health district during the 2009 campaign is above the median.
Standard errors, in (), are clustered at the PSF cluster level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.8: Rural and urban samples

	Rural			Urban		
	Level	*Log*	*Zero spending*	*Level*	*Log*	*Zero spending*
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*
Post × Prevalence	3.665	1.512	−0.144	2.091	5.253*	−0.632*
	(6.118)	(1.949)	(0.246)	(8.414)	(2.669)	(0.330)
Post	1.839	0.643	−0.084	0.395	−0.254	0.031
	(1.783)	(0.633)	(0.077)	(2.334)	(0.421)	(0.048)
Mother FE	Yes	Yes	Yes	Yes	Yes	Yes
N	2339	2339	2339	2211	2211	2211
Mean of dep. var. in 2006	2.02	−4.55	0.70	7.37	−2.76	0.52
Number of clusters	64	64	64	86	86	86

Data: PSF Panel. Rural clusters in columns (1)-(3), urban clusters in columns (4)-(6).
Difference-in-differences regression with mother fixed effects. Linear probability model. The level of observation is a household, composed of a mother and her dependent children.
Dependent variables: health expenditures per capita for children in the household (thousands of CFA francs) in columns (1)-(2) and (4)-(5); dummy for no health expenditures for any child in the household in columns (3) and (6).
Columns (1) and (4): specification in level; columns (2) and (5): log transformation - adding 0.001 to deal with zeros.
Standard errors, in (), are clustered at the PSF cluster level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.9: Controlling for regional trends

	Predictions 1 and 2			Prediction 3	
	Level	Log	Zero spending	Diarrhea	Fever and Cough
	(1)	*(2)*	*(3)*	*(4)*	*(5)*
Post × Prevalence	11.188	4.324**	−0.488*	−0.076	−0.188**
	(6.973)	(2.139)	(0.267)	(0.074)	(0.088)
Post	2.003	−0.336	0.045	−0.039	−0.058**
	(1.950)	(0.390)	(0.044)	(0.025)	(0.025)
Prevalence				0.113*	0.242***
				(0.058)	(0.071)
Constant				0.186***	0.232***
				(0.018)	(0.020)
Mother FE	Yes	Yes	Yes	No	No
N	4550	4550	4550	21251	21251
Mean of dep. var. in 2006	5.24	−3.67	0.61	0.17	0.22

Data: Columns (1)-(3): PSF Panel. Columns (4)-(5): DHS 2005 and DHS 2010, sample of all children under age 5. Difference-in-differences regression with mother fixed effects in columns (1)-(3) and without fixed effect in columns (4)-(5). Linear probability model controlling for regional trends.

Dependent variables: (1)-(2) Health expenditures per capita for children in mother's household (thousands of CFA francs), specification in level in column (1) and log transformation - adding 0.001 to deal with zeros in column (2). (3): Dummy for no health expenditures for any child in the mother's household. (4)-(5): Dummy equal to 1 if the mother did not seek any medical advice or medical treatment in case of diarrhea (column 4), and fever and/or cough (column 5).

Standard errors, in (), are clustered at the PSF or DHS cluster level.

* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.10: Difference-in-differences at the child level

	Full sample			Children younger than 5 in 2009			Children older than 5 in 2009		
	Level	Log	Zero spending	Level	Log	Extensive margin	Level	Log	Zero spending
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*
Post × Prevalence	6.120	3.663***	−0.428***	14.320	4.561***	−0.492***	2.090	3.222***	−0.396***
	(5.807)	(1.120)	(0.135)	(8.880)	(1.287)	(0.155)	(6.197)	(1.170)	(0.140)
Post	0.074	−0.664**	0.081***	−2.638	−1.053***	0.111***	1.399	−0.474*	0.067**
	(1.729)	(0.262)	(0.030)	(2.669)	(0.333)	(0.038)	(2.095)	(0.271)	(0.031)
Child FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
N	8146	8146	8146	2656	2656	2656	5490	5490	5490
Mean of dep. var. in 2006	4.24	−4.69	0.75	4.71	−4.31	0.70	4.02	−4.87	0.76

Data: PSF Panel. Sample of children living with their mother in the first wave.
Difference-in-differences regression with child fixed effects. Linear probability model.
Dependent variables: Columns (1)-(2), (4)-(5), (7)-(8): Individual health expenditures (thousands of CFA francs), specification in level in columns (1), (4) and (7), and log transformation - adding 0.001 to deal with zeros in columns (2), (5) and (8). Columns (3), (6) and (9): Dummy for no individual health expenditures recorded.
Standard errors, in (), are clustered at the PSF cluster level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.11: Robustness tests

	Controlling for sibship structure			Excluding migrants		
	Level	Log	Zero spending	Level	Log	Zero spending
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*
Post × Prevalence	5.651	2.758**	−0.300*	7.280	3.032**	−0.344*
	(5.585)	(1.384)	(0.173)	(6.396)	(1.405)	(0.176)
Post	1.647	0.255	−0.035	0.571	0.148	−0.019
	(1.849)	(0.373)	(0.043)	(1.863)	(0.347)	(0.041)
Mother FE	Yes	Yes	Yes	Yes	Yes	Yes
N	4550	4550	4550	2974	2974	2974
Mean of dep. var. in 2006	5.24	−3.67	0.61	4.18	−3.73	0.61

Data: PSF Panel. Sample in (4)-(6): Mothers residing in the same geographical cluster in both waves. The level of observation is a household, composed of a mother and her dependent children.
Difference-in-differences regression with mother fixed-effects. Linear probability model.
Dependent variable in (1)-(2) and (4)-(5): Health expenditures per capita for children in mother's household (thousands of CFA francs); specification in level in columns (1) and (4), and log transformation - adding 0.001 to deal with zeros in columns (2) and (5). Dependent variable in (3) and (6): Dummy for no health expenditures for any child in the mother's household.
Controls included in (1)-(3): average age of children, number of children and share of children under 5.
Standard errors, in (), are clustered at the PSF cluster level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.