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

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Abstract

This study exploits the introduction of high subsidies for anti-malaria products in Senegal in 2009 to investigate if malaria prevents parents to invest in child health. Building upon the theoretical literature on health investments under competing mortality risks, we argue that there are complementarities between disease-specific investments. We predict that private health investments to fight malaria as well as other diseases should increase in response to anti-malaria public interventions. To test this prediction, we use original panel data from a Senegalese household survey combined with geographical information on malaria prevalence. Our strategy is to compare the evolution of child health expenditures before and after anti-malaria interventions, between malarious and non-malarious regions of Senegal. We find that health expenditures in malarious regions catch up with non-malarious regions, principally at the extensive margin, and also in level and in composition. The same result holds for parental health-seeking behavior against other diseases like diarrhea. We provide evidence that 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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1 Introduction

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

At the beginning of the twenty-first century, there was a series of initiatives coordinated by the international community under the Roll-Back Malaria partnership to start the fight against malaria in Africa. Very large-scale interventions have been implemented to distribute anti-malaria products for free or at highly subsidized prices. On the preventive side, 900 millions of Insecticide-Treated Nets (henceforth ITNs) have been distributed since the early 2000s. Nowadays, an estimated 2/3 of children sleep under an ITN against 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 being treated when sick in 2015, but the coverage is increasing rapidly (World Health Organization and others, 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. Decreasing substantially 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 quantitative but also qualitative, parents spending more on preventative care. Heterogeneity analyses support the idea that anti-malaria campaigns caused the change in spending behavior: effects are stronger (i) in regions where interventions were more intense, and (ii) for younger children, who are most at risk. Finally, we exploit DHS waves conducted roughly at the same time (2005 and 2010) to show that health-seeking behavior against other

[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.

diseases like diarrhea increased more in malarious regions.

Our results could potentially by 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 and rainfall patterns, our estimates are even larger and more significant. Last, our results are qualitatively unchanged once we account for selective migration, attrition and changes in family structure. We further discuss the relative roles of the subsidy component and the information component of anti-malaria interventions.

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 influenced directly 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 might be prohibitive. More generally, our paper fits in the literature on private investments in human capital in developing countries. Jayachandran and Lleras-Muney (2009) show that reductions in maternal mortality risk lead to an increase in girls' educational attainment in Sri Lanka. Oster (2012) finds that reductions in risky sexual behavior in response to HIV epidemic in Africa are larger in areas with lower non-HIV mortality. In the same vein, we examine how health investments respond to changes in a specific mortality risk.

Our second 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 debate. On the pessimistic side, 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 optimistic side, 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 household's 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. To their surprise, and in line with our results, they find that households were *more* likely to buy a bed net 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 on 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", according to Sachs and Malaney (2002), "that the reduction in all-cause mortality with the use of bednets is considerably greater than the reduction in malaria-attributed mortality".

The paper is organized as follows. Section 2 introduces stylized facts on malaria control and infant mortality. Section 3 presents a simple model of investment in child health. Section 4 describes the data and Section 5 explains our empirical strategy. Section 6 provides the main empirical results. Section 7 discusses alternative stories and robustness checks. Section 8 concludes.

2 Malaria control and infant mortality in Sub-Saharan Africa

The impact of Roll-Back Malaria on child health has not been definitively quantified yet. Nonetheless, there is suggestive evidence of a success. Since the start of Roll-Back Malaria, 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 5 years old decreased from 700K per year down to 300K (World Health Organization and others, 2015). Using large household surveys collected in 19 African countries between 2000 and 2015, Cogneau and Rossi (2017) estimate the correlation between the distribution of bednets and the progress in child survival. They find that infant mortality did decrease more where more bednets were distributed, and that the association is stronger for more disadvantaged households. In terms of magnitude, the correlation is large and at the upper end of experimental results in the medical literature.

One explanation, which is not discussed by the authors and is the focus of this paper, is that medical RCTs fail to account for changes in households' health-seeking behavior induced by large-scale public health programs. This explanation is consistent with the stylized fact illustrated in Table 1. Using Demographic and Health Surveys conducted in 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 started to decline also for poor households in highly malarious regions; they are the only ones to 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. Our story 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 before 2002, making health expenses on other diseases worth it. An alternative explanation would be that malaria depresses adult health, 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 patterns in total expenditures that we document in Section 7.

3 Conceptual framework

3.1 Key theoretical insights

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 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.[2]

In Appendix 10.1, we propose a toy 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.

[2]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).

3.2 Application to our context

Before Roll-Back Malaria, 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 and others, 2015). If we exclude neonatal deaths, resulting primarily from prematurity, birth asphyxia and birth trauma, roughly one death in four was caused by malaria.[3] 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). Roll-Back Malaria radically changed the situation by reducing the cost of preventing and curing the disease.

What does it imply in a competing risk framework? Before the intervention, those households who could not afford fighting malaria also gave up on some other health investments. Once treatments become affordable, these other investments become worth it. So the dramatic decrease in the price of anti-malaria products is predicted to raise not only investments against malaria, but also investments against other diseases.[4]

Besides the change in prices, Roll-Back Malaria may also have changed beliefs about malaria mortality risk. 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. We discuss in section 7.3 the relative contribution of prices and information in explaining the changes we observe.

3.3 Testable predictions

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 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 about 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.

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

[4] 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.

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.

4.1 Panel data on household expenditures on child health

Our main dataset is the Poverty and Family Structure[5] (*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 groups or "cells" according to their budgetary arrangements. In particular, mothers and their dependent children[6] belong to the same cell, since the mother is usually the main caregiver and responsible for her children needs and well-being. The survey provides information on non-health expenditures made during the last 12 months at the cell level.

Importantly for our purpose, the survey registers information on private health expenditures paid during the last 12 months before the interview.[7] 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 in the mother's cell. 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 5-6 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

[5] 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.

[6] 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.

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

benefit many of them. That is why our preferred unit of analysis is the mother's cell; we discuss regressions at the child level in robustness tests presented in Section 7.2.1.

The first column of Table 2 shows some descriptive characteristics. Our sample of interest is made up by 1,594 mother's cells that we observe in both waves. On top of that, 788 cells are only observed in the second wave: these women had no dependent child in the first wave. In addition, 573 cells are only observed in the first wave: 368 women had no longer a dependent child in the second wave and 205 women could not be found. Implications of this attrition are discussed in Section 7.2.5.

The main advantage of PSF is to provide a panel so we can estimate regressions with mother fixed effects. The main disadvantage is to register only expenses and not health-seeking behavior broken down by disease.[8]

4.2 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 under age 5 suffering from diarrhea, fever and cough. Parents are asked if they sought treatment when the child was sick.

DHS also collect 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 of DHS is to be a repeated cross-section, so changes over time may capture both changes in behavior and changes in population.

4.3 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.[9] Both PSF and DHS contain GPS information,

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

[9] 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. Table A.2 in the Appendix).

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 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-difference strategy identifies responses to anti-malaria campaigns.

5.1 Temporal variation

In 2008, the Programme National de Lutte contre le Paludisme (PNLP; National Malaria Control Program) initiated a 4-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. Figure 1 shows that funds allocated to fight the disease jumped in 2009 and have remained high until today. Before, in the period 2002-2008, only very targeted and local distributions of bed nets and other malaria-related goods and services took place (President's Malaria Initiative, 2008).

The first nation-wide ITNs distribution campaign started in 2009 and targeted specifically children under 5 and pregnant women. More than 6 millions ITNs were distributed between 2008 and 2010 throughout the country, and no specific areas were singled out (Plan National de Lutte Contre le Paludisme au Sénégal, 2015). For pregnant women and mothers of under-5 children, ITNs could be obtained either for free or at a very subsidized price: maximum of 1 euro, instead of 10-12 euros at the 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 on the importance of using ITNs.[10] 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.

[10] Cf. Figure A.2 in the Appendix for examples of promotional material used in the 2009 campaign.

5.2 Spatial variation

Before anti-malaria campaigns started, there was considerable variation 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 the average, in dark blue on the map) and areas with a high prevalence (above the 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).

Figure 3 shows a map of Senegal with the clusters surveyed in PSF. Triangles are located in high prevalence areas and circles in low prevalence ones. Columns 2 and 3 of Table 2 provide some descriptive statistics, by initial malaria prevalence. Household structure in the first wave is the same everywhere: there are 9 people, the mother is around 34 years old and the average age of children in the cell is 7. But rural and poorer areas tend to be more affected by the disease. In Section 7, we discuss how these differences might threaten our identification strategy and why we believe that the scope for this concern is limited in our context.

5.3 Econometric specification

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

$$Y_{i,t} = \alpha_0 + \alpha_1 \, High \; Prevalence_i \; + \alpha_2 \, Post_t + \alpha_3 \, Post_t \times High \; Prevalence_i + u_i + \epsilon_{i,t} \quad (5.1)$$

$Y_{i,t}$ is the outcome of interest: the annual level of child health expenditures per capita in the mother's cell (prediction 1), a dummy variable equal to one if the cell has no health expenditure (prediction 2), a dummy indicating if a child is sick and left untreated (prediction 3). $High \; Prevalence_i$ indicates whether the household is located in an area initially exposed to high malaria prevalence. $Post_t$ equals one if the survey took place after 2009. Standard errors are clustered at the mother level because this is the unit of observation in our panel.[11]

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 robustness tests, we introduce time-varying controls to account for potential confounders (cf. Section 7). We also use a continuous measure of prevalence instead of a dichotomous variable (cf. Table A.2 in the Appendix) and find comparable results.

[11] Alternatively, it can be argued that standard errors should be clustered at the district level because malaria prevalence is measured at this level. Our results remain the same with such a specification (Table A.1 in the Appendix).

5.4 Identification assumptions

The ideal experiment would be to allocate randomly free anti-malaria treatments in endemic areas and to examine the impact on households' health-seeking behavior, for instance adoption of water chlorine. This experiment is run in Dupas (2014) with another objective: check if subsidizing ITN decreases the willingness to pay for another health product. 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.

In our setting, anti-malaria campaigns targeted the whole country, no area was excluded. We cannot use areas without a campaign 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. Our assumption is that, in the absence of *malaria*, the evolution of health expenses would have been the same for all households. 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.

We provide support for our identification assumption in three ways that we discuss in detail in Section 7. 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 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 differently low and high prevalence areas 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 rural-urban dynamics. We show that they are unlikely to explain our results.

6 Results

6.1 [P1] : Child health expenditures increase more in areas with high initial malaria prevalence

Figure 4 shows descriptive statistics on health expenditures in the two waves of PSF, comparing areas with 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 multiplied by 3 their consumption of health

commodities, up to 5,215, while there was no significant change in low prevalence areas.[12]

In Table 3 column 1, we include mother fixed effects, estimating equation 5.1 with health expenditures as an outcome. 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 (the p-value is 0.13) but the magnitude remains large: 2,600 FCFA, representing a 2.5 times increase from baseline expenditures.

To put these numbers in perspective, Lepine and Nestour (2012) report that, in rural Senegal in 2009, health facilities charged on average 200 and 100 FCFA 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 FCFA (President's Malaria Initiative (2008)). This is consistent with our claim that, in the first wave, households could not afford preventing malaria.

Next, we consider changes in the composition of health expenditures, as illustrated by Figure 5. 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 towards the composition observed in low prevalence areas: less spending on medication and hospitalization, more on consultation. This suggests that parents reallocated part of their expenses from curing episodes of malaria towards medical examination. Another way to distinguish between preventative and curative treatments is to look at sick and non-sick children separately (cf. Figure A.3 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 FCFA in the second wave, while remaining stable in low prevalence areas. The difference-in-difference is significant at 10%. All in all, we find evidence of a catch-up in health expenditures which is not only quantitative but also qualitative.

6.2 [P2] : The proportion of households with no health expenditures decreases more in areas with high initial malaria prevalence

Figure 6 closely mirrors the previous figure. Households in high prevalence areas were 15.7 p.p more likely to make no expenses in health in 2006 than the others. In 2011, they have almost caught up. Between the two PSF waves, the proportion of cells with zero expenditure decreased

[12] Estimation of the differences-in-differences regression without fixed effects can be found in column 1 of Table A.3 in the Appendix. The coefficient on the interaction term is significant at 5%.

by 17.7 p.p whereas a more moderate downturn of 4.7 p.p happened in low prevalence areas.[13]

Estimates of the regression with fixed effects are reported in Table 3, column 2. The coefficient on the interaction term remains of similar magnitude and significance. Around 12% of households started investing in child health in response to anti-malaria campaigns. This amounts to one third of the proportion reporting positive spending in the first wave.

6.3 [P3] : Health-seeking behavior against other diseases increases more in areas with high initial malaria prevalence

Table 4 presents the estimates of equation 5.1 using different specifications to disentangle between preventive and curative behaviors. In columns (1) to (3), we look at the probability of being sick and left untreated among all children under age 5. The first column pools together all diseases. Separated results for diarrhea and fever/cough[14] are in columns 2 and 3 respectively. Between the two waves, the likelihood of being sick and untreated decreased everywhere, and the decline was stronger (by 2-3p.p.) in high prevalence areas.

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 (4) to (7), we look at the probability of being sick. The likelihood of fever, a major symptom of malaria, decreased much more in malarious areas (by 4p.p.), supporting the idea that ITNs distributions were effective in preventing malarious episodes.[15] Trends for diarrhea and cough are not statistically different between areas.

In columns (8) and (9), we restrict the sample to sick children and look at the probability of being left untreated. In the case of diarrhea, parents were 6.7p.p. less likely to seek medical advice or treatment in high prevalence areas in 2005. They entirely caught up between the two waves, suggesting that parents started acting upon diarrhea once they have been relieved from malaria. In the case of fever and cough, the coefficient on the interaction term is close to zero. This may be explained by a strong downward 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 cough only.

We can assess the external validity of our cross-disease result on diarrhea using DHS in other

[13] Estimation of the differences-in-differences regression without fixed effects can be found in column 2 of Table A.3 in the Appendix. The coefficient on the interaction term is significant at 1%.

[14] In the data, we can distinguish between fever and cough for the probability of being sick, but not for the probability of being treated.

[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 low malaria prevalence areas, where people are on average better off.

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.4 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 Kenya and Rwanda than in Senegal.

Overall, we find that, after anti-malaria campaigns, private health investments, in total and against other diseases, have increased more in highly malarious areas than in low prevalence areas. This is consistent with our framework of investments under competing risks.

7 Discussion

7.1 Alternative stories

In this section, we discuss five alternative stories 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 stories do not confound our results, and in fact make them stronger.

7.1.1 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 7 plots the proportion of children sick and untreated in case of diarrhea. There are some fluctuations, 1997 being an especially good year.[16] But these fluctuations are similar everywhere; in particular, the catch-up observed between 2005 and 2010 had not already started in the late 90s.

7.1.2 Total income

The second alternative story argues that total income grew faster in highly malarious areas. It might be for reasons unrelated to malaria control - these regions are different to start with - or specifically because of malaria control, but not through the competing risks channel. Health improvements might have 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 if there is a differential rise in *all* expenditures. We measure total consumption at the cell level, including all individuals, not only the children. Descriptive

[16] We investigated if 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 health data finds no evidence of seasonality of diarrhea in the 1997 Senegalese survey (Pullum, 2008).

statistics in Table 2 indicate that households in highly malarious areas were poorer than the others to start with: 187 vs. 389 KFCFA. 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 have not become richer; they have 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.[17] 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, and slightly more in high prevalence areas. The coefficient on the interaction term is 1,700 FCFA, not significantly different from zero (table available upon request). This is consistent with the competing risk channel and not with the adult health channel.

7.1.3 Access to healthcare

The third alternative story is about access to healthcare. It could have improved more in highly malarious areas, making it easier for parents to spend money on child health. 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. Instead, we focus on key elements that might have changed during our period of interest: health infrastructure and two large-scale child health campaigns, against measles and diarrhea.

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. Results are reported in Column 2 in Table 5. In 2005, access was a greater problem in highly malarious areas, and again, there was no catch-up between the two waves. If anything, access seems to have improved less in high prevalence areas.

Second, two other campaigns were implemented in Senegal between 2005 and 2010. As part of the Millennium Development Goals, a lot of effort was devoted to increase measles immunization coverage in Africa (United Nations, 2015). This would be a concern for us if progress were different in high and low malaria prevalence areas, because we would not know if changes were caused by campaigns against malaria or against measles. But this is not the case. Between 2005 and 2010, in DHS data, the proportion of children under 5 vaccinated against measles rose from 63% to 73% in low prevalence areas, and from 64% to 72% in high prevalence areas. Differences between the two groups are never statistically significant.

The other concomitant campaign aimed at improving access to treatments against diarrhea. In 2010, approximately 6 million zinc dispersible tablets were delivered to Senegal by the UNICEF,

[17] Child health expenditures account for 3% of the cell total consumption.

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. In fact, the opposite happened. The intervention was piloted first in the region of Thies, which received over one fourth of all tablets, and virtually all clusters in this region are classified as low prevalence. 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, as shown in Columns 1, 2 and 3 in Table 6. As expected, coefficients on the interaction terms increase in size and significance.

All in all, accounting for changes in access to healthcare tends to strengthen our results.

7.1.4 Rainfall

The fourth alternative story discusses 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 the year 2011 was particularly rainy, people would spend more on curative treatments in the second wave.

We rule out this story using a geo-coded measure of yearly rainfall provided by the Climate Hazards Group[18] that we were able to merge with the PSF panel, except for one cluster. This allows us to compute positive (flood) and negative (drought) rainfall shocks for each PSF cluster and for both waves.[19] 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 tends to underestimate the causal impact of anti-malaria subsidies on health expenditures. When we control for positive and negative shocks, our coefficients of interest remain comparable in magnitude and are more precisely estimated (cf. Columns 4 and 5 in Table 6).

7.1.5 Geographical dynamics

The last alternative story is that our diff-in-diff estimates capture different dynamics between geographical areas. In particular, one concern is that malaria is more prevalent in rural areas as shown by Table 2. It may be the case that rural areas have 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.

[18] 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

[19] We define as positive (resp. negative) rainfall shocks the observations whose annual rainfall measure is one standard deviation above (resp. below) the district historical mean of annual rainfall calculated over the 1981-2015 period. Alternatively, we can use a continuous measure of rainfall deviation from the historical mean instead of dummies for shocks. This leads to the same conclusion (Table available upon request).

To rule out this concern, we first remove the capital city Dakar from our sample. Columns 1 and 2 in Table A.5 in the Appendix show that low and high prevalence areas are now a bit more similar to start with, and we again observe a significant catch-up. Second, we look at urban and rural areas separately. Columns 3 to 6 show that our findings hold for each sub-sample. More generally, in columns 7 and 8, we account for potentially diverging regional trends by interacting *Post* with dummies for each administrative region of Senegal (see borders in Figure 3). By comparing high and low prevalence clusters in the same region, we find similar results. Therefore, our conclusions are not driven by different geographical dynamics.

7.2 Robustness tests

7.2.1 Sibship structure

In the results presented so far, the unit of observation is the mother's cell. Per capita health expenditures are likely to depend on the cell structure, like the number of surviving children and their age. Mortality is potentially a concern but the number of cells experiencing a child death between the two waves is too small (below 2%) to drive our results. Table 2 indicates that regions with high and low prevalence have similar family structure in PSF1 (same size, same average age of children). But this is no longer the case in PSF2: households in malarious regions are relatively larger and children relatively younger. One may therefore worry that our coefficient of interest captures a differential change in family 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.

We address this concern in two ways. First, we introduce some controls related to the structure of the mother's cell: average age of children, number of children and share of children under 5. Our coefficients remain very stable in magnitude and significance, as shown by columns 1 and 2 in Table A.6 in the Appendix.

Second, we change the unit of analysis from the mother's cell level to the child level. We include all children who were born and living with the mother in PSF1. We follow them in PSF2, whatever the residence status, and examine the evolution of their health expenditures. As shown by Figure A.4 in the Appendix, the same pattern holds: individual expenses increase much more in high prevalence areas. When we include a child fixed effect, the difference-in-difference coefficient is significant at the 5% level and the magnitude remains stable, around 2,500 FCFA (cf. Column 1 in Table A.7 in the Appendix).

7.2.2 Heterogeneity by age

We use the specification at the child level to look at heterogeneity by age. Episodes of malaria do greater harm to younger children, in particular children under age 5. Anti-malaria campaigns are therefore expected to make a bigger difference for them.

In Table A.7 in the Appendix, we split the sample of children depending on their age at the time of the interventions. We find that our results are mostly driven by children younger than 5 in 2009. For them, the coefficient on the interaction term is close to 5,000 FCFA, twice as large as for the whole sample. We find the same result if we use the specification at the cell level and include only children under age 5. This is consistent with our model: parental health-seeking behavior is predicted to change more for children who were most at risk of dying from malaria.

7.2.3 Heterogeneity by intensity of anti-malaria campaigns

We further look at heterogeneity by variation in ITN use. Within malarious areas, the increase in health expenditures should be driven by areas with the largest increase in ITN use. Using data on ITN coverage provided by the Malaria Atlas, we construct a variable indicating whether the variation in ITN use between 2006 and 2011 in the district of observation was higher than the national average (+20pp).

Results are shown in Table A.8 in the Appendix. Private health expenses increased significantly everywhere, but much more in areas where the take-up was stronger. This is another piece of evidence that the change in private expenses was driven by Roll-Back Malaria.

7.2.4 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 even more salient, as shown in columns 3 and 4 in Table A.6 in the Appendix.

7.2.5 Selective attrition

Another issue would arise if the attrition observed in the PSF panel were selected differentially between malarious and non-malarious areas. Table A.9 in the Appendix provides some baseline characteristics for women who were not found in the second wave. A first observation is the limited scope: only 5% of mothers in malarious areas and 12% in non-malarious areas.

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 (comparing columns 3 and 4). 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 (comparing columns 1 and 2). If anything, they seem to be in a situation where switching to positive health spending is likely. All in all, our coefficient of interest is more likely to be underestimated rather than overestimated by attrition.

7.3 Price or information?

If we interpret our difference-in-difference estimates as a causal impact of malaria control efforts, one question remains: which component of the campaign changed behaviors? Our preferred explanation is the strong decrease in price. Another possibility is information.[20] 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 not influenced at all by the framing of marketing messages. Which role plays information 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 was built around ITNs (see pictures in Figure A.2 in the Appendix). 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 to pregnant women or mothers of children under 5, while a larger share of the population benefited from subsidies. In Table 7, we split the sample between pregnant women or mothers with children under 5 in 2009 and the others. Results are the same in both groups, meaning that women who received more information did not respond more to the intervention. It can be either because information does not matter or because it was spread by word of mouth.

Last, we provide support for the price channel by showing that households who 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 cannot afford any health expense. In Table 8, we show total expenditures measured in the first wave, by type of transitions. "Never Invest" are cells making no health expenses in both waves, "Switchers" are cells making no health expenses in PSF1 and some expenses in PSF2, and finally "Always Invest" are cells with some expenses in both waves. "Switchers" are

[20] 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).

poorer than "Always Invest" and wealthier than "Never Invest". Another way to investigate heterogenous responses by income level is estimating an order 2 polynomial of income. We interact $Post$ and $Post \times High\ Prevalence$ with $Income$ and $Income^2$ in the specification with health expenditures per capita as the dependent variable. Coefficients on the triple interaction terms are both significant at 10% and of expected signs: the response increases up to some income levels and then decreases. For clarity of exposition, Figure 8 represents estimates of the difference-in-difference as a function of total expenditures over the support observed in PSF1.

Overall, we do not find conclusive evidence that information played a major role.

8 Conclusion

This paper investigates how private health investments have responded to subsidies for anti-malaria products introduced in Senegal in the late 2000s. We combine panel data from 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 to invest in child health and heavy subsidies proved to be necessary to alleviate this constraint.

An interesting lead for further research would be to examine if changes in spending behavior go hand in hand with changes in perceptions of health agency. Now that 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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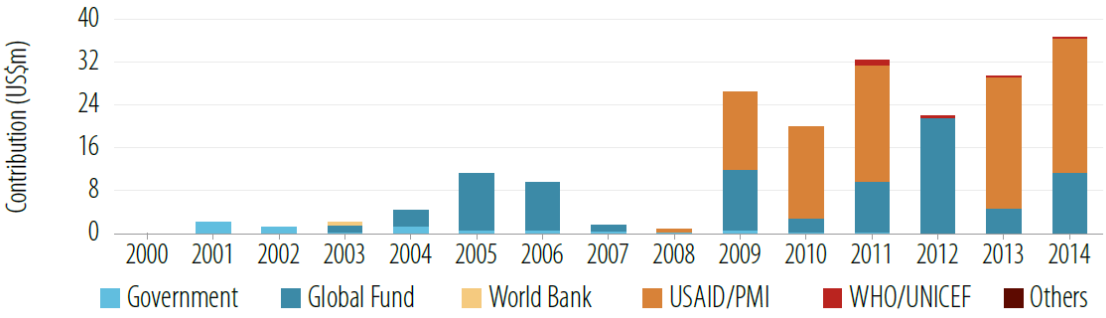
9 Figures and Tables

9.1 Figures

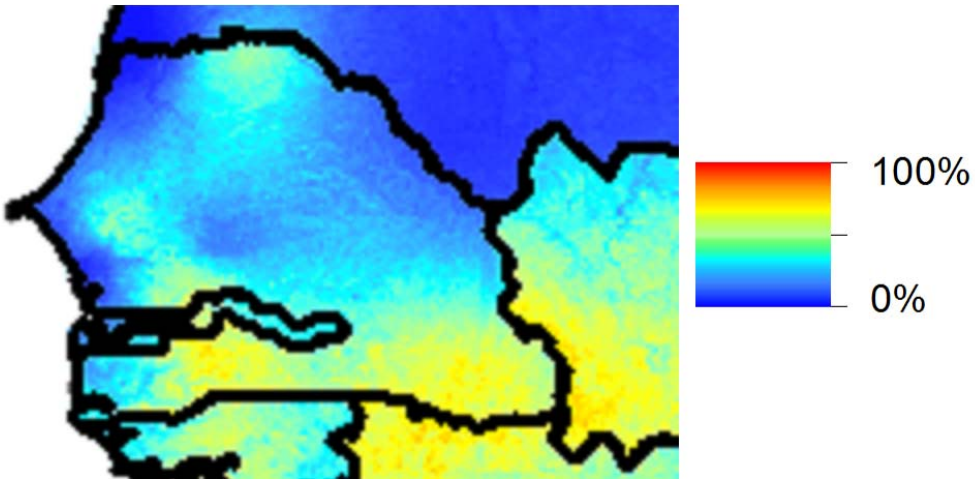
Figure 1: Temporal variation: funds allocated to anti-malaria interventions



Source: World Health Organization and others (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.

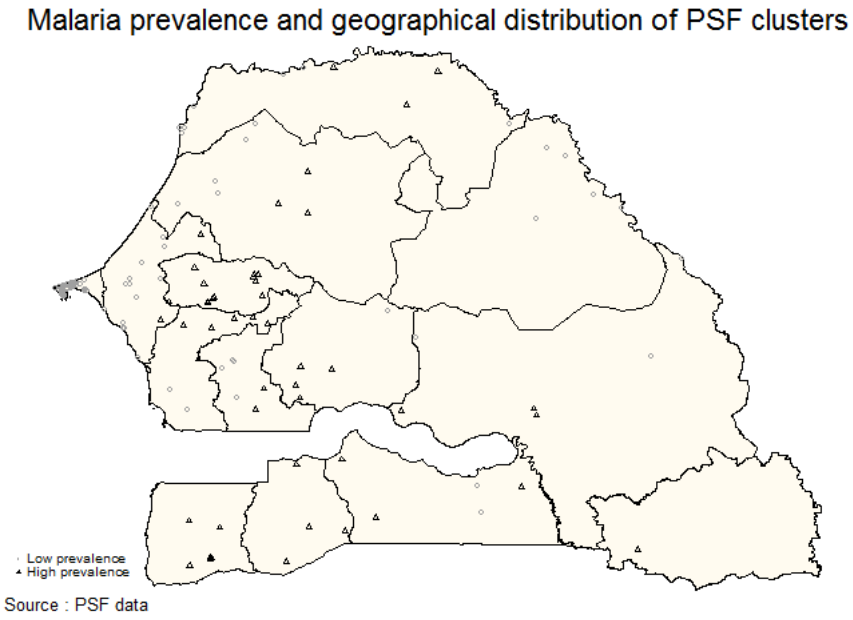
Figure 2: Spatial variation: initial malaria prevalence



Source: Malaria Atlas.

The map shows the proportion of children between age 2 and 10 infected by the parasite 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 the average, in dark blue) and areas with a high prevalence (above the average, in light blue and yellow).

Figure 3: Spatial variation in our sample



We define as high (resp. low) malaria prevalence clusters, clusters whose malaria prevalence in 2000 was above (resp. below) the national average (24%).

Figure 4: Prediction 1: changes in per capita health expenditures

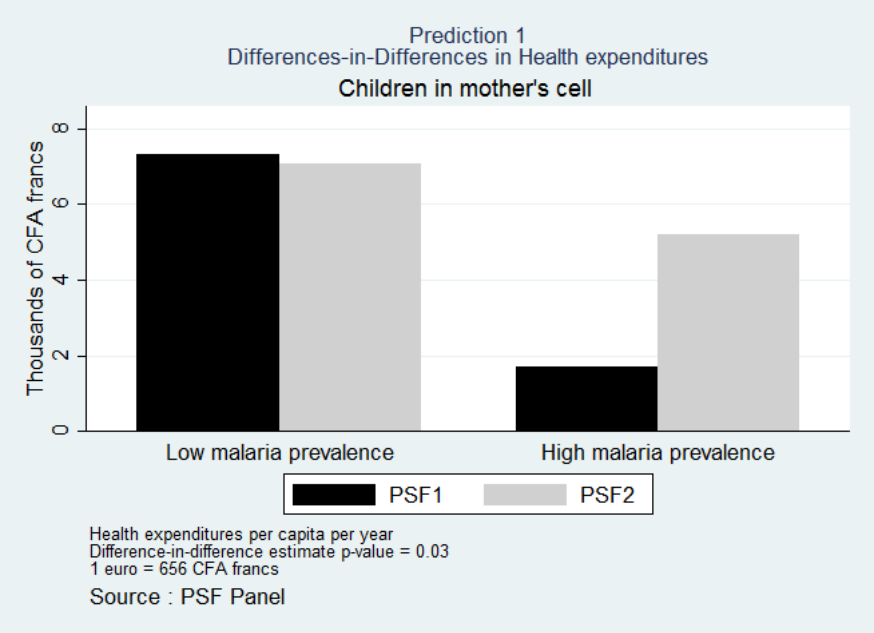
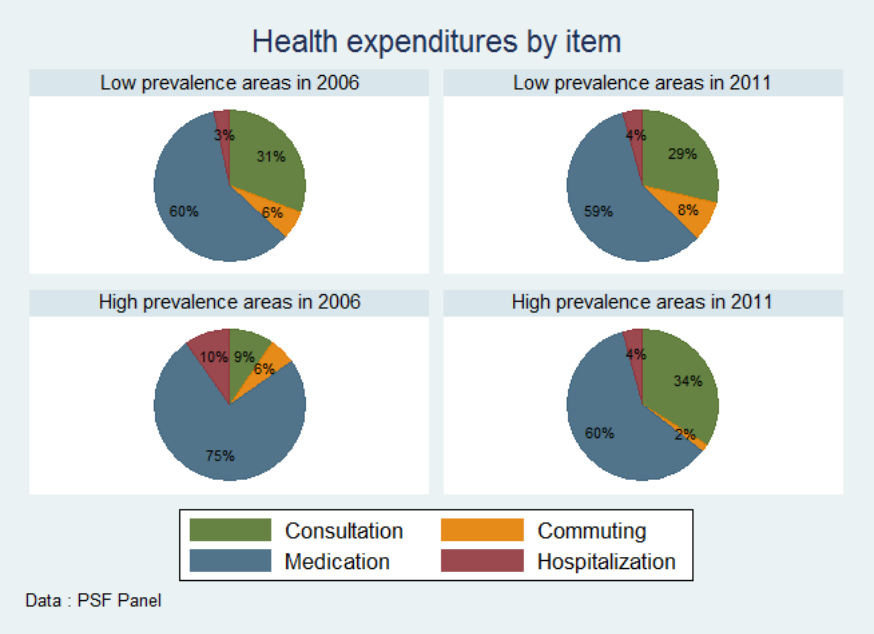


Figure 5: Breakdown of health expenditures by item



The graphs show the average allocation of health expenditures per capita for children in mother's cell by item.

Figure 6: Prediction 2: changes at the extensive margin

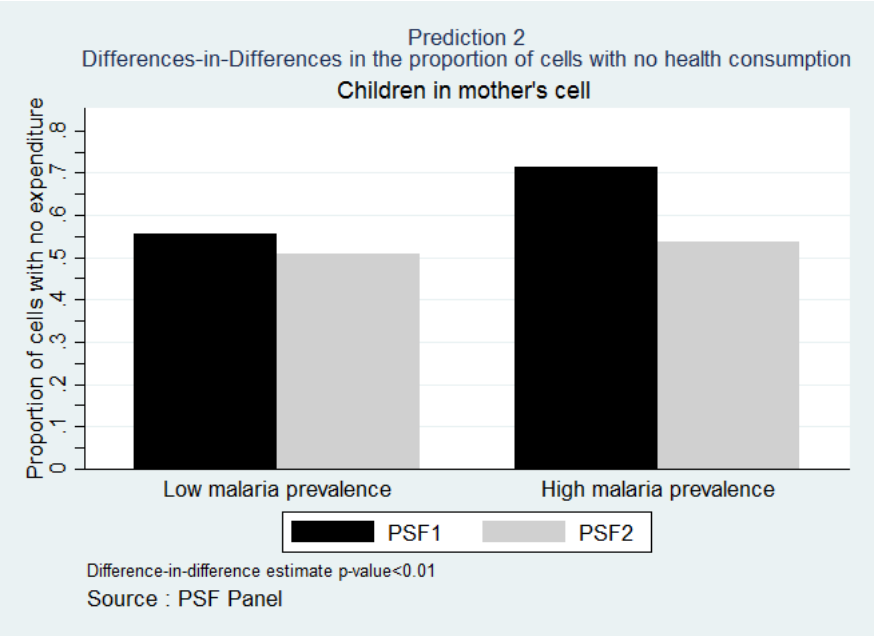
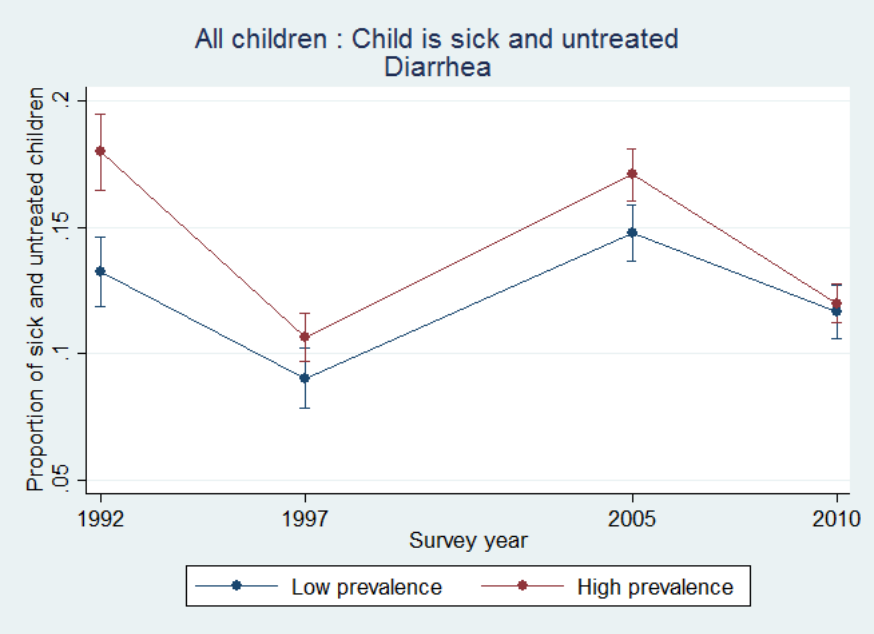
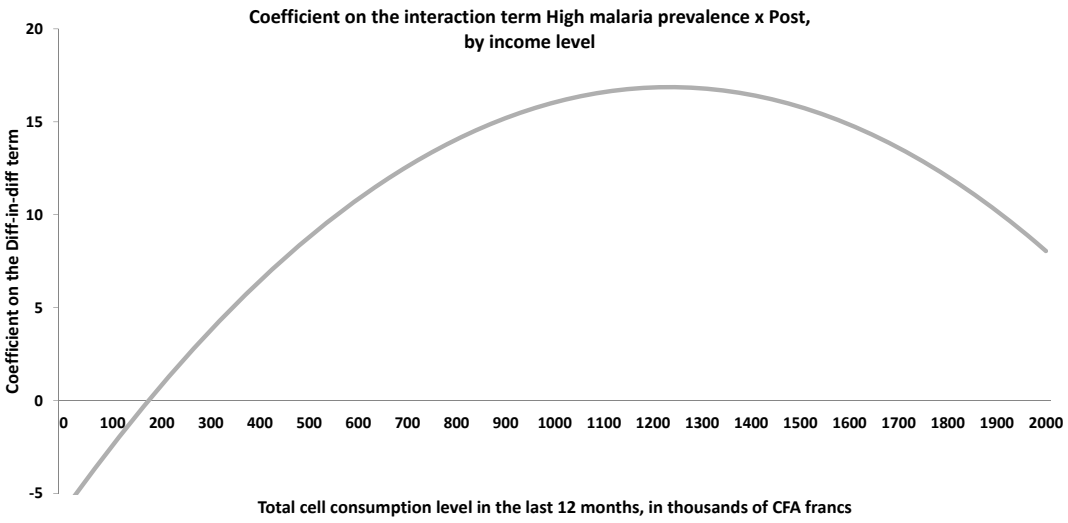


Figure 7: Pre-trends in health-seeking behavior



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 case of diarrhea. Bars represent the 95% confidence intervals.

Figure 8: Heterogeneity by income



The figure shows the estimated difference-in-difference by income level. Specifically, the graph plots the following equation: $y = -5,951 + 0,037x - 0,000015x^2$ where x ranges from the minimum to the maximum values of total annual consumption levels observed in PSF1 (excluding top 1% outliers). The coefficients are obtained by interacting $Post$ and $Post \times High\ Prevalence$ with $Income$ and $Income^2$ in Equation 5.1.
The coefficient on $Post \times High\ Prevalence$ is $-5,951$, not significantly different from 0.
The coefficient on $Post \times High\ Prevalence \times Income$ is $0,037$, significant at 10%.
The coefficient on $Post \times High\ Prevalence \times Income^2$ is $-0,000015$, significant at 10%.

9.2 Tables

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	High prevalence	Low prevalence	pval(diff)
Baseline characteristics in 2006/2007 (PSF1)				
Plasmodium falciparium parasite rate (%) in 2000	0.17	0.36	0.09	0.00
Hh in Dakar region	0.31	0.00	0.49	0.00
Hh in other urban area	0.21	0.21	0.21	0.90
Hh in rural area	0.47	0.79	0.30	0.00
Mother's age	33.75	33.31	34.02	0.16
Cell total consumption (thousands of CFA francs)	313.67	187.33	388.94	0.00
Panel characteristics				
Hh size in PSF1	9.29	9.43	9.20	0.43
Hh size in PSF2	10.47	10.95	10.18	0.03
Average age of children in cell in PSF1	7.45	7.42	7.47	0.80
Average age of children in cell 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 hh in PSF1	1412	503	909	
# of hh in PSF2	1730	655	1075	
# of cells in PSF1	2167	809	1358	
# of cells in PSF2	2382	907	1475	
# of cells in both waves	1594	616	978	

Data : PSF Panel .
(*) : computed only for women found in both waves
The last column shows the p-value of the difference between high and low prevalence areas.

1 euro ≈ 656 CFA francs.

Table 3: Prediction 1 and 2
Differences in Differences in Health expenditures

	Per capita levels *(1)*	*Zero spending* *(2)*
Post	0.842	-0.041^{*}
	(1.353)	(0.021)
High prevalence × Post	2.603	-0.115^{***}
	(1.712)	(0.034)
Mother FE	*Yes*	*Yes*
N	4550	4550
Mean of dep. var. in low prevalence areas in 2006	7.33	0.56
Mean of dep. var. in high prevalence areas in 2006	1.72	0.71

Data : PSF Panel.
Differences-in-differences regression with mother fixed effects. Linear probability model.
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Dummy for no health expenditures for any child in the mother's cell.
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table 4: Prediction 3
Differences in differences in health-seeking behavior

Sample	All children			All children				Sick children	
Dependent variable	*Child is sick and untreated*			*Child is sick*				*Child is untreated*	
	All diseases	*Diarrhea*	*Fever and Cough*	*Diarrhea*	*Fever*	*Cough*	*Fever and Cough*	*Diarrhea*	*Fever and Cough*
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*
High prevalence	0.013	0.023***	0.004	0.011	−0.012	−0.044***	−0.034***	0.067***	0.065***
	(0.010)	(0.008)	(0.009)	(0.009)	(0.010)	(0.010)	(0.010)	(0.020)	(0.018)
Post	−0.052***	−0.031***	−0.034***	−0.017*	−0.062***	−0.054***	−0.068***	−0.117***	0.008
	(0.011)	(0.008)	(0.010)	(0.009)	(0.010)	(0.010)	(0.011)	(0.024)	(0.020)
High prevalence × Post	−0.032**	−0.020**	−0.025**	−0.008	−0.041***	−0.014	−0.031**	−0.061**	0.002
	(0.014)	(0.010)	(0.012)	(0.012)	(0.013)	(0.013)	(0.014)	(0.030)	(0.026)
Constant	0.302***	0.148***	0.218***	0.204***	0.296***	0.272***	0.384***	0.741***	0.574***
	(0.008)	(0.006)	(0.007)	(0.006)	(0.007)	(0.007)	(0.008)	(0.016)	(0.013)
Mother FE	*No*	*No*	*No*	*No*	*No*	*No*	*No*	*No*	*No*
N	21251	21251	21251	21218	21225	21226	21234	4188	6672

Data : DHS 2005 and DHS 2010. Samples: All children under age 5 in colums (1)-(3) and (8)-(9). Sick children under age 5 in columns (4)-(7).
Differences-in-differences regression without mother fixed effects. Linear probability model.
Dep. var in :
Columns (1)-(3) and (8)-(9): Dummy equal to 1 if the mother did not sought any medical advice or medical treatment in case of any disease (column 1), diarrhea (columns 2 and 8), and fever and/or cough (columns 3 and 9).
Columns (4) to (7): Dummy equal to 1 if the child suffered from diarrhea (column 4), fever (column 5), cough (column 6), and fever and/or cough (column 7) in the last two weeks.
Standard errors, in (), are clustered at the mother level.

Table 5: Tests for alternative stories: examining trends in other determinants of health expenditures

	Total expenditures	*Distance to health facilities is a concern*
	(1)	(2)
High prevalence		0.106***
		(0.010)
Post	9.675	−0.018*
	(13.507)	(0.011)
High prevalence × Post	−5.259	0.020
	(16.035)	(0.014)
Constant		0.336***
		(0.008)
Mother FE	*Yes*	*No*
N	4550	19098
Mean of dep. var. in low prevalence areas in 2006	388.9	0.34
Mean of dep. var. in high prevalence areas in 2006	187.3	0.44

Data : (1) : PSF Panel. (2) : Mothers of children under 18 in DHS 2005 and DHS 2010.
Differences-in-differences regression. Linear Probability Model.
Dependent variables : (1): Total expenditures for the whole cell in the last 12 months in thousands of CFA francs. (2): Dummy equal to 1 if distance to the nearest facility is a major problem when the respondent is sick and want to get medical advice or treatment.
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table 6: Tests for alternative stories: controlling for potential confounders

	Campaigns against diarrhea			*Rainfall shocks*	
	All diseases	*Diarrhea*	*Fever and Cough*	*Per capita levels*	*Zero spending*
	(1)	(2)	(3)	(4)	(5)
High prevalence	0.013	0.023***	0.004		
	(0.010)	(0.008)	(0.009)		
Post	−0.055***	−0.035***	−0.033***	0.873	−0.034
	(0.011)	(0.008)	(0.010)	(1.367)	(0.021)
High prevalence × Post	−0.038***	−0.029***	−0.023*	3.094	−0.096***
	(0.015)	(0.011)	(0.013)	(1.904)	(0.037)
Constant	0.302***	0.148***	0.218***		
	(0.008)	(0.006)	(0.007)		
Mother FE	*No*	*No*	*No*	*Yes*	*Yes*
N	21251	21251	21251	4548	4548
Mean of dep. var. in low prevalence areas in 2006	0.30	0.15	0.22	7.33	0.56
Mean of dep. var. in high prevalence areas in 2006	0.31	0.17	0.22	1.72	0.71

Data (1), (2) & (3): DHS 2005 and DHS 2010. Sample of children under age 5. (4) & (5) : PSF Panel.
Differences-in-differences regression. Linear Probability Model.
Dependent variables : (1), (2) & (3) : Dummy equal to 1 if the mother did not sought any medical advice or medical treatment in case of any disease (column 1), diarrhea (columns 2), and fever and/or cough (columns 3). (4): Health expenditures per capita for children in mother's cell (thousands of CFA francs). (7) : Dummy for no health expenditures for any child in the mother's cell.
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table 7: What is the role of information?

	Mothers targeted specifically by information campaigns		Other mothers	
	Per capita levels	Zero spending	Per capita levels	Zero spending
	(1)	*(2)*	*(3)*	*(4)*
Post	1.083	-0.092^{***}	0.384	0.056
	(1.148)	(0.025)	(3.260)	(0.037)
High prevalence × Post	2.406	-0.111^{***}	2.960	-0.104^{*}
	(1.771)	(0.041)	(3.607)	(0.063)
Mother FE	*Yes*	*Yes*	*Yes*	*Yes*
N	2749	2749	1228	1228
Mean of dep. var. in low prevalence areas in 2006	5.99	0.54	6.42	0.59
Mean of dep. var. in high prevalence areas in 2006	1.74	0.69	0.84	0.67

Data : PSF Panel. Sample restricted to mothers residing in the same geographical district in both waves.
Columns (1) & (2): Sample of mothers of children under 5 or pregnant women, at time of the 2009 campaign. These mothers were targeted specifically by information campaigns. Columns (3) & (4) : Sample of other mothers.
Differences-in-differences regression with mother fixed effects. Linear probability model.
Dep var : (1) & (3) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) & (4) : Dummy for no health expenditures for any child in the mother's cell.
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table 8: Who started to invest between the two waves?

	Never Invest (1)	Switchers (2)	Always Invest (3)
Consumption level in PSF1	256.34	274.66	346.82
s.e	12.65	17.43	22.53
Observations	540	425	340

Data: PSF Panel. Table constructed on the balanced sample of mothers.
Mean of total cell consumption level in the last 12 months, in thousands of CFA Francs.
"Never invest" (resp. "Always Invest") are cells with zero spending (resp. some spending) on child health in both waves. "Switchers" are cells with zero spending in the first wave and some spending in the second wave: they started to invest in child health between the two waves.
P-values of the difference in means : (1)-(2) : p-value = 0.38 ; (2)-(3) : p-value =0.01 ; (1)-(3) : p-value < 0.01.

10 Appendix

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

10.1.1 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 who 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	$s_m.v - p_o.\theta$	$v - (p_m + p_o).\theta$

10.1.2 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.

On 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 $\theta_l < s_l.\theta_h$.[21] 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.

[21] 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.

10.1.3 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.1). 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, a 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.

10.1.4 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 perceived survivals 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 raise the number of switchers.

10.2 Figures

Figure A.1: Health investments (x_m^*, x_0^*) before and after subsidizing anti-malaria products

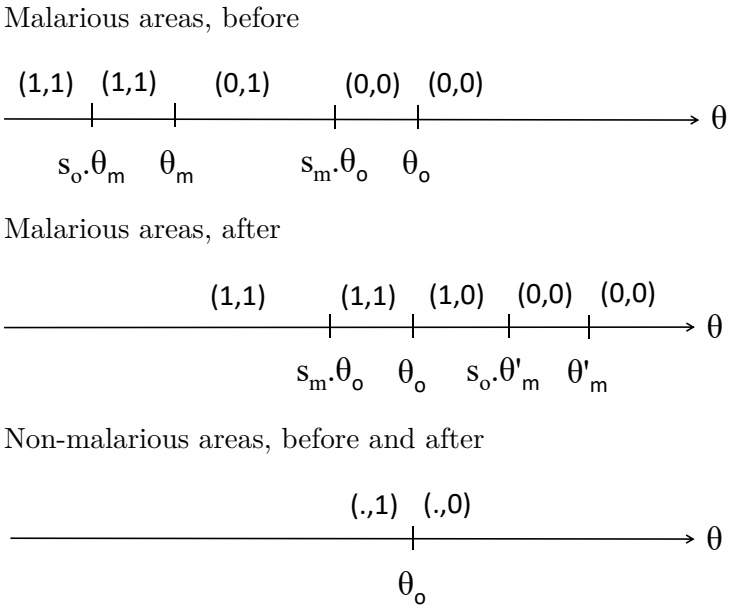


Figure A.2: Examples of promotional material used during the 2009 campaign in Senegal

The Roll-Back Malaria campaign in Senegal was called Xeex Sibbiru ("Let's fight malaria" in Wolof) and promoted the use of ITNs via various channels: posters, certificates given to families who picked up an ITN, singing competitions etc. The main message stressed the importance to sleep under an ITN every night.

Figure A.3: Health expenditures conditional on sickness

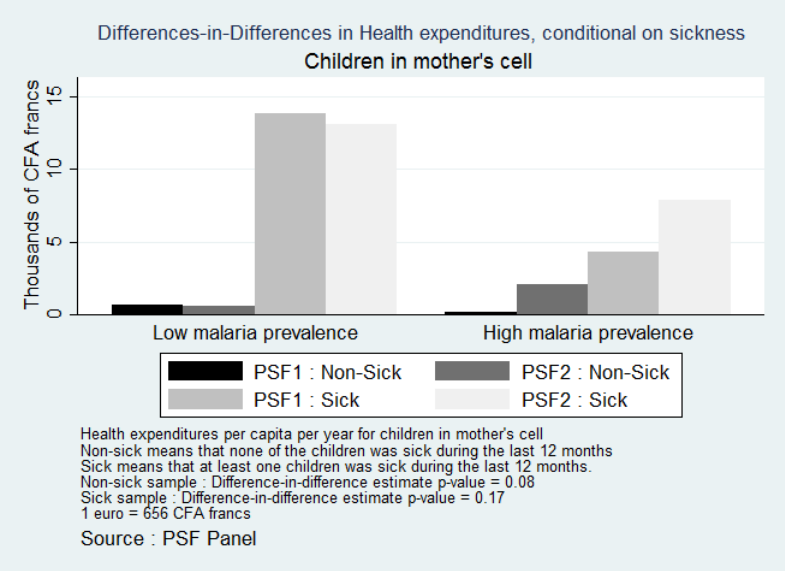
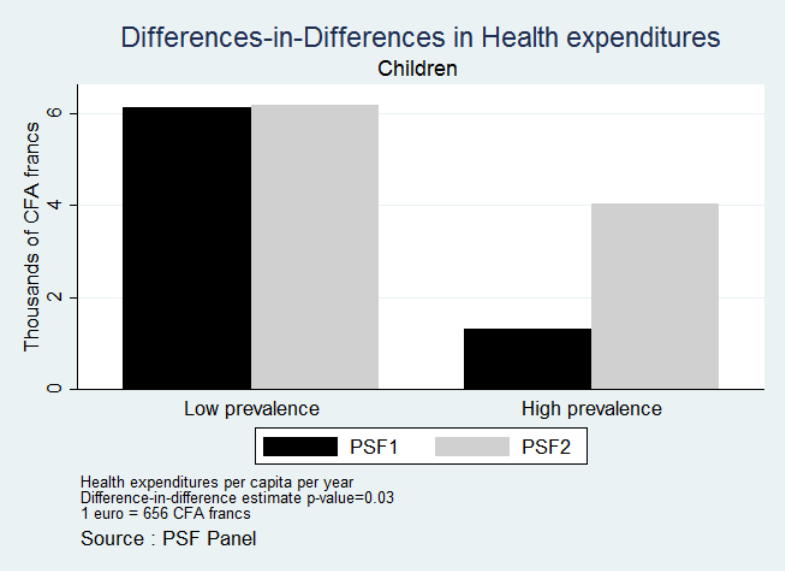


Figure A.4: Child-level analysis: changes in individual health expenditures



10.3 Tables

Table A.1: Prediction 1 and 2
Clustering at the district level

	Per capita levels *(1)*	*Zero spending* *(2)*
Post	0.842	−0.041
	(1.465)	(0.033)
High prevalence × Post	2.603	−0.115**
	(1.910)	(0.055)
Mother FE	*Yes*	*Yes*
N	4550	4550
Mean of dep. var. in low prevalence areas in 2006	7.33	0.56
Mean of dep. var. in high prevalence areas in 2006	1.72	0.71

Data : PSF Panel.
Differences-in-differences regression with mother fixed effects. Linear probability model.
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Dummy for no health expenditures for any child in the mother's cell.
Standard errors, in (), are clustered at the district level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.2: Prediction 1 and 2
Using alternative measures of malaria prevalence

	Prevalence in 2006		*Continuous measure of the 2000 prevalence*	
	Per capita levels *(1)*	*Zero spending* *(2)*	*Per capita levels* *(3)*	*Zero spending* *(4)*
Post	0.657	0.007	0.546	−0.015
	(1.506)	(0.022)	(1.634)	(0.027)
High prevalence × Post	2.637	−0.204***		
	(1.763)	(0.033)		
Prevalence × Post			6.392	−0.345***
			(4.916)	(0.106)
Mother FE	*Yes*	*Yes*	*Yes*	*Yes*
N	4550	4550	4550	4550
Mean of dep. var. in low prevalence areas in 2006	8.03	0.52		
Mean of dep. var. in high prevalence areas in 2006	1.53	0.74		
Mean of dep. variable in 2006			5.24	0.61

Data : PSF Panel.
Differences-in-differences regression with mother fixed effects. Linear probability model.
Dep var :in col. (1) and (3) : Health expenditures per capita for children in mother's cell (thousands of CFA francs). Dep var in col. (2) and (4) : Dummy for no health expenditures for any child in the mother's cell.
In col (1) and (2) : We use the malaria prevalence in 2006 instead of 2000 to construct the high/low prevalence areas.
In col (3) and (4) : "Prevalence" is the proportion of children aged 2-10 infected by malaria in 2000 (measured on a scale 0 to 1).
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.3: Regressions corresponding to Figures 4 and 6

	Per capita levels *(1)*	*Zero spending* *(2)*
High prevalence	-5.615^{***}	0.157^{***}
	(1.286)	(0.021)
Post	-0.244	-0.047^{***}
	(1.416)	(0.018)
High prevalence × Post	3.739^{**}	-0.129^{***}
	(1.694)	(0.029)
Constant	7.335^{***}	0.556^{***}
	(1.235)	(0.013)
Mother FE	*No*	*No*
N	4550	4550

Data : PSF Panel.
Differences-in-differences regression without fixed effectss. Linear probability model.
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Dummy for no health expenditures for any child in the mother's cell.
Standard errors, in (), are clustered at the mother level.
* p $\leq$ 0.1, ** p $\leq$ 0.05, *** p $\leq$ 0.01.

Table A.4: External validity : estimating the cross-disease response in other African countries

	Senegal *(1)*	*Kenya* *(2)*	*Rwanda* *(3)*
High prevalence	0.023^{***}	0.059^{***}	0.040^{***}
	(0.008)	(0.010)	(0.011)
Post	-0.031^{***}	-0.040^{***}	-0.023^{***}
	(0.008)	(0.006)	(0.006)
High prevalence × Post	-0.020^{**}	-0.043^{***}	-0.057^{***}
	(0.010)	(0.011)	(0.013)
Constant	0.148^{***}	0.088^{***}	0.105^{***}
	(0.006)	(0.006)	(0.004)
Mother FE	*No*	*No*	*No*
N	21251	23271	14800
Mean of dep. var. in low prevalence areas in 2006	0.15	0.09	0.11
Mean of dep. var. in high prevalence areas in 2006	0.17	0.15	0.15

Data : Senegal : DHS 2005 and DHS 2010. Kenya : DHS 2003 and DHS 2014. Rwanda : DHS 2005 and DHS 2010.
Sample: 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.
Differences-in-differences regression without mother fixed effects. Linear probability model.
Dependent variable : Dummy equal to 1 if the mother did not sought any medical advice or medical treatment in case of diarrhea
Standard errors, in (), are clustered at the mother level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.5: Geographical dynamics

	Excluding Dakar		Rural		Urban		Controlling for regional trends	
	Per capita levels	*Zero spending*	*Per capita levels*	*Zero spending*	*Per capita levels*	*Zero spending*	*Per capita levels*	*Zero spending*
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*
Post	−0.260	−0.086***	1.820*	−0.093***	0.284	−0.011	2.598	0.019
	(1.953)	(0.028)	(1.048)	(0.035)	(2.039)	(0.026)	(1.827)	(0.031)
High malaria prevalence × Post	3.704*	−0.070*	1.791	−0.054	2.350	−0.189***	1.413	−0.117**
	(2.217)	(0.039)	(1.631)	(0.046)	(2.242)	(0.072)	(1.712)	(0.046)
Mother FE	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
N	3243	3243	2339	2339	2211	2211	4550	4550
Mean of dep. var. in low prevalence areas in 2006	5.86	0.65	2.26	0.70	9.74	0.49	7.33	0.56
Mean of dep. var. in high prevalence areas in 2006	1.72	0.71	1.87	0.70	1.05	0.76	1.72	0.71
Number of clusters	94	94	64	64	86	86	150	150
% of high prevalence clusters	51%	51%	60%	60%	12%	12%	32%	32%

Data : PSF Panel.
Differences-in-differences regression with mother fixed effects. Linear probability model.
Dep var : (1), (3), (5) & (7) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2), (4), (6) & (8) : Dummy for no health expenditures for any child in the mother's cell.
Standard errors, in (), are clustered at the mother level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.6: Robustness tests

	Controlling for sibship structure		Excluding migrants	
	Per capita levels	*Zero spending*	*Per capita levels*	*Zero spending*
	(1)	*(2)*	*(3)*	*(4)*
Post	1.854	−0.055**	0.966	−0.049**
	(1.512)	(0.026)	(1.442)	(0.022)
High malaria prevalence × Post	2.421	−0.108***	2.812	−0.102***
	(1.684)	(0.034)	(1.794)	(0.036)
Mother FE	*Yes*	*Yes*	*Yes*	*Yes*
N	4550	4550	2974	2974
Mean of dep. var. in low prevalence areas in 2006	7.33	0.56	6.07	0.57
Mean of dep. var. in high prevalence areas in 2006	1.72	0.71	1.18	0.69

Data : PSF Panel. Sample in (3) & (4) : Mothers residing in the same geographical district in both waves.
Differences-in-differences regression with mother fixed-effects. Linear probability model.
Dep var in (1) & (3) : Health expenditures per capita for children in mother's cell (thousands of CFA francs). Dep var in (2) & (4) : Dummy for no health expenditures for any child in the mother's cell.
Controls included in (1) & (2) : average age of children, number of children and share of children under 5
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.7: Differences in Differences in Health expenditures at the child level

	Full sample	Children younger than 5 in 2009	Children older than 5 in 2009
	(1)	*(2)*	*(3)*
Post	0.392	−1.558	1.338
	(1.043)	(1.948)	(1.227)
High prevalence × Post	2.411**	4.810**	1.249
	(1.207)	(2.289)	(1.408)
Child FE	*Yes*	*Yes*	*Yes*
N	8824	2867	5957
Mean of dep. var. in low prevalence areas in 2006	6.12	7.02	5.69
Mean of dep. var. in high prevalence areas in 2006	1.32	1.06	1.44

Data : PSF Panel. Sample of children living with their mother in PSF1.
Differences-in-differences regression with child fixed effects. Linear probability model.
Dep var : individual health expenditures (thousands of CFA francs).
Standard errors, in (), are clustered at the child level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.8: Heterogeneity analysis by variation in ITN use

	Per capita levels *(1)*	*Zero spending* *(2)*
Post	1.215*	−0.106
	(0.627)	(0.071)
High Δ in ITN use × Post	2.631*	−0.058
	(1.383)	(0.077)
Mother FE	*Yes*	*Yes*
N	1717	1717
Mean of dep. var. in low Δ in ITN use areas in 2006	2.62	0.73
Mean of dep. var. in high Δ in ITN use areas in 2006	1.55	0.71

Data :PSF Panel. Sample restricted to high malaria prevalence areas.
Difference-in-difference regression with mother fixed effects. Linear probability model.
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Dummy for no health expenditures for any child in the mother's cell.
High Δ in ITN use: dummy equal to one if the average ITN use variation between 2006 and 2011 within the cluster of observation was higher than the national average (+20pp).
Standard errors, in (), are clustered at the mother level.
* p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table A.9: Attrition: characteristics of mothers not found in the second PSF wave

	High prevalence		Low prevalence	
	Non-attrited	Attrited	Non-attrited	Attrited
Mother's age in PSF1	34.71	30.60	35.49	33.77
Cell total consumption (thousands of CFA francs) in PSF1	182.88	198.34	362.21	485.41
Average age of children in cell in PSF1	7.54	5.22	7.61	6.46
Health exp. for children per capita in PSF1	1.76	0.97	6.58	12.90
# of cells	767	42	1195	163

Data : PSF Panel.

The Table shows the average characteristics reported in the first wave for women who were found (non-attrited) and were not found (attrited) in the second wave.