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

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

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

Pauline Rossi* and Paola Villar† ‡

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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 seminal paper of Dow et al. (1999), we develop a simple model of health investments under competing mortality risks, in which people allocate expenses to equalize lifetime across all causes of death. 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, at the extensive and intensive margins, and both in level and in composition. The same result holds for parental health-seeking behavior in case of other diseases like diarrhea. We provide evidence that these patterns cannot be explained by differential trends in total income or access to healthcare or child morbidity between malarious and non-malarious regions. Our results suggest that behavioral responses to anti-malaria campaigns magnify their impact on all-cause mortality for children.

***JEL* Classification: D1, H51, I1, O15**
Keywords: Health investments, Malaria, Africa

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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 ruined efforts to prevent other diseases. 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 Therapy (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 the leading 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 risks from all causes of death. They develop a model of private health investments in which there are complementarities between investments in the prevention of cause-specific mortality risks. We adapt their framework to contexts where malaria prevails over all other causes and show that *not* investing in health might be optimal for poor households. Our model predicts that private health investments should take off 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, at both the extensive and intensive margins, 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

[1] Another preventive intervention is to have public agents spray the inside of dwellings with an insecticide (Indoor Residual Spraying, henceforth IRS). This type of intervention historically eradicated malaria in many places. Nowadays, it is less promoted and covers less than 5% of the population at risk in Africa.

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 in case of other diseases like diarrhea increased more in malarious regions.

Our results could potentially by driven by differential trends in total income, access to healthcare or child morbidity. We provide evidence that this is not the case. When we account for household expenditures, health infrastructure, other large-scale public health campaigns, prevalence of child diseases 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 argue that the subsidy component of anti-malaria interventions, rather than the information component, was the main vehicle for change. Households who started investing in health in response to the interventions were (i) not specifically targeted to receive information and (ii) in the middle of the wealth 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.

We are the first to use data on private health expenditures to validate the model of Dow et al. (1999). In their paper, they 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. This conceptual framework 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. Carneiro et

al. (2012) do not find empirical support for crowding-out effects either. They examine the relationship between a private (ITN) and a public (indoor residual spraying) 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 disease-specific interventions are wasted because of competing mortality risks. Our results suggest that it is not the case for anti-malaria campaigns. On the contrary, people reallocate resources to fight other diseases, generating important spillovers effects on all-cause mortality. 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 remainder of 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 accounting for the stylized facts. 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 the Demographic and Health Surveys conducted in African countries since 2000, 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 A simple model of private health investment decisions

We start by adapting the model of Dow et al. (1999) to explain the stylized facts described above. We further derive three predictions that we will test using our data from Senegal. A full description of the model can be found in Subsection 10.1 in the Appendix.

3.1 Key theoretical insights

Consider an individual who has to allocate wealth across her lifetime. Health investments allow the individual to extend the lifetime at the expense of consumption, generating a trade-off between quantity and quality of life.[2] Dow et al. (1999) argue that, under competing mortality risks, the production function of overall lifetime is Leontief. This implies that disease-specific investments are complementary. Therefore, the optimal allocation of investments equalizes lifetime across all causes of death. In this framework, a public subsidy related to a specific disease affects private incentives to fight not only this disease, but also other causes of deaths.

3.2 Adaptation to contexts where malaria is the leading cause of death

Dow et al. (1999) only discuss interior solutions, whereas corner solutions play a crucial role in our context. Before Roll-Back Malaria, the most immediate cause of death was malaria. The disease accounted for 17% of deaths among children aged under five in Sub-Saharan Africa in

[2] In our adaptation, just as in Dow et al. (1999), we consider altruistic parents facing a similar trade-off: investing in child health raises the life expectancy of children but reduces current consumption.

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] The disease is clearly perceived by parents as the main threat to their children - e.g. Tarimo et al. (2000) in Tanzania and Deressa and Ali (2009) in Ethiopia.

In a competing risk framework, this implies that health investments should target first malaria. Other health expenses should take up only once the risk of dying from malaria reaches the risk of dying from the second cause of death. Depending on wealth, there are three optimal allocations:

1. No investment: people are too poor to afford treatment against malaria. Treatments against other diseases are wasted due to competing risks.
2. Positive investment in malaria only: people are wealthy enough to afford some treatment against malaria, but the disease remains the leading cause of death.
3. Positive investment in malaria and other diseases: people are wealthy enough to reduce the mortality risk from malaria down to the point where treatments against other diseases are worth it.

Poor people living in malarious environments are trapped in a corner solution. If the price of treatments against other diseases decrease, there is no impact on their investments. This is consistent with the trends in mortality discussed in Section 2.

3.3 Testable predictions

First of all, this theoretical framework matches the pattern of expenses documented by the literature on malarious countries. Malaria is by far the first component of out-of-pocket health expenditures, representing between 20% and 40% of the total (Mugisha et al., 2002; Onwujekwe et al., 2000). Moreover, this proportion is higher among poorer households (Onwujekwe et al., 2010).

Can we go one step further and use Roll-Back Malaria to test the model of Dow et al. (1999)? The intervention reduced dramatically the price of anti-malaria treatments, making them affordable to a large share of the population. People are predicted to move from the case "no investment" to the cases "investment in malaria" and "investment in all diseases". Therefore private investments to fight malaria and other diseases should increase after Roll-Back Malaria.[4]

Empirically, as described in the next section, we observe total expenditures on child health and health-seeking behavior in case of other diseases. To formalize, denote q treatments bought by households at price p, and Q treatments provided for free; malaria is subscripted by m

[3] Other major causes were upper and lower respiratory infections (influenza, diphtheria, pneumonia, bronchiolitis, ears infection), diarrhoeal 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 anti-malaria treatments decreases.

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 in case of other diseases should increase.

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

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

[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.

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 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 diseases.[8] That is why we complement our empirical analysis with another household survey described below.

4.2 Repeated cross-sections on child health status

We exploit the Demographic and Health Surveys (DHS hereafter) conducted in Senegal in 2005 and 2010-11 to measure trends in child morbidity and document health-seeking behavior. DHS report cases of children under age 5 suffering from diarrhea, and suffering from fever and/or cough (the two symptoms are considered altogether). Parents are asked if they sought treatment for the sick child and what type of treatment.

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

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

[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.

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. It is not possible to include mother fixed effects so that 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, 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).

[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.1).

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-5s 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. 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.

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 areas tend to be more affected by the disease. As a consequence, households in high prevalence areas are poorer. 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 parents look for medical advice or treatment when their child is sick (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.[10]

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.1) and find comparable results.

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.

To test our identification assumption, we cannot look at pre-trends: in the past, there was malaria in malarious regions. The ideal test would be to look at trends once malaria has been eradicated, to check that initial malaria prevalence is not correlated with future dynamics. In our context, we cannot implement this test because malaria is far from being eradicated yet.

[10] 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 (Tables available upon request).

Instead, we provide support for this assumption in two ways. First, we account for composition effects by using panel data. Indeed, mother fixed effects allow to disentangle the effect of public subsidies from that of changes in population characteristics. What we need to discuss are changes in the environment that could have affected differently low and high prevalence areas. This is our second test: we examine differential trends in other determinants of child health expenses: total income, health infrastructure, other health campaigns, rainfall patterns, child morbidity and rural-urban dynamics. We show in Section 7 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.[11]

When we include mother fixed effects, estimating equation 5.1 with health expenditures as an outcome, results are qualitatively similar. As shown by Table 3 column 1, 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

[11] Estimation of the differences-in-differences regression without fixed effects can be found in column 1 of Table A.2 in the Appendix. The coefficient on the interaction term is significant at 5%.

and hospitalization, more on consultation. This suggests that parents reallocated part of their expenses from curing episodes of malaria to medical examination. Another way to distinguish between preventative and curative treatments is to look at sick and non-sick children separately (cf. Figure A.2 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 by 17.7 p.p whereas a more moderate downturn of 4.7 p.p happened in low prevalence areas.[12]

Estimates of the regression with fixed effects are reported in Table 3, column 2. The coefficient on the interaction term remains of similar magnitude (-11.6 p.p.) and significance (at the 1% level).

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

Table 4 presents the estimates of equation 5.1 without fixed effects, using a dummy indicating if parents sought treatment when their child was sick. The first column pools together all diseases. Separated results for diarrhea and fever/cough are in columns 2 and 3 respectively. Before 2009, children in high prevalence areas were less likely to benefit from either medical advice or treatment, whatever the disease. In the case of diarrhea, they almost entirely caught up between the two waves, supporting the idea 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. Indeed, fever is a symptom of malaria and children under 5 suffering from fever in areas with high initial prevalence are probably not the same before and after anti-malaria campaigns. Ideally, we would like to run the regression for cough only but we cannot distinguish between the two symptoms in DHS data.

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

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

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.3 in 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 - lower in Kenya, higher in Rwanda - which itself is negatively correlated with the initial level of health-seeking behavior. Senegal has therefore an intermediate position in terms of magnitude.

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 model 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: total income, access to healthcare (infrastructure and campaigns), rainfall, child morbidity and geographical dynamics. We explain why these stories do not confound our results, and in fact make them stronger.

7.1.1 Total income

The first 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 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.[13] 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

[13]Health expenditures account for only 3% of the cell total consumption.

increased everywhere between the two waves, and slightly more in high prevalence areas. The coefficient on the interaction term is 1.7, not significantly different from zero (table available upon request). This is consistent with the competing risk channel and not with the adult health channel.

7.1.2 Access to healthcare

The second 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. To examine this mechanism, we start by considering changes in health infrastructure and then turn to other child health campaigns.

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 more in low prevalence areas.

Second, two large-scale health campaigns were implemented in Senegal during our period of interest, against measles and diarrhea. 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 6M zinc dispersible tablets were delivered to Senegal by the UNICEF, and they were only distributed in a few regions (Derosena, 2011). If these regions were predominantly highly malarious areas, this may explain the change in health-seeking behavior in case of 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.

7.1.3 Rainfall

The third 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[14] 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.[15] 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).

7.1.4 Child morbidity

The fourth alternative story relies on selection into illness. If health expenses include mostly curative treatments, they may be a proxy for child morbidity. Our results could reflect a deterioration of child health in high prevalence areas rather than a change in health-seeking behavior in case of illness.

To test this explanation, we check in DHS that morbidity trends are not worse in high prevalence areas. Trends for diarrhea, cough and/or fever are shown in Column 3 in Table 5. They are not statistically different between malarious and non-malarious areas, and if anything, tend to be better in malarious ones.[16]

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

[14] 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

[15] 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).

[16] In levels, parents are less 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 higher in low malaria prevalence areas, where people are on average better off.

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.

One way to rule out this concern is to look at urban and rural areas separately. Columns 1 to 4 in Table A.4 in Appendix shows that our findings hold for each sub-sample. More generally, in columns 5 and 6, 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.5 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.3 in 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.6 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.6, 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

To shore up our results, we 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.7 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.5 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.8 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 in the "investment in both diseases" case. 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.[17] Carneiro et al. (2012) 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. In our case, we document a complementarity between a disease-specific public spending and total private spending, including expenditures to fight other diseases. This cross-disease effect is hard to explain with the imperfect information story.

A second argument in favor of prices is provided by Dupas (2009). The author examines the determinants of private investments in malaria prevention. She finds that demand is very sensitive to price, but not influenced at all by the framing of marketing messages.

A third test supporting the price channel is that households who started investing in health after the campaign are in the middle of the income distribution. This is consistent with our model: the very poor remain in the case "no investment" (prices are still too high) and the very rich were already in the case "investment in all diseases". In Table 7, 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 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

[17] 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).

income levels and then decreases. For clarity of exposition, Figure 7 represents estimates of the difference-in-difference as a function of total expenditures over the support observed in PSF1.

Last, 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 8, 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. Although we cannot rule out information spillovers, this is suggestive evidence that subsidies played a larger role than information.

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. Trends in total income, access to healthcare and child morbidity 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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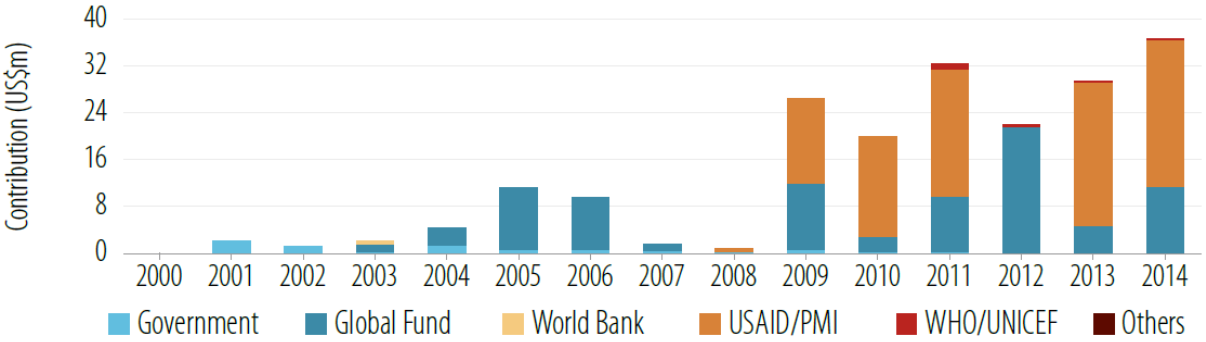
9 Figures and Tables

9.1 Figures

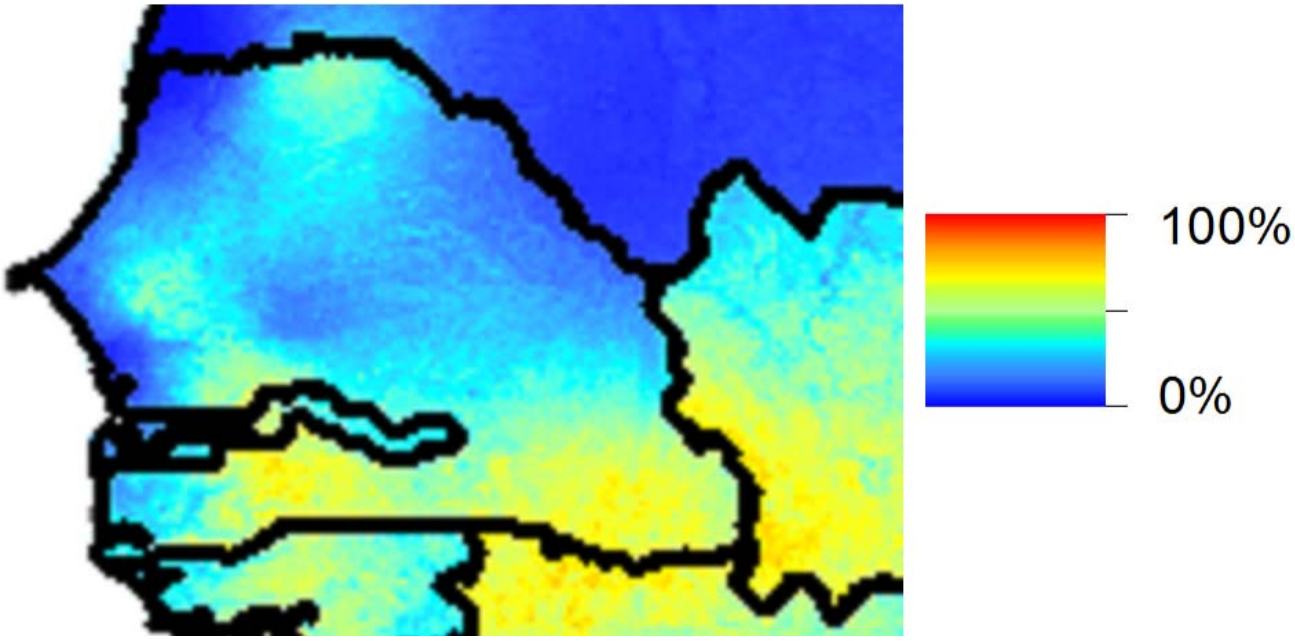
Figure 1: Temporal variation: funds allocated to anti-malaria interventions



Source: World Health Organization and others (2015)

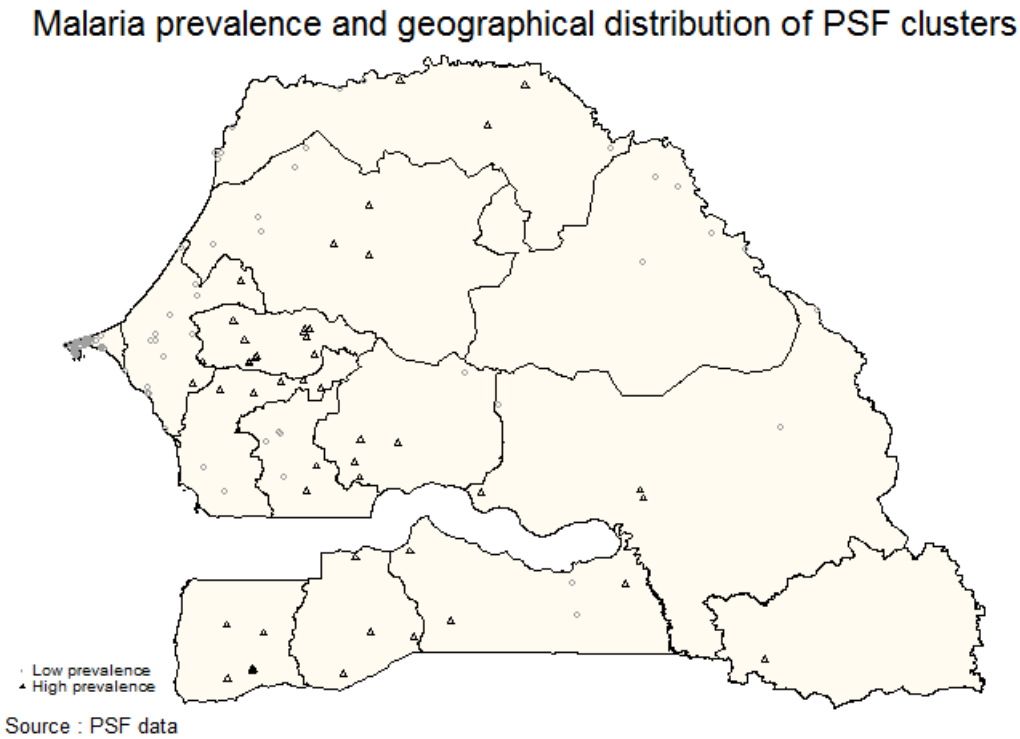
The figure shows the amount of funds allocated to anti-malaria interventions in Senegal. There is a jump in 2009, which coincides with the first nationwide distribution of bednets and the free delivery of curative treatments in public health facilities. We have data on private health investments before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



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

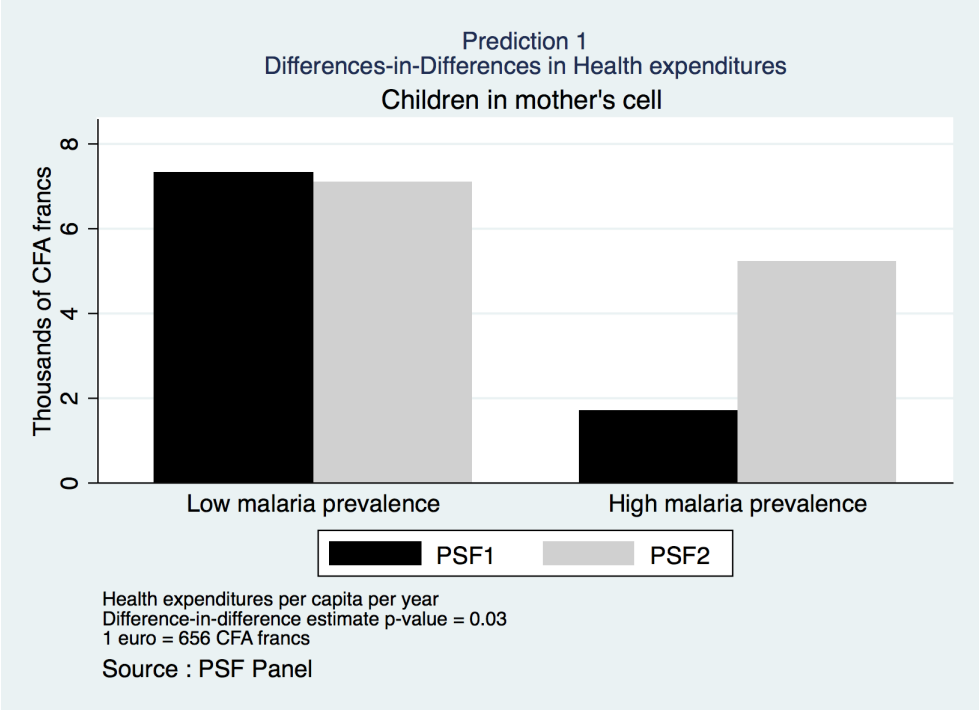
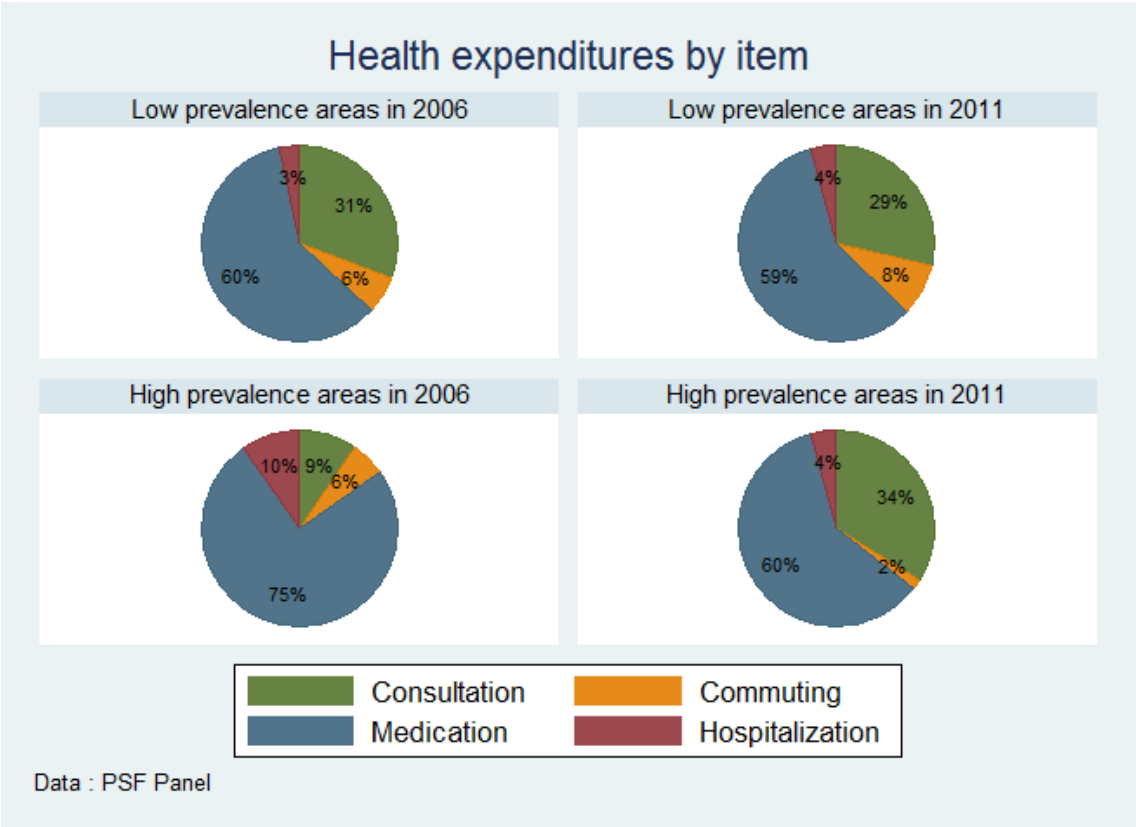


Figure 5: Breakdown of health expenditures by item



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

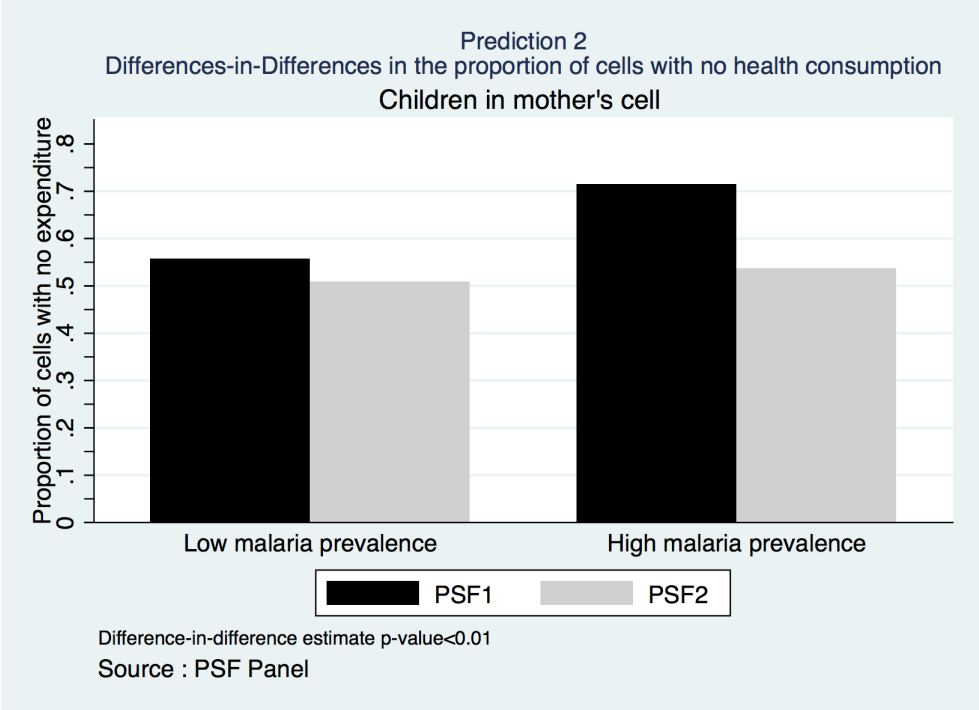
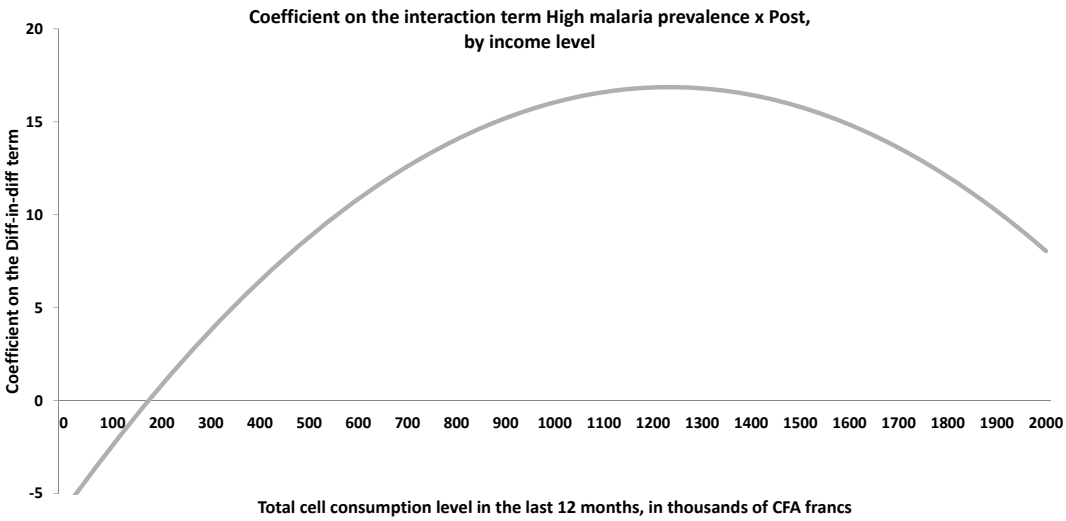


Figure 7: Heterogeneity by income



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

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

	All diseases (1)	*Diarrhea* (2)	*Fever and Cough* (3)
High prevalence	-0.075^{***}	-0.070^{***}	-0.065^{***}
	(0.016)	(0.020)	(0.018)
Post	0.017	0.135^{***}	-0.008
	(0.018)	(0.024)	(0.020)
High prevalence × Post	0.034	0.060^{**}	-0.002
	(0.023)	(0.030)	(0.026)
Constant	0.416^{***}	0.266^{***}	0.426^{***}
	(0.012)	(0.016)	(0.013)
Mother FE	*No*	*No*	*No*
N	8466	4188	6672
Mean of dep. var. in low prevalence areas in 2006	0.42	0.27	0.43
Mean of dep. var. in high prevalence areas in 2006	0.34	0.20	0.36

Data : DHS 2005 and DHS 2010.Sample: children under age 5 who have been sick in the last two weeks before the survey.
Differences-in-differences regression without mother fixed effects. Linear probability model.
Dep. var: dummy for seeking any medical advice or medical treatment when the child suffered from any disease (column 1), diarrhea (column 2), fever or cough (column 3).
Standard errors, in (), are clustered at the mother level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

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

	Total expenditures	*Distance to health facilities is a concern*	*Child is sick*
	(1)	(2)	(3)
High prevalence		0.106^{***}	-0.032^{***}
		(0.010)	(0.011)
Post	9.675	-0.018^{*}	-0.082^{***}
	(13.507)	(0.011)	(0.012)
High prevalence × Post	−5.259	0.020	−0.017
	(16.035)	(0.014)	(0.015)
Constant		0.336^{***}	0.484^{***}
		(0.008)	(0.008)
Mother FE	*Yes*	*No*	*No*
N	4550	19098	20630
Mean of dep. var. in low prevalence areas in 2006	388.9	0.34	0.48
Mean of dep. var. in high prevalence areas in 2006	187.3	0.44	0.45

Data : (1) : PSF Panel. (2) : Mothers of children under 18 in DHS 2005 and DHS 2010. (3): Children under 5 years of age 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. (3): Dummy equal to 1 if the child had either fever, cough or diarrhea in the last two weeks.
Standard errors, in (), are clustered at the mother level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

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	-0.075^{***}	-0.070^{***}	-0.065^{***}		
	(0.016)	(0.020)	(0.018)		
Post	0.024	0.141^{***}	−0.002	0.873	−0.034
	(0.018)	(0.025)	(0.020)	(1.367)	(0.021)
High prevalence × Post	0.053^{**}	0.079^{**}	0.014	3.094	-0.096^{***}
	(0.026)	(0.034)	(0.029)	(1.904)	(0.037)
Constant	0.416^{***}	0.266^{***}	0.426^{***}		
	(0.012)	(0.016)	(0.013)		
Mother FE	*No*	*No*	*No*	*Yes*	*Yes*
N	8466	4188	6672	4548	4548
Mean of dep. var. in low prevalence areas in 2006	0.42	0.27	0.43	7.33	0.56
Mean of dep. var. in high prevalence areas in 2006	0.34	0.20	0.36	1.72	0.71

Data (1), (2) & (3): DHS 2005 and DHS 2010. Sample of children under age 5 who have been sick in the last two weeks before the survey. (4) & (5) : PSF Panel.
Differences-in-differences regression. Linear Probability Model.
Dependent variables : (1), (2) & (3) : Dummy for seeking any medical advice or medical treatment when the child suffered from any disease (column 1), diarrhea (column 2), fever or cough (column 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.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table 7: 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.

Table 8: What is the role of information?

	Mothers targeted specifically by information campaigns		Other mothers	
	Per capita levels *(1)*	Zero spending *(2)*	Per capita levels *(3)*	Zero spending *(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.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

10 Appendix

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

We build upon the Dow, Philipson and Sala-i-Martin model (Dow et al., 1999) of health investments under competing mortality risks. In this theoretical framework, an individual has to allocate her exogenous wealth W across the lifetime of T years, T being known by the individual. If we assume an increasing and strictly concave utility function $U(.)$, as well as no discounting, the optimal solution of the consumption component is constant (perfect smoothing). The maximization of the indirect utility function $V(W,T)$ corresponds then to the maximization of $V(W,T) = TU(\frac{W}{T})$, and the annual consumption level is equal to $\frac{W}{T}$. Therefore, at any given level of wealth, the individual faces a trade-off between quantity of life (T), and quality of life ($U(\frac{W}{T})$).

Costly health investments allow the individual to extend T and hence to convert quality of life into quantity of life. The cost of reaching T years of life is represented by $c(T)$. The explicit expression of the indirect utility becomes then $V(W - c(T), T) = T \times u(\frac{W-c(T)}{T})$. We assume here that $u(.)$ is a CRRA utility function of parameter $\theta \in]0,1[$. The first order condition (FOC) of the maximization program on T is $\frac{w-c(T)}{T} = c'(T) \times \frac{1-\theta}{\theta}$, and simply reflects that the marginal increase in utility of an additional year must equalize its marginal cost.

Model with 1 disease Let us start with a framework where only one cause of death prevails. The overall lifetime T can be decomposed into two components: $T = \alpha_o + q_o$, where α_o is the lifetime under no health investment, and q_o is the level of health inputs invested to avert the disease. We further assume that $c(T) = p_o.(T - \alpha_o)$, where p_o is the unit price of the disease-specific input. Substituting $c(T)$ into the FOC expression, we obtain the optimal level of health investment, q^*:

$$q^* = \begin{cases} \theta \times \frac{w}{p_o} - (1-\theta) \times \alpha_o, & \text{if } w > B \\ 0, & \text{otherwise} \end{cases}$$

Where $B = \frac{(1-\theta)}{\theta}\alpha_o p_o$. In the one-disease case, predictions are straightforward and intuitive. Health investments increase with wealth and decrease with the cost of treatments.

Model with 2 diseases Now, assume a context where two diseases exist : malaria (subscripted by m) and another competing disease (or a composite disease, subscripted by o). Following the previous notations, α_d represents the baseline lifetime with no investment in disease d, q_d is the d-specific level of investment whose unit price is p_d, for $d \in \{m, o\}$. As shown in (Dow et al., 1999), competing mortality risks lead to a Leontief production function of overall

lifetime T so that $T = min(\alpha_m + q_m; \alpha_o + q_o)$.

Specific to our context is the assumption that $\alpha_m < \alpha_o$, reflecting the fact that malaria is the leading cause of death. This generates ample room for corner solutions in environments where most people are poor. There are three types of optimal allocations: one where individuals invest in none of the two diseases, one where they invest in malaria only, and finally one where they invest in both causes [18].

Case 1 – Strictly positive investments in both diseases ($q_m > 0$ and $q_o > 0$): The optimal allocation of wealth is the one that equates the disease-specific lifetimes, so that $T^* = \alpha_m + q_m^* = \alpha_o + q_o^*$. Moreover, the total cost of the two health inputs to reach T is $c(T) = p_m.(T - \alpha_m) + p_o.(T - \alpha_o)$. Using again the FOC, and replacing T and $c(T)$ with the latter expressions we obtain :

$$\begin{cases} q_m^* = \frac{\theta w - p_o(\alpha_m - \theta\alpha_o) - p_m(1-\theta)\alpha_m}{p_o + p_m} \\ q_o^* = \frac{\theta w - p_m(\alpha_o - \theta\alpha_m) - p_o(1-\theta)\alpha_o}{p_o + p_m} \\ \text{and } q_m^* > q_o^* > 0 \text{ if and only if } w > \bar{B}(p_m) \end{cases}$$

Where $\bar{B}(p_m) = \frac{p_m.(\alpha_o - \theta\alpha_m) + p_o.\alpha_o.(1-\theta)}{\theta}$. One important thing to note here is that $\frac{\partial \bar{B}(p_m)}{\partial p_m} > 0$, so that the incentive threshold is positively linked to the anti-malaria inputs price. In addition, health investments present negative cross-price effects, which derive from the complementarity of health inputs. Moreover, $T^* = \theta \times (\frac{w + \alpha_o.p_o + \alpha_m.p_m}{p_o + p_m})$, the optimal overall lifetime increases as p_m and/or p_o decrease.

Case 2 – Strictly positive investment in malaria only ($q_m > 0$ and $q_o = 0$): Here the cost function is $c(T) = p_m \times q_m$. Optimal lifetime T^* is such as $T^* = \alpha_m + q_m^* \leq \alpha_o$.[19] In this case, the solution of the FOC is :

$$\begin{cases} q_m^* = \frac{\theta w}{p_m} - \alpha_m(1-\theta)) \\ q_o^* = 0 \\ \text{and } q_m^* > q_o^* = 0 \text{ if and only if } \underline{B}(p_m) < w \leq \bar{B}(p_m) \end{cases}$$

Where $\underline{B}(p_m) = \frac{p_o.(\alpha_m - \theta\alpha_o) + p_m.\alpha_m.(1-\theta)}{\theta}$. A decrease in p_m lowers $\underline{B}(p_m)$ and raises q_m^* as well as $T^* = \theta \times (\frac{w}{p_m} + \alpha_m))$. These quantities are however totally inelastic to p_o.

[18] The model rules out the possibility that individuals invest only in the other disease, because malaria is the leading cause of death and health investments are complementary. Any investment in the other disease without investing in malaria would then come to naught.

[19] α_o is the natural upper limit of T since we are in the case where the investment in o disease is null.

Case 3 – No investment ($q_m = 0$ and $q_o = 0$): Individuals in this situation are those whose wealth is too low relative to price levels to afford any investment. Hence, individuals are subject to the baseline malaria mortality and $T^* = \alpha_m$.

$$\begin{cases} q_m^* = 0 \\ q_o^* = 0 \\ \text{and } q_m^* = q_o^* = 0 \text{ if and only if } w \leq \underline{B}(p_m) \end{cases}$$

Comparative statics: what happens when the price of anti-malaria products drops? Figure A.1 shows how health investments q_o and q_m change in response to a drop in the price of anti-malaria products p_m, for different levels of wealth. A decrease in p_m has two main effects.

First, the own-price and cross-price effects induce an increase in health investments for a given wealth level : both q_m and q_o increase in Case 1, q_m increases in Case 2, and there is no change for Case 3. In Figure A.1, this corresponds to the slope change of the q_d functions, $d \in \{m, o\}$.

Second, the reduction in prices has also an effect on the thresholds. Both $\underline{B}$ and $\bar{B}$ shift to the left of the wealth axis. A share of individuals whose income was too low to be in Case 1 ($w < \bar{B}$) or Case 2 ($w < \underline{B}$) are now wealthy enough to invest in both causes of mortality (if $w \geq \bar{B}'$) or at least in malaria (if $w \geq \underline{B}'$).

Combining both mechanisms, we predict that q_m and q_o should increase when the price of anti-malaria products drop, at the intensive and at the extensive margins.

10.2 Figures

Figure A.1: Health investment levels before and after subsidizing anti-malaria products

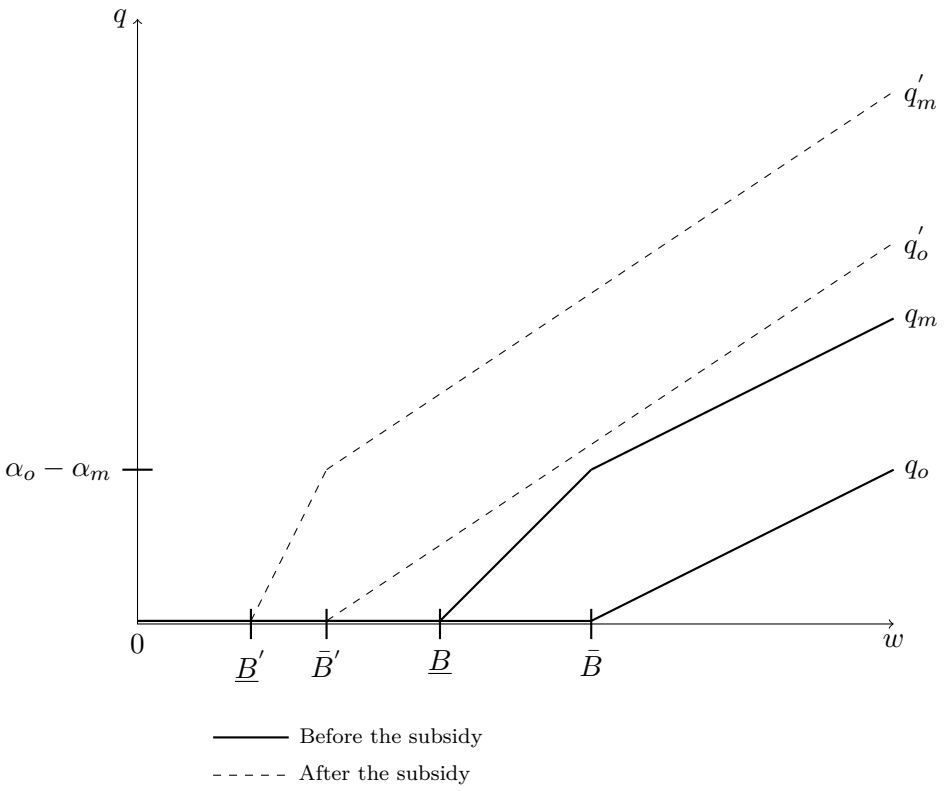


Figure A.2: Health expenditures conditional on sickness

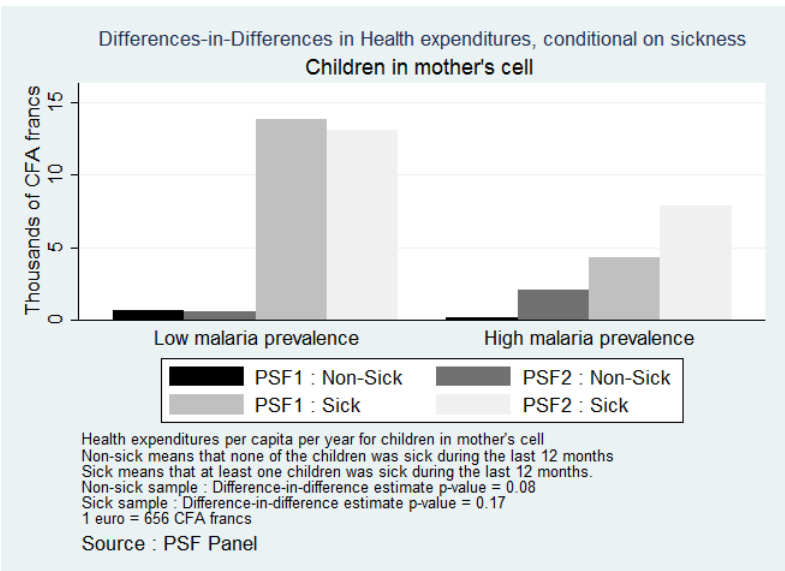
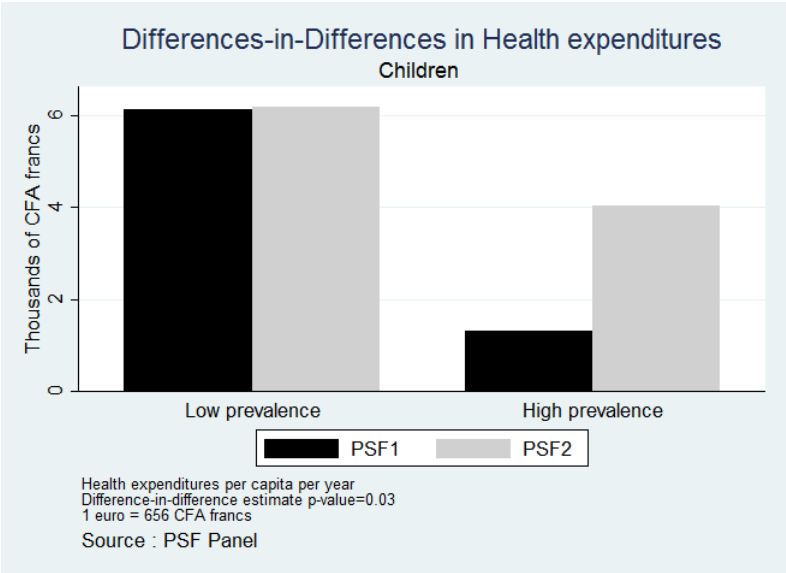


Figure A.3: Child-level analysis: changes in individual health expenditures



10.3 Tables

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

	Prevalence in 2006		*Continuous measure of the 2000 prevalence*	
	Per capita levels	*Zero spending*	*Per capita levels*	*Zero spending*
	(1)	*(2)*	*(3)*	*(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.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.2: Regressions corresponding to Figures 4 and 5

	Per capita levels	*Zero spending*
	(1)	*(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.3: External validity : how has health-seeking behavior in case of diarrhea changed in response to anti-malaria campaigns in other African countries?

	Senegal	*Kenya*	*Rwanda*
	(1)	*(2)*	*(3)*
High prevalence	-0.070^{***}	-0.022	-0.110^{***}
	(0.020)	(0.036)	(0.028)
Post	0.135^{***}	0.294^{***}	0.178^{***}
	(0.024)	(0.029)	(0.023)
High prevalence × Post	0.060^{**}	0.028	0.169^{***}
	(0.030)	(0.041)	(0.049)
Constant	0.266^{***}	0.335^{***}	0.220^{***}
	(0.016)	(0.026)	(0.015)
Mother FE	*No*	*No*	*No*
N	4188	3521	2017
Mean of dep. var. in low prevalence areas in 2006	0.27	0.34	0.22
Mean of dep. var. in high prevalence areas in 2006	0.20	0.31	0.11

Data : Senegal : DHS 2005 and DHS 2010. Kenya : DHS 2003 and DHS 2014. Rwanda : DHS 2005 and DHS 2010.
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.
Sample: children under age 5 who have been sick in the last two weeks before the survey.
Differences-in-differences regression without mother fixed effects. Linear probability model.
Dep. var: dummy for seeking any medical advice or medical treatment when the child suffered from diarrhea.
Standard errors, in (), are clustered at the mother level.
* $p \leq 0.1$, ** $p \leq 0.05$, *** $p \leq 0.01$.

Table A.4: Geographical dynamics

	Rural		Urban		Controlling for regional trends	
	Per capita levels	*Zero spending*	*Per capita levels*	*Zero spending*	*Per capita levels*	*Zero spending*
	(1)	*(2)*	*(3)*	*(4)*	*(3)*	*(4)*
Post	1.820^{*}	-0.093^{***}	0.284	-0.011	2.598	0.019
	(1.048)	(0.035)	(2.039)	(0.026)	(1.827)	(0.031)
High malaria prevalence × Post	1.791	-0.054	2.350	-0.189^{***}	1.413	-0.117^{**}
	(1.631)	(0.046)	(2.242)	(0.072)	(1.712)	(0.046)
Mother FE	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
N	2339	2339	2211	2211	4550	4550
Mean of dep. var. in low prevalence areas in 2006	2.26	0.70	9.74	0.49	7.33	0.56
Mean of dep. var. in high prevalence areas in 2006	1.87	0.70	1.05	0.76	1.72	0.71
Number of clusters	64	64	86	86		
% of high prevalence clusters	60%	60%	12%	12%		

Data : PSF Panel.
Differences-in-differences regression with mother fixed effects. Linear probability model.
Dep var : (1), (3) & (5) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2), (4) & (6) : 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.5: 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 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.6: 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.7: Heterogeneity analysis by variation in ITN use

	Per capita levels	*Zero spending*
	(1)	*(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.8: 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.