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

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Abstract

Do behavioral responses magnify or weaken public health interventions in Africa? We examine this question in the context of high subsidies for anti-malaria products introduced in Senegal in the late 2000s. Building upon the seminal paper of Dow et al. (1999), we develop a simple model of health investments under competing mortality risks, in which there are complementarities between investments in the prevention of cause-specific mortality risks. 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 exploit 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 increased more in malarious regions, in proportions and in levels, and both at the intensive and extensive margins. 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 nor in child morbidity between malarious and non-malarious regions. Our results suggest that anti-malaria campaigns generate important spill-overs effects that magnify their impact on all-cause mortality for children.

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1 Introduction

Most public programs induce people to change their behaviors. Whether these responses undermine or magnify the intended impact of programs is a long-lasting debate in public economics. We investigate this question in the context of public health interventions in developing countries. More specifically, we examine how health-seeking behavior has changed in response to the introduction of high subsidies for anti-malaria products in Sub-Saharan Africa.

Malaria has been eradicated from various parts of the world during the twentieth century. Today, the burden weighs mostly on Sub-Saharan Africa, where we find the largest prevalence rates and the most dangerous form of malaria. 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, 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 they are sick in 2015, but the coverage is increasing rapidly (World Health Organization and others, 2015).

In this paper, we argue that behavioral responses magnify the impact of Roll-Back Malaria because, in our context, there is a complementarity between public and private health expenditures. 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. Building upon this seminal paper, we develop a simple model of private health investments in which there are complementarities between investments in the prevention of cause-specific mortality risks. In Sub-Saharan Africa, malaria was the leading cause of child death in 2000, accounting for 17% of deaths among children aged under five (World Health Organization and others, 2015). In such an environment, we argue that poor households had few incentives to invest in child health. Anti-malaria products were too expensive, and expenses to fight other diseases were likely to be wasted because malaria was just around the corner. By decreasing substantially the price of preventive and curative treatments, Roll-Back Malaria made it profitable to invest in health, not only to avoid malaria but also other causes of death.

Our model predicts that private health investments should increase in response to anti-malaria public interventions. To test this prediction, we exploit original panel data from a Senegalese

[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.

household survey providing detailed information on health expenses. Malaria control efforts in Senegal took off between the two waves of the panel (2006-2007 and 2011), providing a perfect setting to analyze households' responses. Our empirical strategy is to document changes in expenditures patterns between the two waves, comparing malarious and non-malarious regions of Senegal.

We find that child health expenditures were initially lower and increased more, in proportions and in levels, in malarious regions. Moreover, the proportion of households with zero health expenditures was initially higher and decreased more in malarious regions. The change in spending behavior was stronger in regions where anti-malaria campaigns were more intense, supporting the idea that public health interventions caused that change. We further show that households who start investing in health in response to anti-malaria interventions are in the middle of the wealth distribution. Rich people had already started to invest before the campaign, and very poor people still cannot afford any health expense. Last, we find that health-seeking behavior in case of other diseases like diarrhea increases more in areas with high initial malaria prevalence.

Our results are not driven by a larger increase in total income in malarious regions. Indeed, when we consider other types of expenditures, we find that they have increased slightly less in malarious regions, suggesting that households have reallocated part of their income to health. Our results cannot be explained either by worse trends in child morbidity in malarious regions. Using DHS waves conducted roughly at the same time (2005 and 2010), we show that the prevalence of child diseases decreased everywhere in the same way between the two waves. Last, our results are qualitatively unchanged once we account for selective migration, attrition and changes in family structure.

Our paper makes three contributions. First, it relates to the literature on behavioral responses to health subsidies in Africa. 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. 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. More generally, the question is whether public and private health spending are complements or substitutes. The standard presumption in the public economics literature is substitutability, many studies being concerned about crowding-out effects. But in the context of health in developing countries, empirical evidence supporting this presumption is scarce. Carneiro et al. (2012) examine the relationship between a private (ITN) and a public (IRS) investment to fight malaria in Eritrea. To their surprise, they find that households were *more* likely to buy a bed net when public health agents had sprayed their own dwellings with insecticide.

Second, we contribute to the theoretical strand of the literature on household health behavior in developing countries. In their paper, Dow et al. (1999) provide empirical support to their model by showing that birth outcomes improve after child vaccination campaigns in Sub-Saharan Africa. The evidence is at best suggestive, because we do not know what additional interventions were embedded in the campaigns, and they might have influenced directly maternal health. To test properly the model, data on health outcomes is not enough. That is why we exploit data on health expenses. We find evidence of complementarities, which helps explaining 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.

Third, 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 that magnify the impact of anti-malaria interventions on all-cause 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 (2016) 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 much larger than the impact of ITNs on child mortality estimated in the medical literature. The authors argue that the discrepancy can be explained by two reasons: first, medical RCTs lack external validity ; second, the correlation captures an endogenous placement of ITNs and a bunching of health interventions, in addition to the causal impact of malaria control on mortality.

Another potential 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 Figure 1. Using the Demographic and Health Surveys conducted in African countries since 2000, the figure shows 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, mortality decreased gradually in regions with low initial prevalence, for both rich and poor households. 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, at a speed similar to the other groups. These remarks are confirmed by Table 1. We estimate the linear trend in child mortality before and after the start of anti-malaria campaigns for the four sub-groups mentioned above. Before the campaigns, child mortality was decreasing everywhere except for poor households in highly malarious regions. After the campaigns started, mortality decreases even more, but the break in trends is only significant for poor households in highly malarious regions, who eventually caught up with the others.

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 expenditures patterns that we document below, as will be discussed.

3 A simple model of private health investment decisions

We start by discussing how the seminal model of Dow et al. (1999) explains the stylized facts described above. We further derive three predictions that we will test using our data from Senegal.

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. 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 Application to our context

In our context, before Roll-Back Malaria, the most immediate cause of child death was malaria. In the 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

Can we go one step further and predict the impact of Roll-Back Malaria using this theoretical framework? The intervention reduced dramatically the price of anti-malaria treatments, making them affordable to a large share of the population. People moved from the case "no investment" to the cases "investment in malaria" and "investment in all diseases". We predict that, after Roll-Back Malaria:

P1 Private investments in child health should increase.

P2 The proportion of households with no investment should decrease.

P3 Private investments to fight other diseases should increase.

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[2] (*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 (about 15,000 individuals). 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 considerable and the second wave covers about 3,200 households (about 28,000 individuals).

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[3] 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 health expenditures related to consultation, hospitalization, treatment, and commuting to health facilities that were made during the last 12 months before the interview. 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.

The attrition rate of our panel is 26%. This rate is actually made of two types of events : 16% is due to the fact that some mothers, despite being found in PSF2, had no longer a dependent child in her cell, and 10% because the mother was not found in PSF2. Implications of this attrition is discussed in Section 7.3.3.

The main advantage of PSF is to provide a panel so we can estimate regressions with fixed effects.

[2]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.

[3]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.

The main disadvantage is to register expenses, whereas our predictions are about investments. In other words, we would need data on quantities and we only observe quantities time prices. PSF contains questions about health status of children and health-seeking behavior, but they are not perfectly comparable between the two waves of the panel.[4] 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 having diarrhea, fever and/or cough. Parents are asked if they sought treatment for the ill child and what type of treatment. The main drawback of DHS is to be a repeated cross-section. It is not possible to include individual 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 in 2000 (Bhatt et al., 2015). 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 campaigns. To account for time-varying determinants of expenditures, we exploit another source of variation, comparing malarious and non-malarious regions of Senegal.

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 2 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).

[4] Those questions were asked in the household questionnaire and they were different in the two waves. Unlike questions on health expenses that were asked in the expenditures questionnaire and were the same.

The first nation-wide ITNs distribution campaign started in 2009 and targeted specifically children under 5 and pregnant women. More than 10 millions ITNs were distributed between 2008 and 2011 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 3 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).

Table 2 shows some descriptive characteristics of the PSF sample, by initial malaria prevalence. Our sample is made of 1,594 cells in 1,118 households in PSF1. Malaria prevalence in 2000 is not randomly distributed. In particular, rural areas tend to be more affected by the disease. As a consequence, households in high prevalence areas are poorer. Since rural and urban areas have different dynamics, we checked that our results hold for the two sub-samples separately (cf. Table 11 in Appendix).

5.3 Econometric specification

We proceed to a standard differences-in-differences analysis:

$$Y_{i,t} = \alpha_0 + \alpha_1 \, High_i \; + \alpha_2 \, Post_t + \alpha_3 \, Post_t \times High_i + u_{i,t} \tag{5.1}$$

$Y_{i,t}$ 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). $High_i$ indicates whether the household is located in an area exposed

to high malaria prevalence in 2000. $Post_t$ equals one if the survey took place after 2009. Standard errors are clustered at the mother level. In robustness tests, we introduce time-varying controls to account for changes in family structure (cf. section 7.3.1).

To test predictions 1 and 2, we use the PSF panel. Since all individuals are observed twice, before and after anti-malaria campaigns, equation 5.1 gives the same estimates as an individual fixed effect regression. To test prediction 3, we exploit repeated cross-sections in DHS, in which case we are not able to include an individual fixed effect.

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 assumption is that, in the absence of Roll-Back Malaria, the evolution of health expenses would have been the same for all households. When we use panel data, composition effects are not a threat. What we need to discuss are changes in the environment that could have affected differently low and high prevalence areas.

Unfortunately, we cannot check if this assumption holds before Roll-Back Malaria because we only have two waves. Instead, we examine the trends in other determinants of child health expenses: total expenses and child morbidity. We show in section 7 that they cannot explain our results.

6 Results

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

Figure 4 shows the results of equation 5.1 using health expenditures as an outcome. In the first wave, households in high prevalence areas spent less: on average 1,465 CFA francs per child per year against 6,141 in low prevalence areas. Between the two waves, they multiplied by 3.4 their consumption of health commodities, up to 4,910, while there was no significant growth in low

prevalence areas.[5]

[FIGURE 4 ABOUT HERE]

When we separate preventive and curative treatments, the same pattern holds for both types of expenses.[6]

One worry with health expenditures is that they capture both changes in quantities and changes in prices. However, since the price of anti-malaria treatments decreased substantially, an increase in total expenditures implies an increase in quantities.

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

Figure 5 closely mirrors the previous figure. Households in high prevalence areas were 13.2 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 15.6 p.p whereas a more moderate downturn of 4 p.p happened in low prevalence areas.[7]

[FIGURE 5 ABOUT HERE]

To shore up our results, we look at heterogeneity by ITN use variation. Within malarious areas, the increase in health expenditures should be driven by areas with the largest increase in ITN use. We construct a variable indicating whether the average ITN use variation between 2006 and 2011 within the district of observation was higher than the national average (+20pp). Results are shown in Table 9 in the Appendix. Before the campaign, there was no significant difference between regions who would later receive many or few bednets. After the campaign, private health expenses increased significantly everywhere, but much more in areas who received more bednets. This is another piece of evidence that the change in private expenses was driven by Roll-Back Malaria.

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

Table 3 presents the estimates of equation 5.1 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

[5]Estimation of the differences-in-differences regression can be found in column 1 of Table 6 in the Appendix. The p-value of the estimate for the interaction term is 0.13.

[6]Table available by the authors upon request.

[7]Estimation of the differences-in-differences regression can be found in column 2 of Table 6 in the Appendix. The p-value of the estimate for the interaction term is 0.01.

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.

[Table 3 ABOUT HERE]

Overall, we find that, after anti-malaria campaigns, private health investments, in total and against other diseases, have increased in highly malarious areas, whereas they remained stable in low prevalence areas. This is consistent with a complementarity between public and private health spending.

7 Robustness

7.1 Alternative stories

In this section, we discuss two alternative stories that may generate the same patterns and we explain why they are not plausible in our context.

7.1.1 Adult health

One explanation is the adult health channel. Indeed the health improvements induced by malaria control might also have benefited adults, in particular mothers and breadwinners. This could have led to a positive income effect, 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 look for a differential rise in *all* expenditures. We measure total consumption at the cell level, including all individuals, not only the children. Results are shown in Table 4. Households in highly malarious regions are poorer and do 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.

7.1.2 Child morbidity

Another possible explanation 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 Figure 6. They are decreasing and perfectly parallel.

7.2 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. 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.

Another 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 last test supporting the price channel is that household 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 5, 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".

[Table 5 ABOUT HERE]

7.3 Robustness tests

7.3.1 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 children and their

age. 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. Tables 7 and 8 show that our coefficients remain very stable in magnitude and significance.

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 7 in Appendix, the same pattern holds: individual health expenses increase much more in high prevalence areas. The difference-in-difference coefficient is now significant at the 5% level.

7.3.2 Geographical mobility

One potential concern is that we define the area of residence - high or low prevalence - using PSF1, and women might have migrated between the two waves. Migration could explain our results if people migrate from high to low prevalence areas, and spend more on health in low prevalence areas. This could be the case if people migrate to cities for instance.

The scope for this concern is limited because 93% of our sample stayed in the same city block or in the same village (see Table 2). If we exclude migrants, results are even more salient, as shown in Table 10 in the Appendix.

7.3.3 Selective attrition

Another issue would arise if the attrition observed in the PSF panel were selected differentially between malarious and non-malarious areas. As explained in section 4, attrition comes from two types of events: 16% of mothers had no longer a dependent child and 10% could not be found.

The first type of attrition stems from the sample definition. Mothers who no longer live with their children, or whose children are older than 18 in PSF2, are not included in our sample despite being interviewed. We checked that, in both low and high prevalence areas, those mothers are indeed older and have older children than those included in the sample.

The second type of attrition arises when mothers were not found in the second wave. Table 12 provides some baseline characteristics for these women by type of region. A first observation is the limited scope: only 6% of mothers in malarious areas and 14% 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 (cf. Table 12, Table 2 and Figure 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. 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.

8 Conclusion

This paper investigates how private health investments have responded to malaria control programs 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, first, private health expenditures were multiplied by 3.4 in malarious areas, while they remained stable in non-malarious ones. Second, households in malarious regions were 16 p.p. less likely to make any health expenses before the intervention, and they almost caught up after. Third, parental health seeking behavior when children suffer from other diseases like diarrhea increased more in malarious regions than in non-malarious ones (by 6 p.p.) We provide further evidence that these patterns cannot be explained by differential trends in total income nor in child morbidity between regions. These private responses to a public intervention are consistent with a model of health investments under competing mortality risks.

By showing that public and private health spending may be complements, our study has strong implications for health policies in Africa. It suggests that public health interventions do not crowd out private investments in child health. Households may well take over from public actors and sustain the recent progress in child survival.

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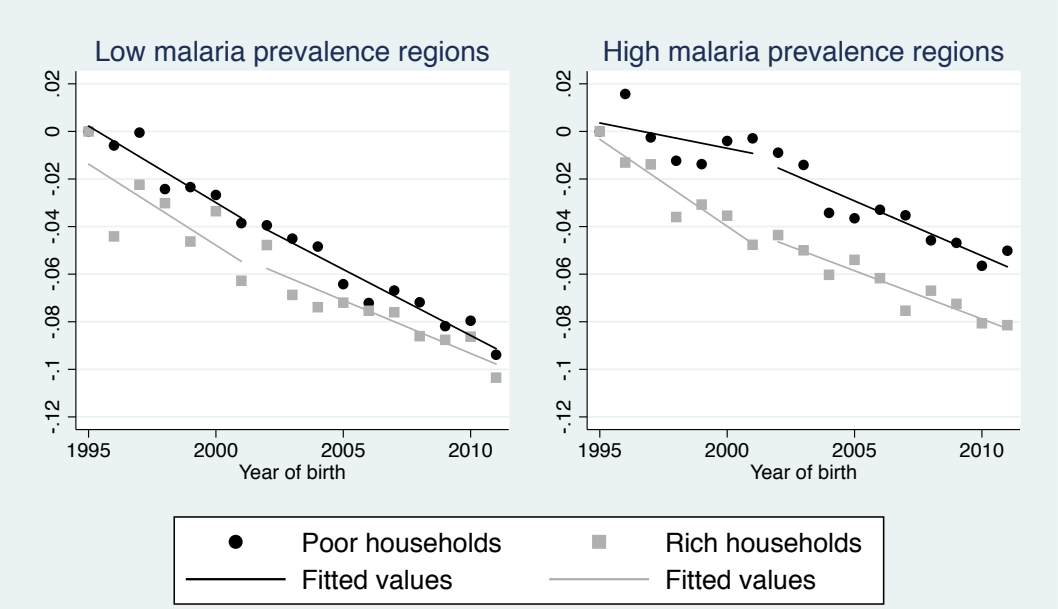
9 Figures and Tables

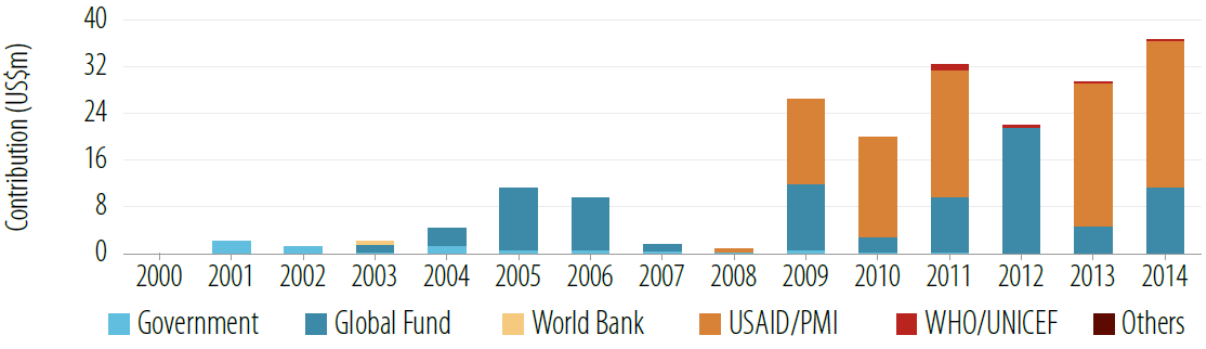
9.1 Figures

Figure 1: Trends in Child mortality



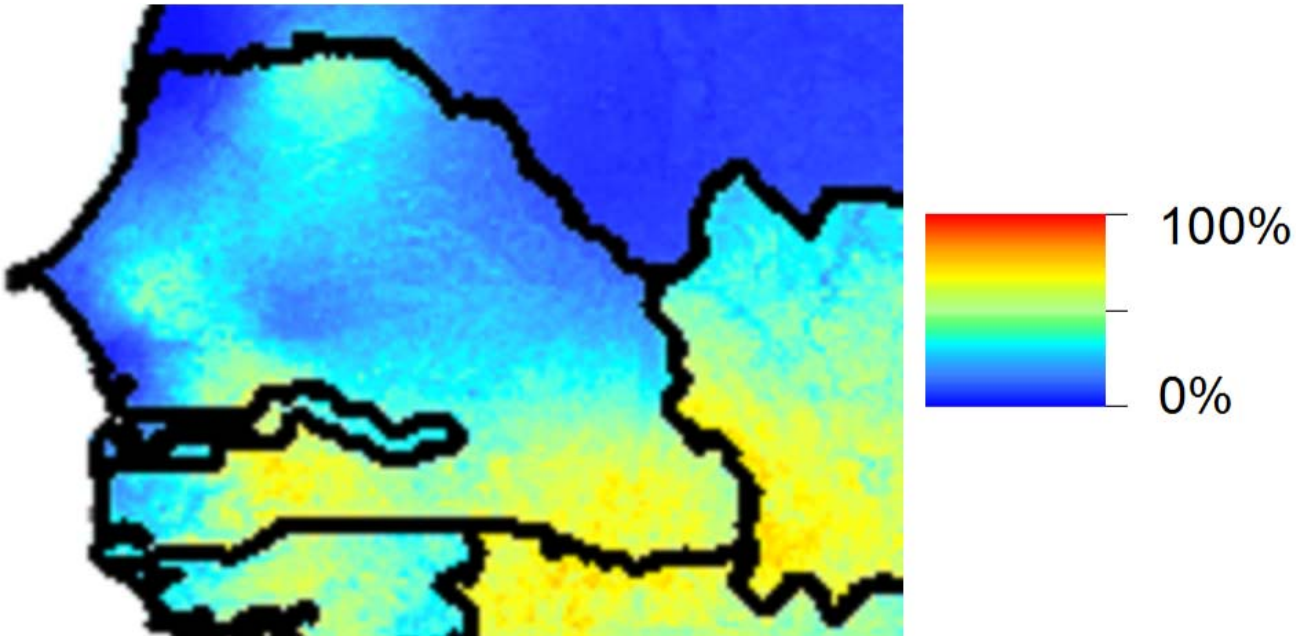
Data: DHS in 19 African countries (cf. Cogneau and Rossi (2016) for a comprehensive description of the dataset).
The graphs plot the coefficients from a within-mother regression of child mortality on year of birth fixed effects.
The graph on the left corresponds to births in regions with a high initial malaria prevalence (>50% of children aged 2 to 10 are infected by the parasite)
The graph on the right corresponds to births in regions with a low initial malaria prevalence <50%).
We use the ownership of durable goods to split the sample of households into the poorest half (in black) and the richest half (in grey).
The solid lines represent the linear fit before (1995-2001) and after (2002-2011) the start of anti-malaria campaigns.
The reference category is 1995. We kept only children born at most 10 years before the survey to perform the estimation.

Figure 2: Funds allocated to anti-malaria interventions in Senegal



Source: World Health Organization and others (2015)

Figure 3: 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. The national average is 24%.

Figure 4: Changes in Health expenditures

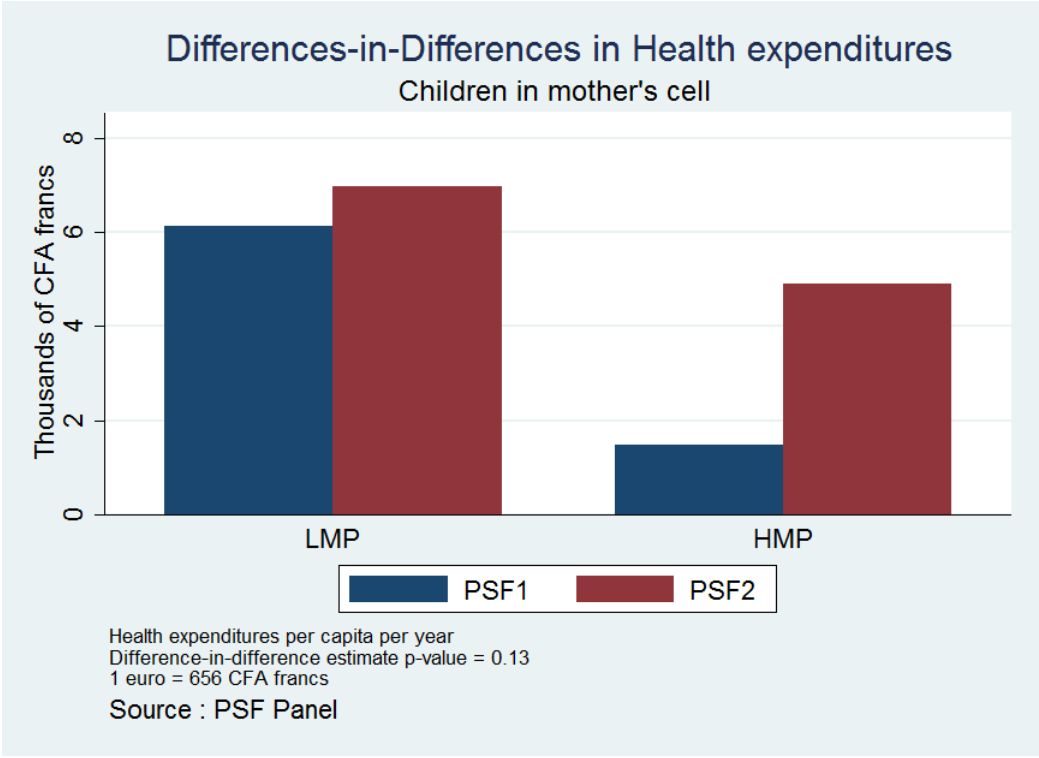


Figure 5: Changes in the % or cells with no expenditures

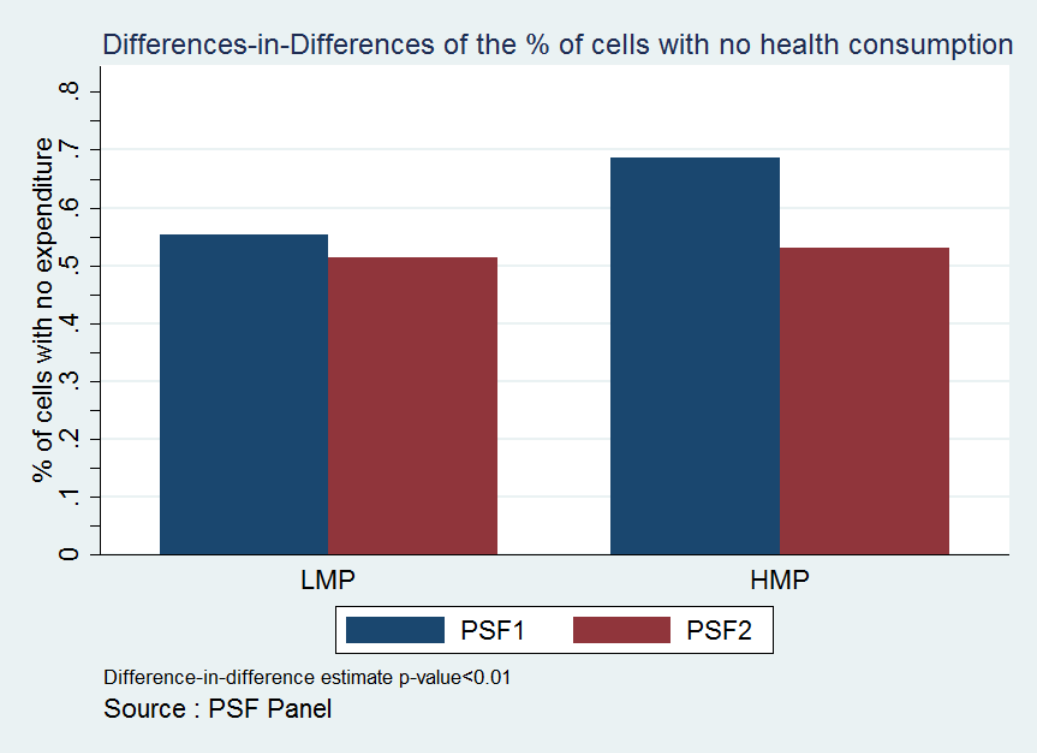
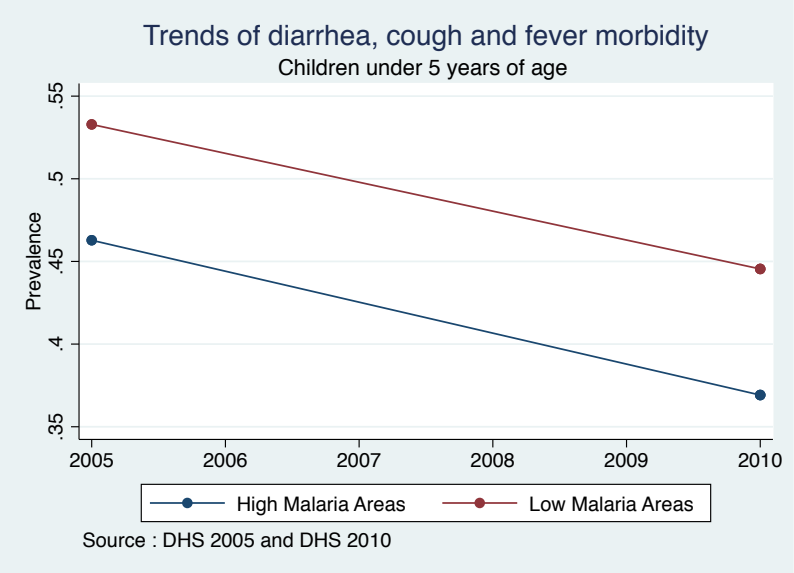


Figure 6: Trends in Morbidity - Children under 5



9.2 Tables

Table 1: Child mortality trends, by wealth

	Malarious areas Poor	*Malarious Areas Rich*	*Non-malarious areas Poor*	*Non-malarious areas Rich*
Linear trend before 2002	-0.0014	-0.0038^{***}	-0.0054^{***}	-0.0044^{***}
	(0.0015)	(0.0013)	(0.0007)	(0.0010)
Linear trend after 2002	-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 19 African countries (cf. Cogneau and Rossi (2016) for a comprehensive description of the dataset.
The table presents estimates of the linear trend in child mortality before and after the start of anti-malaria campaigns in 2002 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 ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01.

Table 2: Mean differences between high and low malaria prevalence areas

	Full sample	High prevalence	Low prevalence	pval(diff)
Plasmodium falciparum parasite rate (%) in 2000	0.18	0.36	0.09	0.00
Hh in Dakar region	0.27	0.00	0.44	0.00
Hh in other urban area	0.20	0.19	0.21	0.37
Hh in rural area	0.52	0.81	0.35	0.00
Hh size in PSF1	9.81	9.73	9.85	0.71
Hh size in PSF2	10.80	11.31	10.49	0.03
Mother in same malaria prevalence cluster btw 2 waves	0.93	0.93	0.93	0.94
Mother's age in PSF1	33.75	33.31	34.02	0.16
Cell total consumption (thousands of CFA francs) in PSF1	285.82	180.48	352.16	0.00
Cell total consumption (thousands of CFA francs) in PSF2	293.58	184.90	362.03	0.00
Average age of children in cell in PSF1	6.79	6.77	6.80	0.89
Average age of children in cell in PSF2	8.88	8.60	9.05	0.04
# of clusters	148	48	100	
# of hh in PSF1	1118	426	692	
# of hh in PSF2	1227	469	758	
# of cells	1594	616	978	

Data : PSF Panel
1 euro $\approx$ 656 CFA francs

Table 3: Health decisions when child is sick : Medical advice or treatment sought

	All diseases *(1)*	*Diarrhea* *(2)*	*Fever and Cough* *(3)*
HMP	-0.075^{***}	-0.070^{***}	-0.065^{***}
	(0.016)	(0.020)	(0.018)
Post=1	0.017	0.135^{***}	-0.008
	(0.018)	(0.024)	(0.020)
HMP × Post=1	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)
N	8466	4188	6672

Data : DHS 2005 and DHS 2010. Children under 5 years of age.
LPM. Outcome : Did you seek any medical advice or medical treatment for your child she was sick?
Sample of children sick the last two weeks
S.e. in (). * p $\leq$ 0.1, ** p $\leq$ 0.05, *** p $\leq$ 0.01.

Table 4: Changes in cell total expenditures, by low/high prevalence region

	Total expenditures
HMP	-171.682^{***}
	(14.616)
Post=1	9.871
	(13.510)
HMP × Post=1	−5.454
	(16.040)
Constant	352.164^{***}
	(12.660)
N	3188

Data :PSF Panel. Dependent variables : total expenditures for the whole cell in the last 12 months in thousands of CFA francs
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level

Table 5: Health decisions and cell consumption levels in 2006/2007

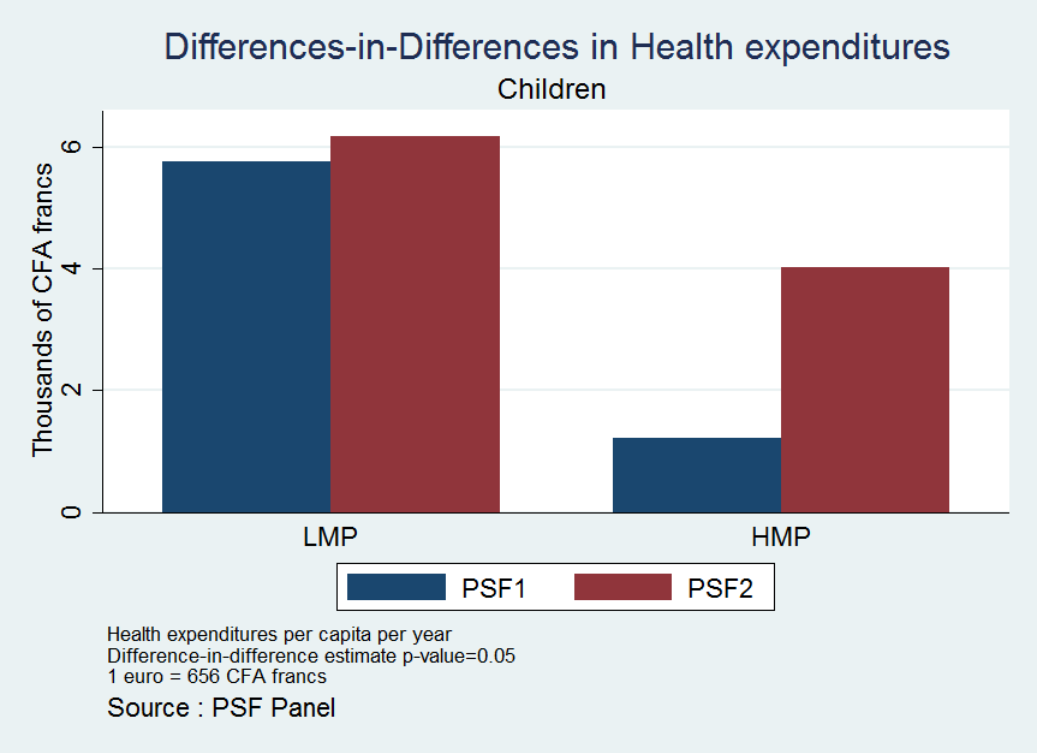
	Never Invest (1)	Switchers (2)	Always Invest (3)
Consumption level in PSF1	261.51	276.02	347.28
s.e	12.73	17.50	22.34
Observations	541	424	340

PSF Panel.
Mean of total cell consumption level in the last 12 months, in thousands of CFA Francs
P-values of the difference in means : (1)-(2) : p-value = 0.49 ; (2)-(3) : p-value =0.01 ; (1)-(3) : p-value < 0.01.

10 Appendix

10.1 Figures

Figure 7: Changes in Health expenditures



10.2 Tables

Table 6: Differences in Differences in Health expenditures by high and low malaria prevalence regions

	Health exp. levels *(1)*	*% Cells with no exp.* *(2)*
HMP	-4.676^{***}	0.132^{***}
	(1.196)	(0.025)
Post=1	0.832	-0.040^{*}
	(1.354)	(0.021)
HMP × Post=1	2.613	-0.116^{***}
	(1.713)	(0.034)
Constant	6.141^{***}	0.554^{***}
	(1.148)	(0.016)
N	3188	3188

Differences-in-differences regression
Data : PSF Panel.
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Cell has no health expenditure recorded
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level

Table 7: Differences in Differences in Health expenditures by high and low malaria prevalence regions

	Health exp. levels					
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*
HMP	-4.676^{***}	-1.872^{***}	-4.680^{***}	-4.654^{***}	-4.670^{***}	-1.868^{***}
	(1.196)	(0.711)	(1.194)	(1.200)	(1.201)	(0.709)
Post=1	0.832	0.700	1.122	0.863	1.014	0.938
	(1.354)	(1.370)	(1.482)	(1.357)	(1.518)	(1.527)
HMP × Post=1	2.613	2.690	2.559	2.616	2.575	2.624
	(1.713)	(1.712)	(1.727)	(1.712)	(1.734)	(1.728)
Constant	6.141^{***}	0.392	7.017^{***}	6.605^{***}	5.657^{***}	0.936
	(1.148)	(1.003)	(1.069)	(1.237)	(1.676)	(2.540)
Controls						
Consumption levels	*No*	*Yes*	*No*	*No*	*No*	*Yes*
Average age of kids in cell	*No*	*No*	*Yes*	*No*	*No*	*Yes*
# of kids in cell	*No*	*No*	*No*	*Yes*	*No*	*Yes*
Share of kids under 5 in cell	*No*	*No*	*No*	*No*	*Yes*	*Yes*
N	3188	3188	3188	3188	3188	3188

Differences-in-differences regression
Data : PSF Panel.
Dep var : Health expenditures per capita for children in mother's cell (thousands of CFA francs).
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level

Table 8: Differences in Differences in % of cells with no health exp, by high and low malaria prevalence regions

	% of cells with no health expenditure recorded					
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*
HMP	0.132^{***}	0.112^{***}	0.133^{***}	0.139^{***}	0.132^{***}	0.111^{***}
	(0.025)	(0.025)	(0.025)	(0.024)	(0.025)	(0.025)
Post=1	-0.040^{*}	-0.039^{*}	-0.059^{***}	-0.031	-0.055^{***}	-0.051^{**}
	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)
HMP × Post=1	-0.116^{***}	-0.117^{***}	-0.112^{***}	-0.115^{***}	-0.113^{***}	-0.111^{***}
	(0.034)	(0.034)	(0.034)	(0.034)	(0.034)	(0.034)
Constant	0.554^{***}	0.596^{***}	0.496^{***}	0.685^{***}	0.595^{***}	0.767^{***}
	(0.016)	(0.019)	(0.021)	(0.023)	(0.020)	(0.058)
Controls						
Consumption levels	*No*	*Yes*	*No*	*No*	*No*	*Yes*
Average age of kids in cell	*No*	*No*	*Yes*	*No*	*No*	*Yes*
# of kids in cell	*No*	*No*	*No*	*Yes*	*No*	*Yes*
Share of kids under 5 in cell	*No*	*No*	*No*	*No*	*Yes*	*Yes*
N	3188	3188	3188	3188	3188	3188

Differences-in-differences regression
Data : PSF Panel.
Dep var : Cell with no health expenditure recorded (thousands of CFA francs).
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level

Table 9: Differences in Differences in Health expenditures in high malaria prevalence regions, by ITN use intensity

	Health Expenditures	*% of cells with no expenditures*
High Δ in ITN use=1	0.532	−0.056
	(0.448)	(0.050)
post=1	1.215^{*}	−0.106
	(0.628)	(0.071)
High Δ in ITN use=1 × post=1	2.631^{*}	−0.058
	(1.384)	(0.077)
Constant	1.014^{***}	0.734^{***}
	(0.217)	(0.046)
N	1232	1232

Data :PSF Panel. Dependent variables : expenditures for the whole cell in the last 12 months in thousands of CFA francs
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level.

Table 10: Health expenditures variations by high and low malaria prevalence regions
"Non-migrants sample"

	Health exp. levels (1)	*% Cells with no exp.* (2)
HMP	-4.887^{***}	0.115^{***}
	(1.235)	(0.025)
Post=1	0.966	-0.049^{**}
	(1.442)	(0.022)
HMP × Post=1	2.812^{+}	-0.102^{***}
	(1.795)	(0.036)
Constant	6.066^{***}	0.570^{***}
	(1.224)	(0.016)
N	2974	2974

Differences-in-differences regression.
Data : PSF Panel. Sample : Non-migrants
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Cell has no health expenditure recorded
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level.

Table 11: Differences in Differences in health expenditures by high and low malaria prevalence regions
Rural and Urban samples

	Rural *Health exp. levels* (1)	Rural *% Cells with no exp.* (2)	Urban *Health exp. levels* (3)	Urban *% Cells with no exp.* (4)
HMP	−0.720	0.006	-7.309^{***}	0.237^{***}
	(0.595)	(0.032)	(1.793)	(0.048)
Post=1	1.820^{*}	-0.093^{***}	0.269	−0.010
	(1.049)	(0.035)	(2.041)	(0.026)
HMP × Post=1	1.791	−0.054	2.366	-0.190^{***}
	(1.632)	(0.046)	(2.244)	(0.072)
Constant	2.273^{***}	0.673^{***}	8.345^{***}	0.486^{***}
	(0.440)	(0.025)	(1.780)	(0.020)
N	1732	1732	1456	1456

Differences-in-differences regression
Data : PSF Panel.
Dep var : (1) Health expenditures per capita for children in mother's cell (thousands of CFA francs). (2) : Cell has no health expenditure recorded
Robust s.e. in (). * p ≤ 0.1, ** p ≤ 0.05, *** p ≤ 0.01. Clustered at the mother level.

Table 12: Mean differences between high and low malaria prevalence areas for attrited observations (mothers not interviewed in PSF2)

	High prevalence	Low prevalence	pval(diff)
Mother's age in PSF1	31.62	33.20	0.33
Cell total consumption (thousands of CFA francs) in PSF1	236.60	481.82	0.01
Average age of children in cell in PSF1	5.99	6.21	0.77
Health exp. for children per capita in PSF1	0.82	12.98	0.31
# of cells	50	179	

Data : PSF Panel