

Micro-loans, Insecticide-Treated Bednets and Malaria: Evidence from a Randomized Controlled Trial in Orissa (India)

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Abstract

We describe findings from the first large-scale cluster randomized controlled trial in a developing country that evaluates the uptake of a health-protecting technology, insecticide-treated bednets (ITNs), through micro-consumer loans, as compared to free distribution and control conditions. Despite a relatively high price, 52% of sample households purchased ITNs, highlighting the role of liquidity constraints in explaining earlier low adoption rates. We find mixed evidence of improvements in malaria indices. We interpret the results and their implications within the debate about cost sharing, sustainability and liquidity constraints in public health initiatives in developing countries.

JEL: I1,I3.

Key words: Malaria, Bednets, Microfinance, Public Health.

*We are very grateful to a number of colleagues, collaborators and institutions for making this study possible. In particular: BISWA and especially Khirod Chandra Malick, Dipti Pattnaik and Asish Sahoo for invaluable help in designing the micro-loan products and for facilitating access to study villages; Anup Roy, Benita Sarah Matthew, Deepak Nayak, Projjal Saha, Sudhansu Behera and all survey monitors for outstanding project supervision and implementation; Dr. Madan Mohan Pradhan, at the National Vector Borne Diseases Control Programme, Orissa, for help at various stages of the interventions; Annie Duflo and the Center for Micro Finance for invaluable help in making this study possible; Ravi and Saurabh Singhal of Biotech International for their generous donation of ITNs for the study and Binax for donating part of the materials necessary for measuring hemoglobin; Dr K S Sharma and his team at the Malaria Research Centre Field Station in Rourkela for their essential help in the validation study of the rapid diagnostic tests; the Dwight Mount Division of Parasitic Diseases, Centers for Disease Control and Prevention, for testing ITNs for insecticide levels; the Center for Micro Finance (Chennai, India), the Stanford Presidential Fund for Innovation in International Studies, the Stanford Center for International Development, the Stanford OTL Research Incentive Fund, RAND Corporation and the Duke Arts & Sciences Committee on Faculty Research for financial support; Jason Blevins, Patricia Foo and Kristin Johnson for outstanding research assistance; James Berry, Pascaline Dupas, Seema Jayachandran, Leigh Linden, Andrea Locatelli, Grant Miller, Wendy Prudhomme O'Meara, Elisa Sicuri, Duncan Thomas, Paul Wise, many participants to seminars and conferences and especially Marianne Bertrand (the Editor) and anonymous referees for constructive comments and suggestions that substantially improved the manuscript. This study was partially funded by Award Number R03AI078119 from the National Institute of Allergy and Infectious Diseases and by the Marie Curie International Incoming Fellowship FP7-PEOPLE-2011-IXF, Proposal ID 298904. The authors are solely responsible for the content of this paper. Tarozzi (corresponding author), Department of Economics and Business, Universitat Pompeu Fabra and Barcelona GSE, alessandro.tarozzi@upf.edu. Mahajan, Department of Economics, UCLA, aprajit@gmail.com. Blackburn, Stanford University School of Medicine, Division of Infectious Diseases and Geographic Medicine, blackburn@stanford.edu. Kopf, dan.kopf@gmail.com. Krishnan, shardha@gmail.com. At the time of their involvement in the project, Kopf and Krishnan were employed by the Centre for Micro Finance (CMF), part of the Institute for Financial Management and Research (IFMR, Chennai, India). Yoong, National University of Singapore and RAND Corporation, joanne_yoong@nuhs.edu.sg.

1 Introduction

A number of recent empirical studies have demonstrated that without free distribution or substantial subsidies, the adoption of highly beneficial health-protecting technologies remains very low among the poor in developing countries. Demand has been shown to be remarkably price-elastic, with even small levels of cost-sharing leading to huge declines in adoption. In Kenya, [Kremer and Miguel \(2007\)](#) found that a 20% co-pay for drugs to eliminate intestinal worms reduced uptake from 75 to 19%. In urban Zambia, [Ashraf et al. \(2010\)](#) estimated a price elasticity of -0.6 for the demand of a relatively inexpensive water disinfectant, effective for the prevention of waterborne diseases that are especially dangerous to young children. [Kremer et al. \(2009\)](#) documented only a 10% uptake when a similar product was offered at half-price in Kenya. In rural Kenya, [Cohen and Dupas \(2010\)](#) found that a remarkable 90% subsidy reduced adoption of insecticide treated bednets to 10%, relative to 99% achieved with free distribution. In rural Zambia, subsidization did not increase bednet ownership rates among the poorest households ([Agha et al. 2007](#)).

Liquidity constraints have been hypothesized to be a key reason for such low adoption rates, because several health products require investing sums that may be non-negligible for poor households ([Dupas 2012a](#)). Free provision or heavy subsidization are thus being advocated by some quarters, especially in the presence of externalities in adoption such as in the case of insecticide treated nets ([WHO 2007](#)) or de-worming drugs ([Kremer and Miguel 2007](#)).¹ However, budget constraints often impose serious limits on the ability of public health campaigns to protect all those at risk. In addition, there has been much recent debate on the sustainability of development initiatives, with advocates citing cost-recovery as a crucial criterion for evaluating poverty reduction, health and education programs ([Alam and Ahmed 2010](#), [Sarriot et al. 2011](#), [Smith 2010](#)). Cost-sharing may also help targeting subsidies towards users with higher marginal benefits, although liquidity constraints will limit such objectives if those at risk are also less able to pay.

When heavy subsidization is not possible but liquidity constraints are a key determinant of low demand, micro-loans may offer a promising option in the search for sustainable public health initiatives. This paper describes findings from the first large-scale cluster randomized controlled trial (RCT) in a developing country context that evaluates the adoption and health impacts of a health-protecting technology offered with micro-consumer loans, relative to free distribution or control conditions. Specifically, we evaluate the effectiveness of micro-loans

¹See e.g. [Hammer \(1997\)](#), [Gersovitz and Hammer \(2004, 2005\)](#) for an examination of economic approaches towards health and infectious diseases in particular.

at increasing ownership and use of insecticide treated bednets (ITNs), and ultimately at reducing the burden of malaria in areas of rural Orissa (India) where the disease is endemic.

Transmitted by *Anopheles* mosquitoes, malaria represents an enormous global health burden, with a worldwide incidence of 300-660 million cases annually, 80 million in India alone.² One third of the human population is estimated to live in areas at risk for the most severe form of malaria, caused by *Plasmodium falciparum* (Snow et al. 2005). The negative association between the disease and economic growth and the accumulation of human capital has been long recognized, although studies that convincingly document and quantify causal links are relatively recent within the economics literature.³ Numerous randomized trials have shown that with high coverage and/or high usage rates ITNs are efficacious at reducing malaria-related morbidity and mortality, as documented in the extensive survey in Lengeler (2004). However, ITN adoption in most malarious areas remains very low and public health agencies frequently have insufficient resources to provide universal ITN coverage. In such a context, a more sustainable approach focusing on cost-recovery may be desirable, but it may lead to the exclusion of vulnerable individuals who do not have access to sufficient funds.

Our field experiment was conducted in 141 villages in rural Orissa, in collaboration with BISWA (Bharat Integrated Social Welfare Agency), a micro-lender with a large presence in the state. After a baseline household survey, completed in the spring of 2007, we randomly assigned villages to three equally sized groups. A control group received no further interventions, while lender clients in a second group received at no cost a number of ITNs depending on household composition. Clients from the third group were offered contracts for the purchase of ITNs and re-treatments, using consumer loans with a one-year repayment period. The ITN offer price was *not* subsidized and included a mark-up to cover delivery and overhead costs to BISWA. The price was not negligible, corresponding approximately to 3-5 times the local daily agricultural wage.

This paper has two specific aims. First, we evaluated to what extent the offer of small loans for purchasing ITNs led to increases in ownership, even among poor households. To the best of our knowledge, this is the first large-scale cluster RCT to evaluate the efficacy of a public health program where a health-protecting technology was provided at full cost but allowing for repayment over time, as compared to both control conditions or free distribution. Fink and Masiye (2012) describe the result of a later study in Zambia, where bednets were

²Snow et al. 2005, Korenromp 2005. For a comprehensive survey of the disease, including its epidemiology, pathology and treatment see White (2009).

³See Gallup and Sachs (2001), Sachs and Malaney (2002), Malaney et al. (2004), Hong (2007a), Hong (2007b), Barreca (2010), Bleakley (2010), Cutler et al. (2010), Lucas (2010), Kitchens (2012).

offered on credit to farmers with access to agricultural loans. [Devoto et al. \(2012\)](#) study adoption of piped water contracts offered on credit in urban Morocco, but focus on how information and counseling affected loan applications open to all study subjects. We also evaluate the cost-effectiveness of micro-loans when compared to free distribution, taking into account that we observed partial repayment rates. In order to further gauge the role of liquidity constraints as a barrier to demand, we also studied uptake of ITNs offered for cash, although this intervention was conducted at a later time.

Second, we evaluated the impact of the alternative ITN delivery mechanisms on different malaria indicators. Our data include results from thousands of blood tests that allow us to estimate changes in malaria *prevalence* (the fraction of infected individuals at a given point in time) as well as in hemoglobin levels. We also study changes in malaria *incidence* (the number of illness episodes over a period of time), although these were respondent-reported. The impact of ITNs distributed free of cost on malaria indices has been studied extensively, although all but one of the 22 studies reviewed in [Lengeler \(2004\)](#) were ‘efficacy’ trials, that is, conducted under highly controlled conditions generally leading to high coverage and use rates. In contrast, our study could be seen as an ‘effectiveness’ trial, carried out on a large scale and without potentially invasive surveillance of malaria indices and ITN usage. In addition, our program did not seek universal community-level coverage but only targeted BISWA-affiliated households. This led to low coverage, a condition that may be important for public health interventions where externalities are important. Ours is also the first large-scale RCT that analyzes the impact of ITNs on malaria indices in India.⁴

The rest of the paper is organized as follows. Section 2 describes the study area, the RCT design and the data. Section 3 describes the impacts on ITN adoption and (self-reported) usage and examines the role of liquidity constraints on demand for ITN. Section 4 discusses the impacts on malaria indices as measured both through blood tests and recall data, after clarifying the features of each indicator and their inter-relationship. This section also discusses the findings in light of the epidemiological and public health literature on the impact of ITNs on malaria indices. Section 5 briefly considers cost-effectiveness of free ITN provision versus sales on credit. Finally, Section 6 summarizes and interprets the results and highlights limitations. Because of space constraints, we will refer the reader to the [Appendix](#) for a number of details and additional results.

⁴A number of studies have been conducted in Orissa and elsewhere in India but they lack an appropriate control group and/or have insufficient sample size, see [Lengeler \(2004, p. 16\)](#) for references.

2 Location, Study Design and Data

This study was carried out in 141 villages from five districts in rural Orissa, the most highly malaria-endemic state in India (Kumar et al. 2007), and conducted in collaboration with BISWA, a micro-lender with a large local presence. Study locations were selected by stratified random sampling from a list of 878 villages with BISWA presence.⁵ A pre-intervention baseline survey was completed in May-June 2007 for a random sample of 1,844 households. Within each village, 15 households were randomly selected from all those with preexisting BISWA accounts as of November 2006, regardless of whether they had an active loan at that time (all were selected if fewer than 15 were present in the BISWA rosters).

Two key malaria indicators (malaria prevalence and hemoglobin levels, Hb) were measured with rapid diagnostic tests (RDTs), and the results were immediately communicated to the subjects. These tests require very small blood samples and deliver results within minutes (see Appendix A.2 for details). Individuals targeted for blood tests included all pregnant women, children under the age of five (U5) and their mothers, and one randomly selected adult (age 15-60). The malaria RDT detects current or recent infections accurately (up to 2-4 weeks prior to the test), but does not indicate the level of parasitemia. The test can also distinguish infections due to different *Plasmodium* species, but because almost all infections in our sample were due to the most severe form of malaria (caused by *P. falciparum*), we only present pooled results. Malaria prevalence is thus a cross-sectional estimate of the fraction of tested individuals with the illness at a given point in time. Anemia, defined here as Hb levels below 11 grams per deciliter of blood, is a common health condition in developing countries and can be severely worsened by Malaria (White 2009). A significant change in anemia rates in U5 is often one of the most sensitive indicators of changes in malaria incidence (Hawley et al. 2003, ter Kuile et al. 2003). For each individual we also recorded respondent-diagnosed illness episodes during the previous six months, which allows us to construct measures of malaria *incidence* during the period. Unlike prevalence, this index is a ‘flow’ variable that measures the overall burden of disease in the study population over a period of time. In Section 4 we discuss at length the relative merit of these two indicators as well as their relationship in epidemiological models of malaria transmission.

After the completion of the baseline, the 141 villages were randomly assigned to three study groups of 47 villages each. We label the three arms (described in detail later) as “MF” (microfinance), when nets were offered for sale on credit, “Free”, when the intervention called

⁵Appendix A.1 includes the details of the sample selection, and documents how study villages were on average larger and with better amenities than the overall population of villages in the five districts.

for free distribution of ITNs, and “Control” when neither intervention was introduced. In Table 1, we report selected summary statistics from the baseline, together with tests for balance across treatment groups. The null of equality of means across arms is not rejected at standard significance levels in 20 of 23 variables, suggesting overall good inter-arm balance, although there are exceptions that we discuss below.

Average total expenditure was low, about 1.5 USD per person per day in purchasing power parity terms, and approximately 20% of households were below the official poverty line for rural Orissa (see the caption of Table 1 for details). Despite all sample households being affiliated with BISWA, more than half said they would find it difficult or impossible to borrow Rs 500, which is approximately the price of two program ITNs (see below). Two-thirds of households had at least one net, 95% of which had been purchased from the market. The mean (median) price paid was Rs 79 (60). The number of *treated* nets owned was significantly lower, ranging from 0.02 ITNs per head in control areas to about 0.05 in Free and MF villages. Despite the low ownership rates in all three arms, the null of equality is rejected at the 5% level. More than 10% of tested individuals resulted positive for malaria, while more than half were anemic.⁶ Malaria prevalence was marginally higher in treatment areas, 11.5% in MF and 12.3% in Free, versus 10.8% in Control villages, although the differences are not significant (p-value= .838). Self-reported malaria incidence was also higher in MF and Free areas (0.12 episodes per person in the previous six months) relative to Control (0.09), and in this case the null of equality is rejected at the 5% level. Estimated malaria-related health expenditures were similarly higher in treatment areas, although the null of equality is not rejected at standard levels (see table captions for details about the estimation of the malaria costs). Overall, these rates document the poor health status of the study population and suggests potentially large health gains from a reduction in the malaria burden.

In September-October 2007 we revisited the study villages and carried out a public information campaign (IC), after gathering all BISWA members in a village. The IC included a brief presentation about malaria and its transmission, the importance of ITN use, a demonstration of how to hang and use nets properly and advice on re-treatment. In treatment communities, the IC also included an explanation of the intervention to be rolled out.

In the 47 Free villages, all households with at least one BISWA member (regardless of inclusion in our baseline sample) received a number of free nets as a function of family composition, up to a maximum of four. The nets were of very good quality and significantly

⁶Malaria prevalence was similar across genders and age groups, while Hb levels vary widely by age and gender (a common finding in developing countries), see Appendix A.3 for details.

sturdier than most of the pre-existing ones. They were treated with deltamethrin, an insecticide demonstrated to be effective against *Anopheles* mosquitoes in Orissa. Nets were treated on the spot by trained personnel.⁷ Individuals were also informed that our team would return after six and twelve months to re-treat the nets at no cost.

In MF communities, ITNs were offered through micro-loan contracts and, as in Free communities, only BISWA clients were targeted. ITNs could also be purchased for cash. The micro-consumer loans were offered by BISWA separately and in addition to any other loan already outstanding. There was no movement of funds at the time of purchase: if a household decided to buy ITNs, these were delivered after being treated as described above and repayment was scheduled to be completed within one year. Field workers clarified that default on ITN loans would be treated similarly to defaults for other BISWA loans and that purchase decisions would not affect their access to regular BISWA loans beyond that determined by repayment behavior.

ITN distribution and recording of loan contracts were to be completed 2-3 days after the IC.⁸ The time interval between the IC and the purchase decision was introduced to ensure that the households had an opportunity to consider the offer carefully. A second visit was conducted approximately one month later, where ITNs were offered again with the same contracts. No ITNs were offered after this second visit.

ITNs were offered for sale with two alternative loan contracts, both at BISWA's standard interest rate, 20% per year. The two contracts allowed buyers to choose between the purchase of an ITN, or (for a higher price) a bundle which also included two re-treatments to be completed at no additional cost six and 12 months later. The price of nets ranged from 173 to 259 Rupees, depending on contract choice or net size (single or double). For perspective, at the time of the intervention daily wages for agricultural labor were around Rs 50, and one kilogram of rice cost approximately Rs 10. Our project team re-visited MF and Free villages in March-April and September-October 2008 for the re-treatment of the bednets, which was completed by study personnel in a central location within villages. Re-treatment was offered at no additional cost, except for buyers who did not choose the bundled contract in MF areas who were offered re-treatment for cash, at Rs. 15 (18) per single (double) net.⁹

A detailed post-intervention survey was conducted shortly after the second re-treatment,

⁷See Appendix A.4 for specifics about bednets, insecticide, and the treatment procedure.

⁸In reality, loan management was not carried out uniformly across the study areas by BISWA personnel.

⁹Because of space constraints, in this paper we ignore the contract choice. Tarozi et al. (2011) show that, as expected, re-treatment rates were significantly lower among buyers who choose to purchase the ITNs without the two re-treatments included in the price.

between December 2008 and April 2009. The content of the survey instrument was similar to the baseline questionnaire and again measured ITN ownership and usage, and health status. Malaria prevalence and Hb levels were measured by similar methodology to the baseline survey. A longitudinal data set was created by re-contacting all baseline households whenever possible. Additional funding also enabled us to increase the number of biomarkers collected by attempting to test all household members for malaria and Hb, rather than only specific demographic groups as at baseline.

Attrition at follow-up was limited and mostly due to temporary migration or inability to find respondents despite repeated visits. Of the 1,844 initial households, 1,768 (96%) were re-interviewed. The null of equal attrition rates among arms is not rejected at standard levels, and neither bednet ownership nor the results of the biomarkers at baseline are statistically or substantively significant predictors of attrition (see Appendix A.5 for details).

In describing the impacts of the interventions, we rely on intent-to-treat (ITT) estimates, that is, we focus on post-intervention differences in outcomes between experimental arms regardless of actual program uptake. We estimate all regressions using Ordinary Least Squares (OLS), with statistical inference robust to intra-village correlation of residuals.

3 Impacts on ITN Ownership and Usage

We first evaluate the impact of the intervention on ITN uptake, ownership and usage. In communities with free distribution, almost all sample households (96%) received at least one ITN, with an average of 2.7 nets per household, about one every two people (Table 2, columns 1 and 2). In MF villages, ITN acquisition was substantively and statistically significantly lower, with 309 of 589 sample households (52%) purchasing at least one ITN (1.2 nets per household, or one ITN every four people). We also find considerable heterogeneity in purchase rates across villages, with no uptake among sample households in five communities. Almost all buyers chose to purchase on credit, with only ten choosing to pay in cash. Despite the gap relative to free distribution, the 52% purchase rate was remarkable, given the non-trivial cost of the ITNs. The high uptake contrasts sharply with the very low cash purchase rates for health products documented among poor households in earlier studies such as [Cohen and Dupas \(2010\)](#).¹⁰ In Section 3.2.1 we discuss the findings from an additional study where we show that in a comparable set of BISWA communities demand for bednets offered only for cash was very low and highly elastic with respect to price.

¹⁰Note also that the highest offer price for long-lasting ITNs in [Cohen and Dupas \(2010\)](#) was \$1.35 (using PPP conversion rates), that is, just above 10% of the least expensive ITN offered in our intervention. At this low price, they estimate a purchase rate of approximately 40%.

Next, we assess the change in overall bednet ownership (regardless of acquisition mode or treatment status of the nets) between the baseline and the follow-up survey. Column 3 of Table 2 shows the results of a differences-in-differences (DD) model where the dependent variable is the change in the number of bednets owned by the household, and the regressors are an intercept and dummies for households in MF and Free communities. We observe an increase of 0.3 bednets per household in control areas but, consistent with the results on ITN uptake, the overall increase was three times as large in MF communities, and six times as large with free distribution.¹¹ Free distribution led to a coverage of 0.63 nets per person, which is close to the figure of two nets every three persons which has been taken to represent full coverage in some contexts (see for instance [ter Kuile et al. 2003](#)).

The increase in net ownership in intervention areas was lower than the number of nets delivered. The gap was on average 0.8 nets in Free and 0.3 nets in MF. In the latter communities, the average gap is reduced to 0.1 if we exclude two outlier villages where a number of BISWA members purchased more than 15 ITNs each for resale purposes. If we exclude these two villages, in both Free and MF areas there was a 0.2 reduction in BISWA-provided ITNs relative to the time of the intervention (results not shown). These ITNs had been sold or otherwise lost or disposed of. In MF villages (again, excluding the two outliers), we also observe a 0.1 increase in nets purchased from sources other than BISWA. Conversely, in Free villages the additional 0.6 gap is explained by a *decline* in the number of non-BISWA nets. This is consistent with the hypothesis that older, worn out nets had been disposed of and replaced by the new high-quality ITNs distributed by our program.

Overall, we find that a large majority of ITNs distributed through the program were retained. In addition, the surveyors were instructed to ask permission to see all nets that the respondent listed as being owned by the household, and the presence of 90% of the nets was confirmed in this way.

Information on bednet usage also confirmed large increases in intervention communities relative to controls. Both at baseline and follow-up, we recorded whether household members slept under a bednet the night before the interview, and whether the net had been treated in the previous six months. In control areas, the proportion of members who slept under a bednet changed from 13 to 18%, an increase likely due to the follow-up survey being completed during a period of more intense mosquito activity (column 4 of Table 2). The usage rate increased by an additional 9 percentage points (pp) in MF and 38 pp in Free

¹¹[Tarozzi et al. \(2011\)](#) also includes, for all outcomes, the results of all regressions estimated in levels using only information from the follow-up survey. Because observed characteristics were overall balanced across arms, these estimates are always very similar and we do not report them for brevity.

communities. Two interesting patterns emerge when we look separately at changes of treated and untreated nets (column 5 and 6). First, there was no increase in ITN usage rates in control areas, which signals the absence of any cross-arm contamination due to imperfect implementation of the study design or to the presence of other ITN distribution programs in the area. Second, we find again evidence that the new, good quality ITNs supplied by our program displaced non-treated nets, especially in areas with free distribution, where the fraction of members who used an untreated net decreased by 8 pp relative to Control (the decline is significant at the 1% level).¹²

Bednet usage during the previous night and the actual presence of the net in the dwelling were also recorded independently in a census of sleeping spaces. Surveyors listed all sleeping spaces used by the household (including outdoors), recorded which members slept there the previous night, asked whether the space was protected by a net, and noted down the source and price of the net and of any recent re-treatment. Surveyors asked to see all nets reported as having been used. We use these alternative data to construct a new dummy for previous-night usage of a treated net, and one for whether the net had been observed by the surveyor and recognized as an ITN distributed through our program. The results are virtually identical to the earlier ones, and while it is possible that misreporting was common to both sets of responses, the remarkable degree of consistency across sections makes it unlikely.¹³ Even though previous-night usage rates are likely a noisy indicator of consistent usage, the results discussed so far show that the intervention increased ITN adoption substantially, but that free distribution was much more successful than micro-loans at doing so.

A related question is whether the price of ITNs sold on credit generated a *screening* effect, defined (as in [Ashraf et al. 2010](#)) as higher usage rates conditional on ownership, relative to what observed with free distribution. This form of screening is often used as an argument in favor of cost-sharing. [Ashraf et al. \(2010\)](#) find that households who agreed to purchase a water purification product at higher prices were more likely to use the product, at least in the short term, while [Cohen and Dupas \(2010\)](#) cannot reject the null that women who received free ITNs were as likely to use them as others who paid subsidized but positive prices. In

¹²We also collected information on “regular” usage during the peak mosquito season. The seasonality of malaria transmission has been documented in neighboring areas ([Sahu et al. 2003](#), [Sharma et al. 2006](#)). Regular usage rates are substantially higher than previous-night usage, but the cross-arm gradient is similar. The results, omitted for brevity, are available in [Tarozzi et al. \(2011\)](#), where we also document that changes in usage were very similar between genders, but larger for younger individuals, especially in Free areas.

¹³In addition, such concordance is not simply due to all members being reported as either having or not having used nets the night before. The correlation between the two separate reports is still very high (0.87) if we use only information from households where there is intra-family variation in reported usage.

contrast, we find that while in MF areas 31% of ITNs had been used the night before, the fraction was 14 pp *higher* in Free villages, and the difference is statistically significant at any standard level, see column 8 of Table 2.

3.1 Correlates of ITN Purchases on Credit

In Table 3, we look at correlates of ITN purchases in MF villages. While these results are descriptive and do not imply causal associations between the predictors and the decision to purchase, they provide useful information on two key issues. The first is whether the sales on credit led to selection into ownership of households with relatively high expected benefits from ITNs. The second is whether purchase decisions are consistent with the presence of credit and/or liquidity constraints, which would help rationalize the high uptake of ITNs sold on credit. To analyze these points, we estimate a Linear Probability Model where the binary dependent variable is equal to one if the household purchased at least one ITN (marginal effects calculated from a probit model, not reported, are almost identical).

Variables that describe the demographic structure of the household (including presence of U5s) are not significant, either individually or jointly (p-value= 0.6276). However, we find strong associations between demand and proxy measures of perceived benefits from ITNs. First, conditional on other covariates, households where everyone used a net prior to the intervention were 21 pp *more* likely to purchase nets relative to others where no one did. This is consistent with bednets being an experience good, with past usage perhaps associated with higher perceived benefits (Dupas 2012b). Second, an increase from zero to the median monetary cost of malaria episodes in the 6 months before the interview increased demand by 9 pp ($0.019 \times 590^{1/4}$). Third, a history of any malaria-related deaths in the previous five years increase the predicted probability of purchase by 10 pp. However, deaths were rare (only nine respondents reported any) and the coefficient is not significant. Fourth, both self-reported malaria episodes and prevalence as measured by our blood tests are among the strongest predictors of purchase. Moving from a household with no self-reported malaria incidence to one where every member had been sick increases the probability of purchase by 27 pp. Similarly, an increase from 0 to 100% in the fraction of blood tests administered to the household that were positive for malaria predicts a 20 pp increase, and both coefficients are significant at the 1% level. In contrast, we find that anemia levels are not correlated with demand for ITNs, as in Cohen and Dupas (2010). This is consistent with anemia being a poor indicator of perceived marginal benefit from ITNs, perhaps because among poor households low Hb levels are often caused by a number of epidemiological and nutritional factors besides exposure to malaria (de Benoist et al. 2008).

3.2 The Role of Liquidity Constraints in Demand for ITNs

The strong association between willingness to pay for ITNs and malaria risk overall indicates that, despite the possibility of delayed payment, non-negligible ITN prices led to significant selection. This finding suggests that credit and/or liquidity constraints (that is, lack of *ability* to pay) were key factors in explaining the low ITN ownership rates observed at baseline. Several pieces of evidence support this hypothesis. First, only 10 of the 309 buyers in the sample chose the available option to purchase for cash. Second, households with lower monthly expenditures were *more* likely to purchase ITNs, despite controlling for ownership and usage of pre-existing nets: a 10% increase in per capita expenditure predicts a 1.2% decrease in the probability of purchase, with the slope significant at the 5% level. Poorer households may have found the opportunity to purchase ITNs on credit more appealing. Third, we have shown that bednets were already present in the area, although few bednets were treated and our ITNs were overall of better quality relative to those available in local markets. Hence, high purchase rates were unlikely to be merely the result of ITNs being a new product, not available outside of the intervention.

In principle, an alternative explanation for the purchases on credit was the presence of alternative investment opportunities for their cash that yielded a return higher than the BISWA interest rate (20% annually). However, in that case (and in the absence of investment ceilings) one would have expected households to be maximizing their BISWA borrowing. Although we cannot rule out this possibility completely, we find that only about 14% of households had a current BISWA loan at follow up (excluding the ITN loan). Another possibility is that the preference for purchase on credit relative to cash was due not to liquidity constraints but to buyers having present-biased preferences. A purchase on credit could have been seen as a way to start enjoying the benefits of ITNs while postponing the associated costs. However, we find that an indicator of present-biased preferences predict neither the decision to purchase nor the choice of cash vs. credit.¹⁴

To further probe the hypothesis that the relaxing of liquidity constraints were a crucial factor leading to high demand, we conducted a follow up study between February and April 2012, where bednets were offered *only* for cash to BISWA households. The presence of significantly lower levels of demand in this context would support our hypothesis. Although the different timing means that we cannot rule out the possible role of time-specific factors

¹⁴The results are available upon request from the authors. The indicator is a measure of whether the respondent exhibited “preference reversals” in a set of intertemporal choices, similarly to [Ashraf et al. \(2006\)](#). See the caption of Table 3 for additional details.

on demand, we argue that a number of key factors were likely to a priori bias the results against such a finding.

3.2.1 Design of a Follow-up Study of Cash Sales

We carried out the new intervention in 40 BISWA villages (“Cash” villages hereafter). Of these, 25 were selected at random from the 47 previous control villages, where no ITN sales had been conducted, while the additional 15 were newly sampled from our original sampling frame. The 25 previous control villages (“PC”) were included because new villages (“New”) were exposed to neither a comparably intense malaria and ITN-focused questionnaire nor to blood tests. If these factors were important in increasing demand in MF villages, the inclusion of only New villages might have biased results in favor of our hypothesis about the centrality of liquidity constraints. Again in order to avoid biasing the results towards finding low demand, the 40 sample villages were selected randomly after excluding communities where BISWA was no longer operational and/or where public health programs or NGOs had initiated bednet disbursement programs after the post-intervention survey in 2009. Due to funding constraints, a household-level survey was not completed for this follow-up study. However, data from the 2001 Census of India show that community-level characteristics in the 40 Cash villages were overall very similar to those of the other villages originally included in the study, and that they were also similar between PC and New villages, see Appendix Table A.9 for details.

In cooperation with the micro-lender, field workers identified all members of BISWA self-help groups in the 40 Cash villages. An information campaign similar to that in 2007 was then conducted, discussing malaria and bednets, and describing the sale that would take place in the following days. Each BISWA household was then provided a voucher, that is, a slip of paper with the household’s name and the price of the ITN printed on it. The vouchers were distributed to facilitate the calculation of the fraction of BISWA households purchasing nets (the total number of vouchers distributed being the denominator). Then, as in MF communities, sales were completed during two separate visits to the village scheduled in the following days. During each visit, BISWA members who wanted to purchase bednets did so by paying cash, on the spot, after returning the voucher.¹⁵

A key difficulty in generating comparability between the Cash and MF arm was that the nets sold in 2007 required periodic re-treatment with insecticide to maintain efficacy. As funding and timing issues did not allow us to schedule the re-visits 6 and 12 months after

¹⁵If the voucher had been lost, a new one was created on the spot and the purchase recorded.

the sale, we substituted the nets with OlysetTM long-lasting insecticidal bednets (LLINs). In these nets, the insecticide permethrin is incorporated into the fabric itself, so periodic re-treatment is not necessary. The use of these LLINs has been recommended for prevention of malaria by the WHO since 2001 (WHO 2001). Olyset nets have been shown to maintain their insecticidal properties even after four years in field conditions, thus guaranteeing significantly longer protection relative to the program nets delivered at the time of initial distribution. An additional advantage is that the mesh of these LLINs is wider than in most traditional bednets, so their usage in hot weather causes less discomfort because of better air circulation. Both the increased life-span and the wider mesh indicate that the LLINs were a higher quality product than the ITNs sold in the 2007 intervention.

Although these LLINs were significantly more expensive than the ITNs marketed in 2007, we priced them at a subsidized level to enhance comparability with the earlier prices in the MF intervention. Potential buyers were informed about the market price of the LLINs (it was also printed on the LLIN packaging) which was about twice as high as our offer prices. The LLINs were then either sold at the same *nominal* price as the ITNs sold in 2007, or at the same *real* price, calculated by inflating the nominal price in 2007 using a price index for rural Orissa. Randomization of prices was done at the village level so that all households in a given village were offered the LLINs at the same price. Census data show that village characteristics were overall balanced in high and low price communities, see column 3 in Appendix Table A.9.

On the one hand, the Cash arm involved the sale of better bednets at a real price either below or identical to that relevant in 2007, with even the largest price being heavily subsidized (and advertised as such). These factors would have likely increased demand relative to what we would have observed had we implemented this Cash arm at the same time as the sales on credit, biasing a comparison with sales on credit against our hypothesis. On the other hand, the different timing of the cash sales relative to the original sale on credit implies that we cannot control for confounding time-variant factors such as changes in malaria risk.

3.2.2 Demand for Bednets with Cash Sales

Vouchers were distributed to a total of 1,728 households, and 187 of these (10.8%) purchased a total of 275 LLINs (0.159 per household on average, or 1.5 among buying households), see Table 4 for details. As a reminder, when ITNs were offered on credit, we found that 52% of BISWA households purchased at least one. The difference is significant at the 1% level.¹⁶

¹⁶Taking into account that the Cash and MF samples were independent, the t-ratio can be calculated simply as $(.52 - .108)/\sqrt{.05^2 + .019^2} = 7.7$.

Demand was very similar between PC and New villages, and we cannot reject the null that the fraction of buyers was the same between the two (p-value= 0.72). We also find that while 15% of households redeemed vouchers in villages where the LLINs were sold at the lower price, demand was 50% lower when the price was increased by 20% relative to 2007 to take inflation into account, a difference that is statistically significant at the 10% level. This implies a very high elasticity of demand equal to 2.5. This is important, because it shows that households in our study areas were very sensitive to price variations when faced with cash payments, consistent with the earlier literature that documented high elasticities of demand for health products in samples of poor African households.¹⁷

One last interesting observation emerges by looking at demand in PC villages as a function of whether the household had been included earlier in the sample (rows F and G). Among the 282 households who had been included in the baseline survey, 12% purchased LLINs. The proportion was lower but similar (9%) among other households who had not been part of the baseline survey. The null of equal demand between the two groups cannot be rejected at standard levels. This is potentially important, because it suggests that the earlier exposure to the survey and the RDTs had only a marginal impact on demand. In principle, exposure to the IC, to the malaria-focused questionnaire and to the RDT results may have encouraged ITN adoption regardless of the offer of delayed repayment. In Appendix A.6 we explore this possibility more thoroughly, making use of data from two additional data sources: first, the purchasing behavior in MF villages of BISWA households that had not been randomly selected for inclusion in the baseline survey; second, information on ITN usage and attitudes towards malaria from 25 additional villages surveyed at follow-up (in 2008-09), where no IC or survey had been carried out earlier. Overall, we conclude that the IC was not a plausible confounder, while exposure to the survey and RDTs may have increased demand in MF villages but can only explain a small fraction of the relatively high uptake.

¹⁷The difference in demand between MF and Cash interventions is even more remarkable if one takes into account the way we measured take-up. In the MF arm, this was measured as a ratio where the denominator was the number of households included in the baseline survey, conducted a few months before the IC and the sale, while the numerator was the number who purchased at least one ITN. In contrast, in the Cash arm the denominator was the number of households who received a voucher during the IC. So, a BISWA household who did not attend the IC/sale and hence did not purchase any net would have been included in the denominator and counted as not buying in MF villages, but it would have been excluded from the calculation in Cash villages, thereby biasing estimated demand for cash *upwards* relative to MF. If we estimate demand by replacing the number of households who received vouchers in the denominator (1,728) with the number of BISWA households in the 40 villages listed in the rosters provided by the micro-lender (1,971), overall demand declines from 10.8% to 9.5%.

4 Malaria Indicators: Descriptions and Impacts

Next, we analyze whether the large increase in ITN ownership and usage in treatment areas was reflected in improvements in malaria indices, namely malaria prevalence and incidence and Hb levels. Before doing so, we clarify the nature, inter-relationship and measurement of the two most direct indicators of malaria burden: prevalence and incidence. This is important, both because the two indicators are not equally good measures of the direct economic burden of malaria, and because empirical findings and epidemiological models of malaria transmission suggest that they may not respond equally to public health interventions.

First, recall that in our data malaria *prevalence* measures the fraction of tested individuals with the disease at a given point in time, estimated from rapid diagnostic blood tests (RDTs). This is a key malaria index, also because it represents the frequency of individuals who may transmit the disease to others through *Anopheles* bites.¹⁸ However, in areas of intense transmission such as our study locations, individuals who test positive are frequently asymptomatic due to partial immunity acquired from repeated infections (Laishram et al. 2012).¹⁹ Accepted epidemiological models are consistent with acute episodes of malaria occurring only when a host experiences either high parasite density or in the presence of other risk factors (e.g. Ross et al. 2006). Although asymptomatic cases of malaria may not lead to significant direct costs to households, they remain of great concern for public health, because they complicate considerably attempts to eradicate or mitigate the disease.

Despite this, malaria *incidence*, that is, the total number of infections per person in the study population over a period of time, is often considered to be a more comprehensive measure of disease burden than prevalence. Measuring incidence accurately, however, requires repeated, regular re-assessments over short periods of time and is therefore expensive and invasive. Indeed, six of the 22 ITN trials reviewed in Lengeler (2004) measured only prevalence. In our study malaria incidence was estimated from detailed recall information about illness of household members during the previous six months.²⁰ We recorded all malaria as well as fever episodes that led to absence from work or school, or to consultation with health workers or hospitalization, noting all the related monetary costs as well as the number of days of work or school lost, see the caption of Table 6 for details.

¹⁸In addition, the completion of the life cycle of *Plasmodium* requires the infection of a host, so that malaria cannot spread in mosquitoes alone (White 2009).

¹⁹For instance, McMorrow et al. (2011, Fig. 1), using data from Malaria Indicator Surveys in 2007-2009, show that among children in Kenya, Mozambique, Senegal and Zambia who tested positive the ratio of asymptomatic to symptomatic ranged between 1.4 and 6.9.

²⁰Pilots suggested that longer recalls led to significant respondent fatigue.

Self-reports are likely affected by recall error and misdiagnosis that may be non-random, see [Strauss and Thomas \(1998, Section 4\)](#). [Das et al. \(2012\)](#) found that longer recall periods led to lower reported morbidity per unit of time in a sample of individuals in Delhi, India, and that the ability to recall was correlated with socio-economic status. In our sample, less than 1% of individuals were reported as having had malaria within a month of the post-intervention survey, while RDTs detected the presence of the malaria parasite in 21% of the tested individuals (see below). Errors of inclusion were also common, given that only 28 of the 63 individuals reported to have malaria tested positive with the RDT. In principle, errors of recall or diagnosis may have been correlated with treatment, with unclear implications for bias: for instance, the distribution of ITNs may have made the disease more salient, pushing respondents to over-report illnesses, or it may have led to a decrease in the perceived malaria risk, with opposite effects. Despite these limitations, in [Appendix A.7](#) we show that respondent-diagnosed recent malaria episodes were strongly correlated with the results of the RDTs, and that this association was not differential across experimental arms. Overall, this suggests that self-reported incidence was thus a valuable if imperfect indicator of past malaria episodes. In addition, one advantage of respondent-diagnosed illness episodes is that they likely identify (unlike RDT-based prevalence) illnesses that were severe enough to be recognized by the household, and thus potentially more important from the viewpoint of the economic burden they imposed on the household.

In epidemiological models, prevalence and incidence are strictly linked by a relationship that depends on frequency of infection and recovery time. However, the two indicators are distinct and they may respond differently to health interventions, even when both are accurately measured with blood tests. For instance, [Beier et al. \(1999\)](#) show that significant reductions in prevalence are usually achieved only with large reductions in the entomological inoculation rate (EIR, the number of infective bites per person/year), an indicator strictly related to incidence.²¹ Intuitively, an anti-malaria intervention may succeed in reducing the number of infective bites (the intensive margin) while barely affecting the probability of receiving *some* infective bites (the extensive margin). Indeed, substantive differences in estimated impacts of ITNs on malaria prevalence vs. incidence have been found in several earlier studies. Among the 22 ITN impact studies reviewed in detail in [Lengeler \(2004\)](#), only seven measured both prevalence and incidence of malaria, but in all those cases the protective power of ITNs was found to be larger when looking at the latter. Two studies actually found

²¹As few as 1-10 infective bites per person/year have been associated with prevalence rates ranging from about 10 to 80%, [Beier et al. \(1999, Fig. 2\)](#). In malaria-endemic areas, EIR above 100 are common. In locations close to our study districts, [Sharma et al. \(2006\)](#) documented EIR in the range of 3-114.

substantial improvements in *P falciparum* incidence while documenting *higher* prevalence in treated areas, although the increases were not significant at standard levels.

Empirical studies and epidemiological models of malaria transmission and ITN usage also suggest that the malaria burden is best reduced when a large fraction of the population has access to ITNs and when the nets are used regularly. Regular usage provides private benefits by limiting the number of infective bites, but a high ITN coverage rate can also be key, when it leads to substantial externalities achieved through declines in the number of mosquitoes (Binka et al. 1998, Hawley et al. 2003). In particular, Killeen et al. (2007) describe a rich epidemiological model calibrated using data from a number of field studies. One of their key results is that a user protected for 90% of the time is predicted to reduce the EIR by 60% relative to a non-user if no other ITNs are used around him, but the reduction becomes close to 100% if everyone else in the community is also regularly using ITNs. In the previous section we have demonstrated that ITN usage increased substantially in our study areas, especially with free distribution, although usage rates remained low, with less than half of ITNs reported in use the previous night. In addition, our ITN distribution programs only targeted BISWA households, so that even in Free villages ITN coverage remained relatively low (about 20% on average). These factors may thus have limited the impact of our interventions, especially on malaria prevalence, and especially in MF areas, where less-than-universal uptake limited ITN coverage and where we also recorded, surprisingly, usage rates lower than in Free areas, even conditional on ownership.

4.1 Results

We first look at the data based on RDTs, that is, malaria prevalence and Hb levels. As a reminder, at follow-up all members of sample households were targeted for testing of both malaria and Hb, while only a subset were at baseline. At follow-up, 75% of individuals were successfully tested, while 19% were not because they were absent during the visits and 6% refused. Both refusal and absence were balanced across experimental arms (see Appendix A.8). The ITT estimates of the program impact on RDT results are reported in Table 5, where we show results of regressions both in levels (using all tests completed at follow-up) and in DD form (for the fewer individuals tested in both surveys).

At follow-up malaria prevalence was 18.3% in control areas, 22.7% in Free and 22% in MF communities (column 1). Malaria prevalence was therefore about 20% *higher* in intervention communities, although the null of no difference between each intervention arm and control areas cannot be rejected at standard levels.²² The estimates are sufficiently precise that we

²²Given that ITN ownership and usage are higher in Free and MF villages relative to controls, these results

can also reject the null hypothesis of large reductions in malaria prevalence in intervention relative to control areas. The lower bound of the 95% confidence interval for the difference between Free and control is -0.022 , which corresponds to a 12% reduction in prevalence relative to control areas. Similarly, the corresponding lower bound for the difference between MF and controls (-0.025) would imply a 13% lower prevalence than in control villages. Several earlier RCTs evaluating the impacts of ITN adoption found reductions in prevalence substantially larger than these lower bounds (see [Lengeler 2004](#), Appendix 8 and 9), although we have discussed above that some studies found no improvements.

The higher prevalence in Free and MF areas could have been explained in part by pre-intervention differences. The figures in [Table 1](#) show that before the intervention malaria prevalence in Free and MF villages was respectively 7% and 14% higher relative to control areas, although the differences were not significant at standard levels. However, the DD estimates, which only include individuals tested both before and after the intervention, are similar to the results in levels ([Table 5](#), column 2). Relative to baseline, malaria prevalence in control areas increased from 11 to 17.3%. The overall increase in prevalence was expected, because the baseline survey was completed during the hot and dry months of spring, when malaria prevalence is lower, and the follow-up survey during winter, when malaria prevalence is generally higher in Orissa ([Sharma et al. 2006](#)). Consistent with the results in levels, the increase in prevalence was 5 pp higher in Free communities and 6 pp higher in the MF arm, although again the differences are not significant at standard levels.²³

Looking now at Hb levels, when we use all follow-up data, mean Hb levels was 11.4 g/dl in control and Free villages, and 11.5 in MF communities. The estimated impacts are therefore close to zero and not significant at standard levels (column 3). When we look at the DD estimates, we find that mean Hb increased by 0.28 g/dl in control areas, 0.32 in MF and 0.50 in Free villages.²⁴ The DD between Free and control areas is then 0.22 g/dl, or about 14% of a (baseline) standard deviation and is significant at the 5% level. The magnitude is small but not negligible. For perspective, among the nine ITN efficacy trials reviewed in [Lengeler \(2004\)](#) that measured Hb, impacts ranged from 0.2 to 1.8 g/dl, with a mean impact of 0.67 g/dl although [D'Alessandro et al. 1995](#), an effectiveness study such as ours, found

also lead to a *positive* association between malaria prevalence and ITN usage or ownership, if one estimates the relationship with instrumental variables using treatment status as instrument.

²³When we calculate mean changes in malaria prevalence within villages, we find that prevalence declined in only 11 of 47 control, 9 of 47 Free and 8 of 47 MF villages, while we observe increases in prevalence in 20 control, 27 Free and 30 MF communities, and no change in the remaining locations.

²⁴The increases in Hb, despite the higher malaria rate, was perhaps due to better nutrition at follow-up, conducted in months when our data indicate that income was seasonally higher for many households.

an impact substantively lower than in our case (0.1 g/dl).²⁵ Thomas et al. (2006) find that an iron-supplementation program that specifically aimed at reducing anemia rates increased Hb levels by 0.18 g/dl among adult males, and 0.12 g/dl among adult females.

When we look at anemia prevalence, defined as Hb < 11g/dl, we find that it was 38.4% in control areas, 39.4% in Free and 38.9% in MF villages (column 5). Anemia was thus close to identical across arms, with differences not significant at standard levels. The DD results show similar patterns (column 6). The relative improvement in mean Hb in Free villages is reflected in a 2.4 pp reduction in anemia relative to control, but in this case the DD is not significant at standard levels. We also find that the lack of improvements in malaria and anemia prevalence was common to all demographic groups (see Appendix A.9 for details).

In principle, the absence of any improvement in malaria prevalence may also have been caused by measurement error, but this is unlikely in our context. First, random misclassification of a binary dependent variable leads, by construction, to negative correlation between the error and the true value of the variable. As long as the true and the mis-measured values are positively correlated (as they likely are in our case) this leads to attenuation bias (Hausman et al. 1998, eq. 15). As prevalence tended to be *higher* in treatment areas, misclassification would more likely have led to *underestimation* of the differences. Second, at the beginning of the study, the reliability of the RDTs was successfully checked by testing a limited number of blood samples with or without malaria infection. On the other hand, during the field work RDT results were *not* confirmed with microscopy and a degree of subjectivity does exist in interpreting the results of the RDTs, which are read on a test strip located on a card where a reagent is added to the blood sample. The presence of recent infection with *Plasmodium* is signaled by the appearance of darker lines on the white strip. Although high concurrency between test readers (including non-trained ones) has been documented in clinical trials of the RDT (see Appendix A.2), a degree of subjectivity is hard to rule out completely, because the lines can sometimes be difficult to detect when parasitemia is low. In addition, if parasitemia was declining in treatment villages over the course of the study, the likelihood of fainter, harder-to-detect test lines may have increased in these areas, which would most plausibly have led to *overestimating* the reduction in prevalence.

To probe further the degree of subjectivity in our context, we carried out a small validation study in collaboration with the Malaria Research Centre (MRC) Field Station in Rourkela (Orissa). The results showed very high sensitivity (> 90%) and specificity (74 to

²⁵Several studies report the results as ‘packed cell volume’. This can be estimated by multiplying by three the Hb level expressed in grams per deciliter of blood.

85%), see Appendix [A.10](#) for details. Further, we checked whether systematic differences in the interpretation of the malaria RDT played a role in the results by re-estimating program impacts with the inclusion of tester fixed effects (see columns 7 and 8 of Table 5). The differences among experimental arms become slightly smaller, but they remain positive and not significant at standard levels.

Finally, measurement error was unlikely to be a problem for the Hb testing, which also showed mixed evidence of differential changes across experimental groups. Although erroneous testing cannot be ruled out entirely, measuring Hb simply requires reading a number from the display of a small diagnostic piece of equipment. In addition, the strong cross-sectional correlation between malaria infection status and Hb levels supports the reliability of the malaria RDTs. When we regress Hb on a dummy for a positive malaria test, the slope ($= -0.19$) is significant at the 1% level.

We also evaluate the hypothesis that malaria indices remained high due to behavioral changes that may have compensated for the benefits of ITNs. The increased availability of ITNs in Free and MF villages may have reduced the use of alternative prophylactic measures such as indoor or outdoor wall spraying with insecticide, mosquito coils, or the control of drainage pools. We tested this hypothesis using data on knowledge and practices collected during the post-intervention survey, but the differences between arms are generally small and show no systematic pattern, see Appendix [A.11](#) for details. A potentially important exception is wall spraying which, like ITNs, is widely considered an effective mean of reducing malaria risk ([World Health Organization 2007](#)). In control areas, 40% of households had the inner walls sprayed after 2007, while the proportion was 37% in Free and 30% in MF communities. The proportions who had the outer walls sprayed in the three groups were respectively 53, 48 and 44%. Although the null of equality is not rejected, the magnitude of the differences is relatively large. We then re-estimate the ITT including dummies for recent wall spraying among the regressors. Spraying is potentially endogenous, but here we are only interested in evaluating whether differences in spraying rates help explain the lack of health benefits in intervention villages. In columns 9 and 10 of Table 5 we show that this leaves the estimated impacts on malaria prevalence almost identical (the results for Hb are similar too and are available upon request). Overall, then, we find no evidence that our results are due to changes in household risk-coping behavior.

Similarly, the lack of effect on malaria or anemia prevalence cannot be explained by the presence of other ITN distribution programs, possibly sponsored by the Government or by other NGOs. First, the results on net ownership in Table 2, which showed large increases

in ITN ownership rates in treatment versus control areas, included nets from all sources. Second, we find that the number of nets received from non-BISWA sources was very small and not significantly different across all arms, see Table A.12 in the appendix for details.

An additional concern is the possibility that malaria and anemia prevalence did not improve either because the bednets had not been treated appropriately with deltamethrin (the insecticide), or because the local population of *Anopheles* mosquitoes was or became resistant to the chemical. We cannot address these concerns directly, because our data include neither systematic measurements of the insecticide concentration on the ITNs nor information on number, behavior and susceptibility to insecticide of local *Anopheles*. We argue, however, that these factors were unlikely to be central. First, all bednet treatments were conducted by our trained personnel, using appropriate procedures and chemical concentrations, and tests run on a small number of ITNs at the end of the study were consistent with adequate treatment, see Appendix A.4 for details. Second, none of a number of recent studies carried out in Orissa and Madhya Pradesh (another Indian state) point to ITNs as a plausible cause of resistance to deltamethrin, see Appendix A.12. In addition, resistance to insecticides is unlikely to develop over a relatively short period of time in a situation such as ours, where ITN use was largely limited to a study population (BISWA members) that always represented a minority of the village.

Next, we analyze our data on respondent-reported malaria incidence. Although recall error make these data less reliable than RDTs, we have argued above that (abstracting from such concerns) incidence is perhaps the best measure of disease burden. In Table 6 we show the estimated ITT program impacts on a number of self-reported malaria indicators. We only discuss the DD estimates because, unlike the RDTs, some of the self-reported outcomes were not balanced at baseline, suggesting higher malaria burden in Free and MF areas relative to Control, see Table 1. We first look at the fraction of individuals with episodes of malaria that are still ongoing or recent (within a month). These figures can be interpreted as self-reported prevalence and (as we noted before) are remarkably low, likely suggesting that most malaria cases identified by RDTs were asymptomatic.

In Control areas only 0.7% of individuals were reported as having had malaria during the last month and, consistent with the RDT-based prevalence results, the null of equality across arms cannot be rejected. In contrast, measures of incidence over the previous six months show beneficial impacts of ITNs, both in Free and in MF villages. In control areas, mean incidence at follow-up was 0.115 episodes per person over six months, an increase of 0.025 relative to baseline likely due to the seasonal pattern of the disease. However, the DD for

both Free and MF indicate a relative *decline* in incidence of about the same magnitude, and the coefficient is significant at the 5% level in Free villages, and at the 1% level in MF. The magnitude of these impacts is large relative to control conditions, with a relative risk ratio of $0.56 = (.115 - .051)/.115$, broadly consistent with the reduction in uncomplicated clinical episodes observed in earlier efficacy studies, see [Lengeler \(2004, Appendix 6,7\)](#). In addition, and given that the average household had about 5.5 members, these estimates indicate a relative decline of $2 \times 0.05 \times 5.5 = 0.55$ episodes of malaria per household per year.

An important caveat is that a fraction of these malaria cases were in fact likely misdiagnosed fever episodes. Recall that we find that RDT results and respondent-diagnosed recent malaria cases only coincided in 44% of cases, with similar rates of concurrence among experimental arms (see also [Appendix A.7](#)). The actual impact on symptomatic malaria incidence was thus likely smaller than suggested by the results in column 2 of [Table 6](#). In [Appendix A.13](#) we show that the estimates are about 40% smaller (but still significant at standard levels) if we assume that only 44% of incidence was correctly diagnosed, while also considering that a fraction of respondent-reported fever cases were likely misdiagnosed cases of symptomatic malaria. Regardless of this adjustment, the estimates are substantively large, given that at baseline the average monetary cost of a malaria episode was close to Rs 1000, about 30% of total monthly household expenditure. The estimates are also large relative to the price of our program ITNs, which cost at most Rs 259. Note also that the estimates are ITT, surely a lower bound of the average benefit for the treated.

The changes in incidence were also reflected in the costs borne by households due to self-reported malaria cases ([columns 3-7](#)). In both arms, the DD show an average reduction per household of about two days of school or work lost due to malaria, relative to an endline average of 5.8 days in Control areas. In control areas, we estimate that malaria cost Rs 863 per household during the previous six months, about 3% of total household expenditure during the same period. Relative to Control areas, free distribution of ITNs was associated with a Rs 194 lower expenditure due to malaria, while the reduction was Rs 269 in MF villages, although only the latter is significant, at the 10% level. Such reductions correspond respectively to 23% and 31% of estimates of total malaria costs over a six month period in control areas at endline.²⁶ The figures in [column 5](#) show that a large parts of these costs

²⁶The estimates in [columns 3-7](#) (as those in [column 2](#)) do not distinguish between correctly self-diagnosed malaria cases and other sickness episodes incorrectly diagnosed as malaria. We are interested in impacts on costs regardless of whether they were actually due to malaria because respondents sought treatment based on their perceptions (hence perceptions are what matters) and the ITNs may plausibly have led to improvements in overall health as well due to decreased malaria burden. Note also that the impacts on

were due to doctors and drugs. Finally, we find that the interventions were associated with a relative decline of about 0.1 in the number of malaria episodes that forced the households to incur debt or reduce consumption to cope with the necessary costs. These DD are significant at the 10% or below and are large relative to the levels in Control villages.

4.2 Interpretation and Discussion

Overall, self-reported malaria incidence rates are thus in stark contrast with prevalence as measured with RDTs, both in terms of levels and in terms of changes over time. Reporting error on malaria incidence correlated with the intervention could explain at least in part these findings, but as we described earlier substantive differences in impacts on prevalence vs. incidence are not unique to our study. Our findings are thus consistent with the hypothesis that the increase in ITN usage reduced infective bites enough to reduce the case-incidence of acute malarial episodes, but not enough to reduce the overall prevalence of malaria.

Low usage and coverage rates may have contributed to these findings. First, despite the substantive increases in ITN usage documented in Table 2, only 45% of the program ITNs were in use the night before the follow-up survey in Free areas, and about one third fewer were in use in MF villages. Second, it must be recalled that only BISWA clients received free ITNs or the offer of ITNs for sale on credit. Although BISWA had a large presence in the study area, we estimate that on average only 20% of people lived in households with at least one BISWA affiliate and thus were eligible for inclusion in the study. Even in villages where nets were distributed for free, ITN coverage was therefore low, nowhere larger than 50% and with only four villages where it surpassed 30%. With a 20% coverage and a frequency of usage equal to the 45% cross-sectional usage rate, the epidemiological model of Killeen et al. (2007) (leaving all other calibrated parameters unchanged) predicts a reduction in infective bites of about 40% for users and significantly less for non-users, see Appendix A.14 for details. Such declines, while substantive, may have been too small to be detected by measures of prevalence (Beier et al. 1999). Of course these calibrations have to be taken with caution, because the model in Killeen et al. (2007) depends on a number of parameters—such as species, number and feeding habit of *Anopheles*—that we do not observe in our data.

Our data are not ideal to study the role of community-level ITN coverage on malaria prevalence, both because the fraction of the population treated in intervention villages (BISWA households) was always small and because random assignment of treatment was

malaria *and* fever pooled together are qualitatively similar to those for malaria only but the magnitudes are larger (see Appendix Table A.14 for details).

not stratified by BISWA degree of presence in the village. Perhaps for these reasons, we find no clear association between changes in prevalence and estimates of ITN coverage achieved via free distribution, see Appendix A.15 for details. We also investigate the extent of within-village externalities, by using geo-coded data from a sub-sample of 11 villages, four Control and seven Free. We estimate a model where malaria status is regressed on the number of total and BISWA neighbors within different radii, both interacted with the Free dummy. The point estimates suggest substantial externalities at short ranges, with lower prevalence in Free villages associated with increases in the number of BISWA neighbors within 5-20 meters (a proxy for local ITN coverage). This is consistent with the existence of clusters of ITN-related protection, and with the argument that higher coverage rates may have led to declines in prevalence. However, the small sample leads to very imprecise estimates that are not significant, see Appendix A.16 for details.

To help rationalize the lack of improvement in malaria prevalence, it is also useful to compare our study design with that of the 22 ITN efficacy studies reviewed in [Lengeler \(2004\)](#). Fourteen were, like ours, clustered randomized trials, while in the remaining eight ITNs were randomly assigned within community. Among the clustered RCTs, the largest impacts of ITNs were found where community-level coverage was very high. In all trials, the number of ITNs distributed was sufficient to ensure that a majority of sleeping spaces were protected by nets in treatment communities. In addition, six of the seven clustered RCTs that measured impacts on malaria prevalence achieved close to universal coverage. The one exception is [D'Alessandro et al. \(1995\)](#), which is also the only 'effectiveness' study surveyed in [Lengeler \(2004\)](#). That is, while all other studies evaluated benefits of ITNs under ideal trial conditions ('efficacy'), this study focused on sentinel sites for the evaluation of a public health program in The Gambia. After one year, they observe substantial improvements in malaria indices among children in treated areas. However, they also show that prevalence was actually *higher* (71 vs. 45%) in treatment relative to control areas in one of the five sentinel sites, despite no evidence of resistance to insecticide. As a likely key explanation they mention "low usage of nets by children in this area" (p. 482). More evidence about the importance of high coverage is also found in [Kroeger et al. \(1999\)](#), a study carried out in Nicaragua where the fraction of individuals reported as sleeping under ITNs ranged between 5 and 70%. They find that declines in incidence were smaller in areas with lower coverage, with no improvements detected when coverage was $< 16\%$.

The studies with intra-community assignment of ITNs (and hence low community-wide coverage rates) found 40-60% declines in malaria incidence. However, as in our case, preva-

lence *increased* in treated areas in two of the studies. Moreover, in these studies usage rates were reported to be very high (70% or above), while in our context less than half of the ITNs were reported as being used the previous night.²⁷ It is possible that such high usage rates were achieved because these studies (unlike ours) involved intense monitoring of net usage and/or health outcomes, including a combinations of nightly surprise visits and frequent (sometimes daily) health checks. Such a study design could have induced behavioral responses such as increased compliance with regular ITN usage.

In sum, taking both the biomarkers and the self-reports as broadly correct, we conclude that our intervention reduced the incidence of severe malaria cases sufficiently to lead to declines in malaria-related expenditures that were large relative to control conditions. On the other hand, we also found no improvements in prevalence, and comparisons with the existing literature on ITN efficacy and malaria epidemiology suggest that this was likely the result of relatively low usage rates and population coverage.

5 Cost Effectiveness Analysis

The cost-effectiveness comparison between free distribution and sales on credit would be trivially in favor of micro-loans if repayments were complete and take-up comparable between arms, but neither condition held in reality. Together with the cost of the ITN themselves (which included delivery at the BISWA headquarters in Sambalpur), distribution expenses included costs for labor and for transporting the ITNs from Sambalpur to the villages. Based on field expenses, we estimate a transport cost of Rs. 500 per day and wages of Rs. 150 per day per worker, and about 1.5 days to cover a village. If our intervention were scaled up through a micro-finance network, these delivery costs could be lower if the delivery operations were scheduled using the MFI's existing labor and transportation resources. Insecticide treatment costs were Rs. 10-13 per net (depending upon size). Dividing the total cost thus obtained by the number of ITNs distributed in each arm lead to a cost per ITN delivered of Rs. 305 in MF villages and Rs. 225 with free provision.

Turning to revenues, recall that the price charged to buyers already covered BISWA's costs in administering the loan. However, at the time of the follow-up survey, about 1.5 years after the sale, sample households in MF communities had repaid on average 64% of ITNs, and we assume that no further payments were made afterwards. The low repayment rates were largely due to some BISWA program officers not putting effort into enforcing

²⁷Unlike our project, all these eight studies were also carried out within relatively small geographical areas, with the exception of one where the study population was spread across one district.

repayments, especially in certain districts. This was despite what was conveyed to BISWA members at the time of the sales, when they were informed that loan defaults would have been treated as for any other BISWA loan. Note that our data are not consistent with households anticipating that repayment would be scarcely enforced in some areas. This is important, because if households had anticipated such enforcement behavior, the high demand for ITNs on loan could have been a mere by-product of ‘sales’ actually perceived as free or highly subsidized distribution. However, we find a *positive* correlation between demand for ITNs and share of the loan repaid across study districts.

Overall, there are thus two key drivers of the differences in cost per ITN in the two arms. On the one hand, about four times as many ITNs were delivered in Free villages, thus lowering considerably the incidence of fixed costs per ITN distributed. On the other hand, in MF villages a substantial fraction of costs were recouped through repayments. These calculations yield a cost per ITN of about Rs. 150 in MF villages and Rs. 225 in Free villages. Sales on credit, despite repayment and fixed cost concerns, were thus considerably more cost-effective in the sense that for a fixed budget, 50% ($=100 \times [(1/150) - (1/225)]/(1/225)$) more nets can be distributed relative to free disbursal. However, given that purchase rates were well below 100%, covering the same number of households under MF would require ITNs to be distributed across more villages. For instance, the 4,000 nets that we distributed in total in the 47 Free communities would have required reaching about 200 villages if they had to be sold on credit. An important corollary is that cost effectiveness was achieved at the expense of significantly lower within-village ownership rates relative to free distribution. To the extent that externalities from mass distribution are an important source of ITN protective efficacy, cost-recovery may be suboptimal since it will likely result in lower ITN densities.

An alternative way to evaluate the two delivery schemes is to consider their relative cost of reaching “high benefit” households, defined crudely as those where any of the members had malaria at baseline. By this metric, 60% of all households were high benefit. In MF villages, such households were 17 pp more likely to purchase an ITN. These considerations would tilt cost-effectiveness further in favor of microloans, implying that the cost of reaching a high benefit household using microloans was Rs 227 and the corresponding figure with free distribution was Rs 375, that is, two-thirds higher.

6 Discussion, Limitations and Conclusions

Liquidity constraints have been hypothesized to be a key reason behind the low adoption rates of beneficial preventative health products among the poor in developing countries.

In this paper, we implement a randomized controlled trial to argue that micro-consumer loans may provide a feasible and cost-effective method to increase adoption in situations where existing markets and public health interventions have not been successful at ensuring adequate coverage of ITNs, which are one of the most efficacious malaria prevention methods. In a treatment arm composed of 47 villages in rural Orissa (India), our program succeeded in selling about 1,100 ITNs on credit to clients of a micro-lender over a few months, despite the relatively high price of the ITNs, about 3-5 times the daily agricultural wage in the study area. This increased ITN ownership substantially relative to control areas, with 52% of sample households purchasing at least one net.

These purchase rates are substantially higher than in earlier studies that found very low cash purchases of health products among the poor, despite heavy subsidization (Ashraf et al. 2010, Cohen and Dupas 2010, Kremer and Miguel 2007 and Kremer et al. 2009). Consistent with these studies, we also find that the demand for bednets in our study areas was significantly lower (11%) and highly price-elastic when households had to pay upfront in cash. However, cash sales were conducted at a later time with respect to the sales on credit, so we cannot exclude the presence of time-varying confounders. On the other hand, we have described a number of factors that were likely to bias demand for ITNs offered for cash *upwards* relative to what we observed in the sales on credit. We also provide additional evidence that liquidity constraints played a key role in explaining the high adoption rates of ITNs sold on credit, including the fact that only a handful of buyers chose to pay cash, despite the option being available at the time of the sales on credit.

We also found clear evidence of selection into purchase, with indicators of past exposure to malaria strongly associated with demand. In contrast, in a sample of women in rural Kenya, Cohen and Dupas (2010) found no correlation between low levels of Hb levels and willingness to pay for ITNs. This is possibly due to Hb being a noisy indicator of malaria exposure, and indeed we also find no association. However, an alternative hypothesis is that demand among at-risk women was reduced by positive correlation between malaria risk and liquidity constraints, that is, by a negative correlation between willingness to pay and ability to do so. In our setting, liquidity constraints were relaxed by the loan offer, and so correlates of demand were less likely to confound willingness and ability to pay.

Despite a two-thirds repayment rate of loan at the time of the follow-up survey, 1 to 1.5 years after the sales, we estimate that sales on credit reduced the estimated cost of reaching a household at risk (defined as one where any of the members had malaria at baseline) by about 50% relative to free distribution. Such considerations may be important for public health

programs that aim at maximizing the number of at-risk beneficiaries within the constraints of a given budget. In situations where funding is only sufficient to offer protection to high-risk individuals—such as pregnant women and young children—micro-loans may perhaps help in approaching universal coverage by increasing adoption among individuals—such as working adults—for whom episodes of clinical malaria may still lead to substantive economic costs. However, these factors must be weighted against the lower product coverage achievable with cost-sharing relative to free distribution, even when the product is offered on credit. This is a potentially serious drawback in the presence of externalities.

When we estimate the program impacts on malaria indices, the results are mixed. First, we find no evidence of substantial improvements in malaria or anemia *prevalence* (the fraction of the population affected by the condition) when measured from blood samples. We find an improvement (significant at the 5% level) in mean Hb in areas with free distribution, but only when we use differences-in-differences estimates. In contrast, we find substantial and statistically significant improvements in malaria *incidence* (the number of cases over a period of time) in areas where nets were either donated or sold on credit, although these results are based on recall data and were not clinically validated. Incidence was thus surely measured with error, and our data suggest that a large majority of malaria cases detected with RDTs were asymptomatic, a common finding in malaria endemic areas (such as Orissa) where repeated exposure to the disease generates partial immunity. Fever cases misdiagnosed as malaria were also likely common, although we have argued that recall data remain a useful if biased proxy of *symptomatic* malaria cases, severe enough to be recognized within the household.

The relative reduction in malaria incidence was also associated with lower health expenditures, fewer days of work or school lost due to malaria, and fewer episodes forcing the household to incur debt or lower consumption to pay for the related costs. The ITT estimates suggest an average yearly saving in malaria-related health expenditure about twice as large as the most expensive ITNs sold through our program. Given that the average impacts on the treated was likely higher, and that our high quality ITNs should have lasted at least 2-3 years, these figures suggest that liquidity constraints imposed substantial health-associated costs on the households. In addition, these figures ignore the welfare gains that arose directly from enjoying better health.

To reconcile the absence of improvements in prevalence and the substantial decline in self-diagnosed incidence, we go back to the numerous earlier field trials of ITN efficacy and to accepted epidemiological models of malaria transmission. Consistent with our results,

we find that studies that reported both malaria prevalence and incidence systematically found larger improvements using the latter measures, and some studies (like ours) actually found increases in prevalence after the introduction of ITNs in treatment areas. Substantive declines in prevalence have been argued to be unlikely to emerge without large declines in the number of infective mosquito bites. In turn, such declines are only likely to arise when ITN coverage is high and/or ITN usage is very regular. By design, our study only targeted BISWA-affiliated households, with the consequence that even with free distribution ITN coverage rates rarely surpassed 30% within the village. In addition, although a large majority of ITNs were retained by households, usage was relatively low at the time of the follow-up visits, with 45% of free ITNs reported as having being used the night before and (somewhat surprisingly) lower usage rates for nets purchased on credit.

Even taking the reduction in self-diagnosed incidence at face value, the lack of improvements in RDT-assessed prevalence remain a concern, because it suggests that the potential reservoir for infection remained unabated in study areas. This is important, because although asymptomatic cases may not lead to substantive health or economic costs for the individual affected, sub-clinical malaria can still be transmitted to others, so that it remains “a major hurdle for malaria elimination, as infected hosts serve as silent reservoirs” (Laishram et al. 2012, p. 9, see also Vinetz and Gilman 2002). Unfortunately, our study was not designed to measure entomologic indicators such as anopheline density, biting rates and behaviors. We were thus not able to assess directly any program impacts on these key channels through which ITNs exert their protective effect.

We also emphasize that low coverage and irregular usage are likely to mimic more closely the result of actual public health interventions (‘effectiveness’) than studies carried out under ideal trial conditions (‘efficacy’). From this perspective, the results of our trial should also be of relevance for the public health literature, given that almost all the results surveyed in Lengeler (2004) are “from randomized controlled trials where the intervention was deployed under highly controlled conditions, leading to high coverage and use rates. [...] While the difference between efficacy and effectiveness is likely to be small for certain medical interventions (such as vaccination or surgery), it can potentially be large for preventive interventions such as ITNs” (p. 10). Importantly, the unique features of our study design also imply that our results should *not* be interpreted as contradicting such earlier studies on the efficacy of ITNs. Our findings on the health impacts of ITNs should rather be seen as complementing the existing literature and suggest that public health interventions which only achieve the distribution of a relatively limited number of ITNs and/or do not ensure regular

usage may fail to achieve the desired effects. Much more may be needed, and efforts should include ensuring high village-wide coverage, providing incentives for regular use, and possibly adding complementary interventions such as indoor residual spraying, case management and environmental measures.

We conclude by emphasizing a number of additional factors that may limit the external validity of our results. First, although our study area comprised 141 villages from a very wide geographical area, the study population was not a representative sample of the five districts where we operated. Our study villages were selected because BISWA already had a presence there, and only BISWA clients were eligible for the intervention. Therefore, our study does not identify the impacts of introducing sales of ITNs on credit in a population with no access to BISWA's credit network. Extending sales to non-BISWA clients within our study communities could have increased the overall coverage of ITNs within the village, but our data are silent about this, and the implications on the expected repayment rates are unclear. Second, ITN adoption among sample households may have been raised beyond what achievable by micro-loans only by the information content of the information campaign, survey, and blood tests that preceded the sales. Should a micro-loan program such as ours be scaled up, at least some of these factors would be unlikely to be replicated. On the one hand, we do find evidence that the results of the blood tests (immediately divulged to the individuals or the guardian) affected demand for ITN offered on credit, and we also observe higher demand among households included in our baseline survey relative to non-sample households. On the other hand, we have argued that exposure to the information campaign was not a plausible confounder, and that being part of the baseline survey can explain at most part of the success of the micro-loan program at increasing ITN adoption.

One additional limitation is that the bulk of the study was conducted with standard ITNs, and not with the long-lasting insecticidal nets (LLINs) that are being increasingly used in many mass distribution campaigns, particularly in Africa. We chose the inclusion of ITNs for the study because LLINs were not available in the area—and to the best of our knowledge remain so—and we favored a product that was available locally in case local NGOs wanted to implement similar interventions. We only adopted LLINs when we implemented cash sales, after the conclusion of the main study, but funding constraints did not allow us to evaluate health impacts. The choice of LLINs for free distribution or sale on credit may have provided a more reliable insecticide concentration on the ITNs in the field, given that they are factory pre-treated, more wash resistant, and do not need to be re-treated every six months. In the paper we argue that the guidelines followed for the re-treatment of the

standard ITNs adopted in the main study, as well as the choice of insecticide, should have guaranteed their effectiveness. Despite this, we cannot exclude the possibility that LLINs may have led to better health impacts.

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Table 1: Baseline Summary Statistics and Randomization Tests

	(1) Control	(2) Free	(3) MF	(4) p-value	(5) s.dev.	(6) Obs.
1. Scheduled Caste/Tribe/Other Backward Castes	0.9 (0.013)	0.933 (0.013)	0.912 (0.021)	0.421	0.256	1838
2. Household size	5.5 (0.103)	5.6 (0.117)	5.3 (0.086)	0.138	2.22	1844
3. No. children U5 in household	0.499 (0.033)	0.506 (0.030)	0.487 (0.026)	0.892	0.704	1844
4. Male household head	0.952 (0.009)	0.941 (0.011)	0.932 (0.010)	0.368	0.235	1843
5. Household head has some schooling	0.72 (0.018)	0.706 (0.027)	0.714 (0.021)	0.908	0.452	1843
6. Household head has secondary education or above	0.084 (0.016)	0.075 (0.013)	0.114 (0.015)	0.123	0.287	1809
7. Expenditure per head (2007 Rs per day)†	22.3 (0.928)	21.2 (0.827)	24.2 (1.101)	0.085*	16.2	1844
8. Poor (expenditure per head < official poverty line)†‡	0.195 (0.025)	0.24 (0.031)	0.196 (0.024)	0.463	0.408	1844
9. Difficult/impossible for household to borrow Rs 500	0.529 (0.026)	0.536 (0.029)	0.529 (0.025)	0.980	0.499	1842
10. Ratio Debt/total yearly expenditure	0.47 (0.082)	0.389 (0.048)	0.400 (0.040)	0.685	1.01	1843
11. Household has at least one net	0.654 (0.030)	0.628 (0.029)	0.68 (0.023)	0.373	0.476	1844
12. Nets (per capita)†	0.287 (0.020)	0.264 (0.018)	0.311 (0.018)	0.167	0.3	1836
13. ITNs (per capita)†	0.021 (0.006)	0.046 (0.013)	0.055 (0.014)	0.027**	0.146	1831
14. Expenditure for self-diagnosed malaria last 6 months*	565 (77)	725 (72)	686 (85)	0.298	1689	1844
15. Used net last night†	0.131 (0.022)	0.116 (0.019)	0.162 (0.017)	0.195	0.295	1844
16. Used ITN last night†	0.019 (0.006)	0.022 (0.007)	0.03 (0.010)	0.617	0.134	1840
17. Use regularly nets during “mosquito season” †	0.564 (0.032)	0.512 (0.030)	0.572 (0.028)	0.304	0.453	1844
18. Price paid for bednets (2007 Rs)+	82.9 (8.2)	83.5 (8.4)	72.9 (6.6)	0.510	63	579
19. Malaria prevalence (RDT)	0.108 (0.016)	0.115 (0.018)	0.123 (0.018)	0.838	0.32	2557
20. Hemoglobin (RDT)	11.0 (0.087)	10.7 (0.096)	11.0 (0.087)	0.132	1.91	2528
21. Anemia prevalence (Hb< 11 g/dl) (RDT)	0.527 (0.024)	0.569 (0.025)	0.504 (0.020)	0.121	0.499	2528
22. Self-diagnosed malaria episodes last 6 months	0.093 (0.009)	0.124 (0.012)	0.125 (0.012)	0.045**	0.328	10062
23. Self-diagnosed malaria/fever episodes last 6 months	0.218 (0.015)	0.238 (0.015)	0.258 (0.017)	0.196	0.446	10062

Source: Data from 1844 households included in the pre-intervention household survey (April-May 2007). For each variable, columns 1-3 show the experimental arm-specific means and the corresponding standard errors (in parenthesis) adjusted for intra-village correlation. Column 4 reports p-values for a test of the null hypothesis that the means are identical across the three experimental arms, with asterisks denoting significance at the 10 (*), 5 (**), and 1% (***) level. Column 5 contains the standard deviation of the variable calculated over the whole sample and column 6 indicates the number of non-missing observations. The unit of observation of the variables is the household in rows 1-17, a bednet in row 18 and an individual in rows 19-23. The results in rows 19-21 include only information from individuals for whom RDTs were conducted. The means for variables denoted † were weighted by household size. + Mean bednet prices are estimated as arm-specific means of prices paid for bednets owned by households at baseline, imputing a zero if the net had been received free of charge. * For each malaria episode, we noted all the related monetary costs as well as the number of days of work or school lost. Health expenditures were elicited using an itemized list that included doctor fees, drugs and tests, hospitalization, surgery, costs of lodging and transportation (including those for any caretaker), lost earnings from days of lost work, and cost of non-household members hired to replace the sick at work. ‡“Poor” is a dummy equal to one if per capita monthly household expenditure is below a poverty line equal to Rs 381 = 326 × (373/319.5), where 326 is the official poverty line for rural Orissa in 2004-05, and 373 and 319.5 are the Consumer Price Index for Agricultural Laborers in May-June 2007 and July 2004-June 2005 respectively. According to the 2005 International Comparison Group Global Report, the purchasing power parity exchange rate was Rs 14.67 per 1 USD, see <http://siteresources.worldbank.org/ICPINT/Resources/icp-final-tables.pdf>.

Table 2: Bednet Acquisition and Ownership

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Intervention (Fall 2007)				Follow-up (Winter 08-09)			
Dependent variable	ITNs delivered	Any ITN Delivered	Bednets owned	Slept under bednet	Slept under ITN	Slept under untreated net	Slept under BISWA net (observed)	Fraction of BISWA ITNs used
Free (β_{Free})	2.65 (0.07)	0.96 (0.02)	1.56 (0.109)	0.38 (0.036)	0.46 (0.031)	-0.08 (0.026)	0.47 (0.030)	0.45 (0.021)
MF (β_{MF})	1.19 (0.21)	0.52 (0.05)	0.57 (0.106)	0.09 (0.034)	0.13 (0.026)	-0.04 (0.026)	0.13 (0.022)	0.31 (0.029)
Intercept (Control) (β_0)			0.30 (0.072)	0.05 (0.019)	0.003 (0.007)	0.05 (0.016)	0.002 (0.002)	
Difference: $\hat{\beta}_{Free} - \hat{\beta}_{MF}$	1.46	0.43	0.99	0.29	0.33	-0.05	0.34	0.14
p-value ($H_0 : \beta_{Free} - \beta_{MF} = 0$)	0.0000	0.0000	0.0000	0.0000	0.0000	0.1046	0.0000	0.0002
Unit of analysis	Household Level	Household Level	Household DD	Individual DD	Individual DD	Individual DD	Individual Level	Household Level
Regression type								
Observations	1199	1199	1759	7707	7647	7647	8018	891
R-squared	0.55	0.81	0.12	0.091	0.199	0.007	0.241	0.03
no. clusters	94	94	141	141	141	141	141	89
Baseline mean of dependent variable	N/A	N/A	1.58	0.13	0.02	0.11	N/A	N/A

Notes: Standard errors (in parenthesis) are robust to intra-village correlation. All regressions estimated with OLS. In columns 1, 2 and 8 the estimated model is $y = \beta_{MF}MF + \beta_{Free}Free + u$, where y is the dependent variable, and MF and $Free$ are dummies for the two experimental arms. These regressions use only observations from Free and MF villages. In columns 3 to 7, the model is $y = \beta_0 + \beta_{MF}MF + \beta_{Free}Free + u$, so that β_{MF} and β_{Free} are differences relative to controls. The row labeled 'regression type' indicates whether y is the level of the variable indicated in the column header or its change between baseline and follow-up survey. In column 7, the dependent variable is a dummy equal to one when an individual was reported as having used a treated net the night before the interview, and when the net had been observed by the surveyor and identified as one distributed through our program. In column 8, the dependent variable is the household-specific ratio between the number of BISWA ITNs in use the night before the survey and the number of BISWA ITNs delivered to the household during the intervention. This last regression is thus only estimated including households that received at least one ITN.

Table 3: Correlates of ITN purchase

Dependent variable: at least one ITN purchased		
Log(monthly total expenditure per head)	-0.116	(0.053)**
Debt towards BISWA (per head, quartic root)	-0.005	(0.009)
Cost of malaria episodes last 6 months (per capita, quartic root) ¹	0.019	(0.011)*
% members who slept under net last night	0.209	(0.093)**
% members who slept under ITN last night	-0.053	(0.279)
# nets owned by household	0.007	(0.026)
# nets treated last 6 months	-0.033	(0.036)
% members using nets during peak season	-0.035	(0.079)
Any malaria-related deaths last 5 yrs	0.101	(0.141)
Expected cost of a malaria episode (quartic root) ²	0.014	(0.019)
% tested members positive for malaria	0.202	(0.080)**
% members with self-reported malaria episodes last 6 months	0.272	(0.116)**
Subjective $P(\text{malaria} \mid \text{untreated net}) - P(\text{malaria} \mid \text{ITN})$ ³	-0.066	(0.106)
Subjective $P(\text{malaria} \mid \text{no net}) - P(\text{malaria} \mid \text{ITN})$ ³	-0.140	(0.142)
Observations	513	
R-squared	0.11	

Notes: OLS estimates of a linear probability model with a binary dependent variable = 1 if the household purchased at least one ITN in fall 2007. Standard errors in parenthesis are robust to intra-village correlation. Statistical significance is indicated with * (10% level), ** (5%) and *** (1%). The regressors were measured at baseline (spring 2007). Only panel households from MF villages are included. Sample size is smaller than the 589 panel households in MF villages because 76 observations (13%) have at least one regressor missing. Also included in the model are the following regressors, none of which is significant at standard levels: intercept, age, gender and schooling of household head, household size, number of members younger than 5 years old, or 5 to 14, or older than 60, measures of risk aversion and intertemporal preferences. To reduce the influence of outliers among regressors measured in Rupees, values are transformed into logarithms or, when zeros are present, using the quartic root, which has a shape similar to the logarithm for positive numbers (Thomas et al. 2006). Risk aversion is measured by an indicator equal to one when the respondent chose a no-risk lottery from a list of different lotteries (played with real monetary payoff), differing in the expected value and variance of the reward. We evaluated time preferences with 12 questions where the respondent had to choose between an earlier reward and a later but larger one. The regression includes a dummy equal to one when the respondent always chose the earlier reward, and a variable recording the number of “preference reversals” implicit in the choices, which arise when an individual chose a reward at date t over a larger one at date $t + s$ but preferred the later reward when the two dates were shifted by an equal time period.

¹ Includes all actual expenses for in-patient and out-patient care, drugs, transportation and lost household earnings. ² Expected total cost of a malaria episode for a working adult male, including all items listed above.

³ The probabilities were elicited by asking respondents to express the likelihood of an event by choosing an integer between zero (impossible event) and ten (certainty).

Table 4: Results of Cash Intervention

	Villages	# LLINs	Any LLIN
A. All villages	40	0.159 (0.033)	0.108 (0.019)
B. Earlier Control villages (PC)	25	0.162 (0.043)	0.103 (0.022)
C. New villages	15	0.152 (0.048)	0.119 (0.037)
D. Low price	20	0.226 (0.069)	0.149 (0.039)
E. High price	20	0.100 (0.026)	0.073 (0.019)
F. Baseline households	25	0.199 (0.061)	0.121 (0.026)
G. Non-baseline households	25	0.151 (0.046)	0.098 (0.025)
Tests (p-values)			
		$H_0: B=C$	0.8779
		$H_0: D=E$	0.0944
		$H_0: F=G$	0.4638

Source: authors' calculations from 2012 data from Cash villages. All standard errors and tests are robust to intra-village correlation of residuals. The 25 PC villages are a subset of the 47 Control villages initially included in the study, while the 15 New villages had not been selected before. 'Sample households' are households in PC villages that had been earlier selected as sample households for the 2007-2009 study. The lower prices were Rs 200 for a single LLIN and 250 for a double, while the higher prices were respectively Rs 240 and 300. The price paid by our research team to Sumitomo, the manufacturer, was about twice as large.

Table 5: Impact of Intervention on RDT-based Health Indices

	(1)		(2)		(3)		(4)		(5)		(6)		(7)		(8)		(9)		(10)	
	+ve Malaria		Hemoglobin		Anemic (Hb < 11g/dl)		Robustness Checks		Tester FE		IRS Dummies		+ve Malaria		Robustness Checks		IRS Dummies			
	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD	Follow-up	DD
Free distribution= 1	0.037 [0.030]	0.054 [0.040]	-0.033 [0.105]	0.222 [0.107]**	0.01 [0.022]	-0.024 [0.033]	0.021 [0.026]	0.038 [0.036]	0.04 [0.035]	0.062 [0.039]	0.021 [0.023]	0.038 [0.036]	0.04 [0.035]	0.038 [0.036]	0.04 [0.035]	0.062 [0.040]	0.04 [0.035]	0.062 [0.039]	0.04 [0.035]	0.062 [0.040]
Micro-loans= 1	0.044 [0.035]	0.063 [0.039]	0.023 [0.094]	0.046 [0.123]	0.005 [0.021]	0.035 [0.035]	0.023 [0.029]	0.046 [0.036]	0.005 [0.021]	0.035 [0.035]	0.023 [0.029]	0.046 [0.036]	0.035 [0.035]	0.046 [0.036]	0.035 [0.030]	0.055 [0.040]	0.035 [0.030]	0.055 [0.040]	0.035 [0.030]	0.055 [0.040]
Constant	0.183 [0.022]***	0.063 [0.028]**	11.433 [0.064]***	0.277 [0.075]***	0.384 [0.012]***	-0.111 [0.024]***	0.379 [0.043]***	0.227 [0.047]***	0.384 [0.012]***	-0.111 [0.024]***	0.379 [0.043]***	0.227 [0.047]***	0.185 [0.025]***	0.227 [0.047]***	0.185 [0.025]***	0.064 [0.031]**	0.185 [0.025]***	0.064 [0.031]**	0.064 [0.031]**	
Dummies for Spraying	No	No	No	No	No	No	No	No	No	No	No	No	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Tester Fixed Effects	No	No	No	No	No	No	No	No	No	No	No	No	Yes	Yes	No	No	No	No	No	No
Observations	7154	1897	7149	1869	7149	1869	7154	1897	7149	1869	7154	1897	7154	1897	7154	1897	7154	1897	7154	1897
No. clusters (villages)	141	141	141	141	141	141	141	141	141	141	141	141	141	141	141	141	141	141	141	141
R-squared	0.0022	0.0037	0.0001	0.0036	0.0001	0.0021	0.0467	0.0415	0.0001	0.0021	0.0467	0.0415	0.0051	0.0051	0.0051	0.0041	0.0051	0.0051	0.0041	0.0041
Free=MF (p-value)	0.833	0.8289	0.6058	0.1568	0.8474	0.0937*	0.9502	0.8200	0.8474	0.0937*	0.9502	0.8200	0.8893	0.8893	0.8893	0.8584	0.8893	0.8893	0.8584	0.8584
Free=MF=0 (p-value)	0.3538	0.228	0.8749	0.1025	0.9043	0.2437	0.6479	0.3971	0.9043	0.2437	0.6479	0.3971	0.3899	0.3899	0.3899	0.2407	0.3899	0.3899	0.2407	0.2407

Notes: Data from baseline (Spring 2007) and post-intervention household surveys (Winter 2008-09). All results are OLS estimates with individual-level observations. Standard errors (in brackets) are robust to intra-village correlation. Asterisks indicate significance at the 10 (*), 5 (**), and 1% (***) level. Estimates in columns 2, 4, 6, 8 and 10 (DD) only include tests from individuals tested both at baseline and at follow-up. In columns 7 and 8 we include fixed effects for the individuals who carried out the blood tests during the study. In columns 9 and 10 regressors also include dummies for inner walls having been sprayed in 2008/09, a similar dummy for spraying of outer walls and two dummies = 1 when information about spraying is missing for inner or outer walls respectively.

Table 6: Impact of Intervention on Self-reported Malaria Indices

	(1)	(2)	(3)	(4)		(5)	(6)	(7)
	Malaria previous month	Number of episodes	Days of work or school lost	Health expenditures		Doctors & drugs	# episodes paid for with debt	# episodes paid for with lower consumption
Free distribution= 1	-0.004 [0.004]	-0.048 [0.018]**	-1.9 [1.2]	-194 [180.1]	-86 [100.1]	-0.11 [0.05]**	-0.12 [0.06]**	
Micro-loans= 1	-0.002 [0.004]	-0.051 [0.018]**	-2.4 [1.1]**	-269.3 [143.4]*	-187.2 [76.5]**	-0.11 [0.05]**	-0.09 [0.05]*	
Constant (Control)	-0.001 [0.003]	0.025 [0.013]*	1.5 [0.8]*	238.2 [110.2]**	168.8 [54.6]**	0.07 [0.04]*	-0.18 [0.03]**	
Endline level (Control)	0.007	0.115	5.8	862.8	486.8	0.22	0.07	
Unit of observation	Individual	Individual	Household	Household	Household	Household	Household	
Observations	8684	8684	1768	1768	1768	1768	1768	
Free=MF=0 (p-value)	0.6065	0.0087***	0.0862*	0.1713	0.053*	0.0326**	0.077*	
Free=MF (p-value)	0.5873	0.8557	0.7043	0.6578	0.3105	0.942	0.6285	

Notes: Data from baseline (Spring 2007) and post-intervention household surveys (Winter 2008-09). All results are OLS estimates of difference-in-differences models. All outcomes refer to malaria episodes diagnosed as such by the respondent. Monetary values are in 2008-09 Rupees and are at the household level. In column 4, “all” health expenditures were elicited using an itemized list that included doctor fees, drugs and tests, hospitalization, surgery, costs of lodging and transportation (including those for any caretaker), lost earnings from days of lost work, and cost of non-household members hired to replace the sick at work. Standard errors (in brackets) and tests are robust to intra-village correlation. Asterisks indicate significance at the 10 (*), 5 (**), and 1% (***) level.

A Appendix - NOT FOR PUBLICATION

A.1 Selection of Sample Villages and their Representativeness within the Study Districts

The villages included in our sample were selected from a list of 878 villages where BISWA operated in 2007. These villages were spread across 318 *panchayats* (administrative unions of villages) in 26 blocks across the five districts of Bargarh, Balangir, Keonjhar, Khandhamal and Sambalpur (see Figure A.1). We selected 150 villages for the study, stratified as follows: 33 from Balangir, 48 from Bargarh, 30 from Keonjhar, 9 from Khandhamal and 30 from Sambalpur (the allocation was approximately proportional to the number of BISWA communities in each district). Villages were drawn using a pseudo-random number generator, with a selection algorithm that ensured the inclusion of a multiple of three villages from each block. Blocks where the Government of Orissa was planning to initiate free distribution of nets were excluded from the sampling frame. While the study locations were thus chosen to minimize this risk, the sampling scheme was designed to preserve the balanced structure of the sample across treatment groups in case the state Government initiated any unanticipated distribution. Data collected during the post-intervention survey show that indeed distribution of nets from the Government (or from other NGOs) was extremely limited in study areas, see Tables 2 and A.12. After the baseline survey, but before the intervention, nine of the 150 villages were found to have no actual BISWA activity and were then excluded from the study. Data from these villages are excluded from the analysis.

In Table A.7, we evaluate the characteristics of communities in our sample relative to other communities in the five study districts, by using data from the 2001 Census of India on a broad range of village-level characteristics. Overall, the five study districts included a population of 8,991 villages. Although the data used in this paper have been collected from 2007 onwards, the time gap relative to the 2001 census is short enough that a comparison between sample and non-sample villages should be informative.

The results show that the null hypothesis of equality of means between sample and non-sample villages is strongly rejected for most village characteristics (column 6). Sample villages are relatively large (both in terms of area and population), with mean total population more than twice as large as in non-sample villages. Sample villages also appear to be closer to towns, although not to a large extent. Mean distance from the nearest town is 35 kilometers among non-sample villages and 1-10 kilometers less in sample villages. Amenities are overall significantly better in sample villages as reflected, for instance, in the higher proportion of villages with schools, health centers, a post office, a telephone connection and electricity. Interestingly, sample villages are also characterized by significantly larger fractions of land devoted to rice cultivation. This may have implication on malaria prevalence, because rice fields are often an ideal breeding ground for larvae of *Anopheles* mosquitoes.

We also test the null hypothesis that village characteristics are on average equal in the three experimental arms (column 7). This is useful, because the randomization tests in Table 1 only evaluated balance in household-level characteristics among villages included at baseline. In a list of 26 variables, the test of equality across groups is only rejected, at the 10% level, for the presence of a middle school in the village.

A.2 Details of Blood Tests

The RDTs were conducted using fingerprick samples of less than 0.5 ml of blood for each test. Malaria prevalence was determined using the Binax Now malaria RDT. This test is well validated in comparison to blood smears for the diagnosis of malaria. The RDT detects both current and recent infections, up to 2-4 weeks prior to the test. The result of the RDT is read on a test strip, located on a card, where a reagent is added to the blood sample. Recent infection is detected when the presence of *Plasmodium* antigens in the blood (histidine-rich protein 2, or HRP2) is signaled by the appearance of darker lines on the white strip. High concurrency between test readers (including non-trained ones) has been documented in clinical trials of the RDT (Khairnar et al. 2009). The test does not indicate the level of parasitemia, and only delivers a positive/negative result for malaria infection, besides showing whether that infection is due to *P. falciparum*, to one of the other *Plasmodium* species, or to both (Moody 2002, Farcas et al. 2003, van den Broek et al. 2006, Khairnar et al. 2009). The test has been shown to have both good *specificity* and *sensitivity*. Both these concepts are defined assuming that the “null hypothesis” of the test is that the individual does not have malaria. The specificity is calculated as the fraction of negative cases correctly diagnosed as such (that is, it is equal to one minus the probability of a Type-I error). The sensitivity is the fraction of positive cases correctly diagnosed as such (that is, one minus the probability of a Type-II error).

Hemoglobin levels were tested with the HemoCue 201 Hb analyzer, a portable, accurate system for measuring Hb. The test, like the one used to detect malaria prevalence, requires less than 0.5 ml of blood and delivered results in approximately 15 minutes.

A.3 Gender and Age variation in Malaria and Anemia Rates at Baseline

In Figure A.2 we show malaria and anemia prevalence by gender and age group. Women were 3 pp more likely to test positive for the parasite, and the difference is significant at the 5% level. There is overall little variation in prevalence by age group, although when we disaggregate the data into single-year age bins we find that prevalence follows an inverted U-shape pattern with respect to age (results not shown).²⁸ Such age patterns are commonly observed in malarious areas, because very young children have initially some immune protection from the mother (and are more often protected by bednets, when available) although such immunity is gradually lost and subsequently replaced by their own semi-immunity acquired through repeated exposure to the disease, so that malaria prevalence usually peaks for children of age 2-10 years (see Smith et al. 2007 for a review of the evidence). Sharma et al. (2006) found similar non-monotone age gradients in incidence and prevalence in Sundargarh, a district of Orissa that shares borders with two of our study districts.

There was substantial variation in anemia rates by gender and age. Approximately 80% of tested U5, of either gender, were anemic. Anemia rates declined significantly among adults aged 15 to 45, but prevalence remained extremely high (60%) among women, while it was less than 12% among men. Prevalence increased again among older adults, where it

²⁸The same inverted U-shaped patterns was also found at follow-up, results not shown.

characterized about three-quarters of women and one quarter of men. Similar patterns for anemia for different ages and genders are common in developing countries (see for instance [Thomas et al. 2006](#)), and are also present in data from Orissa collected as part of the Indian National Family and Health Survey in 2004-05, which showed an anemia prevalence of 65% among U5, 34% among women 15-49 and only 8% among men in the same age group.

A.4 Details of Bednets and Treatment with Insecticide

The nets were of uniform quality, composed of white polyester multifilament, mesh size 156, and 75 denier. They had a bottom reinforcement of 28 cm, with single nets measuring 180×150×100 cm and double nets measuring 180×150×160 cm. A total of 6,750 single and 3,250 double nets were supplied by Biotech International Limited, who generously donated 5,000 single and 2,500 double nets.

The bednets were treated on the spot at the time of delivery by trained personnel, following rules recommended in [World Health Organization 2002](#), using K-Othrine flow, which contains deltamethrin, a highly effective pyrethroid. The subsequent re-treatments after six and 12 months were done similarly by our trained collaborators, using the same guidelines. While wearing gloves, the field worker dipped the washed net into a bucket where water had been mixed with the appropriate quantity of insecticide. After being soaked for a few minutes, the net was removed from the bucket and was laid flat on a plastic sheet or mat in the shade to dry. The concentration of the insecticide was determined based on the manufacturer's instructions: 10 ml of insecticide to 500 ml of water for single nets and 15 ml-750 ml for double nets. The chemical concentration made re-treatment optimal after six months.

The study design did not incorporate the systematic use of 'bioassays', that is, procedures to test rigorously the insecticidal power of treated bednets. However, at the conclusion of the study, samples from four ITNs gathered from Free villages were tested through gas chromatographic analysis, and two of the nets still had concentrations of deltamethrin around the concentrations recommended by the WHO (15-25 mg/m²), while the other two bednets had lower concentrations. Although the number of ITNs tested is obviously very small, the results do not signal obvious shortcomings with the re-treatment operations, given that the bednets had been last re-treated 6-7 months earlier, and that it is not unexpected to find low insecticide concentrations six months after re-treatment (particularly if the ITN has been washed multiple times).

Pyrethroids have been widely used for bednet impregnation with encouraging evidence about the lack of side-effects on human health ([World Health Organization 2005](#)). In Orissa, synthetic pyrethroids have been in use since 1999, and tests performed in 2002-03 in several districts (including our study districts Balangir, Khandhamal and Keonjhar) showed high rates of susceptibility to deltamethrin of *Anopheles culicifacies* and *A. fluviatilis*, the two most common malaria vectors in the state ([Sharma et al. 2004](#)). The insecticidal efficacy of deltamethrin compound has also been confirmed in Sundargarh, which borders the study district Sambalpur ([Yadav et al. 2001](#), [Sharma et al. 2006](#)).

A.5 Attrition

At follow-up, the survey team attempted to re-contact all households included in the baseline survey. The survey protocol called for at least three attempts, although a handful of households were re-contacted after 4 or 5 visits. Refusals accounted for only 13 of 76 lost households. As a result, attrition was limited, and of the 1,844 initial households, 1,768 (96%) were re-interviewed. Attrition was 5% in MF and control villages and 3% in Free communities (see Table A.8, column 2). The null of equal attrition rates among arms is not rejected at standard levels, regardless of whether we use individual or joint tests. There was little correlation between attrition and household characteristics at baseline, including RDT results and bednet ownership and usage (columns 3 and 4). The only regression coefficients that are individually statistically significant indicate that households with an older and better educated head were less likely to exit the panel. On the other hand, we cannot reject the joint null that all the included slopes are equal to zero (p-value=0.14).

We also investigated whether significant changes in household composition took place between the baseline and the follow-up survey, as well as whether such changes were balanced across experimental arms. This was potentially important for two reasons. First, changes in availability of ITNs may have arisen from changes in the number and age of household members (for instance, young children often share a sleeping space with their parents). Second, malaria and anemia prevalence at baseline differed across age and gender groups (see Figure A.2), so that changes in the demographic structure of the household may have confounded aggregate changes in such health measures calculated over all household members. We looked at both entry into or exit from panel households and to changes in the relative weight of different demographic groups. This analysis was possible because enumerators filled a complete household roster both at baseline and at follow-up, so that we can separately identify new members as well as individuals who left the household because of death or relocation. We find that these factors did not plausibly drive any of the results in the paper. We omit the detailed analysis for brevity but the interested reader can find it in the appendix of [Tarozzi et al. \(2011\)](#).

A.6 The Information Campaign and Household Survey as Possible Confounders

In principle, the relatively high ITN adoption rates observed with micro-loans may have been explained at least in part by the information campaign (IC) and household survey *cum* RDTs that preceded the sales. These factors may have made the malaria problem more salient, leading to high demand regardless of the possibility to pay over time rather than in cash. There is indeed growing awareness within field-based development economics that surveys may themselves constitute ‘interventions’, see e.g. [Zwane et al. \(2011\)](#). In this section we argue that although such confounders likely played a role, they cannot plausibly explain more than a fraction of the high demand observed with micro-loans.

As a first point, we note that confounders were also present in the recent seminal studies that documented very steep demand curves among poor populations in developing countries. ITN sales in [Cohen and Dupas \(2010\)](#) took place at ante-natal visits, during which the importance of ITNs was discussed and hemoglobin levels were measured (p. 14). In [Ashraf](#)

et al. (2010), the baseline survey also included a number of questions on water use practices and Clorin adoption, as well as measurements of the concentration of chlorine in households' drinking water supply (pp. 2389-2391). In this experiment, the water disinfectant Clorin was sold during door-to-door marketing visits. The de-worming project studied in Kremer and Miguel (2007) was carried out with teacher training, teacher and NGO-led school lessons, and a number of classroom educational materials (pp 1013-1015). In addition, in that study the huge drop in demand for drugs observed after the introduction of cost-sharing was also observed in areas where pupils had been tested for intestinal worms infections and had been part of the de-worming campaign. From this perspective, our study design was comparable to that of these earlier studies and we show that, if anything, it could perhaps be singled out for its unusual ability to study the impact of such behavioral components.

A.6.1 The IC as a possible confounder

We first discuss the IC, which we argue was not a plausible key confounder. First, the IC was a simple one-time presentation about malaria, the means by which it is transmitted and the importance and rationale for ITN use, a demonstration of how to hang nets properly, and advice on proper use and re-treatment. Such presentation usually lasted less than one hour, and a large majority of households were already familiar with the IC content, with the major exception of the importance of treating bednets regularly. For instance, at the time of the baseline survey, 96% of respondents stated (un-prompted) that malaria was transmitted by mosquitoes, while 95% stated that bednets can prevent the disease (although less than 3% explicitly mentioned 'ITNs' rather than 'bednets'). Second, the IC conducted before the sales on credit in 2007 and the one before the cash sales in 2011 were very similar, and yet the resulting demand was significantly different. Third, we demonstrated that in control areas there was virtually no change in ITN usage between baseline and follow-up (Table 2, column 5). During the same period there was only a small increase (0.3 bednets per households) in the number of bednets owned, suggesting that the IC did not change behavior or perceptions of malaria risk substantively. Fourth, additional evidence comes from a household survey conducted at the same time as the follow-up survey—in Winter of 2008-09—in 25 villages that had not been part of the initial study.

These 25 villages were added specifically to allow for the separate identification of any impact of the IC and/or of the survey itself on behavior. These 25 'follow-up only' villages ('FUO' hereafter) were selected from the same randomly sorted lists used for the selection of the communities at baseline. In other words, we did not complete a new randomization, but we selected the "next 25 villages" from the same randomization done in 2007. The similarity of the new village relative to those included since baseline was confirmed by comparing the village characteristics included in Table A.7 (measured during the 2001 Census) between the 25 FUO villages and the 141 study villages where the baseline survey had been conducted. The null of equality is rejected for only three of the 26 characteristics (results available upon request). In each FUO village, 15 households were selected regardless of BISWA affiliation, using simple random sampling from publicly available census lists formed in 2002 as part of the 'Below Poverty Line census' by the Government of Orissa. Because BISWA had a strong presence in the study areas, the sample ended up including BISWA households in almost all villages (21/25).

When we compare sample households in Control areas to BISWA households in FUIO villages, we find that the number of bednets was very close between the two groups, and the null of equality cannot be rejected at standard levels: the mean was 0.36 per person in Control and 0.32 in FUIO, and the p-value for the test of equality is 0.3248. Consistent with this result, the survey-elicited subjective probability of someone falling ill with malaria within a year when always sleeping under an ITN was 0.16 in both sub-samples.²⁹ Overall, then, the data do not support the hypothesis that the IC affected behavior or perceptions about malaria substantively.

A.6.2 The baseline survey and RDTs as possible confounders

The baseline survey included a long list of questions about malaria and bednets. In addition, the results of the RDTs were available on the spot, a few minutes after the blood sample was taken, and individuals were immediately informed about the outcome of the test. These factors may have made the disease more salient, possibly increasing the willingness to pay for ITNs regardless of the possibility being offered to delay payment. Indeed we have shown that demand was significantly higher among households where at least one member tested positive to the blood test. We argue, however, that these factors cannot plausibly explain more than a fraction of relatively high demand for ITNs on credit when compared with earlier studies that found very little demand for health-protecting technologies when these were not offered for free.

First, we have discussed before how comparable confounders (including health tests) were also present in earlier studies that found very low demand for health products. In principle, such confounders may have been more important in our empirical context, but it is not clear why this should be the case.

Second, we have shown that, by comparing outcomes in Control areas with those of BISWA households in FUIO villages, we found no evidence that the joint impact of the IC, the baseline survey and RDTs increased bednet ownership or changed perceptions about the effectiveness of ITNs. Even so, we cannot rule out the possibility that demand would have been higher in the New villages in the Cash arm if we had filled the same questionnaire and conducted the same RDTs in these communities (these elements could not be added to the supplemental arm due to time and funding constraints). In PC villages, however, both potential confounders had been present, albeit more than four years prior to the Cash intervention. As we pointed out earlier, there is no difference in ITN adoption between PC and New villages which at least suggests that the surveys and RDTs had no longer term effects on take-up. In addition, within PC villages, demand is very similar (and low) when we directly compare households who had been exposed to the survey and RDTs, and others who had not (see rows F and G of Table 4). Recall also that attrition between baseline and follow-up was very limited, so almost all sample households in PC villages had been exposed to a lengthy questionnaire and RDTs both at baseline, in 2007, and at follow-up, in 2008-09.

To probe this issue further, we can use data about ITN purchases in MF villages among BISWA households *not* included in the pre-intervention survey, among whom biomarkers

²⁹These subjective probabilities were elicited by asking respondents to place a number of marbles ranging from 0 to 10 into a cup, with the number increasing in the probability of the event taking place in the future. Similar methodologies have been adopted in several studies, see [Delavande et al. \(2010\)](#) for a review.

were not collected. At the time of the MF sales, in 2007, surveyors recorded the number and type of ITNs purchased by all BISWA members, regardless of their inclusion in our sample. Our data do not include the total number of ‘BISWA households’ in study villages, but this number can be estimated from the lists of BISWA *members* supplied by the micro-lender at the beginning of the study. The latter figure is not the correct one to be used for the estimation of demand among non-sample households, for two reasons. First, some households had more than one member affiliated to BISWA (on average 1.11). Second, a fraction of individuals listed as BISWA members were found not to be such during the field work, or had migrated, or were otherwise excluded from the study population. In this way, we estimate that every 100 members listed by BISWA corresponded to about 79 BISWA households. Let n and n_s denote respectively the total number of buyers in MF villages and the number of buyers among sample households. Let also m denote the initial number of BISWA members provided by the micro-lender, and let m_s denote the number of baseline sample households in the same villages. We thus calculate demand among non-sample BISWA households as $(n - n_s)/(0.79m - m_s) = 0.28$. Uptake was then about twice as large as that observed among BISWA members who were offered LLINs for cash at the same nominal price (.149, see Table 4), and about four times as large as that observed when the price was kept constant in real terms (.073). In addition, as described earlier, these figures likely attenuate the differences in demand between Cash and MF, because the voucher system implies that a BISWA member who was not present during the voucher distribution would not be counted in the demand estimation, rather than being counted as not having purchased.

Another key factor points to the fact that the 28% take up rate among ‘non-sample’ BISWA households in MF communities is artificially biased downwards relative to demand among sample households. That is, field reports indicate that more effort was put into ensuring attendance of sales meetings for sample relative to non-sample households. In fact, during the first sale session, 78% of baseline households attended the sale, while only 56% did among non-baseline households. Similarly, during the second session, conducted 1-2 weeks afterwards, attendance rates were 62 and 40% for the former and latter group respectively. Of course, attendance itself may have been influenced by the inclusion in the baseline.

To summarize: we argue that while the IC and the baseline surveys may have played a role in increasing take-up, the effects are not sufficient to explain the overall take-up rates.

A.7 Respondent-reported Malaria Incidence *versus* RDT Results

As we mention in Section 4, our data on malaria incidence are derived from respondent reports and not from blood tests. Such reports may be noisy indicators of actual incidence and may also suffer from bias potentially differential across experimental arms. For instance, the distribution of ITNs may have made the disease more salient, pushing respondents to over-report illnesses or it may have led to a decrease in the perceived malaria risk, with opposite effects on program impacts. In this section we provide evidence in support of the view that, despite these concerns, incidence data in our data set were a valuable source of information on malaria burden.

First, note that reported incidence can be validated against the RDTs only for very recent malaria episodes, because the RDTs we used in the field can only detect malaria episodes that are still ongoing or that took place no more than 2-4 weeks earlier (see Appendix A.2).

Let the binary variable $S_i = 1$ if individual i was reported as having had malaria in the month preceding the survey, and let $M_i = 1$ if the individual tested positive for malaria when tested with a RDT. In our post-intervention sample, there is a total of 63 individuals for whom $S_i = 1$ and for whom we observe M_i . Among these 63 individuals, 28 (44%, 95% C.I. 0.32-0.57) also have $M_i = 1$. As we discuss in the paper, most malaria cases detected by the RDTs were apparently asymptomatic and thus not mentioned by respondents, but despite this the self-reported information about recent malaria incidence is strongly correlated with the RDTs. To show this we estimate with OLS the following model, using all individuals for which M_i and S_i are non-missing

$$S_i = \beta_0 + \beta_M M_i + u_i.$$

The estimated intercept is $\hat{\beta}_0 = 0.006$ while $\hat{\beta}_M = 0.012$ and is significant at the 1% level (p-value= 0.006, adjusted for clustering at the village level, $n = 7, 153$). In other words, self-reported recent incidence was three times as large for individuals who tested positive relative to others who did not.

That respondents were able to recognize symptomatic malaria episodes is also confirmed by the fact that the results are very different if we estimate a regression such as the one above using as dependent variable a dummy = 1 if the individual was only reported as having had ‘fever’ during the last month. In this case, the intercept is 0.03 while $\hat{\beta}_M = 0.002$ and is not significant at any standard level (p-value= 0.715, adjusted for clustering at the village level, $n = 7, 153$).

Another key observation is that the link between S_i and M_i does not appear to be differential across experimental arms, so there is no compelling evidence that the intervention changed perceptions about malaria incidence conditional on actual malaria infection. The fractions $\hat{P}(M_i = 1 | S_i = 1)$ are 42% in Control areas (8/19), 45% in Free (10/22) and 45% in MF (10/22). The fractions are thus almost identical, and the null of equality cannot be rejected (p-value= 0.9724 for the joint null of equality. The individual differences are also not significant).

A.8 Post-intervention RDT Success Rates

In the post-intervention survey, all members of households re-contacted after the baseline were targeted for blood tests. Our testers were able to successfully test 75% of members in panel households, while 19% could not be tested because they were not present at the time of the visits and only 6% because consent was not given, see columns 1 and 4 in Table A.10. The figures in columns 2 and 5 show that absence and refusal were almost identical across experimental arms. Conversely, we find differences in testing success across different age groups (columns 3 and 6). Almost one third of adult males (15-45, the omitted category in the regressions) could not be tested because of absence during the visits, probably because they were more likely to be off to work. Testing rates among all other demographic groups were substantively and statistically significantly higher, especially among U5 of either gender and among women 15 years old and above. For these groups, testing rates were close to 90%. The testing rates are very close between boys and girls, and the null of equality between genders cannot be rejected for both U5s and 5 to 15 year old children. Refusal rates were

highest among women over 45 (8%) and girls U5 (9%). Refusal rates were 3 pp lower among U5 boys relative to girls but the null of equality between genders cannot be rejected at standard significance levels.

A.9 Changes in Malaria Indices by Demographic Group

Was the lack of health benefits shared by all demographic groups? The bars in Figure A.3 show malaria and anemia prevalence for each experimental arm by gender and age group, together with 95% confidence intervals.

Among adult males (age 15 or above), malaria prevalence was $\sim 15\%$ and almost identical across arms (panel A). Among U5s, prevalence was 11% in control villages but about twice as large in intervention communities: 18.4% in Free and 19.8% in MF villages. However, the estimates are imprecise, and the difference relative to control is not significant at standard levels, although the p-values are relatively small (below 0.2). Details of the test statistics are available upon request from the authors. Prevalence among males is highest among 5-14 boys, where in each arm it is ~ 15 pp higher than for younger children, so that the differences among groups are almost identical in these two age groups.

These patterns change when we look at females (panel B), although again differences between arms are never significant at standard levels. Among females, we observe almost identical prevalence across arms among the youngest girls ($\sim 15\%$) and higher prevalence in intervention villages in older age groups. In each experimental arm, the highest prevalence is observed among females of age 5 to 59.

Overall, these results document remarkable differences in malaria prevalence across subgroups, but these differences are largely concentrated between genders or across age groups rather than across experimental arms. Note also that, consistent with the baseline results, we do not observe prevalence rates monotonically declining with age. The relatively low prevalence among U5s is actually driven by very low rates among children less than two years old (results not in the figure). Of a total of 263 children in this latter age group, only 12 (4.6%) tested positive, while prevalence jumps to 23.3% among the 412 two to four years old tested. Overall, in our sample malaria prevalence peaks among 5 to 10 years old, and then gradually declines with age. These patterns are similar among experimental arms.

Consistent with the baseline results, the results for anemia (panels C and D) show large systematic gaps across gender-age groups. In particular, these results confirm the U-shape of anemia prevalence with respect to age for both genders, as well as the significantly higher anemia rates among females 5 and older relative to males of the same age. Like for malaria, however, the differences in anemia prevalence between arms are small and never significant at standard levels.

A.10 RDT Validation Study

In July 2009, we carried out a small validation study after the conclusion of the follow-up survey in collaboration with the Malaria Research Centre (MRC) Field Station in Rourkela (Orissa), which confirmed the accuracy of the RDTs. A total of 205 blood samples were independently collected from the MRC team from individuals with malaria symptoms from three villages. The RDT cards were interpreted by three different blinded readers, including

two of the testers who were part of the field team during our study, and the most senior survey monitor in our research team. These results were then compared with thick and thin blood smears read with microscopy by the MRC team for the same samples, with the smear result accepted as the correct infection status. The results showed very high sensitivity ($> 90\%$ for each of the three readers, see Table A.11 for details). The fraction of correctly identified negatives (specificity) ranged from 74 to 85%.

The lower specificity (higher prevalence) measured by the RDTs relative to microscopy was not surprising, given that these tests may detect the presence of the *P. falciparum* antigens up to 2-4 weeks after parasitemia has cleared (Humar et al. 1997). The RDT results were overall very similar but not identical between readers (pairwise correlations ranged from 0.78 to 0.88). In columns 9 and 10 of Table 5, we show that the ITT estimates for malaria prevalence remain almost identical if we include tester fixed effects in the regressions.

A.11 Changes in Other Prophylactic Behavior

In Table A.12, we look at differences among experimental arms in knowledge about causes of malaria (panel A), precautions one can take against it (panel B) and wall spraying between baseline and follow-up (panel C). The survey instrument asked respondents—without prompting—to list all possible causes of malaria, and then asked “[w]hat are the best precautions you can take to protect yourself from getting malaria.” In each arm, 85% or more of respondents list mosquito bites as a cause of malaria. Overall, households in intervention communities appear to be about as knowledgeable regarding causes of malaria as those in control areas, although the test of equality is rejected at the 10% level (but not at the 5%) for three of the four causes of malaria, and in each of these cases it is one of the experimental arms that suggests the best knowledge. There was no systematic variation in malaria-avoiding behavior among groups (panel B). Bednets are by far the most commonly listed precaution, mentioned by 82-87% of respondents (with the highest proportions in intervention villages). The next two most common precautions are “avoid contaminated environment” (16-21%) and “avoid drinking contaminated water” (5-8%). For all the fourteen indices, the test of equal means is not rejected at the 5% level, although the null is rejected at the 10% in two cases, and the joint null of equality for all behaviors is rejected (p-value = 0.0421). However, the differences are not consistent with risk-averting behavior being more common in control villages, and indeed in several cases they indicate the opposite (for example, use of smoke or long sleeves, or cleaning of drainage pools).

In panel C we analyze differences in residual spraying of indoors or outdoor walls. Although the null hypothesis of equal proportion among treatment groups cannot be rejected at standard levels, the magnitude of the differences between control and intervention areas is large. The reason why the null is not rejected despite the large differences is that the intra-village correlation for these two variables is very large (0.41 and 0.63 for inner and outer spraying respectively). Our data do not tell us if these differences were driven by household decisions, or if instead they resulted from choices made by public health officials who may have scheduled wall spraying taking into account our intervention. To evaluate whether differences in spraying rates help explain the lack of health benefits in intervention villages, we re-estimate the ITT model for malaria prevalence including dummies for recent wall spraying among the regressors, but this leaves the estimated impacts almost identical

(see columns 9 and 10 of Table 5).

A.12 Changes in Local *Anopheles* Behavior or Resistance to Insecticide

In principle, changes in the characteristics of the local *Anopheles* population may explain the lack of improvements in malaria and anemia prevalence. First, *Anopheles* mosquitoes may have been resistant to deltamethrin, the insecticide used to impregnate study bednets, or they may have developed resistance during the course of the study. Second, the reduction in malaria transmission may have been hampered if local *Anopheles* took a sufficiently high fraction of blood meals outside of the sleeping hours, when individuals were less likely to be protected by ITNs. In principle, a large increase in the fraction of individuals protected by bednets, as well as the excito-repellent property of deltamethrin, could lead to changes in peak biting hours, or in indoors vs. outdoors feeding habits. The increased difficulty in finding blood meals during the sleeping hours could force mosquitoes to increase biting at times when individuals are not protected by ITNs. Our project did not collect information on the local *Anopheles* population, before or after the intervention, so we cannot address these concerns directly. However, a number of factors make these hypotheses unlikely to hold.

First, recent studies carried out in Orissa suggest that local *Anopheles* biting patterns and susceptibility to deltamethrin made ITNs a promising protective tool against malaria. In Keonjhar, one of our study districts, [Sahu et al. \(2009\)](#) found that biting activity of the main local malaria vectors was concentrated between 2100 and 0300 hours, regardless of the season. [Sharma et al. \(2004\)](#) describes tests performed in 2002-03 in several Orissa districts (including our study districts Balangir, Khandhamal and Keonjhar). The tests showed high rates of susceptibility to deltamethrin of *Anopheles culicifacies* and *A. fluviatilis*, the two most common malaria vectors in the state. The insecticidal efficacy of deltamethrin compound has also been confirmed in Sundargarh, which borders the study district Sambalpur ([Yadav et al. 2001](#), [Sharma et al. 2006](#)). The field work for these studies was conducted a few years before our project, but a very recent study in Sundargarh, conducted in 2009-2010, found that synthetic pyrethroids were still highly effective against both *A. culicifacies* and *A. fluviatilis*, despite the fact that study areas had been exposed to either large-scale spraying with pyrethroids or to large-scale free distribution of bednets treated with deltamethrin, the same synthetic pyrethroid adopted in our study ([Sharma et al. 2012](#)). Another recent study, carried out in 2009 in Madhya Pradesh, central India, found some evidence of resistance to deltamethrin, but even in areas that had been sprayed regularly in the previous 5-10 years, the researchers documented about 75% mortality rates in the local population of *A. culicifacies* when exposed to the chemical ([Mishra et al. 2012](#)).

Second, although the emergence of resistance to insecticides such as DDT and pyrethroids has been documented following widespread use in agriculture or wall spraying, there is as yet little evidence of resistance developing *as a consequence* of the introduction of ITNs. Even in situations where resistance is present, ITNs have been documented to retain some protective efficacy ([Enayati and Hemingway 2010](#)). The only exception we are aware of is [Trape et al. 2011](#). In this study, the authors found that the introduction of deltamethrin-treated LLINs

in one village in Senegal led initially to sharp reductions in malaria incidence and prevalence, but that resistance to the insecticide became widespread in about two years. This led to an *increase* in malaria morbidity relative to before LLINs distribution among adults and older children. However, unlike in our study, nets were distributed to all villagers, and ownership and usage rates remained around 60-80% throughout the study period (and were close to 100% at the onset of the study).

Third, the literature is overall inconclusive about the impact of ITNs on *Anopheles* biting patterns, with only a fraction of the evidence pointing to changes in mosquito behavior that may have reduced the efficacy of nets (Takken 2002, Pates and Curtis 2005). After the distribution of permethrin-treated bednets to all inhabitants of one hamlet in Papua New Guinea, Charlwood and Graves (1987) observed a relative increase in biting during the evening, although the number of *Anopheles* in the area decreased substantially. Similar results were also found after mass distribution of ITNs in five villages in Tanzania (Magesa et al. 1991) and in locations where ITNs were distributed to cover *all* beds in Kenya (Mbogo et al. 1996) and Benin (Moiroux et al. 2012). Note that in all these studies ITNs had been delivered to ensure universal coverage, a situation in stark contrast with our case.

In sum, the existing evidence points to the likely efficacy of deltamethrin-treated ITNs in our study areas, and the literature suggests that the relatively low coverage of ITNs at the community level would have been unlikely to produce the emergence of either insecticide resistance or changes in biting patterns that may have reduced the benefits of the intervention.

A.13 Impacts on Self-reported Incidence Adjusted for Misdiagnoses

In Appendix A.7 we have shown that only 44% of the individuals reported as having had malaria in the same month as the interview tested positive to malaria. Some of these individuals may have recovered from malaria by the time blood samples were taken, but it is likely that the discrepancy is at least partly explained by misdiagnoses. In malarious areas, while asymptomatic cases are common, it is also common to attribute to malaria other fever episodes not caused by this disease (see for instance Adhvaryu 2012, Cohen et al. 2012). In such case, the figures in column 2 of Table 6 could confound changes in symptomatic malaria cases with changes in other symptomatic fever episodes. In addition, our data show that respondents were also misdiagnosing some malaria episodes as ‘fever’. This can be seen looking at the RDT results among individuals reported as having had fever during the same month as the interview. Among these 221 individuals, we find that 22% tested positive to malaria (95% C.I. 0.16-0.29).

In this section we use these considerations to construct a procedure to adjust the impacts on incidence in column 2 of Table 6 in a way that takes misdiagnosis into account. Note that we are *not* interested in estimating the program impacts on ‘true’ malaria incidence (regardless of whether an episode was recognized by the respondent), but rather we aim at estimating impacts on *symptomatic* malaria incidence. We argue in the paper that the latter is of interest because it measures cases severe enough to be perceived and to lead to illness-related costs recognized by the respondent. Recall that our data include both self-reported

malaria cases and self-reported fever cases. Suppose that $M_{A,ti}$ represents the number of malaria cases during the previous six months *reported* for individual i from experimental arm A ($A = Free, MF, Control$) interviewed at time t (where $t = 0$ denotes baseline and $t = 1$ denotes follow-up), while $F_{A,ti}$ is the corresponding figure for self-reported fever incidence. We assume that errors of diagnoses for symptomatic cases happen at the same rate over time and across different experimental arms (see Appendix A.7 for some evidence in support of this assumption). Consistent with the estimates above, we then assume that only 44% of self-reported malaria cases are actually malaria, but also that 22% of self-reported fever cases were actually malaria. We can thus estimate the mean number of actual malaria episodes at time t in a given treatment arm as $0.44 \times \bar{M}_{A,t} + 0.22 \times \bar{F}_{A,t}$, where $\bar{M}_{A,t}$ and $\bar{F}_{A,t}$ are respectively malaria and fever incidence as measured in our raw recall data.

Next, let $\hat{\beta}_{Y,T}^{DD}$ denote the estimated difference-in-difference impact on outcome $Y = M, F$ for treatment $T = MF, Free$ versus control areas. From column 2 of Table 6 we estimate that $\hat{\beta}_{M,Free}^{DD} = -0.048$ and $\hat{\beta}_{M,MF}^{DD} = -0.051$. Similarly, when we estimate the same model using F as dependent variable we obtain (results not shown in the table) $\hat{\beta}_{F,Free}^{DD} = -0.032$ (s.e. 0.033, so not significant) and $\hat{\beta}_{F,MF}^{DD} = -0.056$ (s.e. 0.031, significant at the 10% level). Because the DD is a linear combination of time and arm-specific means, and under the previously stated assumption that mid-diagnosis errors of symptomatic illness are non-differential across arms and over time, the adjusted DD for actual symptomatic malaria incidence can be finally calculated as $0.44 \times \hat{\beta}_{M,T}^{DD} + 0.22 \times \hat{\beta}_{F,T}^{DD}$, $T = MF, Free$. The final estimates are thus $-0.048 \times 0.44 - 0.032 \times 0.22 = -0.028$ (s.e. 0.012) in Free and $-0.051 \times 0.44 - 0.056 \times 0.22 = -0.035$ (s.e. 0.011) in MF areas.³⁰ Such estimates are thus about 60% as large as those in column 2 of Table 6, although both remain significant and substantive in magnitude.

A.14 Epidemiological Models of ITN Use

Current advances in epidemiological models of malaria transmission may help explaining the link between ITN usage and coverage and changes in malaria indices in our study areas. In particular, Killeen et al. (2007) describe a complex model that describes how malaria infection is affected by several factors, including mosquito numbers, biting patterns and mortality (also in relation to ITN presence) and above all ITN coverage and usage. The model simulates the protective power of ITNs for both users and non-users, by calibrating 16 exogenously determined factors (largely borrowed from earlier studies), and then showing how ITN protection varies with changes in coverage and usage. The protective power of bednets is measured as a relative risk (RR) of entomological inoculation rates (EIR), that is, the number of infective bites per year calculated relative to a situation where no ones uses nets. A useful feature of this study is that the authors also provide a spreadsheet that can be used to analyze how changes in any of the exogenous factors affect the RR. The spreadsheet is available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1904465/bin/pmed.0040229.sd001.xls>.

Panel A in Figure A.4 shows one of the key results in Killeen et al. (2007): in a scenario where about 60% of the population always uses ITN, individuals without an ITN (the dashed

³⁰To take into account that both 0.22 and 0.44 were estimated, we calculate the standard errors using 1,000 block bootstrap replications, using the village as the block.

line) are as protected as an individual who sleep regularly under an ITN in a community where no one else does (the continuous line). A key assumption to produce these results is that the index individual using the net does so very regularly. Specifically, panel A (identical to one of the graphs in Figure 3 of [Killeen et al. 2007](#)) is produced assuming that the individual uses the net for 90% of the potential time of exposure. In our empirical context, we have argued that information on previous night usage of ITNs is reliable, but even so our data indicate that only 45% of the program ITNs were in use the night before the follow-up survey in Free villages, and about 30% were in use in MF communities. If we assume that the frequency of usage is equal to such cross-sectional usage rates, while leaving all other parameters in the epidemiological model unchanged, the association between relative transmission intensity and coverage for users becomes as described in panel B of Figure A.4. Even under such scenario ITNs would provide some protection, but if less than 20% of the village population always uses ITNs (as surely is the case in a large majority of our study areas), then the RR remains close to 0.6-0.7.

These results formalize the intuition that sleeping under an ITN, even when done irregularly, should be expected to decrease the number of infectious bites to some extent. However, whether the decline is sufficient to produce a decline in malaria prevalence (our key biomarker) is not obvious. The link between EIR and prevalence is studied in [Beier et al. 1999](#), who analyze data from 31 studies throughout Africa where both outcomes could be estimated. They find that, after excluding two clear outliers the data are tightly concentrated around a linear regression relationship between malaria prevalence and the logarithm of annual EIR ($R^2 = 0.712$). Malaria prevalence predicted by the linear fit is $24.68 + 24.2 \log_{10} EIR$, with the standard errors of intercept and slope equal respectively to 3.06 and 5.42.

Although there is no direct information from Indian locations, the authors point out that “[w]hile malaria stratification according to ecologic zones is an important element of malaria control, it is important to note that the fundamental relationships between EIR and the prevalence of *P. falciparum* infection will likely hold across diverse ecosystems in Africa.” Together with the very tight distribution of the scatterplot around the regression line linking EIR to prevalence (see their Figure 2), this suggests that a similar relationship will also likely hold outside of the African continent. In areas neighboring our study districts, [Sharma et al. \(2006\)](#) documented EIR in the range of 3-114 infective bites per year, depending on location, well inside the relevant range considered in [Beier et al. 1999](#).

In our study areas, prevalence rates was about 20%, with village-specific prevalence ranging from 0 to about 60% and 95% of the 141 study villages showing prevalence below 0.53. Looking at Figure 2 of [Beier et al. \(1999\)](#), this suggests that the EIR in the area was likely between 1 and 10, but it also suggests that a 30-40% decline in EIR may have barely affected prevalence, given that EIR in the 1 to 10 range are associated with a very wide spectrum of prevalence rates. Using the words in [Beier et al. \(1999\)](#), “it may not be possible to achieve dramatic decreases in prevalence of *P. falciparum* infection at sites in Africa unless control measures reduce EIRs to levels well below 1 infective bites per year” (p. 111). Our results suggest that similar arguments will hold in other malaria-endemic areas outside of the African continent, such as our study areas in Orissa.

A.15 Malaria Prevalence and ITN Coverage

Recall that only BISWA clients received free ITNs or the offer of ITNs for sale on credit. Although BISWA has a large presence in the study area, we estimate that on average only 20% of people lived in households with at least one BISWA affiliate and thus were eligible for inclusion in the study.³¹ It is now accepted that the externalities offered by mass adoption of ITNs are a key factor for ITN efficacy, although the relative role of personal versus mass protection of ITNs is not yet well understood (Binka et al. 1998, Hawley et al. 2003, Killeen et al. 2007). Reductions in malaria indices have been documented among non-users of ITNs living within a few hundred meters of communities covered by mass distribution of ITNs. In our intervention, study villages were scattered spatially over a very broad geographical area (see Section 2), so cross-village externalities are not plausible. Here, we look at the relationship between village-level coverage and changes in malaria prevalence in our study area.³²

As a first step, we estimated village-specific changes in malaria prevalence in all intervention communities. We then plot the results against a measure of village-wide ITN coverage, calculated as the ratio of the total number of ITNs distributed to BISWA households (regardless of their inclusion in the survey sample) and village population counts from the 2001 Indian Census. Although not up-to-date, the population counts are a good proxy for current population, and if anything, in most cases 2001 population would underestimate current population, so that our estimates may overstate true coverage. The results are displayed separately for MF and Free communities in the two panels at the top of Figure A.5. Each graph also shows the fitted values of two OLS regressions, one where we include data from all villages (the continuous line) and the other where we exclude the very few villages where the ITN coverage ratio was larger than 0.35 (the dashed lines).

When we include all Free villages, there is a *positive* association between malaria prevalence at follow-up and program coverage. The estimated slope (0.59) is actually significant at the 1% level. However, the results are driven by the three outlier villages with coverage > 0.35 , and when we exclude them the slope becomes negative but very close to zero (-0.02) and not significant at standard levels (p-value = 0.966). In MF villages (panel B), where substantially fewer ITNs were distributed, the slope of the regressions are negative but we cannot reject the null that slopes are zero at standard levels, although when we include all villages the slope is almost significant at the 10% level (p-value = 0.103).

Because the ITN coverage achieved in MF communities was endogenously determined by household purchase decisions, its association with changes in malaria prevalence should not

³¹We estimated the fraction by making use of village population data from the 2001 census of India, together with estimates of the total number of individuals living in households with at least one BISWA member. Let \hat{s}_v and \hat{b}_v denote respectively average household size and average number of BISWA affiliates in BISWA households in village v , both estimated using baseline survey data. Let also m_v be the number of BISWA members in the village, as provided by the micro-lender. Then, if we denote by p_v the village population from the census, our estimate of the fraction who lives in BISWA households is $\hat{s}_v(m_v/\hat{b}_v)/p_v$.

³²We could not study the link between prevalence and *density* of ITNs throughout the village (e.g. the number of ITNs per squared hectare), because we do not have information on village size. The Indian Census reports the area covered by each village, but it does not report the size and the distribution of the areas covered by dwellings. In Section A.16 we look at the link between prevalence and coverage within the village using data from a subset of communities where we collected GIS information for all households.

be interpreted as necessarily causal. In contrast, in communities with free distribution, the number of ITNs delivered was decided by our research team based on household size and composition. This produced variation in ITN coverage resulting only from the distribution of BISWA affiliation and household composition within the community. Even so, BISWA affiliation could be associated with village characteristics related to malaria prevalence, although if we regress malaria prevalence at baseline on ITN coverage the slope is close to zero (0.03) and not significant (p-value = 0.720). On the one hand, the fact that the dependent variable in panels A and B is the *change* in prevalence, eliminates any possible spurious correlation due to time-invariant (observed or unobserved) village-level characteristics. On the other hand, there may be other unobserved differences in trends correlated with both ITN coverage and malaria prevalence. To address this concern, in panel C of Figure A.5 we look at the relationship between changes in prevalence and the fraction of the population affiliated to BISWA in control villages (“BISWA penetration”). No ITNs were distributed in these communities, but by construction BISWA penetration is very strongly correlated with the measure of ITN coverage that would have been observed if ITNs had been distributed as in Free communities. Indeed, the correlation between the two variables in Free villages is 0.95. The graph in panel C shows no clear association between changes in malaria prevalence and BISWA penetration. This suggests, albeit indirectly, that the lack of an association between changes in prevalence and ITN coverage in Free villages (panel A) is unlikely to be caused by differential trends in prevalence across communities with varying degrees of BISWA penetration.

As an additional check, we use Control and Free villages to estimate an OLS regression of the village-level change in prevalence on BISWA penetration, the Free dummy, and the interaction between the two variables. If ITN coverage were causing declines in malaria prevalence in our sample, we would expect the coefficient on the interaction to be negative. Consistent with the results in panel A, we find instead that the coefficient is positive and significant when we include all 94 villages (= 1.8, p-value= 0.009), and close to zero and not significant (= 0.25, p-value= 0.770) when we exclude the three villages with coverage larger than 0.35. Overall, we conclude that in our sample the coverage of the intervention did not appear to be systematically related to the changes in malaria prevalence.

A.16 Within-village Externalities

In Section A.15 we found no direct support for the link between ITN coverage and malaria prevalence. In principle, it is still possible that such a link existed within villages, with more protection provided in clusters with a denser concentration of ITNs. Although the baseline and follow-up surveys did not include geo-coding of household locations, such information was recorded later in a subset of 11 study villages, including four Control and seven Free villages. The geo-coding was completed in February-June 2012, during the implementation of the supplemental Cash arm described in Section 3.2.1. Unfortunately, time and funding constraints did not allow us to conduct a complete mapping of the whole study area. In this section we show that the available data provide some evidence of within-village externalities, although the estimates are very imprecise and the null of no effect can never be rejected.

In each of the 11 villages, surveyors visited all households, regardless of BISWA affiliation,

and recorded for each latitude and longitude using GPS hardware.³³ Surveyors also recorded whether the household belonged to BISWA at the time of the baseline survey, in 2007. Although the GPS survey was carried out a few years later, we were able to find nearly all of the original surveyed households and field observations suggested that few households had moved within the village so we are reasonably confident that the 2012 GPS coordinates are accurate measures of households’ 2007-2009 locations.

We then constructed measures of population density within pre-specified radii of our sample households. Concretely, for each sample household (an ‘index’ household) we constructed the total number of neighbors (P) and the number of BISWA households (B) within a given radius. The number of BISWA households matters because they all received ITNs in the Free villages, so that B provides a good proxy for the potential ITN coverage around the index household. Controlling for total population in the neighborhood is important, because B is by construction strongly correlated with population density around the index household, and this in turn may be correlated with unobserved characteristics that could be linked to health. On average households had 16 neighbors within a 20-meter radius, of whom 7 were BISWA members.

We thus estimate the following model for the malaria indicator M_{iv} of individual i in village v :

$$M_{iv} = \alpha_v + \alpha_P P_{iv} + \alpha_B B_{iv} + \tau_P P_{iv} \times Free_v + \tau_B B_{iv} \times Free_v + \epsilon_{iv},$$

where α_v is a village fixed effect, and $Free_v$ is the usual dummy for Free villages. The inclusion of Control villages allows to interpret the estimates of τ_P and τ_B as causal, because any correlation between malaria infection and population density regardless of ITN presence will be captured by α_P and α_B . In particular, if there are externalities from being surrounded by households with ITNs, we expect $\tau_B < 0$, that is, after controlling for total density P , an increase in the number of BISWA neighbors should be associated with lower malaria prevalence in Free relative to Control villages. In contrast, we do not have clear predictions for the sign of τ_P , which measures the impact of population density regardless of ITN coverage. Note that the interpretation of τ_B as measuring externalities needs to be taken with caution, given that the number of BISWA neighbors, even when controlling for overall density, was not randomly determined, and may proxy for other unobserved location characteristics.³⁴

Overall, we have malaria infection status for 611 individuals, but because identification relies on individual variation in neighbors interacted with treatment status, we cluster standard errors at the village level. Because we have only 11 villages, we estimate standard errors using block bootstrap, using the village as the block in each iteration. We only focus on relatively short radii, because several of the villages are small, and using a radius of 50 meters or more would generate collinearity between the measures of density and the village fixed effects, reducing further the already small number of observations. Table A.15 displays the results, which show some evidence of externalities at short ranges, from 5 to

³³For each location, two independent measurements were taken, and both were recorded. This double measurement allowed to detect a handful of measurement errors, but otherwise the vast majority of measurements were almost identical, so the results remain virtually unchanged if we use either one or the other sets of records.

³⁴So, for instance, our specification is not identical to that in Miguel and Kremer (2004) or Dupas (2012b), because in both these studies the fraction of treated neighbors was randomized by design.

20 meters, although the estimates are very imprecise and the null of no correlation is never rejected at standard levels. The estimated τ_B becomes close to zero for distances of 30 or 40 meters, but the point estimates are relatively large if we look at households immediately around the index households. For instance, If we compare two households in Free villages with an average number of total neighbors within a 10m radius (5.7), but with 0 versus 2.6 (the average) BISWA members among them, the predicted probability of malaria will be $2.6 \times 0.035 = 9$ percentage points lower in the household with more BISWA neighbors, relative to what would be predicted in Control areas. Of course, the 95% confidence interval is large, so the null of no relationship, or even of a positive relationship between ITN coverage and malaria cannot be rejected.

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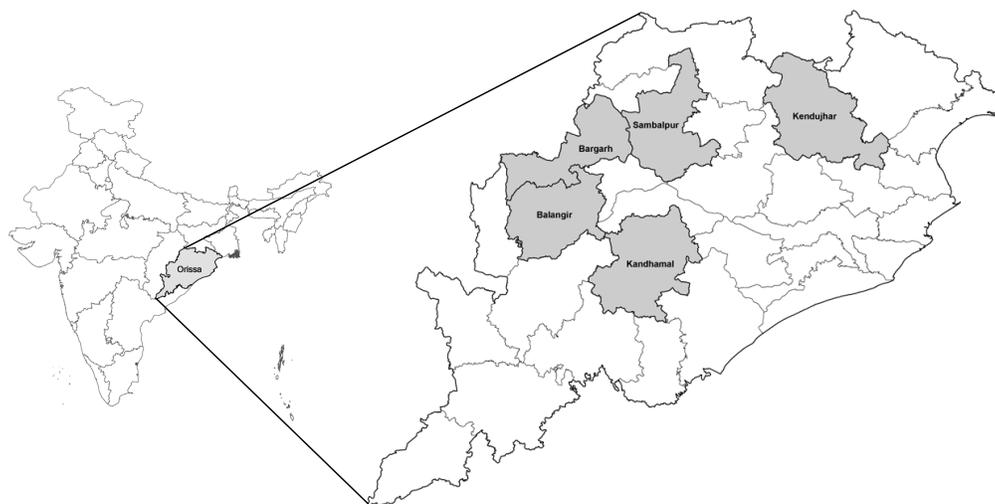


Figure A.1: Study Areas

Notes: Study communities at baseline included 30 villages in Sambalpur, 9 in Khandhamal, 30 in Keonjhar (Kendujhar), 33 in Balangir and 48 in Bargarh. Nine villages were later excluded from the analysis because the baseline survey showed that BISWA had no active presence there (5 villages in Sambalpur and 4 in Balangir).

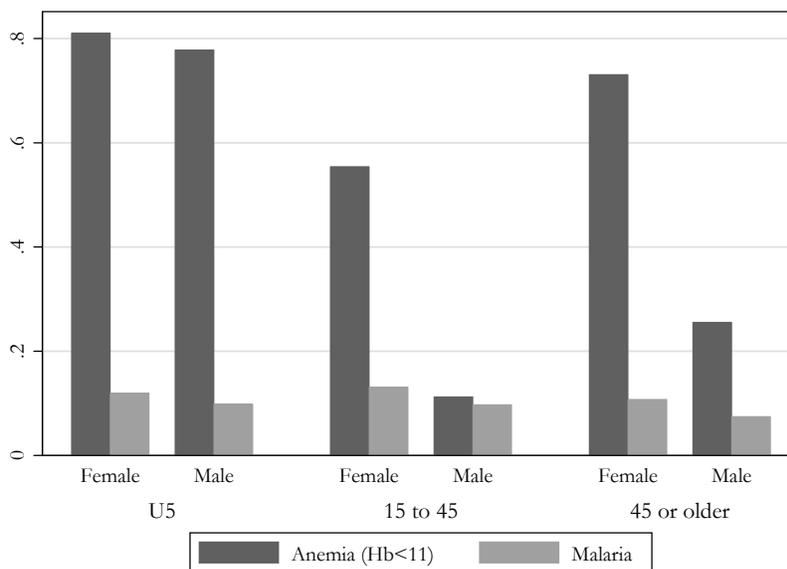


Figure A.2: Baseline Malaria and Anemia Prevalence, by Demographic Group

Notes: Data from Spring 2007 baseline survey. The bars represent the results of blood testing for anemia ($n = 2,532$) and malaria ($n = 2,561$) prevalence.

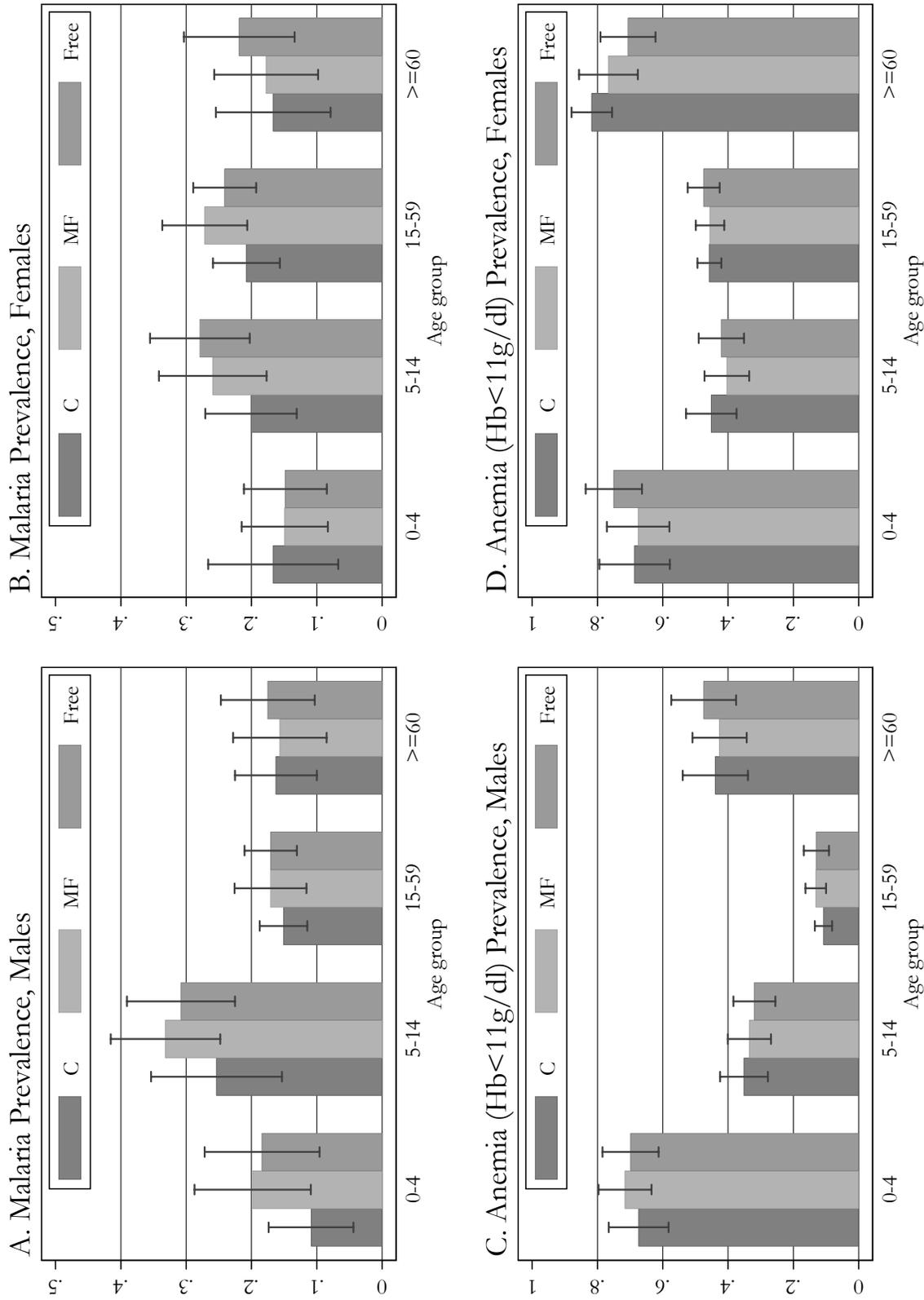


Figure A.3: Post-intervention Malaria and Anemia Prevalence, by Age and Gender
 Notes: Columns show anemia or malaria prevalence in the specific age-gender group, by experimental arm. Each column also displays 95% confidence intervals, robust to intra-village correlation.

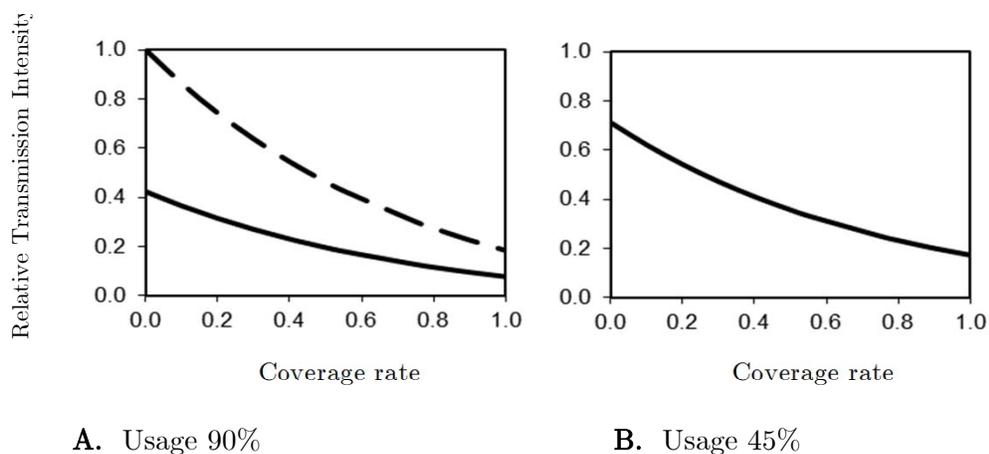


Figure A.4: Figure 1: Protective Power of ITNs vs. Community Coverage

Source: Calculations from the epidemiological model in [Killeen et al. \(2007\)](#). The graphs can be produced using the spreadsheet provided by Killeen et al. at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1904465/bin/pmed.0040229.sd001.xls>. Coverage is defined as the fraction of individuals using an ITN each night, while the relative transmission intensity is the proportional reduction of infectious bites for users (continuous lines) and non-users (dashed line in graph A). The label 'usage' refers to the fraction of time of normal exposure during which the individual is actually protected by the ITN.

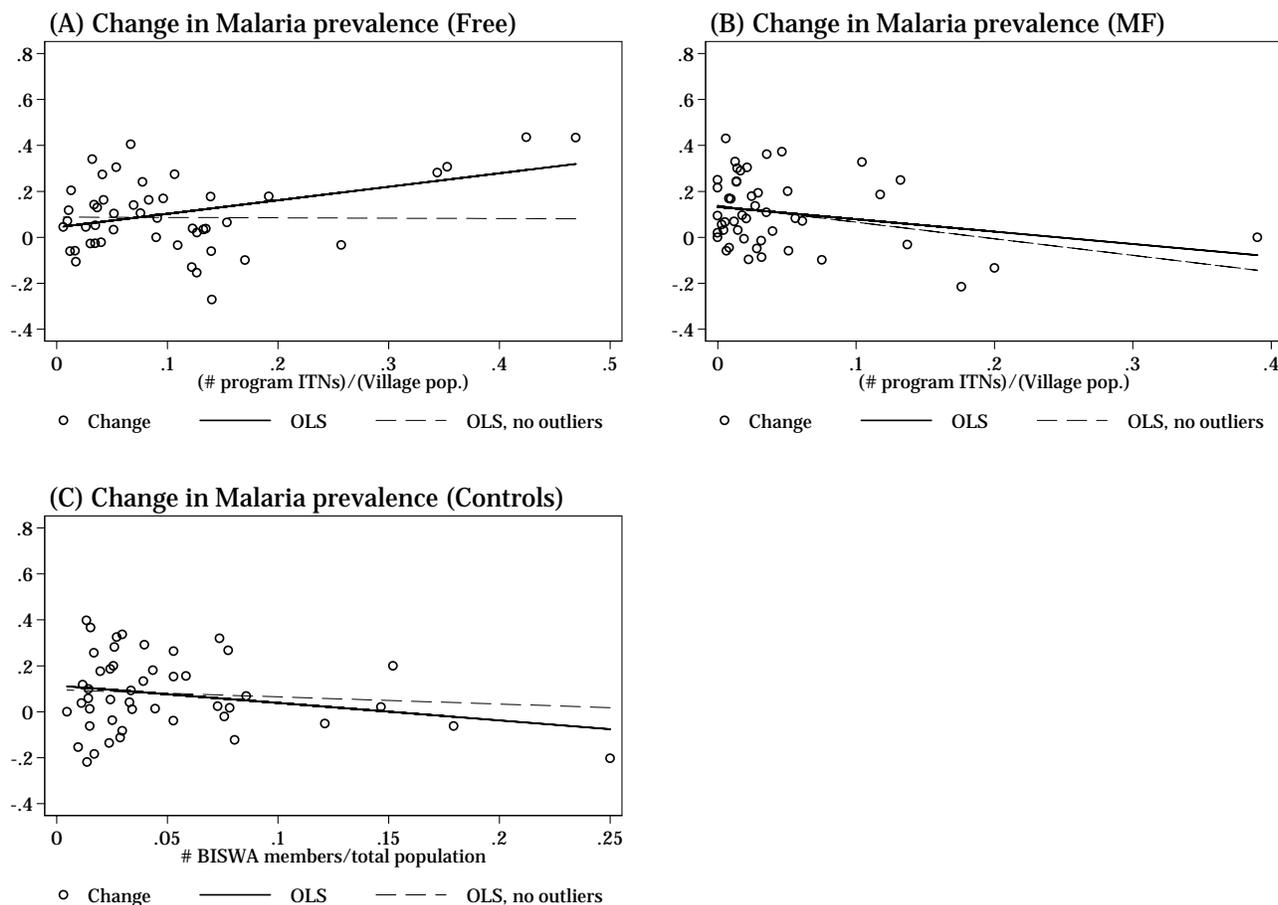


Figure A.5: Malaria Prevalence vs. Intensity of ITNs Distribution

Note: Data from spring 2007 and winter 2008-09. Each circle in the graphs represents a village. The continuous lines in each graph show fitted values of a village-level OLS regression through the points. The dashed lines show fitted values when we exclude villages with coverage larger than 0.35 (graphs A and B) or with more than 20% BISWA membership (graph C). The point estimates of the slopes and the corresponding heteroskedasticity-robust standard errors (in parenthesis), using all villages or excluding outliers respectively, are as follows: (A) .59 (.17)*** and $-.02$ (.37); (B) $-.54$ (.32) and $-.72$ (.54); (C) $-.76$ (.43)* and $-.31$ (.53). Statistical significance is indicated with * (10% level), ** (5%) and *** (1%).

Table A.7: Comparison of Sample Villages vs. Overall Village Population in Study Districts

	(1)	(2)		(3)	(4)	(5)	(6)		(7)
	Not in sample	Means, by village category		Free, $n = 47$	MF, $n = 47$	no. of Villages	H_0 : All equal	Tests (p-values)	H_0 : Exper. arms equal
Area of Village (in hectares)	275.2	413.1	476.4	417.4	417.4	8991	0.000***	0.608	
Number of Households	121.5	261.4	359.0	284.3	284.3	8991	0.000***	0.526	
Scheduled Caste population (%)	0.134	0.164	0.164	0.173	0.173	8630	0.012**	0.921	
Scheduled Tribe population (%)	0.478	0.328	0.372	0.321	0.321	8630	0.000***	0.597	
Females	0.501	0.497	0.496	0.499	0.499	8630	0.128	0.763	
Primary school	0.746	0.936	0.979	0.936	0.936	8991	0.000***	0.432	
Middle school	0.236	0.383	0.596	0.447	0.447	8991	0.000***	0.096*	
Secondary school	0.129	0.319	0.404	0.298	0.298	8991	0.000***	0.523	
Hospital	0.002	0.000	0.021	0.000	0.000	8991	0.001***	0.312	
Number of Primary Health Centres	0.025	0.106	0.064	0.064	0.064	8991	0.132	0.712	
Number of Primary Health Sub Centres	0.105	0.170	0.234	0.213	0.213	8991	0.029**	0.727	
Well Water	0.815	0.830	0.872	0.809	0.809	8991	0.692	0.678	
Tank Water	0.557	0.702	0.723	0.745	0.745	8991	0.000***	0.899	
River Water	0.120	0.106	0.170	0.149	0.149	8991	0.747	0.643	
Canal	0.050	0.128	0.149	0.128	0.128	8991	0.034**	0.943	
Number of Post Office	0.158	0.234	0.383	0.255	0.255	8991	0.003***	0.246	
Number of Telephone connections	0.285	0.532	0.617	0.553	0.553	8991	0.000***	0.682	
Bus services	0.228	0.255	0.298	0.298	0.298	8991	0.499	0.866	
Number of Commercial Banks	0.027	0.064	0.064	0.085	0.085	8991	0.242	0.906	
Number of Agricultural Credit Societies	0.027	0.085	0.106	0.106	0.106	8991	0.043**	0.919	
Approach - Paved Road	0.332	0.383	0.426	0.362	0.362	8991	0.506	0.813	
Distance from the nearest Town (in Kilometers)	34.9	34.3	25.2	26.1	26.1	8991	0.000***	0.445	
Electricity for Domestic use	0.465	0.702	0.575	0.681	0.681	8991	0.000***	0.389	
Electricity of Agricultural use	0.066	0.106	0.064	0.149	0.149	8991	0.346	0.386	
Wet Rice (irrigated) cultivated Area (%)	0.075	0.151	0.188	0.183	0.183	8875	0.000***	0.727	
Dry Rice (not irrigated) cultivated Area (%)	0.422	0.504	0.483	0.510	0.510	8875	0.005**	0.864	

Notes: Data from the 2001 Government of India Census. The point estimates in column 1 indicate means in villages not included in the baseline sample, while estimates in columns 2 to 4 indicate means in study villages that belong to the group indicated in the column header. The figures in column 6 are p-values for the null hypothesis that the mean of the variable in the row is the same across all four village groups. The p-values in column 7 are for the test of equality among the three experimental arms. Statistical significance is indicated as *** (1% level), ** (5%) or * (10%). All tests are heteroskedasticity-robust.

Table A.8: Attrition between Pre and Post Intervention Household Surveys

Dependent variable: Dummy = 1 if household was not re-interviewed at follow-up	(1)	(2)	(3)	(4)
Constant	0.041 [0.005]***	0.05 [0.013]***	0.2 [0.108]*	0.173 [0.109]
Free		-0.023 [0.014]	-0.022 [0.014]	-0.021 [0.013]
Micro-loans		-0.003 [0.015]	-0.001 [0.015]	0.004 [0.015]
log(monthly expenditure/household size)			0.011 [0.012]	0.014 [0.011]
# household members			-0.002 [0.002]	-0.001 [0.002]
Access to electricity			0.011 [0.010]	0.011 [0.010]
BISWA Debt/(Total yearly expenditure) < 0.05			-0.01 [0.016]	-0.021 [0.017]
BISWA Debt/(Total yearly expenditure) > 0.25			-0.006 [0.022]	-0.012 [0.022]
Baseline bednets per head			-0.018 [0.023]	-0.035 [0.021]
% Members who slept under net last night			-0.009 [0.016]	0.002 [0.017]
% Members who sleeps regularly under net			0.001 [0.017]	0.009 [0.017]
Household head is male			0.008 [0.019]	0.025 [0.017]
Household head's age (log)			-0.05 [0.019]***	-0.053 [0.020]***
Household head had any schooling			-0.024 [0.013]*	-0.029 [0.012]**
% malaria +ve in household				-0.005 [0.013]
% anemic (Hb < 11) in household				0.005 [0.011]
Observations	1844	1844	1814	1645
R-squared	0	0	0.01	0.02
H_0 : all coefficients = 0 (p-values)		0.11	0.21	0.14

Notes: OLS estimates. Standard errors (in brackets) are robust to intra-village correlation. Asterisks indicate significance at the 10 (*), 5 (**), and 1% (***) level. All regressions include observations from 141 clusters (villages). The smaller sample size in columns 3 and 4 relative to columns 1 and 2 is due to missing values in one or more regressors.

Table A.9: Tests of Balance for Characteristics of Cash Villages (p-values)

	(1) Cash vs. Other Study villages	(2) Cash: New vs. Control	(3) Cash: Low vs. High price
Area of Village (in hectares)	0.862	0.822	0.196
Number of Households	0.961	0.997	0.077*
SC population (%)	0.378	0.221	0.785
ST population (%)	0.006	0.627	0.476
Females	0.884	0.931	0.964
Middle school in village	0.78	0.752	0.539
Secondary school in village	0.594	0.68	0.108
Primary Health Centre	0.9	0.08*	0.56
Primary Health Sub Centre	0.642	0.262	0.267
Well Water	0.224	0.573	1
Tank Water	0.046**	0.213	1
River Water	0.68	0.283	0.222
Canal	0.589	0.283	0.687
Post Office	0.637	0.933	0.096*
Telephone connection	0.836	0.874	1
Bus services	0.393	0.623	0.194
Agricultural Credit Societies	0.693	0.08*	0.56
Paved Road	0.063*	0.066*	0.214
Distance from the nearest Town (in Kms)	0.18	0.807	0.75
Electricity for Domestic use	0.119	0.378	1
Electricity of Agricultural use	0.719	0.906	0.643
Wet Rice (irrigated) cultivated Area (%)	0.892	0.324	0.518
Dry Rice (un-irr.) cult. Area (%)	0.41	0.342	0.741
# Villages	156	40	40

Notes: all figures are p-values of tests of equality of means of the listed village-level characteristics between villages in the two groups indicated in the column header. All data are from the 2001 Census of India. All tests are heteroskedasticity-robust. Asterisks denote statistical significance at the 10(*), 5(**) or 1%(***) level. The results in column (1) use information from the 40 Cash villages and from the remaining 116 villages surveyed at baseline (47 MF, 47 Free and the 22 Control villages not included in the Cash study as 'PC' villages). The results in column 2 use information from the 25 PC villages and the 15 New villages. The same 40 villages are also used in column 3, where they are split by whether LLINs were sold at lower or higher prices (20 per group).

Table A.10: Post-intervention Malaria Biomarkers: Testing Success Rate in Baseline Households

	(1)	(2)	(3)	(4)	(5)	(6)
	Absent	Absent	Absent	Refusal	Refusal	Refusal
Free		-0.001 [0.018]	-0.001 [0.018]		-0.009 [0.015]	-0.01 [0.015]
MF		0.006 [0.018]	0.005 [0.019]		0.018 [0.016]	0.017 [0.016]
Male, 0-5			-0.212 [0.020]***			0.017 [0.013]
Female, 0-5			-0.205 [0.023]***			0.045 [0.017]***
Male, 5-15			-0.121 [0.018]***			0.017 [0.010]*
Female, 5-15			-0.136 [0.019]***			0.008 [0.010]
Female, 15-45			-0.187 [0.015]***			0.011 [0.006]*
Male, > 45			-0.133 [0.017]***			0.003 [0.006]
Female, > 45			-0.212 [0.018]***			0.036 [0.009]***
Constant	0.194 [0.007]***	0.193 [0.013]***	0.32 [0.018]***	0.057 [0.006]***	0.054 [0.011]***	0.043 [0.012]***
Observations	9589	9589	9555	9589	9589	9555
R-squared	0.0000	0.0001	0.0404	0.0000	0.0023	0.0052
Clusters	141	141	141	141	141	141
Free=MF=0		0.9209	0.9343		0.2303	0.2355
M=F,0-5			0.7449			0.1558
M=F,5-15			0.4402			0.4505
M=F,Over 45			0.0000			0.0010

Notes: Data from post-intervention household survey (Winter 2008-09). Standard errors (in brackets) are robust to intra-village correlation. Asterisks indicate significance at the 10 (*), 5 (**), and 1% (***) level. All figures are OLS estimates of a linear probability model where the dependent variable is indicated in the column header. Both absence and refusal refer to malaria RDTs, but the figures for Hb are almost identical. All regressions include only observations from all members (at the time of the follow-up) of the 1768 households interviewed at baseline and re-contacted during the follow-up survey.

Table A.11: Results of Rapid Diagnostic Tests Validation

	RDT(1)	RDT(2)	RDT(3)
RDT(2)	0.7873		
RDT(3)	0.7844	0.8760	
Microscopy	0.5274	0.6131	0.5968

		Microscopy	
		-ve	+ve
Tester 1 RDT	-ve	129	1
	+ve	45	30

		Microscopy	
		-ve	+ve
Tester 2 RDT	-ve	148	3
	+ve	26	28

		Microscopy	
		-ve	+ve
Tester 3 RDT	-ve	146	3
	+ve	28	28

Notes: Data from July 2009. The results refer to tests of 205 blood samples collected from individuals with malaria symptoms in 3 villages in Rourkela district (Orissa). The figures in the sub-table on top are sample correlations between the results as read by the tester indicated in the column header and the one indicated in the row. The figures in the three sub-tables underneath indicate the details of the sample joint distributions of the test results as read by each tester vs. microscopy. Testers 1 and 2 were part of the field team that conducted blood tests during the follow-up household survey. Tester 3 was the most senior survey monitor in the team.

Table A.12: Knowledge of Causes of Malaria and Risk Mitigating Behavior

	(1)	(2)	(3)	(4)
	Means			Test of equality (p-values)
	Control	Free	MF	
(A) Causes of malaria				
Drinking contaminated water	0.105	0.059	0.073	0.055**
Mosquito bites	0.845	0.892	0.854	0.058*
Contaminated environment	0.116	0.131	0.148	0.447
Don't know	0.037	0.025	0.051	0.065*
(B) Malaria-avoiding behavior				
Nets	0.819	0.866	0.830	0.139
ITNs	0.023	0.023	0.017	0.718
Proper clothing (long sleeves etc.)	0.004	0.008	0.010	0.268
Avoid drinking contaminated water	0.076	0.054	0.058	0.471
Insecticides	0.009	0.008	0.017	0.352
Repellents/mosquito coils	0.030	0.020	0.020	0.554
Smoke	0.016	0.023	0.022	0.622
Clearing stagnant water	0.028	0.021	0.022	0.702
Cleaning drainage system/sewage	0.054	0.075	0.087	0.093*
Avoiding contaminated environments	0.158	0.170	0.211	0.151
Proper diet	0.051	0.039	0.037	0.618
Medicine	0.042	0.033	0.066	0.058*
Other ways	0.035	0.021	0.027	0.469
Don't know	0.035	0.030	0.024	0.608
(C) Residual spraying of walls				
Inner walls sprayed in 2008-09	0.403	0.368	0.296	0.242
Outer walls sprayed in 2008-09	0.531	0.481	0.442	0.580
(D) Number of nets from other sources in 12 months before follow-up survey (per household)				
From Government/health centers	0.051	0.054	0.136	0.321
From NGOs other than BISWA	0.004	0.000	0.019	0.328
Purchased from the market	0.678	0.139	0.511	0.000***

Notes: Data from follow-up survey (winter 2008-09). Only panel households are included ($n = 1,768$). The figures in panels A and B show proportions of respondents who list, un-prompted, the cause/behavior indicated in the row header. The p-values in column 4 are calculated for a test of the joint null hypothesis that means are identical across experimental arms. All tests are robust to the presence of intra-village correlation of residuals. Asterisks in column 4 indicate significance at the 10 (*), 5 (**), and 1% (***) level.

Table A.13: Self-reported Malaria Indices: Baseline and Follow-up Differences in Levels

	(1) Cases within a month of survey		(2) Number of episodes (last 6 months)		(3) Days of work or school lost (last 6 months)		(4) Health expenditures (last 6 months)		(5) Expenditures for drugs/doctors (last 6 months)		(6) Costs paid with debt (last 6 months)		(7) Costs paid lower consumption (last 6 months)	
	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up
Free distribution=1	0.001 [0.002]	-0.001 [0.003]	0.032 [0.015]**	-0.011 [0.019]	0.713 [0.854]	-1.231 [1.086]	189 [121]	-5 [178]	106 [65]	20 [104]	0.082 [0.043]*	-0.033 [0.051]	0.093 [0.053]*	-0.023 [0.021]
Micro-loans=1	0.002 [0.003]	0.001 [0.003]	0.031 [0.015]**	-0.018 [0.017]	0.92 [0.928]	-1.475 [0.985]	163 [131]	-106 [161]	112 [67]*	-75 [86]	0.032 [0.033]	-0.079 [0.045]*	0.09 [0.053]*	0.003 [0.026]
Constant (Control)	0.008 [0.002]***	0.007 [0.002]***	0.092 [0.009]***	0.115 [0.013]***	4.267 [0.572]***	5.779 [0.833]***	625 [86]***	863 [119]***	318 [43]***	487 [63]***	0.151 [0.022]***	0.22 [0.038]***	0.244 [0.031]***	0.069 [0.017]***
Observations	9684	9598	9684	9598	1768	1768	1768	1768	1768	1768	1768	1768	1768	1768
Free=MF=0 (p-value)	0.6992	0.9247	0.0402**	0.5786	0.5499	0.3218	0.2523	0.7572	0.1526	0.5623	0.1557	0.1856	0.111	0.4074
Free=MF (p-value)	0.7803	0.6931	0.949	0.6843	0.8315	0.7806	0.8438	0.5528	0.929	0.3548	0.2648	0.2755	0.9574	0.2688
Self-reported Malaria and Fever cases														
Free distribution=1	0 [0.006]	-0.006 [0.007]	0.021 [0.021]	-0.049 [0.040]	-0.334 [1.055]	-3.213 [1.540]**	8 [141]	-217 [255]	13 [80]	-75 [145]	0.054 [0.060]	-0.135 [0.111]	0.111 [0.089]	-0.057 [0.074]
Micro-loans=1	0.003 [0.006]	0.005 [0.007]	0.041 [0.022]*	-0.069 [0.037]*	1.236 [1.174]	-3.571 [1.369]**	230 [162]	-344 [238]	143 [90]	-193 [132]	0.032 [0.059]	-0.186 [0.117]	0.146 [0.086]*	0.013 [0.080]
Constant (Control)	0.026 [0.004]***	0.036 [0.005]***	0.215 [0.015]***	0.458 [0.028]***	7.847 [0.788]***	13.125 [1.190]***	1,101 [108]***	2,011 [184]***	566 [62]***	1,111 [99]***	0.315 [0.040]***	0.691 [0.088]***	0.613 [0.060]***	0.262 [0.055]***
Unit of observation	9684	9598	9684	9598	1768	1768	1768	1768	1768	1768	1768	1768	1768	1768
Free=MF=0 (p-value)	0.8611	0.2735	0.1791	0.1667	0.3591	0.0327**	0.2735	0.3535	0.211	0.335	0.6571	0.2693	0.2152	0.6081
Free=MF (p-value)	0.6294	0.1137	0.3815	0.5775	0.1621	0.7636	0.1434	0.5858	0.1207	0.3913	0.7137	0.6184	0.7036	0.3625

Notes: Data from baseline (Spring 2007) and post-intervention household surveys (Winter 2008-09). All results are OLS estimates of difference-in-differences models. All outcomes refer to malaria and fever episodes diagnosed as such by the respondent. Monetary values are in 2008-09 Rupees. Regressions in columns 1-4 are estimated at the individual level, while regressions 5-14 are estimated at the household level. Standard errors (in brackets) and tests are robust to intra-village correlation. Asterisks indicate significance at the 10 (*), 5 (**), and 1% (***) level.

Table A.14: Impact of Intervention on Self-reported Malaria and Fever Indices

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Malaria or fever previous month	Malaria and fever episodes in 6 months before interview					
		Number of episodes	Days of work or school lost	Health expenditures		# episodes paid for with debt	# episodes paid for with lower consumption
				All	Doctors & drugs		
Free distribution= 1	-0.011 [0.009]	-0.08 [0.038]**	-2.9 [1.42]**	-225 [251]	-87 [131]	-0.189 [0.101]*	-0.168 [0.115]
Micro-loans= 1	0.002 [0.009]	-0.107 [0.036]***	-4.8 [1.44]***	-575 [214]***	-336 [114]***	-0.218 [0.105]**	-0.132 [0.106]
Constant (Control)	0.01 [0.006]	0.243 [0.028]***	5.3 [1.03]***	910 [166]***	545 [81]***	0.376 [0.078]***	-0.351 [0.075]***
Endline level (Control)	0.036	0.458	13.1	2,011	1,111	0.691	0.262
Unit of observation	Individual	Individual	Household	Household	Household	Household	Household
Observations	8684	8684	1768	1768	1768	1768	1768
Free=MF=0 (p-value)	0.331	0.0126**	0.0045***	0.026**	0.0113**	0.0849*	0.2808
Free=MF (p-value)	0.1653	0.4275	0.1708	0.1336	0.0588*	0.7577	0.7533

Notes: Data from baseline (Spring 2007) and post-intervention household surveys (Winter 2008-09). All results are OLS estimates of difference-in-differences models. All outcomes refer to malaria and fever episodes diagnosed as such by the respondent. Monetary values are in 2008-09 Rupees. Standard errors (in brackets) and tests are robust to intra-village correlation. Asterisks indicate significance at the 10 (*), 5 (**), and 1% (***) level.

Table A.15: Malaria Prevalence and Spatial Distribution of BISWA Households within Villages

	(1)	(2)	(3)	(4)	(5)
	Radius around index household (in meters)				
	5	10	20	30	40
# Households within radius, α_P	-0.016 [0.029]	-0.001 [0.030]	0.001 [0.015]	0.002 [0.010]	-0.002 [0.013]
# BISWA Households within radius, α_B	0.022 [0.068]	0.024 [0.049]	0.010 [0.053]	0.000 [0.018]	0.005 [0.028]
# Households within radius×Free, τ_P	0.028 [0.039]	-0.001 [0.031]	0.001 [0.015]	-0.001 [0.010]	0.002 [0.013]
# BISWA Households within radius×Free, τ_B	-0.053 [0.077]	-0.035 [0.054]	-0.013 [0.054]	0.001 [0.020]	-0.007 [0.028]
Observations	611	611	611	611	611

Notes: Data on malaria infection from 2008-09 post-intervention survey in 11 villages (4 Control and 7 Free). The dependent variable is a dummy variable for malaria infection of the individual, measured using RDTs. Standard errors (in brackets) are calculated using block bootstrap, with 250 replications and using the village as block. None of the coefficients in the table is significant at standard levels. All regressions include village fixed effects.