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

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A Online Appendix

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

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

12% among men. Prevalence increased again among older adults, where it 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/m^2), 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 households 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 FUO 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 FUO, 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

[29]These 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.

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 FUO 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 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 $\hat{P}(M_i = 1 \mid S_i = 1)$ 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 ∼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 (∼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 sub-groups, 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 $\hat{\beta}^{DD}_{Y,T}$ 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 $\hat{\beta}^{DD}_{M,Free} = -0.048$ and $\hat{\beta}^{DD}_{M,MF} = -0.051$. Similarly, when we estimate the same model using F as dependent variable we obtain (results not shown in the table) $\hat{\beta}^{DD}_{F,Free} = -0.032$ (s.e. 0.033, so not significant) and $\hat{\beta}^{DD}_{F,MF} = -0.056$ (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 $0.44 \times \hat{\beta}^{DD}_{M,T} + 0.22 \times \hat{\beta}^{DD}_{F,T}, \quad T = MF, Free$. 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 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%

[30] To 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.

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.

[31] We 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 $\hat{s}_v(m_v/\hat{b}_v)/p_v$.

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 that 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 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

[32] We 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.

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

[33] For 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.

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 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.

Appendix Bibliography

Adhvaryu, A. (2012). Learning, misallocation, and technology adoption: Evidence from new malaria therapy in Tanzania. Working Paper.

Ashraf, N., J. Berry, and J. Shapiro (2010). Can higher prices stimulate product use? Evidence from a field experiment in Zambia. *American Economic Review 100*(5), 2383–2413.

Beier, J., G. Killeen, and J. Githure (1999). Short report: entomologic inoculation rates and plasmodium falciparum malaria prevalence in africa. *The American Journal of Tropical Medicine and Hygiene 61*(1), 109–113.

Binka, F., F. Indome, and T. Smith (1998). Impact of spatial distribution of permethrin-impregnated bed nets on child mortality in rural northern Ghana. *The American Journal of Tropical Medicine and Hygiene 59*(1), 80–85.

Charlwood, J. and P. Graves (1987). The effect of permethrin-impregnated bednets on a population of *Anopheles farauti* in coastal Papua New Guinea. *Medical and veterinary entomology 1*(3), 319–327.

[34] So, 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.

Cohen, J. and P. Dupas (2010). Free distribution or cost-sharing? Evidence from a randomized malaria prevention experiment. *Quarterly Journal of Economics 125*(1), 1–45.

Cohen, J., P. Dupas, and S. G. Schaner (2012). Price subsidies, diagnostic tests, and targeting of malaria treatment: Evidence from a randomized controlled trial. NBER Working Paper No. 17943.

Delavande, A., X. Giné, and D. McKenzie (2010). Measuring subjective expectations in developing countries: A critical review and new evidence. *Journal of Development Economics 94*(2), 151–163.

Dupas, P. (2012). Short-run subsidies and long-run adoption of new health products: Evidence from a field experiment. Working Paper.

Enayati, A. and J. Hemingway (2010). Malaria management: Past, present, and future. *Annual Review of Entomology 55*(1), 569–591.

Farcas, G. A., K. J. Y. Zhong, F. E. Lovegrove, C. M. Graham, and K. C. Kain (2003). Evaluation of the Binax now(r) ict test versus polymerase chain reaction and microscopy for the detection of malaria in returned travelers. *The American Journal of Tropical Medicine and Hygiene 69*(6), 589–592.

Hawley, W. A., P. A. Phillips-Howard, F. O. ter Kuile, D. J. Terlouw, J. M. Vulule, M. Ombok, B. L. Nahlen, J. E. Gimnig, S. K. Kariuki, M. S. Kolczak, and A. W. Hightower (2003). Community-wide effects of permethrin-treated bed nets on child mortality and malaria morbidity in western Kenya. *The American Journal of Tropical Medicine and Hygiene 68*(4 Suppl), 121–7.

Humar, A., C. Ohrt, M. A. Harrington, D. Pillai, and K. C. Kain (1997). Parasight(R)F test compared with the polymerase chain reaction and microscopy for the diagnosis of Plasmodium falciparum malaria in travelers. *The American Journal of Tropical Medicine and Hygiene 56*(1), 44–48.

Khairnar, K., D. Martin, R. Lau, F. Ralevski, and D. Pillai (2009). Multiplex real-time quantitative PCR, microscopy and rapid diagnostic immuno-chromatographic tests for the detection of *Plasmodium* spp: performance, limit of detection analysis and quality assurance. *Malaria Journal 8*(1), 284.

Killeen, G. F., T. A. Smith, H. M. Ferguson, H. Mshinda, S. Abdulla, C. Lengeler, and S. P. Kachur (2007). Preventing childhood malaria in Africa by protecting adults from mosquitoes with insecticide-treated nets. *PLoS Medicine 4*(7).

Kremer, M. and E. Miguel (2007). The illusion of sustainability. *Quarterly Journal of Economics 122*(3), 1007–1065.

Magesa, S., T. Wilkes, A. Mnzava, K. Njunwa, J. Myamba, M. Kivuyo, N. Hill, J. Lines, and C. Curtis (1991). Trial of pyrethroid impregnated bednets in an area of Tanzania holoendemic for malaria. Part 2. Effects on the malaria vector population. *Acta Tropica 49*, 97–108.

Mbogo, C. N. M., N. M. Baya, A. V. O. Ofulla, J. I. Githure, and R. W. Snow (1996). The impact of permethrin-impregnated bednets on malaria vectors of the Kenyan coast. *Medical and Veterinary Entomology 10*(3), 251–259.

Miguel, E. and M. Kremer (2004). Worms: identifying impacts on education and health the presence of treatment externalities. *Econometrica 72*(1), 159–217.

Mishra, A. K., S. K. Chand, T. K. Barik, V. K. Dua, and K. Raghavendra (2012). Insecticide resistance status in *Anopheles culicifacies* in Madhya Pradesh, central India. *Journal of Vector Borne Diseases 49*(1), 39–41.

Moiroux, N., M. B. Gomez, C. Pennetier, E. Elanga, A. Djènontin, F. Chandre, I. Djègbé, H. Guis,

and V. Corbel (2012). Changes in *Anopheles funestus* biting behaviour following universal coverage of long-lasting insecticidal nets in Benin. *Journal of Infectious Diseases*.

Moody, A. (2002). Rapid diagnostic tests for malaria parasites. *Clinical Microbiology Reviews 15*(1), 66–78.

Pates, H. and C. Curtis (2005). Mosquito behavior and vector control. *Annual Review of Entomology 50*, 53–70.

Sahu, S. S., K. Gunasekaran, and P. Jambulingam (2009). Bionomics of *Anopheles minimus* and *An. fluviatilis* (Diptera: Culicidae) in East-central India, endemic for *falciparum* malaria: Human landing rates, host feeding, and parity. *Journal of Medical Entomology 46*(5), 1045–1051.

Sharma, S., A. Upadhyay, B. Kindo, M. Haque, and P. Tyagi (2012). Impact of changing over of insecticide from synthetic pyrethroids to DDT for indoor residual spray in a malaria endemic area of Orissa, India. *The Indian Journal of Medical Research 135*(3), 382–388.

Sharma, S. K., P. K. Tyagi, K. Padhan, A. K. Upadhyay, M. A. Haque, N. Nanda, H. Joshi, S. Biswas, T. Adak, B. S. Das, V. S. Chauhan, C. E. Chitnis, and S. K. Subbarao (2006). Epidemiology of malaria transmission in forest and plain ecotype villages in Sundargarh District and Orissa, India. *Transactions of the Royal Society of Tropical Medicine and Hygiene 100*(10), 917–925.

Sharma, S. K., A. K. Upadhyay, M. A. Haque, O. P. Singh, T. Adak, and S. K. Subbarao (2004). Insecticide susceptibility status of malaria vectors in some hyperendemic tribal districts of Orissa. *Current Science 87*(12), 1722–1726.

Smith, D., C. Guerra, R. Snow, and S. Hay (2007). Standardizing estimates of the *Plasmodium falciparum* parasite rate. *Malaria Journal 6*(1), 131.

Takken, W. (2002). Do insecticide-treated bednets have an effect on malaria vectors? *Tropical Medicine & International Health 7*(12), 1022–1030.

Tarozzi, A., A. Mahajan, B. Blackburn, D. Kopf, L. Krishnan, and J. Yoong (2011). Micro-loans, bednets and malaria: Evidence from a randomized controlled trial in Orissa (India). Working Paper, available at http://www.econ.upf.edu/~tarozzi/TarozziEtAl2011RCT.pdf.

Thomas, D., E. Frankenberg, J. Friedman, J.-P. Habicht, M. Hakimi, N. Ingwersen, Jaswadi, N. Jones, K. McKelvey, G. Pelto, B. Sikoki, T. Seeman, J. P. Smith, C. Sumantri, W. Suriastini, and S. Wilopo (2006). Causal effect of health on labor market outcomes: Experimental evidence. Working Paper.

Trape, J.-F., A. Tall, N. Diagne, O. Ndiath, A. B. Ly, J. Faye, F. Dieye-Ba, C. Roucher, C. Bouganali, A. Badiane, F. D. Sarr, C. Mazenot, A. Touré-Baldé, D. Raoult, P. Druilhe, O. Mercereau-Puijalon, C. Rogier, and C. Sokhna (2011). Malaria morbidity and pyrethroid resistance after the introduction of insecticide-treated bednets and artemisinin-based combination therapies: a longitudinal study. *The Lancet Infectious Diseases 11*(12), 925–932.

van den Broek, I., O. Hill, F. Gordillo, B. Angarita, P. Hamade, H. Counihan, and J.-P. Guthmann (2006). Evaluation of three rapid tests for diagnosis of P. falciparum and P. vivax malaria in colombia. *The American Journal of Tropical Medicine and Hygiene 75*(6), 1209–1215.

World Health Organization (2002). Instructions for treatment and use of insecticide-treated mosquito nets. WHO/CDS/RBM 2002.41, World Health Organization, Geneva, Switzerland.

World Health Organization (2005). Safety of pyrethroid for public health use. WHO/CDS/WHOPES/GCDPP/2005.10, WHO/PCS/RA/2005.1, Communicable Disease Control, Prevention and Eradication WHO Pesticide Evaluation Scheme (WHOPES) & Protection of the Human Environment Programme on Chemical Safety (PCS).

Yadav, R. S., R. R. Sampath, and V. P. Sharma (2001). Deltamethrin treated bednets for control of malaria transmitted by *Anopheles culicifacies* (Diptera: Culicidae) in India. *Journal of Medical Entomology 38*(5), 613–622.

Zwane, A. P., J. Zinman, E. V. Dusen, W. Parienté, C. Null, E. Miguel, M. Kremer, D. S. Karlan, R. Hornbeck, X. Giné, E. Duflo, F. Devoto, B. Crépon, and A. Banerjee (2011). Being surveyed can change later behavior and related parameter estimates. *Proceedings of the National Academy of Sciences 108*(5), 1821–1826.

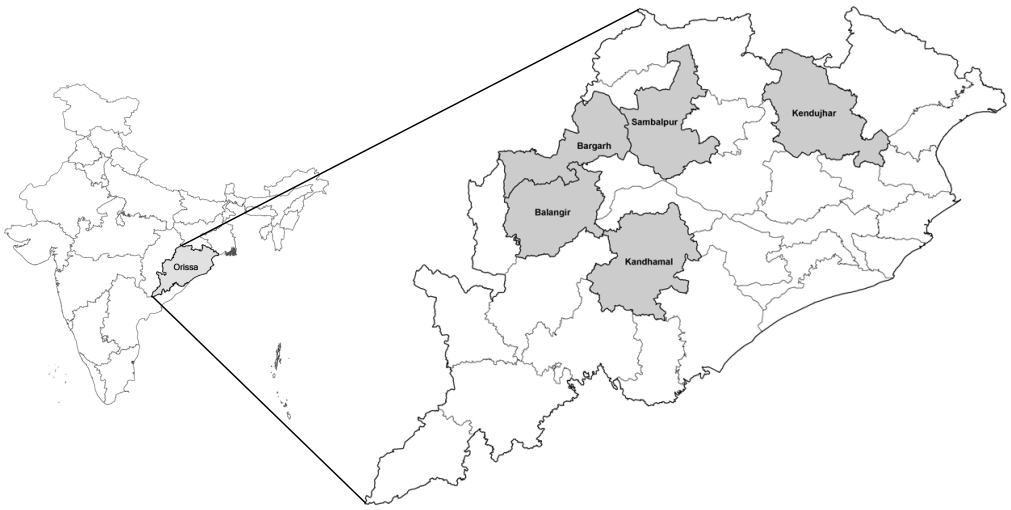


Figure A.1: Study Areas

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).

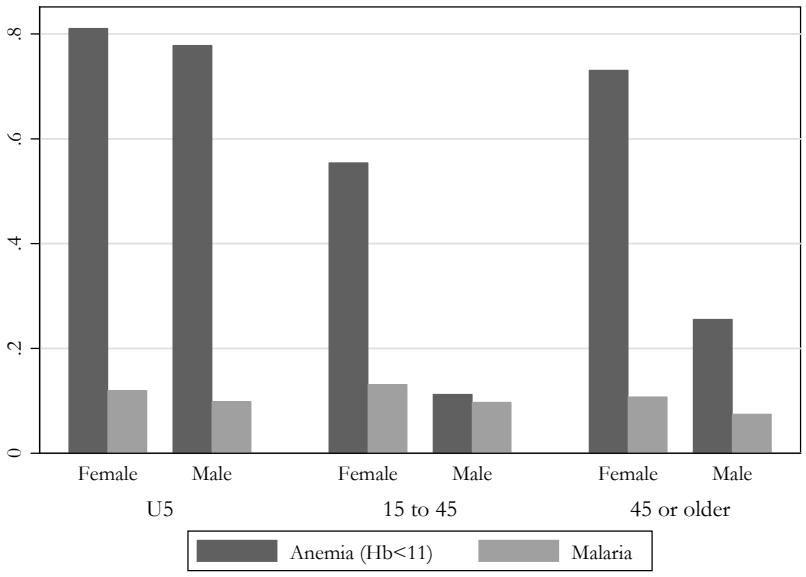


Figure A.2: Baseline Malaria and Anemia Prevalence, by Demographic Group

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.

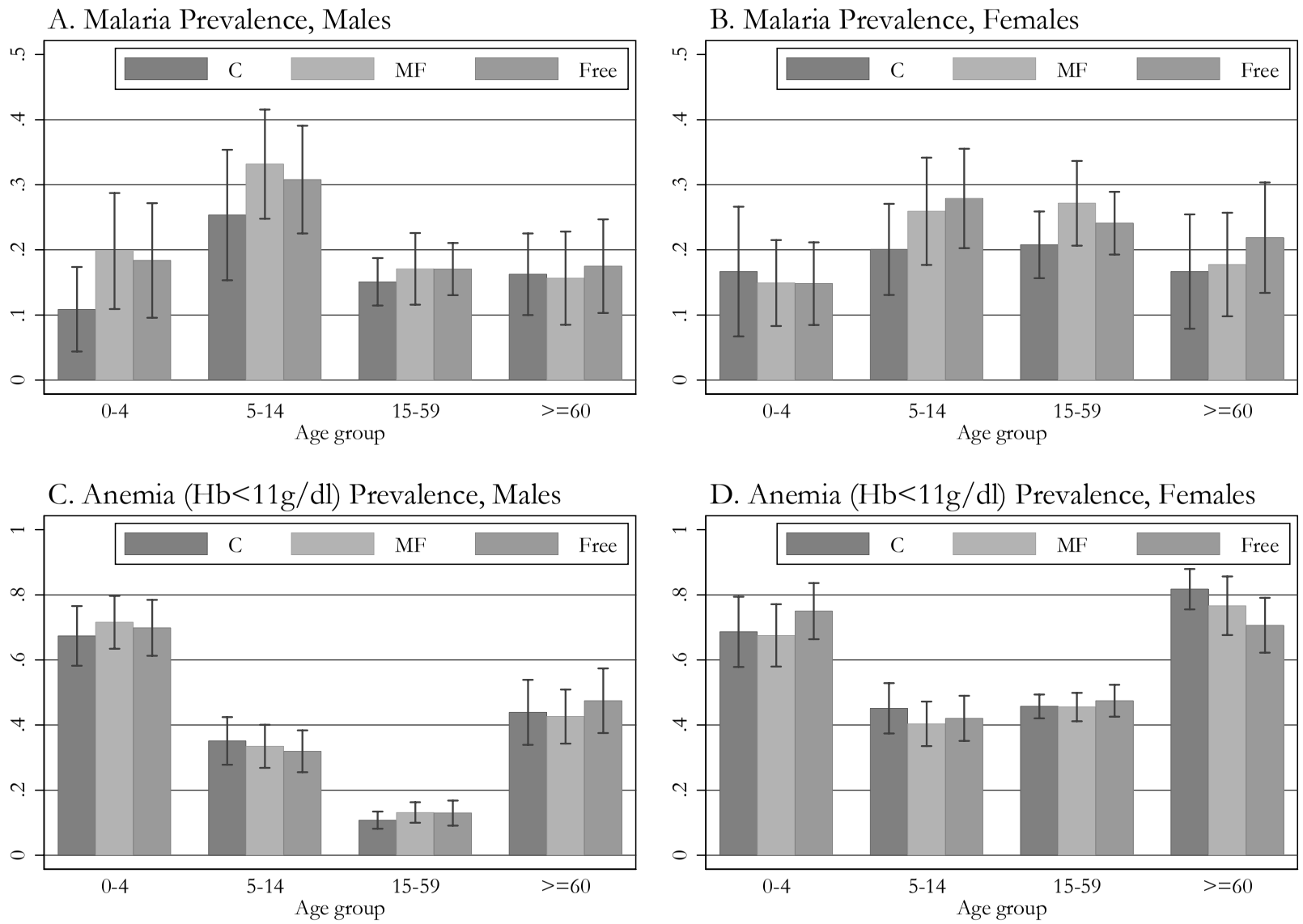
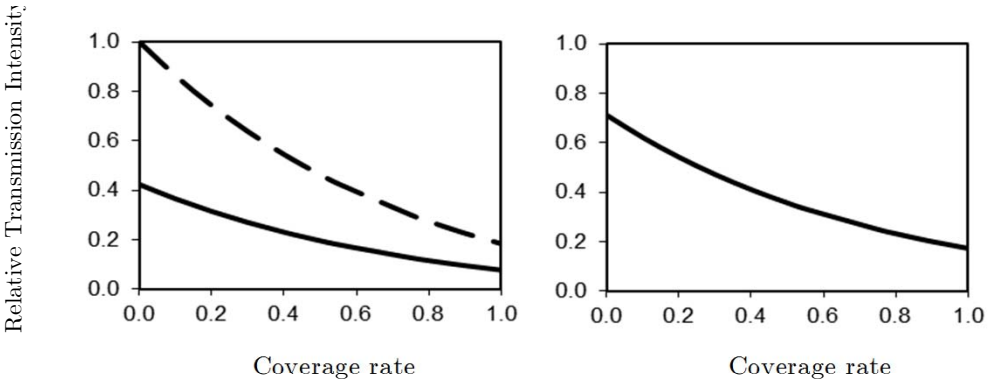


Figure A.3: Post-intervention Malaria and Anemia Prevalence, by Age and Gender

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.



A. Usage 90%

B. Usage 45%

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.

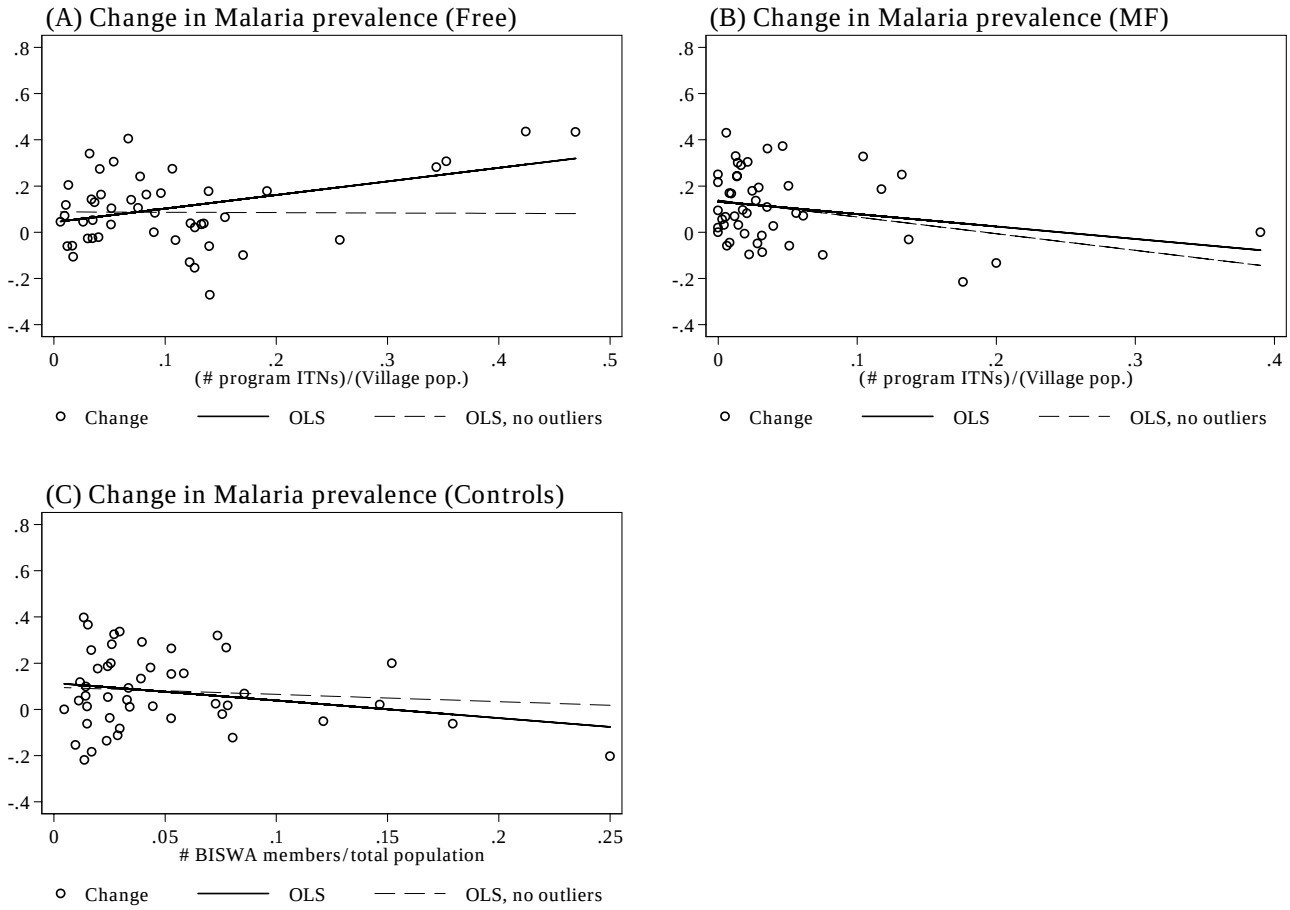


Figure A.5: Malaria Prevalence vs. Intensity of ITNs Distribution

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)
	Means, by village category				no. of	Tests (p-values)	
	Not in sample	Control, $n = 47$	Free, $n = 47$	MF, $n = 47$	Villages	H_0: All equal	H_0: Exper. arms equal
Area of Village (in hectares)	275.2	413.1	476.4	417.4	8991	0.000***	0.608
Number of Households	121.5	261.4	359.0	284.3	8991	0.000***	0.526
Scheduled Caste population (%)	0.134	0.164	0.164	0.173	8630	0.012**	0.921
Scheduled Tribe population (%)	0.478	0.328	0.372	0.321	8630	0.000***	0.597
Females	0.501	0.497	0.496	0.499	8630	0.128	0.763
Primary school	0.746	0.936	0.979	0.936	8991	0.000***	0.432
Middle school	0.236	0.383	0.596	0.447	8991	0.000***	0.096*
Secondary school	0.129	0.319	0.404	0.298	8991	0.000***	0.523
Hospital	0.002	0.000	0.021	0.000	8991	0.001***	0.312
Number of Primary Health Centres	0.025	0.106	0.064	0.064	8991	0.132	0.712
Number of Primary Health Sub Centres	0.105	0.170	0.234	0.213	8991	0.029**	0.727
Well Water	0.815	0.830	0.872	0.809	8991	0.692	0.678
Tank Water	0.557	0.702	0.723	0.745	8991	0.000***	0.899
River Water	0.120	0.106	0.170	0.149	8991	0.747	0.643
Canal	0.050	0.128	0.149	0.128	8991	0.034**	0.943
Number of Post Office	0.158	0.234	0.383	0.255	8991	0.003***	0.246
Number of Telephone connections	0.285	0.532	0.617	0.553	8991	0.000***	0.682
Bus services	0.228	0.255	0.298	0.298	8991	0.499	0.866
Number of Commercial Banks	0.027	0.064	0.064	0.085	8991	0.242	0.906
Number of Agricultural Credit Societies	0.027	0.085	0.106	0.106	8991	0.043**	0.919
Approach - Paved Road	0.332	0.383	0.426	0.362	8991	0.506	0.813
Distance from the nearest Town (in Kilometers)	34.9	34.3	25.2	26.1	8991	0.000***	0.445
Electricity for Domestic use	0.465	0.702	0.575	0.681	8991	0.000***	0.389
Electricity of Agricultural use	0.066	0.106	0.064	0.149	8991	0.346	0.386
Wet Rice (irrigated) cultivated Area (%)	0.075	0.151	0.188	0.183	8875	0.000***	0.727
Dry Rice (not irrigated) cultivated Area (%)	0.422	0.504	0.483	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	0.05	0.2	0.173
	[0.005]***	[0.013]***	[0.108]*	[0.109]
Free		-0.023	-0.022	-0.021
		[0.014]	[0.014]	[0.013]
Micro-loans		-0.003	-0.001	0.004
		[0.015]	[0.015]	[0.015]
log(monthly expenditure/household size)			0.011	0.014
			[0.012]	[0.011]
# household members			-0.002	-0.001
			[0.002]	[0.002]
Access to electricity			0.011	0.011
			[0.010]	[0.010]
BISWA Debt/(Total yearly expenditure)< 0.05			-0.01	-0.021
			[0.016]	[0.017]
BISWA Debt/(Total yearly expenditure)> 0.25			-0.006	-0.012
			[0.022]	[0.022]
Baseline bednets per head			-0.018	-0.035
			[0.023]	[0.021]
% Members who slept under net last night			-0.009	0.002
			[0.016]	[0.017]
% Members who sleeps regularly under net			0.001	0.009
			[0.017]	[0.017]
Household head is male			0.008	0.025
			[0.019]	[0.017]
Household head's age (log)			-0.05	-0.053
			[0.019]***	[0.020]***
Household head had any schooling			-0.024	-0.029
			[0.013]*	[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	-0.001		-0.009	-0.01
		[0.018]	[0.018]		[0.015]	[0.015]
MF		0.006	0.005		0.018	0.017
		[0.018]	[0.019]		[0.016]	[0.016]
Male, 0-5			-0.212			0.017
			[0.020]***			[0.013]
Female, 0-5			-0.205			0.045
			[0.023]***			[0.017]***
Male, 5-15			-0.121			0.017
			[0.018]***			[0.010]*
Female, 5-15			-0.136			0.008
			[0.019]***			[0.010]
Female, 15-45			-0.187			0.011
			[0.015]***			[0.006]*
Male, > 45			-0.133			0.003
			[0.017]***			[0.006]
Female, > 45			-0.212			0.036
			[0.018]***			[0.009]***
Constant	0.194	0.193	0.32	0.057	0.054	0.043
	[0.007]***	[0.013]***	[0.018]***	[0.006]***	[0.011]***	[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
	Control	Free	MF	(p-values)
(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	0.451
Don't know	0.037	0.025	0.051	0.065*
(B) Malaria-avoiding behavior				
Nets	0.819	0.866	0.830	0.141
ITNs	0.023	0.023	0.017	0.718
Proper clothing (long sleeves etc.)	0.004	0.008	0.010	0.269
Avoid drinking contaminated water	0.076	0.054	0.058	0.483
Insecticides	0.009	0.008	0.017	0.354
Repellents/mosquito coils	0.030	0.020	0.020	0.553
Smoke	0.016	0.023	0.022	0.624
Clearing stagnant water	0.028	0.021	0.022	0.700
Cleaning drainage system/sewage	0.054	0.075	0.087	0.095*
Avoiding contaminated environments	0.158	0.170	0.211	0.155
Proper diet	0.051	0.039	0.037	0.616
Medicine	0.042	0.033	0.066	0.059*
Other ways	0.035	0.021	0.027	0.470
Don't know	0.035	0.030	0.024	0.605
(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	0.575
(D) Number of nets from other sources in the 12 months before the follow-up survey (per head)				
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)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
	Cases within a month of survey		Number of episodes (last 6 months)		Days of work or school lost (last 6 months)		Health expenditures (last 6 months)		Expenditures for drugs/doctors (last 6 months)		Costs paid with debt (last 6 months)		Costs paid with 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
	Self-reported Malaria													
Free distribution= 1	0.001	-0.001	0.032	-0.011	0.713	-1.231	189	-5	106	20	0.082	-0.033	0.093	-0.023
	[0.002]	[0.003]	[0.015]**	[0.019]	[0.854]	[1.086]	[121]	[178]	[65]	[104]	[0.043]*	[0.051]	[0.053]*	[0.021]
Micro-loans= 1	0.002	0.001	0.031	-0.018	0.92	-1.475	163	-106	112	-75	0.032	-0.079	0.09	0.003
	[0.003]	[0.003]	[0.015]**	[0.017]	[0.928]	[0.985]	[131]	[161]	[67]*	[86]	[0.033]	[0.045]*	[0.053]*	[0.026]
Constant (Control)	0.008	0.007	0.092	0.115	4.267	5.779	625	863	318	487	0.151	0.22	0.244	0.069
	[0.002]***	[0.002]***	[0.009]***	[0.013]***	[0.572]***	[0.833]***	[86]***	[119]***	[43]***	[63]***	[0.022]***	[0.038]***	[0.031]***	[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	0.021	-0.049	-0.334	-3.213	8	-217	13	-75	0.054	-0.135	0.111	-0.057
	[0.006]	[0.007]	[0.021]	[0.040]	[1.055]	[1.540]**	[141]	[255]	[80]	[145]	[0.060]	[0.111]	[0.089]	[0.074]
Micro-loans= 1	0.003	0.005	0.041	-0.069	1.236	-3.571	230	-344	143	-193	0.032	-0.186	0.146	0.013
	[0.006]	[0.007]	[0.022]*	[0.037]*	[1.174]	[1.369]**	[162]	[238]	[90]	[132]	[0.059]	[0.117]	[0.086]*	[0.080]
Constant (Control)	0.026	0.036	0.215	0.458	7.847	13.125	1,101	2,011	566	1,111	0.315	0.691	0.613	0.262
	[0.004]***	[0.005]***	[0.015]***	[0.028]***	[0.788]***	[1.190]***	[108]***	[184]***	[62]***	[99]***	[0.040]***	[0.088]***	[0.060]***	[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 and fever episodes in 6 months before interview						
	Malaria or fever previous month	Number of episodes	Days of work or school lost	Health expenditures: All	Health expenditures: Doctors & drugs	# episodes paid for with debt	# episodes paid for with lower consumption
Free distribution= 1	-0.011	-0.08	-2.9	-225	-87	-0.189	-0.168
	[0.009]	[0.038]**	[1.42]**	[251]	[131]	[0.101]*	[0.115]
Micro-loans= 1	0.002	-0.107	-4.8	-575	-336	-0.218	-0.132
	[0.009]	[0.036]***	[1.44]***	[214]***	[114]***	[0.105]**	[0.106]
Constant (Control)	0.01	0.243	5.3	910	545	0.376	-0.351
	[0.006]	[0.028]***	[1.03]***	[166]***	[81]***	[0.078]***	[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	-0.001	0.001	0.002	-0.002
	[0.034]	[0.026]	[0.014]	[0.009]	[0.015]
# BISWA Households within radius, α_B	0.022	0.024	0.010	0.000	0.005
	[0.073]	[0.042]	[0.045]	[0.017]	[0.027]
# Households within radius×Free, τ_P	0.028	-0.001	0.001	-0.001	0.002
	[0.040]	[0.029]	[0.014]	[0.009]	[0.015]
# BISWA Households within radius×Free, τ_B	-0.053	-0.035	-0.013	0.001	-0.007
	[0.082]	[0.046]	[0.045]	[0.018]	[0.027]
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.