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Patient vs. provider incentives for malaria care: A cluster randomized controlled trial in Kenyan pharmacies

by

Maria Dieci

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Health Policy

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

Professor William H. Dow, Chair
Professor Paul J. Gertler
Professor Lia C.H. Fernald
Professor Jonathan Kolstad

Spring 2022

Patient vs. provider incentives for malaria care: A cluster randomized controlled trial in Kenyan pharmacies

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by

Maria Dieci

Abstract

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Doctor of Philosophy in Health Policy

University of California, Berkeley

Professor William H. Dow, Chair

A key aim of health policy is to ensure that patients are able to access high quality care when appropriate while minimizing the over-use of unnecessary treatments. Achieving this balance requires aligned decision-making between patients and providers, which can be hindered if incentives are incompatible with this aim. I study how patient and pharmacist decision-making in the context of malaria case management is affected by financial incentives. I test whether incentives for rapid tests and high quality antimalarials (ACTs) only to malaria-positive cases targeted to patients (demand-side), providers (supply-side), or both (hybrid) improve malaria case management and align incentives towards socially-optimal antimalarial use. Using a cluster randomized trial in 140 pharmacies in malaria-endemic zones in Kenya, I randomize patient discounts and pharmacist performance incentives and compare their effectiveness and cost-effectiveness to the status quo standard of care. I find that both patient subsidies and pharmacy incentives for diagnostic testing significantly increase usage of testing and may encourage malaria positive individuals to purchase ACTs, and report results from unadjusted models here. Patient subsidies increase the likelihood that a symptomatic patient takes a rapid test by 25 percentage points ($p = 0.046$), from a control group mean of 8 percent. Provider incentives and the hybrid approach increase the likelihood of rapid test uptake by 20 ($p = 0.008$) and 25 ($p < 0.001$) percentage points, respectively, which is indistinguishable from the demand-side approach. I also find that all arms improve ACT targeting by more than 200%: patient subsidies, pharmacy incentives, and the hybrid arm increase the likelihood that a patient purchases ACTs with a diagnostic test by 7 percentage points compared to a control group mean of 6 percent ($p = 0.244, 0.088, 0.032$, for patient subsidies, pharmacy incentives, and hybrid arms, respectively). I find that both demand- and supply-side incentives reduce the likelihood that a patient purchases malaria treatment without a confirmatory diagnosis by between 17-19 percentage points, compared to 81 percent in the control group. Taken together, these results suggest that appropriately calibrated and targeted financial incentives are promising for improving malaria case management in a

pharmacy setting. Additionally, the fact that supply- and demand-side incentives lead to comparable effects on diagnostic testing and treatment targeting suggests that both patients and pharmacists are effective channels for incentive targeting.

For Harlan and our family.

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Introduction

A key aim of health policy is to ensure that patients are able to access high quality care when appropriate while minimizing the over-use of unnecessary treatments. Achieving this balance requires incentive alignment between patients and providers. In many low-income countries, health care markets are decentralized, making top-down coordination difficult. However, both over- and under-treatment are ubiquitous across disease areas and have negative consequences for patient and community outcomes [1, 2]. For example, studies in both the US and China have found high levels of unnecessary antibiotic prescriptions [3, 4], which affects patient outcomes and contributes to growing rates of drug resistance, which is a societal cost. Studies across sub-Saharan Africa have found that large shares of malaria-negative patients receive antimalarials at health centers and pharmacies [5–8], which contributes to resistance and limits medication supply for those who may need it most.

Malaria is an important clinical area to study how decisions made by patients and providers impact quality of care because it is a clinically well-understood illness, it has a high disease burden, and nearly all deaths and serious illness are preventable through effective, inexpensive medication that can be targeted to patients [9]. Diagnostic tests are widely available across high-burden areas in sub-Saharan Africa and are highly accurate in confirming the presence or absence of malaria parasites in a symptomatic patient. In practice however, few malaria patients are diagnosed prior to getting treated. There are several reasons why diagnostic testing is low: relatively high cost of tests, affordable and accessible antimalarials, and established practices of using symptoms alone to determine malaria status are a few major contributors. Low diagnostic testing contributes to a gap between treatment and need: missed diagnoses result in avoidable illness, and over-prescription of antimalarials can lead to drug resistance. In the context of pharmacies, where over half of malaria patients in Kenya access treatment [10], this may be due to misaligned incentives between patients and pharmacists. Studies in Kenya have found that patients may not ask to be tested because they have prior beliefs about their malaria status [11], the cost of the test is prohibitive [6, 12], or they do not want to wait for the diagnostic test result. Pharmacists may skip testing because they are optimizing patient preferences and profit motivations [3, 13].

I test whether financial incentives can improve coordination between patients and providers to impact malaria treatment and cost-effectively mitigate the social costs of misallocating

antimalarials. Using a cluster randomized controlled trial in 140 pharmacies in malaria-prone counties in Kenya, I evaluate the effect of patient subsidies and provider performance incentives on malaria testing and treatment decisions. I investigate whether financial incentives are more effective at improving diagnostic testing and treatment targeting when they are given to patients through subsidies (demand-side), providers through performance incentives (supply-side), or both. While classical economic theory would suggest that demand- and supply-side incentives should result in the same impact on demand, they may differ if incentives are captured by the pharmacy and not passed through to patients, or if pharmacists encourage increased demand through non-price mechanisms such as improved counseling around the importance of diagnostic testing. I explore these hypotheses in this dissertation. Pharmacies were randomized to a status quo control group or one of three treatment groups: (1) patient subsidies for rapid tests and for artemisinin combination therapies (ACTs) conditional on a positive test; (2) pharmacy incentives for selling rapid tests to diagnose fevers and for selling ACTs conditional on a positive test; and (3) hybrid incentives (patient subsidies and pharmacy incentives) for rapid test use and ACTs for confirmed malaria-positive cases. This design allowed me to evaluate the impact of two-part incentive structures as well as to examine the causal effect of targeting that incentive to the patient (demand-side) or the provider (supply-side). Prior literature that has evaluated the impact of demand-side subsidies on malaria care has found them to be effective at improving testing but not as effective at improving test result adherence [6, 7, 12]. This study is the first to my knowledge to compare supply-side and demand-side incentives at this scale.

The study takes place in Kenya, where over 3.5 million people fall ill with malaria annually, with children, pregnant women and people living near Lake Victoria and on the coast most vulnerable to infection [14, 15]. Over half of malaria patients in Kenya access treatment via pharmacies, often the preferred access point for primary care given pharmacies' convenience and reliable presence even in areas that are underserved by public health care clinics and hospitals [10, 16]. Given that pharmacies play a crucial role in providing access to malaria case management in Kenya, it is essential that they provide appropriate diagnostic testing and low-cost, effective and appropriate medicines for treatment.

I find that both patient subsidies and provider incentives are effective at increasing diagnostic testing uptake and may be effective at improving malaria treatment targeting. Patient subsidies increase the likelihood that a symptomatic patient takes a rapid test by 27 percentage points, from a control group mean of 8 percent. Pharmacy incentives increase the likelihood of rapid test uptake by 20 percentage points, which is indistinguishable from the demand-side approach. This result is consistent with what has been found in prior literature on subsidies for health goods: demand-side subsidies are effective in encouraging adoption of the target behavior when the incentive is appropriately timed. I also find that provider incentives and the hybrid incentives increase the likelihood that a patient purchases ACTs with a diagnostic test by 7 percentage points compared to a control group mean of 6 percent, with directional evidence that demand-side incentives alone have similar effects. I find

that both demand- and supply-side incentives reduce the likelihood that a patient purchases malaria treatment without a confirmatory diagnosis by between 17-19 percentage points, compared to 81 percent in the control group. I find no evidence of intervention effects on the share of malaria patients seeking care at study pharmacies, suggesting that these findings are driven by behavior change rather than patient sorting. When testing whether incentives were passed through to patients, I find suggestive evidence that providers in the pharmacy incentive group did not pass through incentives to patients when compared to the patient subsidy group. This means that incentive pass-through is unlikely to be a key mechanism that explains why demand- and supply-side incentives yield similar effects on uptake of tests and treatment targeting, and instead, other mechanisms such as improved provider counseling to encourage testing, or other forms of supplier-induced demand, may explain this finding.

I complement the analysis on testing and treatment decisions with a cost-effectiveness analysis, which provides a more complete picture of how to efficiently allocate resources. When only considering implementer costs, I find that patient subsidies cost $3.26 for each additional patient that purchases an ACT and is malaria positive, and pharmacy incentives cost only $4.01 for each additional patient that purchases an ACT and is malaria positive, when compared to the status quo standard of care. The hybrid approach (combined patient subsidies and pharmacy incentives) costs $9.15 for each additional patient that purchases an ACT and is malaria positive, when compared to the status quo control group. When also considering the direct (medication) costs of over-treating malaria negative patients and the time costs for patient seeking care, I find that the patient subsidy and the provider performance incentive interventions are cost-saving, relative to the control group. The hybrid approach costs $24 for each additional patient that purchases an ACT and is malaria positive, compared to the status quo control group. This suggests that all interventions are relatively low cost when compared to the status quo, and that patient subsidies and pharmacy incentives may be cost-saving depending on the perspective taken.

This paper makes four contributions. First, it contributes to our understanding of how incentives targeted at the demand-side or the supply-side can affect health decision-making. Financial incentives are well-established tools used around the world to promote a wide range of health behaviors. Demand-side incentives all operate based on the assumption that either price itself is a barrier to adopting a health behavior, or an incentive can nudge people to overcome other nonpecuniary barriers. Price experiments for health treatments have shown that people do not respond uniformly to prices, and instead the nature of the health decision and timing of the benefits matter [6, 7, 17–19]. On the supply side, providers influence patient health decisions using their expertise, preferences, and sometimes biases, which can have significant effects on quality of care. They can act as gatekeepers to reduce unnecessary medical treatments, or promote overuse [3, 13]. Provider performance incentives have focused on improving quality of care, particularly for maternal and child health outcomes [20, 21]. These studies suggest that properly incentivizing providers can lead to improvements in

health care utilization and key health outcomes, but the evidence has been limited to a relatively narrow set of indicators and outcomes. This paper bridges the supply- and demand-side incentive literatures in the context of malaria care by comparing patient subsidies to provider performance incentives for the full cascade of care for suspected malaria.

Second, it adds to the literature on how individualized health information and financial incentives can be combined to change health behavior. I combine financial incentives for diagnostic testing and appropriate malaria treatment with tailored health information obtained from a diagnostic test result, and evaluate the effectiveness of information and incentives on health behavior change for the full cascade of care for malaria case management. Lack of accurate information is another barrier to adoption of desirable health behaviors. Evidence suggests that health information is necessary, but not always sufficient, to change health behaviors. However, information combined with financial incentives has shown more promise in encouraging health behavior adoption [22–24]. Additionally, the quality of the information matters: general health information tends to be less effective in changing individual behaviors than individually tailored messages targeted at the key decision-makers [25]. Studies that have examined whether the information provided by a malaria diagnostic test changes treatment-seeking behavior have found mixed results – information is effective in steering some patients towards appropriate treatment options, especially when coupled with an incentive, but many elect to ignore test results when making treatment decisions [6, 12]. This study combines targeted financial incentives with personalized health information in a way that leverages the two steps of the testing and treatment decision-making process.

Third, this paper contributes to our understanding of how pharmacists make decisions. Pharmacies are important access points to health care in many low- and middle-income country contexts and are under-studied in the literature on provider motivation. Prior studies in Kenya have used vouchers that patients could redeem at participating pharmacies but have not studied the decision of malaria case management from the pharmacist's perspective. There is one other ongoing study, to my knowledge, which tests pharmacy incentives and patient subsidies for malaria testing and treatment [26].

Finally, this paper adds to the evidence on the cost-effectiveness of financial incentives for improving malaria case management. I develop a cost-effectiveness framework to quantify the societal costs of over-treatment and benefits of appropriate treatment targeting from a health systems perspective. This is important to contextualize this program's impact and feasibility for scale. I can compare the cost-effectiveness of this approach to improve malaria care with others that have been evaluated in the literature. The framework that I develop for assessing cost-effectiveness can be extended to other settings that are characterized by diagnostic testing availability and over-treatment that can have negative social consequences.

The remainder of this dissertation is organized as follows: Chapter 1 provides background on malaria in Kenya, clinical malaria care guidelines, the institutional context of the experi-

ment, and existing literature on incentives for malaria care in the Kenyan context. Chapter 2 discusses the theoretical framework and predictions for the main research questions and outcomes. Chapter 3 describes the experimental design and provides information about the intervention implementation, describes the primary and secondary outcomes, and the empirical strategy. Chapter 4 presents experimental results on the main and secondary outcomes, and results from the cost-effectiveness analysis. Chapter 5 presents results for research questions around mechanisms and tertiary outcomes. Finally, the dissertation concludes with a discussion, overview of research and policy implications, and avenues for future research.

Chapter 1

Background

This chapter provides a description of the malaria burden in Kenya and recent policy advances for handling it, and the role of pharmacies. It follows with a description of the standard of care for appropriate malaria case management, and provides institutional context for the pharmacy network used in this study. Finally, it provides an overview of how this experiment fits in with the existing literature on demand- and supply-side incentives for health behavior change, with a focus on incentives for malaria case management.

1.1 Malaria in Kenya

There are an estimated 190 million malaria cases and 391,500 deaths in sub-Saharan Africa per year, with the poor bearing the brunt of this health and economic burden [27–29]. In Kenya, over 70% of the population is at risk of malaria, with children, pregnant women and people living near Lake Victoria and on the coast most vulnerable to infection [14].[1] True malaria rates vary by age, with young children having the highest rates of transmission and adults having much lower rates, conditional on being symptomatic. In Western Kenya, the malaria rate is 54 percent among symptomatic children under 5 and 14 percent among those above the age of 14 [6]. This strong relationship between malaria rates and age is well-established in the malaria literature [30]. Given the high variability of prevalence by age, interventions aimed at increasing diagnostic testing may be particularly cost-effective for detecting (and treating appropriately) adults and older children, who have lower transmission rates.

While the current mortality and morbidity burden of malaria remains high, it has fallen

[1] Historically, the peak rainy seasons in these areas occur between October-December, and March-June, but recent disruptions in weather patterns and El Niño/La Niña phenomena have led to delayed onset of rains and more unpredictable rainy seasons in recent years. Appendix Figure B.1, which plots antimalarial sales volumes over time from pharmacy level microdata from 2018-2020, shows that antimalarial sales spike between November - January and May - July but remain elevated all year in these endemic areas.

over the past decade with improvements in treatment accessibility and prevention [28]. This decline has coincided with targeted global efforts to increase the use of ACTs and decrease the price of these medicines in the public and private sectors. Since 2006, the public health sector in Kenya has offered malaria treatment for free conditional on diagnosis [31], but many patients seek care in the private sector to avoid common barriers such as long wait times, consultation fees, and unreliable medication supply [32]. In fact, over half of malaria patients access treatment via pharmacies, choosing to incur some monetary cost for treatment to get more reliable and faster access to care [6].

Availability of malaria treatment in the private sector is largely due to a subsidy program run by the Affordable Medicines Facility-Malaria (AMFm) between 2011-2016, which provided manufacturer incentives to lower prices of ACTs for patients to less than $1 USD per treatment course. In 2010, the AMFm initiative enacted a 95 percent ACT subsidy to pharmaceutical wholesalers in seven countries, including Kenya, to increase the availability and reduce price in the private sector. This approach was extensively evaluated, and in 2012 was modified to give countries more choice for how to use these funds. This subsidy program — which substantially increased access to antimalarials through Africa's private sector pharmacies and drug sellers — ended in 2016 but resulted in an enduring shift to low cost antimalarials in the private sector [33].

One unintended consequence of manufacturer-level subsidies for antimalarials has been low levels of diagnostic testing and high levels of unnecessary treatment. Prior work in Kenya has found that only 29% of patients who self-report having malaria were diagnosed [6]. One study of 677 malaria-symptomatic children in Homa Bay, Kenya finds that 42 percent of them have laboratory-confirmed malaria, and among the remaining malaria-negative children, more than half of them are also prescribed antimalarials [34]. In another study examining prescribing behavior over a year in a large Kenyan hospital, researchers find that the rate of antimalarial prescription is 4.2 times higher than the number of positive tests reported [35]. This is consistent with recent sales trends in Kenyan pharmacy data: malaria tests (rapid test or microscopy) account for only 10% of antimalarial product sales.[2]

1.2 Malaria case management

Malaria presents with general flu-like symptoms including fever, chills and headache. Because malaria symptoms are non-specific, diagnosis based on symptoms alone is insufficient. Instead, the World Health Organization and the Kenyan National Malaria Guidelines rec-

[2]Despite low levels of testing, pharmacy staff are knowledgeable about appropriate case management: 78% of providers who responded to a clinical vignette about appropriate case management at baseline (N=233) said they would first have the patient who presented with malaria-like symptoms obtain a diagnostic test prior to prescribing treatment.

ommend that all cases of suspected malaria be diagnosed by microscopy or rapid test.[3] Both sets of guidelines limit antimalarial treatment to cases with positive tests, and patients with negative test results should be reassessed for other common causes of fever and treated appropriately [9, 38]. After diagnosis, the recommended treatment for uncomplicated malaria is a three-day course of artemisinin combination therapy (ACT) for children and adults.[4]

Accurate diagnosis of suspected malaria prior to treatment provides private benefits by giving information to individuals about their illness status and social benefits by decreasing overall rates of over-treatment of febrile illness with antimalarials, which can lead to patients getting incorrect treatment regimens for their illness, and more broadly, increased drug resistance and wasted resources [39]. There is not a consensus on how to quantify the private and social costs of over-treatment with antimalarials. For the individual, private costs of over-treatment include costs of unnecessary medication and costs of delayed diagnosis and appropriate treatment. For society, over-treatment can lead to drug resistance, which has costs that parallel costs of antibiotic resistance. These include costs of morbidity and mortality of those infected with drug-resistant strains, resources for appropriate infection control programs, and research and development of new treatments that are effective against drug-resistant strains [40, 41]. Private and social benefits may not perfectly align: the cost of a rapid test is equivalent to the cost of antimalarials, and patients must wait 15 minutes for test results so they may prefer to just purchase treatment and then return to the pharmacy if symptoms persist. The primary social benefit of testing is to prevent drug resistance, which is an emerging problem with antimalarials in sub-Saharan Africa [39]. This is particularly important as individuals may make decisions that seem optimal for themselves in the short run, without internalizing the potential downstream effects of their choices.

1.3 Institutional context: Maisha Meds pharmacy network

Maisha Meds, the study's implementing partner, works with a network of over 600 private sector pharmacies across Kenya. Pharmacies within the Maisha Meds network serve over 200,000 patients each month. In order to be a Maisha Meds pharmacy, pharmacies must be active users of a digital sales and inventory management platform. This platform logs all transactions that occur at the pharmacy, and tracks inventory of medications and pharmacy products. This platform is designed to help pharmacies manage their business, as opposed to a pen and paper based approach to logging sales and inventory. For the purposes of this

[3] Rapid diagnostic tests have a sensitivity of 78% and a specificity of 94%, compared to microscopy which is 57% sensitive and 99% specific [36, 37]. In Kenya, microscopy costs around $1.70 USD (excluding any consultation fees) and rapid tests cost around $1.00 USD (average prices from pharmacy sales data from 2019-2021).

[4] More specifically, this is the treatment course for uncomplicated *P.Falciparum* malaria, for children and adults, excluding pregnant women in their first trimester.

study, Maisha Meds developed a supplementary digital tool to complement the sales tracking system. This tool, called the "Loyalty Program", administers incentives to either patients (subsidies) or pharmacy owners and staff (performance incentives) for recording sales of specific products. For the study, the incentivized products are malaria rapid diagnostic tests and quality-assured ACTs. These products were made available to pharmacies enrolled in the program on consignment, and the program set retail prices for each product in accordance with the specification of the study design. In the patient subsidy arm and in the hybrid arm, discounted retail prices were set for rapid tests and treatments and patients were expected to pay the discounted price set by the program. In the pharmacy incentive arm, there were no discounts set by the program for rapid tests or treatment, and instead the listed retail prices for these products are the average market-level retail price. Pharmacies in the pharmacy incentive arm were told that they could further discount those products to clients if they chose.

Sales for products through the loyalty program are recorded in the digital platform, patients pay the set prices for the diagnostic test and record their malaria test result in the digital platform, and pay for the treatment at the set price (incentivized if the test is positive, otherwise no incentive). Maisha Meds then reimburses pharmacies on a weekly basis for all approved transactions through the Loyalty Program, paying out any incentives and profit margins that are associated with each approved sale. At the beginning of the study, Maisha Meds staff trained all pharmacy owners and staff on how to use the digital platform capabilities, and throughout the study conducted monitoring to ensure compliance with implementation protocols and prevent fraudulent claims. Pharmacies in the study sample were reminded to log all of their pharmacy sales (whether or not they were participating in the Loyalty Program) in the digital point of sale tool, and to offer the Loyalty Program malaria tests to all patients who sought care for malaria-related symptoms or products.

1.4 Demand- and supply-side incentives for health behavior-change

Financial incentives and subsidies are well-established tools used around the world to promote a wide range of health behaviors. Price experiments for health treatments have shown that people do not respond uniformly to prices, and instead the nature of the health decision and timing of the benefits matter significantly when determining whether providing a product for free (or even a negative price) or charging a positive price for it will yield a socially optimal outcome [17, 18].

Disease screening and diagnostic testing are often considered promising applications for incentives, given that hassle costs and procrastination are common barriers to uptake and that small monetary nudges can overcome these barriers. The hassle cost of getting screened or diagnosed may lead people to choose to forego these essential tests (or procrastinate). The

work in this area highlights the importance of non-monetary barriers to take-up, and that incentives can help overcome these barriers. A particularly well-known experiment in Malawi finds that providing patients with a financial incentive increased the likelihood that individuals sought their HIV test results from 35% to 78% [42]. The incentive that was provided was relatively small - an hour's wage - but resulted in a large behavioral response. Demand-side financial incentives have also been shown to increase immunization rates in Udaipur [43], with the authors also citing procrastination or present-bias as an underlying mechanism. Research on financial incentives and malaria diagnostic testing is discussed below in more detail, but overall the evidence suggests that offering tests for free or at a reduced price does increase diagnostic test rates.

All of the studies discussed above consider demand-side incentives rewarding a specific behavior. This experiment offers incentives for the full continuum of care for malaria: the dual testing and treatment decisions.

Provider behavior can be changed using performance incentives in a way that has shown to improve health outcomes, and this experiment is one of the first to evaluate the impact of pharmcist performance incentives on malaria case management. There is a small but growing literature on performance pay schemes that incentivize quality of care in global heath contexts, but this work has been limited to maternal and child health. These studies have shown positive impact of performance incentives on important child health outcomes at birth and in early life. One study on performance pay in India focused on improving health worker attendance [44], with findings suggesting that incentives work in the short term to improve attendance outcomes and children's birth-weight, but they are operationally intensive to administer. In Rwanda, performance incentives for quality results in improved child health outcomes as a result of increased use and quality of pre- and post-natal care [20, 45]. In order to isolate the effect of the incentive, Basinga and coauthors increased the input budget in comparison clinics by the average performance payments made in treatment facilities. In Argentina, a national performance pay program targeting prenatal care and birth outcomes finds that these incentives are effective at improving attendance at prenatal visits, reducing neonatal mortality, and reducing low birth-weight, among other prenatal and birth outcomes [21].

These studies all demonstrate that properly incentivizing providers can lead to improvements in health care utilization and key health outcomes, but the evidence has been limited to a relatively narrow set of indicators and outcomes, as described above. This study expands this literature by looking at performance incentives in the context of malaria care and with pharmacists, an understudied but crucial care provider in many settings around the world. Finally, a systematic review on performance-based-financing in global health highlights that despite the enthusiasm for implementing performance-based-financing programs, there is relatively little rigorous evidence on whether these approaches are successful in improving quality of care and are cost-effective compared to alternative approaches

[46]. Between 2006-2013, the number of African countries using performance-based-financing approaches increased from 4-21, yet there is little evidence from low and middle income countries about whether these approaches are effective or cost-effective. Out of 45 studies selected for screening in this systematic review, only 7 were included in the final set of studies. Among these 7 studies, the evidence on the effectiveness of results-based-financing program was mixed (mostly descriptive analysis, some quasi-experimental work), and none of the studies considered cost-effectiveness analysis. This experiment adds to the evidence base of performance-based-financing on health outcomes by providing rigorous evidence on effectiveness and cost-effectiveness of results-based-financing for malaria care.

Subsidies for malaria case management in Kenya

Over the past decade, there have been many efforts to increase usage of diagnostic testing for malaria in Kenya which have largely focused on using patient subsidies. There are three overarching findings from this literature: (1) subsidizing rapid tests increases diagnosis rates, (2) subsidizing high quality treatment (ACTs) increases patient demand for it, and (3) subsidizing diagnostic tests has mixed results for subsequent treatment decisions. Early efforts examining the effect of subsidizing rapid tests on testing and treatment decisions found that subsidies increase use of diagnostic tests and make some progress on improving treatment targeting but do not close the gap [47, 48].

There are two seminal studies in the last decade that evalued the impact of subsidies on testing and treatment decisions in Kenya. A large randomized controlled trial led by Cohen, Dupas and Schaner experimentally varied subsidy levels for ACTs and cross-randomized a rapid test subsidy in Kenya's malaria endemic areas. This study evaluated the extent of the trade-off between access to high quality antimalarials (ACTs) and appropriate treatment targeting at different subsidy levels, and whether introducing subsidized rapid tests improved treatment targeting [6]. They found that subsidizing treatments increased use, and that relatively more modest subsidy levels improved targeting. They also found that rapid test subsidies increased testing uptake but did not improve treatment targeting. A more recent randomized controlled trial in Kenya by Prudhomme O'Meara and colleagues introduced a free rapid test administered by community health workers and a voucher for subsidized treatment for malaria positive individuals that could be redeemed at a pharmacy [12]. Authors of this study found that the combined subsidy increases diagnostic testing and improves treatment targeting.[5]

All of these papers suggest that price is an important barrier for patients when making decisions about malaria testing and treatment, and that using price subsidies to encourage diagnostic testing can improve uptake and can make some progress in improving treatment

[5]At the time of filing this dissertation, another research group from Duke University, Moi University, and the Clinton Health Access Initiative were conducting a linked field experiment in Nigeria and Kenya comparing pharmacy incentives and patient subsidies for malaria testing and treatment [26].

targeting. However, the size, delivery mechanism and conditionality of the subsidies matter and there is not a consensus yet on the best combination of these elements.

In summary, this study adds to the evidence base on how financial incentives targeted at the demand-side or the supply-side can affect health decision-making. Secondly, it contributes to the literature on how individualized health information and financial incentives can be combined to change health behavior for the full cascade of malaria care, rather for a single health decision. Third, it contributes to our understanding of how pharmacists make decisions, and how results-based-financing mechanisms may affect pharmacist behavior. And finally, it adds to the evidence on the cost-effectiveness of subsidy and results-based-financing approaches in improving malaria case management.

Chapter 2

Theoretical Framework

The primary goal of clinical decision-making is to ensure that patients who seek care are given the diagnosis and treatment recommendations that are best suited to their illness episode. This requires collaboration between the patient and provider, as well as interpretation of often noisy and incomplete signals. For many common illnesses, providers can rely on clinical guidelines that provide a set of decision rules to aid in diagnosis and correct case management. For malaria, clinical guidelines are clear: confirm the malaria diagnosis with either a rapid diagnostic test or microscopy prior to administering antimalarials. Rapid tests for malaria are widely available, affordable, and can be administered by a wide range of health professionals. Despite this, most malaria cases are treated presumptively, without any formal diagnosis. Lack of testing for malaria can be due to various supply- and demand-side barriers that are discussed in more detail elsewhere in this dissertation: provider unwillingness to prescribe tests to patients, lack of patient demand for tests due to relatively high financial and time costs, perceptions of malaria illness status based on symptomatic presentation, and lack of supply of tests are all contributing factors.

Two types of errors can occur when antimalarials are administered without a diagnostic test: overtreatment of malaria-negative individuals with antimalarials, and undertreatment of malaria-positive individuals with ACTs (Figure 2.1). Overtreatment occurs when malaria negative patients still get prescribed antimalarials. This is problematic for two reasons: first, the patient does not receive the appropriate care that she needs given her true underlying illness status.[1] Second, over-prescribing antimalarials can lead to a rise in drug-resistant strains of malaria. This is a well-founded public health concern,[2] and individuals may not make private decisions that align fully with what is best from a societal perspective. Un-

[1] In practice, there are different possible diagnoses given a set of observable symptoms for a malaria-negative patient, including harmless viral infections, and more serious problems that require different treatments to cure.

[2] Chloroquine-resistant P. falciparum has spread to nearly all areas of the world where falciparum malaria is transmitted, making this drug ineffective.[28]

dertreatment occurs when malaria-positive patients do not get prescribed ACTs. This may lead patients with a confirmed need for high-quality medication to take a less effective option. This could in turn delay their recovery and put more strain on the health care system, especially if their illness progresses from an uncomplicated to a complicated case of malaria.

Here, I discuss a simple framework of patient and pharmacist decision-making that illustrates their objective functions,[3] and then illustrate how supply- and demand-side incentives may shift behavior towards improved diagnoses and treatment targeting. This theoretical framework informs my experimental design and main hypotheses comparing demand- and supply-side incentives. Then, I aggregate the individual testing and treatment decisions to the population level and discuss how these parameters are measured in the experimental design.

2.1 Patient and pharmacist decisions

Patients and pharmacists make a series of coordinated decisions, which can be influenced by a variety of factors, to appropriately manage a suspected malaria case. I illustrate this sequence for patients in Figure 2.2.[4] The starting point for this framework is that the patient is symptomatic and has decided to seek care at a pharmacy. The first decision that the patient makes is whether or not to take a diagnostic test, which is represented by Step 1 in the figure. The decision to test depends on factors like availability, the pharmacist's recommendation, cost, and the patient's own beliefs about her illness status. If the diagnostic test is expensive, especially relative to the treatment, the patient may avoid purchase due to low willingness or inability to pay.[5] If the patient does not get diagnosed, she does not gain any additional information about her illness status, and she must decide whether to purchase ACTs or not. If the patient does get diagnosed, she learns her malaria status with a high degree of certainty (Step 2 in the figure). At this point, the treatment choices are the same as if she did not get diagnosed, but the clinically appropriate course of action is clear, as illustrated in Step 3 in the figure. From a public health perspective, the optimal end states are illustrated in the green boxes in the figure: malaria positive patients obtaining ACTs and malaria negative patients not obtaining ACTs or other antimalarials.

Arriving at these ideal outcomes also requires that the pharmacist be willing to recommend testing and appropriate treatments. Pharmacists instead balance patient utility with their own profit motivation when they decide what to prescribe for a given patient encounter.

[3] This conceptual framework and model build from models developed in Lopez et al 2020 [13] and Cohen, Dupas and Schaner [6].

[4] Because this study's target population is febrile patients who seek care at pharmacies, the conceptual framework restricts the scope of the decision to after a patient has already made the decision to seek care in the private sector as opposed to either (a) not seeking care at all, or (b) seeking care at a public clinic.

[5] See Technical Appendix A.1 for the derivation of the patient's testing and treatment decision.

This is represented by the pharmacist utility function below:

$$V_t(U^*, \Pi_t) = \alpha E[U^*] + (1 - \alpha)\Pi_t$$

where $E[U^*]$is the optimal expected utility for patients, Π_t represents pharmacy profits from a particular transaction, and α represents the weight that the pharmacist places on the patient's utility as opposed to profitability, which can be thought of as an altruism parameter measuring pro-patient preferences.

The interventions tested in this study aim to align patient and pharmacist objective functions by using financial incentives that incentivize testing and only treating malaria-positive cases. For patients, this reduces the cost barrier to testing and incentivizes use of ACTs if appropriate. The additional disincentive of unsubsidized medication after a negative test further disincentivizes patients from purchasing antimalarials unnecessarily. By incentivizing pharmacists to diagnose, they may encourage patients to get tested (supplier-induced demand). Additionally, incentivizing patients to make treatment recommendations that are linked to the diagnostic test outcome may also encourage patients to choose treatment options that are based on clinical need, rather than perceived illness status. In the case of malaria care, low rates of testing and high rates of treatment misuse is evidence of behavioral hazard [49]. Supplier-induced demand in this setting can actually encourage use of high value care, and move patients more towards optimal outcomes. For pharmacists, these incentives make diagnostic testing more attractive from the perspective of firm profitability by compensating for any lost medication sales that would come from malaria-negative patients choosing to not purchase antimalarials.

2.2 Theoretical model and predictions for comparing supply/demand incentives

Now, I generate the predictions that inform the hypotheses tested in the experiment when comparing supply and demand-side incentives for testing and treatment targeting.[6] I motivate this first by considering a competitive market environment (standard model), and then a market environment characterized by institutional constraints on price setting.

Perfect competition: standard model

Let the aggregate demand for rapid tests be $x(p) = \sum_i x(p_i)$, where p is the price of rapid test for each purchase i, $x()$ is a function that is continuous and non-increasing at all $p > 0$. Similarly, let aggregate supply of rapid tests be $q(p) = \sum_i q(p_i)$, where $q()$ is a function that is continuous and non-decreasing at all $p > 0$. Finally, let p^* be the solution to $x(p^*) = q(p^*)$, the equilibrium price which equates aggregate supply and demand.

[6] I only illustrate the rapid test case, as the treatment targeting case is parallel.

Suppose that there is a change in p through the introduction of a subsidy of size s. Below are the predictions generated by the introduction of a demand-side subsidy or a supply-side incentive of size s in a perfectly competitive environment:

- *Prediction 1:* In a perfectly competitive market without constraints on price-setting, if patients are fully informed about the health benefit of testing and appropriate malaria treatment so that there is no scope for supplier induced demand, the subsidies would be fully passed through to patients. In this market, supply of tests and treatment is perfectly elastic because pharmacies can procure enough additional tests and treatments to fulfill higher demand induced by the addition of a subsidy/incentive. In this case, the change in equilibrium quantity only depends on the demand elasticity.
- *Prediction 2:* If instead pharmacies have local market pricing power, then the economic incidence of the incentive s will be split between patients and pharmacists. The share of s that is passed through to the patient vs. captured by the pharmacist, and by extension equilibrium quantity, depends on the relative elasticities of supply and demand.
- *Prediction 3:* If pharmacies have local market power and also have scope for supplier-induced-demand - or, increasing demand for tests and treatment through non-price mechanisms - the the presence of a subsidy s increases the profitability of supplier-induced demand, so they will expand this. In this case, the change in equilibrium quantity depends on the effectiveness of supplier-induced demand.
- *Prediction 4:* In any of the three cases above, supply-side and demand-side incentives of the same magnitude increase (decrease) equilibrium quantity (price) by the same amount. If this is the case, I expect to see no difference in rapid test uptake and treatment targeting between demand- and supply-side approaches, or the combination of the two (hybrid approach).

Model predictions with institutional constraints on price-setting

The pharmacy environment in this study is one where there are institutional constraints in place that limit pharmacies' abilities to set their own prices for malaria tests and treatments. In particular, in the patient subsidy and the hybrid treatment arms, discounted prices for rapid diagnostic tests and ACTs are set by the implementation organization and these prices are made known to patients seeking care at the time that they visit participating pharmacies. There are posters in the pharmacies that list the discounted prices, patients receive a text message confirmation with the price they should expect to pay prior to making the decision to be tested, and the discounted prices are listed in the digital tool so they are transparent and visible at every transaction to both the patient and the pharmacist.

If we assume the same model set up as above, but with the additional feature that prices to patients cannot be set by pharmacies in the intervention arms (they are set centrally by the subsidy program), we would expect the different effects on price and quantity than in

the standard model. We can still denote the equilibrium price received by pharmacists in each intervention arm as $p*_t^s$, the equilibrium price paid by patients as $(p*^s - s)_t$, and the equilibrium quantity sold as $q*_t$, where $t \in (d, s, h)$.

This generates the following predictions for how the various interventions would impact prices and quantities:

- *Prediction 5:* With constraints on price-setting and no scope for supplier-induced-demand, then at least some supply-side incentives will be captured by the pharmacy,[7] which will result in a smaller change in equilibrium quantity and price relative to demand-side subsidies of the same magnitude ($q*_s < q*_d$ and $(p^s - s)_d < (p^s - s)_s$). I expect that demand-side subsidies will yield higher uptake of rapid tests and treatment targeting than supply-side incentives.
- *Prediction 6:* With constraints on price-setting and scope for supplier-induced demand (increasing demand for tests and treatment through non-price mechanisms), pharmacies will have more incentive to exercise supplier-induced demand when the subsidy is on the supply side (and they are able to capture more of the subsidy compared to the demand-side subsidy arm). Supply-side incentives will induce pharmacists to sell more rapid tests, which will result in a larger change in equilibrium quantity relative to demand-side subsidies of the same magnitude ($q*_d < q*_s$) [8]. If this is the case, and the effects induced by supplier induced demand are larger than the effects via price-elasticity of demand with demand-side subsidies, I expect that supply-side incentives will yield higher uptake of rapid tests and treatment targeting than demand-side subsidies.
- *Prediction 7*: With the same set up as Prediction 6, and the effects induced by supplier induced demand are smaller than the effects via price-elasticity of demand with demand-side subsidies, I expect that demand-side subsidies will yield higher uptake of rapid tests and treatment targeting than supply-side incentives.
- *Prediction 8*: With constraints on price-setting in the demand-side incentive arm and the hybrid arm, the hybrid intervention will result in a change in equilibrium quantity and price that is in between demand- or supply-side incentives alone if the effects are linear in the incentive amount.
- *Prediction 9*: With constraints on price-setting in the demand-side subsidy arm and the hybrid arm, and if the costs of engaging in supplier-induced demand are increasing in q (the number of tests sold to patients), then the hybrid intervention will result in a change in equilibrium quantity and price that is greater than either the demand- or

[7] The amount of what is captured by the pharmacy depends on the relative elasticities; only if supply is perfectly inelastic will the full subsidy be captured by the pharmacy, if it is upward sloping then some of that will be passed through to patients

[8] Again, the magnitude of the effect size differences in quantity (price) depends on the elasticity of demand in the demand-side subsidy arm, and the degree to which supplier induced demand is able to shift the demand curve out at all prices.

supply-side incentives alone (complementarities).

Depending on the relative elasticities of supply and demand, and the ability of pharmacies to set prices, demand- and supply-side incentives may have different implications for rapid test uptake and treatment targeting. My hypotheses make predictions for both quantity and price, however I will only be able to measure changes in quantity through my experiment. I link the theoretical predictions here to the research questions and hypotheses in Chapter 3.

2.3 Treatment targeting

The ideal outcomes from a public health perspective are that (1) confirmed malaria positive patients choose to be treated with high quality antimalarials (ACTs), and (2) malaria negative patients choose to not be treated with antimalarials, and instead seek further consultation for their symptoms. I write these end state outcomes as conditional probabilities:

$$Pr(a_1)^{m_1} = Pr(a_1)^{m_1 t_1} Pr(m_1)^{t_1} Pr(t_1) + Pr(a_1)^{m_1 t_0} Pr(m_1)^{t_0} Pr(t_0) \tag{2.1}$$

$$Pr(a_0)^{m_0} = Pr(a_0)^{m_0 t_1} Pr(m_0)^{t_1} Pr(t_1) + Pr(a_0)^{m_0 t_0} Pr(m_0)^{t_0} Pr(t_0) \tag{2.2}$$

where for $i \in \{0, 1\}$: a_i is whether the patient takes an ACT, m_i is whether the patient has malaria (true illness status), and t_i is whether the patient takes a rapid test. The superscripts are the conditionality statements: for example, $Pr(a_1)^{m_1}$is the probability that an individual takes an ACT conditional on being malaria positive. Additionally, $Pr(t_0) = 1 - Pr(t_1)$. In order to measure the probability that an individual takes an ACT conditional on being malaria positive (Equation 2.1), I need to measure the probability that (1) an individual takes an ACT, conditional on being tested and malaria positive, (2) an individual is malaria positive conditional on being tested, (3) an individual is tested, (4) an individual takes an ACT, conditional on not being tested and being malaria positive, (5) an individual is malaria positive conditional on not being tested, and (6) an individual not being tested. Equation 2.2 can be interpreted similarly.

Each of these components will be measured in the experimental design and allows for identifying the share of malaria positive individuals who obtain ACTs and the share of malaria negative individuals who do not obtain antimalarials — the two dimensions of treatment targeting.

I expect the interventions to increase rapid test uptake, and appropriate ACT targeting through three mechanisms: (1) subsidized testing and treatment conditional on confirmed positivity, (2) increased incentive for pharmacists to promote testing and ACTs if positive,

and (3) increased supply. Reducing the price of testing is expected to increase the probability of patients choosing to get tested, which will increase the information available to patients and pharmacists when recommending treatment options. Reducing the price of ACTs conditional on a positive diagnosis is expected to increase the probability of malaria-positive patients choosing to take high quality treatment. Providing pharmacists with incentives for prescribing rapid tests is expected to increase the probability of pharmacists offering symptomatic patients a rapid test, and incentives for treatment targeting is expected to increase the probability of pharmacists offering malaria positive patients ACTs. Finally, increased supply of both rapid tests and ACTs will ensure that these products are available when patients seek treatment.

We can differentiate Equations 2.1 and 2.2 with respect to rapid test price (c_t) and treatment price (c_a), to show how changes in price may affect each of the components in these equations[9]:

$$\begin{aligned} P'_{c_t}(a_1)^{m_1} = {} & P'_{c_t}(a_1)^{m_1 t_1} Pr(m_1)^{t_1} Pr(t_1) + P'_{c_t}(m_1)^{t_1} Pr(a_1)^{m_1 t_1} Pr(t_1) + \\ & P'_{c_t}(t_1) Pr(a_1)^{m_1 t_1} Pr(m_1)^{t_1} + P'_{c_t}(a_1)^{m_1 t_0} Pr(m_1)^{t_0} Pr(t_0) + \\ & P'_{c_t}(m_1)^{t_0} Pr(a_1)^{m_1 t_0} Pr(t_0) + P'_{c_t}(t_0) Pr(a_1)^{m_1 t_0} Pr(m_1)^{t_0} \end{aligned} \quad (2.3)$$

$$\begin{aligned} P'_{c_t}(a_1)^{m_0} = {} & P'_{c_t}(a_1)^{m_0 t_1} Pr(m_0)^{t_1} Pr(t_1) + P'_{c_t}(m_0)^{t_1} Pr(a_1)^{m_0 t_1} Pr(t_1) + \\ & P'_{c_t}(t_1) Pr(a_1)^{m_0 t_1} Pr(m_0)^{t_1} + P'_{c_t}(a_1)^{m_0 t_0} Pr(m_0)^{t_0} Pr(t_0) + \\ & P'_{c_t}(m_0)^{t_0} Pr(a_1)^{m_0 t_0} Pr(t_0) + P'_{c_t}(t_0) Pr(a_1)^{m_0 t_0} Pr(m_0)^{t_0} \end{aligned} \quad (2.4)$$

Changing the price of a rapid test affects the end state probabilities in three places: (1) testing, (2) malaria positivity conditional on testing, and (3) treatment conditional on positive test.[10] The interventions are designed to experimentally vary the price of testing and treatment, so I expect to see exogenous variation in each of these probabilities through the experiment. These end state probabilities are inputs to the cost effectiveness analysis, the methodology and assumptions of which are described in Chapter 3.

2.4 Figures

[9] I reproduce only the differentiation with respect to test price, as the result for treatment price follows a parallel structure.

[10] These also allow me to recover the probabilities of not getting treatment conditional on being malaria negative.

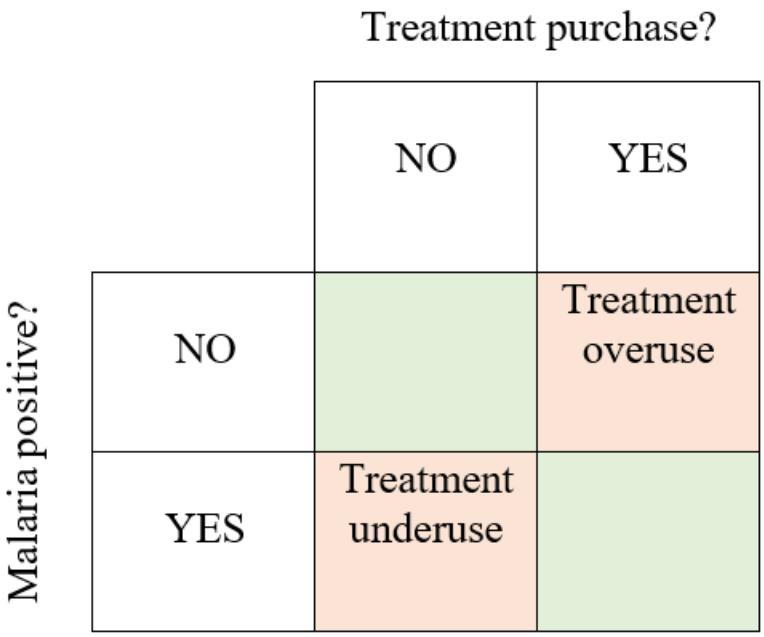


Figure 2.1: Types of errors (Back: 2.1)

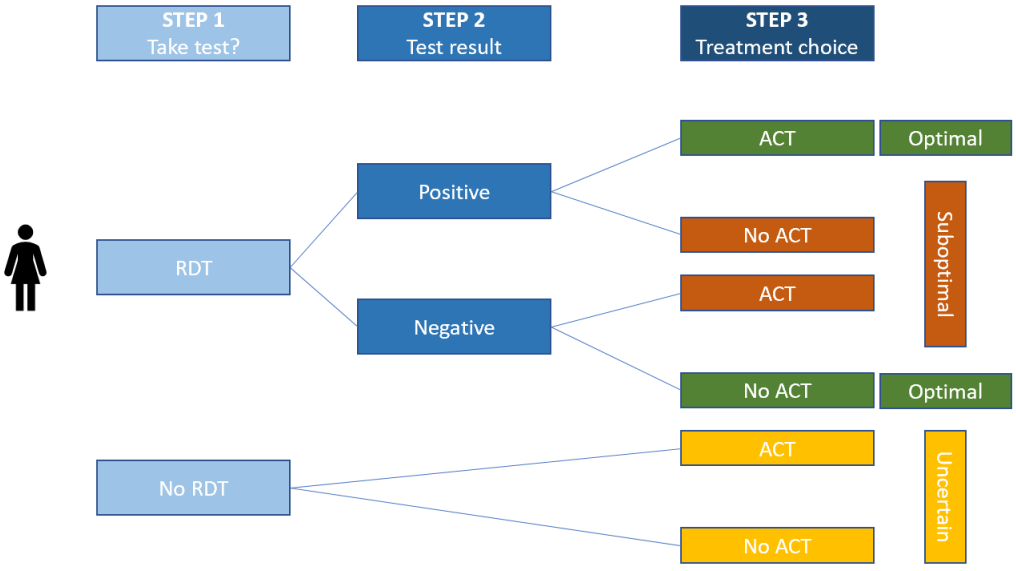


Figure 2.2: Patient decision to test and treat (Back: 2.1)

Chapter 3

Experimental Design and Methods

This chapter provides a detailed description of the study design. I begin with the research questions and hypotheses and continue with a description of the study site, experimental treatments, and experimental design. I then describe the empirical strategy for the study, beginning with a description of the study timeline and data sources, including the key outcome variables and how each is measured. I then proceed with a description of the analysis approach for testing the research hypotheses.

3.1 Research questions and hypotheses

This study attempts to address several research questions, which in turn inform a set of research hypotheses.

1. Do targeted incentives (patient, pharmacy, or both) for rapid diagnostic tests lead to higher malaria diagnosis rates relative to the status quo?

   Hypothesis 1: Targeted incentives for diagnostic tests lead to higher diagnostic testing rates, relative to the status quo.

   I will test this hypothesis by measuring uptake of diagnostic tests across pharmacies with varying subsidized price offers and pharmacy incentives for diagnostic test sales. I will have one treatment group with a high patient subsidy, one treatment group with high pharmacy incentive, one hybrid group with a moderate patient subsidy and pharmacy incentive, and a control group (no subsidy or incentives) in a network of pharmacies that comprise the sample frame over the course of the study period. I hold the total incentive amount fixed across all interventions arms, allowing me to look at the targeting mechanism. This means that the discount/incentive amounts in the patient or provider only arms are not directly comparable to those

tested in the hybrid group that gets both incentives. Prior studies have demonstrated that subsidizing diagnostic tests improves uptake of testing [6, 50]. If this study finds results that are inconsistent with previous findings, then it could be that patients are more aware of the availability and effectiveness of diagnostic tests and have reasons other than price for choosing whether to uptake.

2. Do targeted incentives (patient, pharmacy, or both) for diagnostic testing and diagnosis-conditional incentives for high quality treatments (ACTs) lead to improved treatment targeting, as defined by (a) the proportion of ACTs sold with an accompanying test result and (b) the proportion of antimalarials sold without a test, relative to the status quo?

   Hypothesis 2: Targeted incentives for diagnostic tests and diagnosis-conditional incentives for high quality treatments lead to improved malaria treatment targeting, relative to the status quo.

   I will test this hypothesis by measuring the proportion of all ACTs sold that go to patients with a confirmatory malaria diagnosis in each intervention arm, compared to the control group. Additionally, I will compare the proportion of all antimalarials sold without an accompanying test to measure whether interventions discourage malaria-negative individuals to purchase medication unnecessarily. Prudhomme O'Meara and colleagues (2018) examine the effectiveness of diagnosis-conditional subsidy for ACTs on treatment targeting in Kenya with CHWs administering free rapid tests and then an ACT voucher based on test result. They find that the conditional subsidy improves treatment targeting. This study will be the first to examine the value of a two-part subsidy administered entirely at the point of sale, leveraging the pharmacist role and targeting people who have already made the decision to seek treatment by going to the pharmacy. This study is also one of the first to examine the impact of a pharmacist-directed incentive on these outcomes.[1]

3. Does demand-side targeting of incentives for malaria care to patients lead to differential uptake of diagnostic testing and diagnosis-appropriate malaria treatment as compared to providing a supply-side incentive of the same magnitude?

   Hypothesis 3a: Demand-side subsidies lead to higher rapid test use relative to supply-side subsidies, suggesting firms capture at least some of the incentives when supply-side incentives are given, resulting in incomplete pass through of subsidy to patients, in this setting where pharmacy price-setting is not possible due to prices

[1] There is one other ongoing study, to my knowledge, that is looking at patient vs. pharmacy incentives for malaria care in Nigeria and Kenya [26]

being set externally. This links to Prediction 5 from Chapter 2, if there is no scope for supplier-induced demand, and Prediction 7 from Chapter 2, if there is scope for supplier-induced demand and the effects induced by supplier induced demand are smaller than those induced by the price-elasticity of demand via the demand-side subsidies.

Hypothesis 3b: Demand-side subsidies lead to lower rapid test use relative to supply-side subsidies, suggesting firms encourage sales through supplier-induced-demand when the incentive is administered on the supply-side, in this setting where pharmacy price-setting is not possible due to prices being set externally. This links to Prediction 6 from Chapter 2, where the effects of supplier-induced demand are larger than the effects of price-elasticity of demand via the demand-side subsidy.

Hypothesis 3c: Demand-side subsidies lead to improved malaria treatment targeting relative to supply-side subsidies, suggesting firms capture at least some of the incentives when supply-side incentives are given, resulting in incomplete pass through of subsidy to patients, in this setting where pharmacy price-setting is not possible due to prices being set externally. This links to Predictions 5 and 7 from Chapter 2, following the same logic as Hypothesis 3a.

Hypothesis 3d: Demand-side subsidies lead to worsened malaria treatment targeting relative to supply-side subsidies, suggesting firms encourage sales through supplier-induced-demand when the incentive is administered on the supply-side, in this setting where pharmacy price-setting is not possible due to prices being set externally. This links to Prediction 6 from Chapter 2, following the same logic as Hypothesis 3b.

Hypothesis 3e: Demand-side subsidies and supply-side incentives have no differential impact on testing and treatment targeting. This hypothesis links to Predictions 1-4 in Chapter 2, and would be suggestive of a competitive market environment which allows for pass-through of incentives.

These hypotheses directly relate to the theoretical framework described in Chapter 2, and will provide novel evidence on the extent of pass-through of incentives or the presence of supplier-induced demand in this setting, which is characterized by constraints on price-setting by individual pharmacies.

4. Are there complementarities between demand- and supply-side incentives for rapid test use and malaria treatment targeting?

Hypothesis 4: Combined incentives are more effective at increasing rapid test

use and/or improving treatment targeting than both demand- and supply-side incentives alone, suggesting that the effects of supplier-induced demand are increasing in the number of patients purchasing tests (q) and therefore a supply-side incentive of a smaller magnitude coupled with a demand-side incentive will yield larger effects than either of the two approaches alone. This links to Prediction 9 from Chapter 2, which would suggest that there are nonlinearities in the elasticity of demand and decreasing returns to engaging in supplier-induced demand (cost of supplier-induced demand is increasing in q).

5. Do the incentive interventions affect malaria care seeking behavior overall, compared to the status quo?

   Hypothesis 5: The incentive interventions do not increase or decrease the share of patients purchasing malaria tests or treatments in participating pharmacies, compared to the status quo.

   Understanding how the interventions may affect patient care-seeking behavior is important to determine whether the interventions operate through patient selection (shifting more malaria-care-seeking patients to intervention sites compared to the control) or behavior change at the individual patient level, without selection. I will measure whether the interventions affect the share of malaria patients as a proportion of the total patients served at each pharmacy to determine whether patient selection is occurring in the sample.

6. Is there evidence of incentive pass through in the pharmacy incentive arms compared to the patient subsidy arms?

   Hypothesis 6: Pharmacies do pass through a portion of their incentive on to malaria patients in the form of reduced prices, by adding discounts to malaria tests and ACTs when malaria-positive.

3.2 Study design

Study site

I recruited pharmacies from thirteen counties in malaria endemic and epidemic regions in western Kenya (Figure 3.1).[2] These counties account for 32% of Kenya's population, with a total population 15,231,090 individuals in 3.6 million households. The average household size in these counties is 4.6.

Experimental treatments

This section describes the intervention details and the experimental design. The study flow diagram can be found in Figure 3.2.

The treatment arms incentivize patients, pharmacists, or both to use malaria rapid tests and to use ACTs when confirmed by diagnostic tests. The magnitude of the incentive is held fixed at 200 Kes (~$2 USD) across all three treatment arms.[3] This amount is either given entirely to the patient in the form of a subsidy, entirely to the pharmacy in the form of an incentive divided between the pharmacy owner and attendant, or split between the patient and the pharmacy in the hybrid arm. The four intervention arms are as follows (also in Appendix Table B.1):

1. Control group: pharmacy is an active user of the basic sales and inventory management digital platform, and pharmacy manages their own stock of malaria diagnostic tests and treatments. Patients purchase diagnostic tests and treatment at market prices, and pharmacies stock and price these products according to their business practices.

2. Patient subsidy group: In addition to the features present at control pharmacies, clients who seek care for suspected malaria cases are eligible for a subsidized rapid test (90% subsidy, a 10 Kes copay) and a subsidized ACT (80% subsidy, a 30 Kes copay) conditional on a confirmed positive malaria diagnosis.

3. Pharmacy incentive group: In addition to the features present at control pharmacies, the pharmacy owners receive an incentive to sell the rapid test (90 Kes), and an additional incentive to prescribe ACTs to malaria-positive patients (80 Kes). Pharmacy attendants receive a 30 Kes incentive for recording transaction information in the malaria case management platform and completing the sale of incentivized products.

[2] The counties included in the study are: Bungoma, Busia, Homa Bay, Kakamega, Kisumu, Migori, Siaya, and Vihiga. Study counties that are not part of the lake endemic region, but still have significant levels of malaria burden, are Bomet, Kisii, Nyamira, Kericho, and Nakuru.

[3] The incentive amount is consistent with prior literature, was determined after an extensive pilot phase, and was calibrated to ensure pharmacy profitability would not be adversely affected, compared to the status quo.

4. Hybrid group: In addition to the features present at control pharmacies, the clients are eligible for discounted rapid tests (60% subsidy, a 40 Kes copay) and discounted treatment conditional on a positive test result (60% subsidy, a 60 Kes copay). Pharmacy owners receive an incentive to sell rapid tests (15 Kes), and an additional incentive to prescribe ACTs to malaria-positive patients (15 Kes). Pharmacy attendants receive a 30 Kes incentive for recording transaction information in the malaria case management platform and completing the sale of incentivized products.

The interventions were operationalized by Maisha Meds, a Kisumu-based healthcare technology company that provides sales and inventory management support to small pharmacies and clinics throughout Kenya. All pharmacies in the sample were existing users of the Maisha Meds sales management platform, which records all pharmacy transactions and product stock. The incentive interventions were integrated into this digital platform and managed centrally by the Maisha Meds team. This means that subsidy and incentive amounts are automatically calculated based on the products that are being bought/sold and verified by implementation staff independent of the pharmacies prior to disbursement to ensure implementation fidelity.

In all three treatment arms, pharmacy staff receive training on the malaria case management digital platform and proper rapid test administration, and rapid tests and ACTs are provided on consignment through the program. In all four study arms, pharmacy staff use the basic sales tracking tool to collect details of all pharmacy transactions during the study period and are provided information at the start of the study period about the importance of diagnostic testing for suspected malaria prior to treatment.

Sample selection and pharmacy randomization

Within study counties, all pharmacies that were part of the Maisha Meds network were mapped and screened for eligibility. There is significant heterogeneity in what qualifies as a private sector pharmacy in this setting. To ensure adequate regulatory oversight and homogeneity among study sites, only pharmacies that were registered businesses with Kenya's Pharmacy and Poisons Board at the time of onboarding were eligible to participate in the study. In order to be eligible for the study, pharmacies needed to be licensed and registered, be active users of the Maisha Meds digital sales and inventory management tool, be at least 0.5 km away from other study sites,[4] and be willing to be randomized to one of the study arms.

In total, 140 pharmacies across twelve counties in the malaria endemic and epidemic areas of Kenya's western regions were selected to be part of the study. Pharmacies that met these criteria were sequentially randomly assigned to one of the four arms in waves, stratified on

[4]The average distance between study sites is 6.24 km (range of 0.5 km to 46.2 km).

average monthly malaria product sales volumes (above/below median), urban/rural, location of pharmacy in lake endemic county, and participation in earlier pilot study phase.[5] Figure 3.3 shows the geographic span of the experiment across the target regions in Kenya and the final selection of pharmacies. Because interventions were randomized at the pharmacy level, every person seeking care for suspected malaria is eligible for the same intervention.

3.3 Experiment timeline and data collection

Below is the study timeline[6] and a description of the primary sources of data:

Jun-Dec '21	**Experiment launch**: baseline pharmacy survey with 233 pharmacy owners and staff from all 140 sites; staggered onboarding of 140 pharmacies to intervention and study
Aug '21-Feb '22	**Monitoring of implementation**: implementation team monitors intervention implementation in all pharmacies through regular outreach calls and random site visits; ongoing administrative data collection through digital platform
Oct '21-Jan '22	**Patient exit survey**: survey of random sample of 1654 adult clients who seek care for malaria-like symptoms at study pharmacies
Dec '21-Feb '22	**Standardized patient visits**: 412 visits by skilled enumerators presenting as suspected malaria patients to all pharmacies in the study sample, to obtain data on patient-pharmacist interaction, implementation fidelity, and quality of care
Jan-Feb '22	**Control group testing**: testing of random subset of 230 pharmacy clients at control group sites to obtain test positivity rate
Mar '22	**Pharmacy endline survey**: survey of all pharmacy staff and owners at conclusion of the data collection period

[5] Randomization was done in Stata 16 by the lead investigator.

[6] The study was initially planned to begin in June 2020, but was delayed due to COVID-19. The research and implementation teams followed Kenyan and UC Berkeley CPHS guidelines for conducting research while keeping study staff, implementation staff, and study subjects safe from COVID-19. All personnel and pharmacy staff were required to wear masks, maintain 1 meter distance from each other, and sanitize hands frequently. The research and implementation teams provided adequate PPE and hand sanitizer for all study and implementation personnel. Pharmacies were required by the Kenyan government to have all staff wearing masks, and have hand washing stations for staff and pharmacy clients, and pharmacies in our sample were compliant with these requirements during the study period. The pharmacy onboarding, patient exit survey, standardized patient visits, and control group testing activities were all done in person following appropriate COVID-19 precautions. The pharmacy baseline surveys were conducted over the phone.

I use the following data sources for analysis:

1. *Baseline data:*
    a) Pharmacy owner survey: survey with 120 pharmacy owners from 140 pharmacies about number of staff, pharmacy business operations, patient volumes, pharmacy characteristics, costs and revenues, and knowledge of malaria case management.
    b) Pharmacy staff survey: survey with 103 client-facing pharmacy staff from 140 pharmacies on malaria case management knowledge, worker motivation, and use of the status quo digital platform used to manage sales and inventory.
2. *Administrative data:*
    a) Sales data: continuously collected transaction data including prices[7] and quantities of products purchased, location, date, and time of sale, and pharmacy staff who made the sale for over 50,000 malaria-related patient encounters between June 2021 - February 2022.
    b) Malaria case management data: continuously collected transaction data on all rapid test and treatment purchases made through incentive program, including information on age/gender of patient, rapid test result, prices and quantities of medications purchased, method of purchase (cash/mobile money), location, date, and time of sale, and pharmacy staff who made the sale. Over 8,000 malaria transactions logged between June 2021 - February 2022.
3. *Patient exit survey data:* survey with a random sample of 1654 eligible adult pharmacy clients across all study sites (12.6 clients/site). In order to be eligible, clients must have sought care for malaria symptoms for themselves or a family member present at the pharmacy with them. Trained research staff visited each study pharmacy during an unannounced 5 day period, and screened all patients who exhibited malaria-related symptoms or purchased malaria products for eligibility. There were 1674 possible respondents screened, and 1654 respondents who completed the survey. This survey includes information on quality of care, symptoms, prices and quantities of medications and diagnostic tests purchased, beliefs about their illness status, malaria test result if applicable, and basic demographics.
4. *Testing subsample data:* data on test positivity from testing of random subset of 230 pharmacy clients at control group sites to obtain test positivity rate in a sample unaffected by the interventions (8.5 clients tested/site, 28 sites participated). Additional test positivity data from administrative records from 10 control group pharmacies that

[7]Prices observed in the data are retail prices set by pharmacists in the digital tool.

conducted testing on-site between January-February 2022 and kept records: 2547 test results obtained from this sample.[8]

5. *Endline data:*

   a) Pharmacy owner survey: survey with pharmacy owners at all 140 sites about number of staff, pharmacy business operations, patient volumes, pharmacy characteristics, costs and revenues, and altruistic tendencies.

   b) Pharmacy staff survey: survey with all client-facing pharmacy staff on malaria case management knowledge, worker motivation, use/familiarity with the digital platform used to manage sales and inventory and manage malaria cases, and altruistic tendencies.

Sample size calculations are described below.

3.4 Primary and secondary outcomes

All study outcomes are measured in individuals who sought treatment for suspected malaria (for themselves or a household member present with them at the time of care-seeking) at participating study pharmacies. These outcomes are obtained from the administrative data collected at the pharmacy by the digital tools for tracking sales and managing malaria cases.

There are three primary outcomes:

1. **Rapid test use**: The proportion of rapid test sales recorded at participating pharmacies as a share of total malaria product sales to clients with suspected malaria
2. **ACT targeting, overall**: Proportion of malaria treatments sold at participating pharmacies that are high quality treatments (ACTs) sold with a confirmatory diagnosis (as measured through diagnostic test result) to clients with suspected malaria
3. **ACT without test**: Proportion of high quality treatments (ACTs) sold at participating pharmacies without any diagnosis (as measured through diagnostic test sale)

These primary outcomes are the main outcomes of interest, and will be used to estimate parameters in the cost-effectiveness analysis. These outcomes examine different dimensions of the same two-part question of patient demand for the information provided by rapid tests and treatment choice. Outcome measure 1 (rapid test uptake) examines uptake for a particular illness episode, when the choice of whether to purchase a diagnostic test is salient

[8]These sample sizes were based on availability of testing data from pharmacy administrative records, and testing targets that balanced operational feasibility and research budget.

and can be thought of in terms of how much the patient values the information provided by that test relative to the cost. Connecting these outcomes to the hypotheses described in Chapter 2 and Section 3.1, I predict that that compared to the control group, uptake of rapid tests will be higher at subsidized prices. Comparing the two subsidy levels to each other (T1 and T3), I will learn the price elasticity of demand for rapid tests. Comparing the subsidy to the pharmacy incentives (T1 and T2), I will learn whether incentives are passed through to patients. Outcome measure 2 (ACT with test) examines uptake for a given confirmed malaria case, of a high quality treatment. There are alternative treatment options readily available at pharmacies, so this outcome measures the relative impact of subsidy/incentive on treatment choice.

There are three secondary outcomes:

1. **ACT targeting, over ACTs**: Proportion of high quality treatments (ACTs) sold at participating pharmacies with a confirmatory diagnosis (as measured through diagnostic test result) to clients with suspected malaria. This is an alternative measure of the primary outcome.
2. **Antimalarial sales**: Proportion of sales recorded at participating pharmacies that are antimalarial products
3. **ACT use:** Proportion of high quality treatment (ACT) sales recorded at participating pharmacies as a share of total antimalarial sales

These outcomes provide insight on a fuller picture of how the interventions impact patient volumes and care-seeking behavior at study pharmacies. ACT without a test and antimalarial treatment without a test is an alternative measure of treatment targeting that measures both whether malaria positive patients purchase antimalarials (ACTs) with an accompanying test and whether malaria negative patients elect to not purchase antimalarials, given their test outcome. Antimalarial sales and high-quality treatment use are measures of patient shares overall to answer the question of whether the interventions impacted overall likelihood of malaria-symptomatic patients to seek care at participating pharmacies.

Sample size calculations

The study has two primary outcomes of interest, rapid test uptake and ACT treatment targeting. The primary comparisons are the effect on the primary outcomes of each of the three interventions relative to the control arm and the demand-side intervention to the supply-side intervention. The study is powered to detect meaningful minimum effects for each of these primary comparisons at $\beta = 0.85$. For pairwise comparisons with the control group, the study is powered to detect a minimum detectable effect (MDE) of 0.13 for rapid test uptake and 0.12 for ACT targeting. For treatment arm comparisons, the study is

powered to detect between 0.13-0.19 MDEs for rapid test uptake and 0.12-0.16 MDEs for ACT targeting. Standard formulas for cluster randomized control trials with individual-level binary outcomes were used to calculate the power for each comparison [51]. These power calculations were conducted conservatively, not adjusting for covariates and using the Bonferroni correction to adjust for multiple comparisons.

For a two-tailed test, $\alpha = 0.0063$ ($\alpha = 0.05/8$ to adjust for the 4 main comparisons for each primary outcome). We split our sample of 140 pharmacies into four equal sized groups ($J = 35$) and assume that there will be 100 individuals per cluster in our sample frame (100 eligible pharmacy clients with suspected malaria, and that 75% of these are sold ACTs, for a sample of 75 clients receiving ACTs at each pharmacy). We calculate the coefficient of variation (k) for each primary outcome using ICC (ρ) estimates of 0.17 (rapid test uptake) and 0.15 (ACT targeting) obtained from baseline data for our sample frame. We draw our comparison means using data from the study sample and a range of plausible benchmarks. We present these in Table 3.1. Given these parameters, power of 85%, and assuming an unadjusted model without covariates at levels 1 (individual) or 2 (site), we are powered to detect MDEs of between 0.13-0.19, depending on the comparison group as shown in Table 3.2.

3.5 Empirical strategy

Take-up of the intervention

I measure trial take-up as the subset of eligible pharmacies in the study area that consented to participate in the trial. The total number of eligible pharmacies in the study catchment area was obtained from the administrative records of the implementing partner. Each consenting pharmacy agreed to manage their sales through the digital tool, and to offer incentives (either supply- or demand-side) for malaria testing and treatment if assigned to one of the intervention arms.

I then determine the relationship between trial take-up and eight pharmacy-level characteristics: number of months active on digital sales management tool; baseline (2019-2020) average monthly malaria product sales, high quality malaria treatment (ACT) sales, and rapid diagnostic test sales; participation in earlier study phase, [9] urbanicity, location in a lake endemic county, and pharmacy type. In order to determine whether trial take-up is related to any of these covariates, I conduct pair-wise t-tests comparing these characteristics across pharmacies that declined to participate and those that elected to participate.

[9] 60 pharmacies participated in a Phase 1 of the study between November 2020-February 2021, where different levels of patient subsidies for malaria testing and treatment were randomized to patients. This study phase was stopped because of insufficient take-up and operational complexity, and these sites were balanced across treatment arms in the full study sample.

Treatment effects on testing and treatment targeting

I present all results of the program impact on testing and treatment targeting in terms of comparisons between each intervention arm and the control group (status quo pharmacy care experience). Additionally, I discuss any significant differences between demand-side incentives (T1) and supply-side incentives (T2), and across all three intervention arms (T1, T2, T3) and compare to the minimum detectable effects that the study was powered to detect. All analyses are conducted at the patient level[10], and an intention-to-treat (ITT) framework is used. Some clients at intervention pharmacies refuse to purchase rapid tests and treatments through the intervention platform (at a fixed reduced price, in T1 and T3) and will elect to make other purchases or none. By including all eligible malaria patients in an ITT analysis, rather than only patients who elect to take up the intervention assigned to the pharmacy, I preserve the unbiasedness benefits of randomization. The analyses specified in this section were pre-registered in a pre-analysis plan (AEARCTR-0004705). I discuss any deviations from the pre-analysis plan where relevant in this chapter and the results chapters.

For all binary outcomes, I estimate unadjusted and adjusted logistic regressions using the following regression framework.

$$Pr(Y_{ip}) = expit(\beta_0 + \beta_1 T_{1ip} + \beta_2 T_{2ip} + \beta_3 T_{3ip} + \lambda_s + \epsilon_{ip}) \tag{3.1}$$

where Y_{ip} is a malaria testing or treatment outcome, T_{jip} are treatment assignment indicators for each intervention j for individual i seeking care at pharmacy p, with the control group as the reference category, λ_s are strata fixed effects, and ϵ_{ip} is the error term. The β terms represent the log-odds of the treatment effect of each intervention relative to the control group. I report all results in terms of marginal effects in relation to the control group mean. I also report p-values from Wald tests comparing the marginal effect coefficients of the demand side and supply side interventions, and all three interventions. A p-value of less than 5 percent on these tests indicates that supply and demand-side incentive targeting have differential impact on the outcome of interest, or that the hybrid intervention has a differential impact than either the supply or demand side arms. The estimates produced by Equation 3.1 do not account for baseline pharmacy-level differences in malaria case management between groups, nor do they account for potential confounders that are not completely balanced at baseline (Tables 4.1 and 4.2). I include variables that had significant imbalance with the control group at the 10% level or below at baseline as covariates in this adjusted model ($\boldsymbol{X_p}$), as specified in the pre-analysis plan. I estimate equation 3.2 to adjust for this imbalance and improve precision.

[10]This is equivalent to febrile illness episode level since most patients in our sample only have had one symptomatic pharmacy visit during the study period.

$$Pr(Y_{ip}) = expit(\beta_0 + \beta_1 T_{1ip} + \beta_2 T_{2ip} + \beta_3 T_{3ip} + \lambda_s + \boldsymbol{X}_p + \epsilon_{ip}) \tag{3.2}$$

I report all results from the adjusted regressions in the same way that I do for the unadjusted model. In addition to looking at each intervention separately, I report results from pooling the interventions, to measure overall impact of any incentive program on outcomes of interest. This pooled regression specification was not pre-specified in the analysis plan, and is below:

$$Pr(Y_{ip}) = expit(\beta_0 + \beta_{pooled} \boldsymbol{T}_{ip} + \lambda_s + \boldsymbol{X}_p + \epsilon_{ip}) \tag{3.3}$$

I adjust my p-values for my primary comparisons for the primary outcomes of interest using the Anderson multiple hypothesis correction to control for the false discovery rate [52]. These comparisons are each intervention arm to the control group (3 tests), the supply-side arm compared to the demand-side arm (1 test), and the hybrid and supply-side arms compared to the demand-side arm (1 test) for three outcome variables (15 tests total). Both unadjusted and adjusted p-values are reported. This method does not account for any correlations among p-values, which I do expect. Therefore, it is likely a conservative adjustment - future publications will use an updated method that takes this dependence structure into account, like that developed by Clarke, Romano and Wolf [53].

Cost-effectiveness

In order to analyze the efficiency of each incentive intervention, I conduct a cost-effectiveness analysis from the implementer's perspective and a societal perspective (including program costs, costs incurred by the patient, and direct costs of over-treatment). My CEA metric is the incremental cost per additional patient who takes ACTs and is malaria positive (so, is appropriately treated). To evaluate cost-effectiveness, I calculate the ratio of the change in benefits to the change in costs across each intervention arm compared to the status quo. I look at benefits defined as the change in patients taking ACTs that are malaria positive, and costs defined as (1) incentive costs and (2) the direct medication costs of over-treating malaria negative patients with antimalarials. The framework described below allows me to measure whether the interventions tested in the experiment cost-effectively improve adherence to malaria clinical guidelines and reduce the direct costs of misallocating antimalarials. The cost effectiveness analysis framework relies on the theoretical model described in Chapter 2 and parameters recovered from the experimental design.

Benefits

In Chapter 2, I presented the conditional probabilities for the ideal end state outcomes that will constitute the measure of benefits - patients who take ACTs appropriately (are malaria positive). Only malaria positive patients who take ACTs contribute to the benefits. To

estimate the number of patients who get ACTs appropriately in each of the intervention arms, I use the following equation:

$$Beneficiaries_t = Pr(ACT|positive)_t \times ACT_t \tag{3.4}$$

where $Pr(ACT|positive)_t$ is the probability of purchasing an ACT conditional on being malaria positive, for each intervention arm t, and ACT_t is the number of patients in intervention arm t who purchase ACTs (the share of patients who purchase ACTs[11] multiplied by a hypothetical cohort of 10,000 patients). I can further expand $Pr(ACT|positive)_t$ into a component that applies to patients who were tested for malaria and one that applies to patients who were untested for malaria, as is shown below:

$$\begin{aligned} Pr(ACT|positive)_t = {} & Pr(ACT|positive\&tested)Pr(positive|tested)Pr(tested)+ \\ & Pr(ACT|positive\&untested)Pr(positive|untested)Pr(untested) \end{aligned} \tag{3.5}$$

Each of these probabilities can be found from the parameters that are measured through the experimental design and data collection activities. $Pr(ACT|positive\&tested)$ is directly estimated from the administrative data in the treatment groups, and is the treatment arm specific mean in column 2 of Table 4.7. In the control group, this probability is estimated using the control group mean from column 3 of Table 4.4 (0.057) multiplied by the control group $Pr(positive|tested)$. $Pr(positive|tested)$ is obtained from administrative pharmacy data in all four arms. In the control group, this comes from aggregate reported test positivity rates from 2547 tests done in 10 control group sites that conducted testing between January-February 2022 and kept records. In the treatment groups, this comes from the administrative data collected through the study on individual test results, for patients who tested through the intervention. These parameters are the treatment arm specific means in column 1 of Table 4.7. $Pr(tested)$ is directly estimated from the administrative data in all four arms, and is the treatment arm specific mean in column 2 of Table 4.4. $Pr(ACT|positive\&untested)$ is estimated for all four arms and is the treatment group specific means of $Pr(ACT|untested)$ from column 6 in Table 4.4 multiplied by $Pr(positive|untested)$. $Pr(positive|untested)$ is estimated in the control group using data collected from the lab tech activity which tested a random subset of 230 patients who purchased antimalarials for a suspected illness at 28 control group sites but did not get tested prior between January-February 2022. In the treatment groups, $Pr(positive|untested) = Pr(positive) - Pr(positive|tested)$. $Pr(positive)$ is the unselected (for testing) malaria positivity rate, and is obtained from the control group testing data ($Pr(positive|tested) + Pr(positive|untested)$), and $Pr(positive|tested)$ is directly obtained from Table 4.7, as described above.

[11] Obtained from intervention group specific means from Table 4.5 column 4.

The inputs needed to calculate the number of beneficiaries in each intervention arm can be found in Table 3.3. I will estimate the program benefits for each intervention using these parameters and compare them to the status quo standard of care, as well as to the next best alternative. For details on the sources of each parameter input for the benefits, please see Appendix Tables B.12 and B.13. For details on formulas used to calculate the benefits estimates, please see Appendix Table B.14.

Costs

The costs of the program can be broken down into direct costs of running the program, the direct costs of over-treating malaria negative patients with antimalarials, and other non-programmatic costs to patients of participating in the program. From the implementer perspective, I include the costs of administering the incentive interventions as my primary program cost for Maisha Meds (c_t). I multiply these costs by the number of patients who purchased an incentivized product in each intervention arm ($Patients_t$). The total cost of the incentive interventions (CI_t) is calculated using the below formula:

$$CI_t = c_t \times Patients_t \tag{3.6}$$

where $t \in (0, 1, 2, 3)$ is one of the three treatment arms or the control group.

For the societal perspective, I also include the direct costs of over-treating malaria negative patients in each of the intervention arms, and the time costs to patients for seeking malaria care at pharmacies in each of the intervention arms, in addition to the program implementation costs. In order to estimate the costs of over-treating malaria negative patients, I first estimate the average cost of treatment for patients who did not get tested for malaria and the average cost of treatment for patients who did get tested for malaria. These cost estimates are directly observed from the administrative data, and I have estimates for each of these out of pocket costs for each of my intervention arms. Then I also observe the number of untested patients and number of tested patients in each treatment arm, again from the administrative data. I estimate the likelihood of being malaria negative condition on being untested, and the likelihood of being malaria negative conditional on being tested in each treatment arm. I use parameter estimates obtained from data collection activities for these probabilities. $Pr(negative|untested)$ is estimated in the control group using data collected from the lab tech activity which tested a random subset of 230 patients who purchased antimalarials for a suspected illness at 28 control group sites but did not get tested prior between January-February 2022. $Pr(negative|tested)$ is obtained in the control group from aggregate reported test positivity rates from 2547 tests done in 10 control group sites that conducted testing between January-February 2022 and kept records. In the treatment groups, $Pr(negative|untested) = Pr(negative) - Pr(negative|tested)$. $Pr(negative)$ is the unselected (for testing) malaria negativity rate, and is obtained from the control group testing data $(1 - (Pr(positive|tested)|Pr(positive|untested)))$, and $Pr(negative|tested)$

is directly obtained from the treatment arm specific means in column 1 of Table 4.7 $(1 - Pr(positive|tested))$.

Finally, I calculate the time cost to patients of obtaining care for their malaria symptoms in the pharmacy setting. This is relevant because patients may experience longer visit times if they elect to be tested for malaria, which may enter into their calculus of whether or not to be tested. I obtain estimates of total time spent at pharmacy seeking care from the patient exit survey data (in minutes) for each intervention arm, and multiply that by an estimate of the local hourly wage to obtain a monetary measure of the time cost for care-seeking. the total social cost of the interventions is calculated using the below formula:

$$TotalCost_t = \underbrace{c_t \times Patients_t}_{\text{Program costs}} + \underbrace{CostOverTx_t \times PatientsOverTx}_{\text{Costs of over treatment}} + \underbrace{CostTime_t}_{\text{Time cost to patients}} \quad (3.7)$$

where $t \in (0, 1, 2, 3)$ is one of the three treatment arms or the control group.

The inputs needed to calculate all cost parameters can be found in Table 3.3. For details on the sources of each parameter input for the costs, please see Appendix Tables B.12 and B.13. For details on formulas used to calculate the cost estimates, please see Appendix Table B.14.

Incentive pass-through

I use self-reported price data from standardized patient (SP) exit surveys to test whether incentives are passed through to patients in the form of reduced prices in the pharmacy incentive arms (T2 and T3). This analysis is exploratory and was not pre-specified in the analysis plan. A team of 12 SPs conducted 411 anonymous visits at 137 study sites, for a total of 3 visits/site (3 sites were not visited by SPs because they were closed during the SP data collection period, which was from December 2021 - February 2022). As part of the exit surveys, SPs were asked what items they purchased from the pharmacy for their visit that day, as well as the itemized prices of each product. Additionally, they were asked the total price of all items purchased, as well as whether the pharmacist offered them any discounts on any of the products purchased.

I analyze the extent of incentive pass-through in the provider-side incentive arms using two dependent variables. First, I construct a binary variable that indicates whether the SP

reported that the pharmacist offered them a discount on a malaria test or treatment. I analyze the intervention effects on this outcome using logistic regression, modeled below:

$$Pr(Discount_{ip}) = expit(\beta_0 + \beta_1 T_{1ip} + \beta_2 T_{2ip} + \beta_3 T_{3ip} + \phi_j + \epsilon_{ip}) \tag{3.8}$$

This equation includes SP fixed effects (ϕ_j), and the standard errors are clustered at the pharmacy level. The β terms represent the log-odds of the treatment effect of each intervention relative to the control group. I report all results in terms of marginal effects in relation to the control group mean. To test for pass-through, I compare the coefficients on the marginal effect of the demand-side intervention (T1) to both the supply-side intervention (T2) and the hybrid intervention (T3). If I am able to reject that either $\gamma_{T2} = \gamma_{T1}$ or $\gamma_{T3} = \gamma_{T1}$, then that is evidence of incentive pass through.

In addition, I look at the extent of pass-through by using the actual reported prices paid for malaria tests and treatment. I analyze the intervention effects on this outcome using linear regression, modeled below:

$$log(Price_{ip}) = \beta_0 + \beta_1 T_{1ip} + \beta_2 T_{2ip} + \beta_3 T_{3ip} + \phi_j + \epsilon_{ip} \tag{3.9}$$

This equation includes SP fixed effects (ϕ_j), and the errors are clustered at the pharmacy level. To test for pass through, I compare the coefficients on the demand-side intervention (β_1) to both the supply-side intervention (β_2) and the hybrid intervention (β_3). If I am able to reject that either $\beta_2 = \beta_1$ or $\beta_3 = \beta_1$, then that is evidence of incentive pass through.

3.6 Ethical review

This study was granted clearance by the ethics committees at University of California, Berkeley (Protocol number 2019-11-12739) and Strathmore University in Kenya (Protocol number SU-IERC0609/19). Additionally, it was approved by Kenya's National Commission for Science, Technology and Innovation (License number NACOSTI/P/21/13580). Prior to enrolling pharmacies in the study, trained field staff obtained informed consent from pharmacy owners about the study activities, which included using the digital point of sale platform, implementing the incentive interventions if applicable, and participating in survey-based data collection. All survey data collection activities were conducted by trained field staff and data were recorded digitally on secure tablets. All providers and patients gave informed consent prior to participating in any survey data activities. For the standardized patient component of the study, all SPs were hired as field staff and participated in a 3-week training, 2-week pilot, and refresher trainings during fieldwork in order to mitigate any potentially harmful events, such as unsafe injections, invasive tests, and consuming any medicines during encounters in the pharmacies. Similar to other SP studies, we obtained a waiver of provider informed consent to conduct the SP study. Providers were told that there would be unannounced monitoring during the study period, but they were not informed by whom. The

waiver was obtained because the study posed no more than minimal risk of participation to the SPs or the pharmacy staff, and obtaining informed consent about the SP visits during the study period would compromise the scientific validity of the study objectives and increase the risk of SP detection. Appendix 3 includes the SP case scenario for the malaria visits. Survey instruments are available upon request.

3.7 Figures

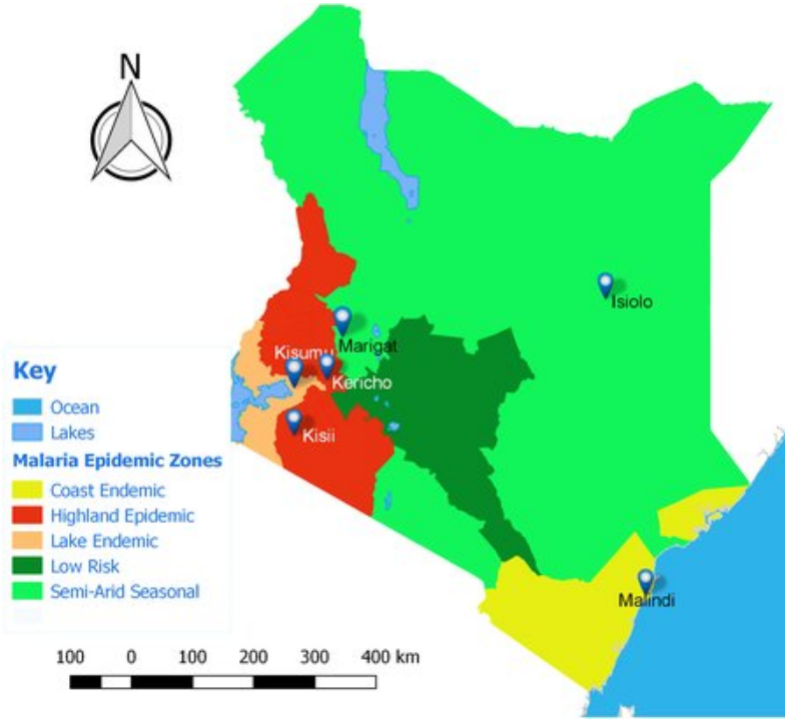


Figure 3.1: Malaria zones in Kenya, source: [54] (Back: 3.2)

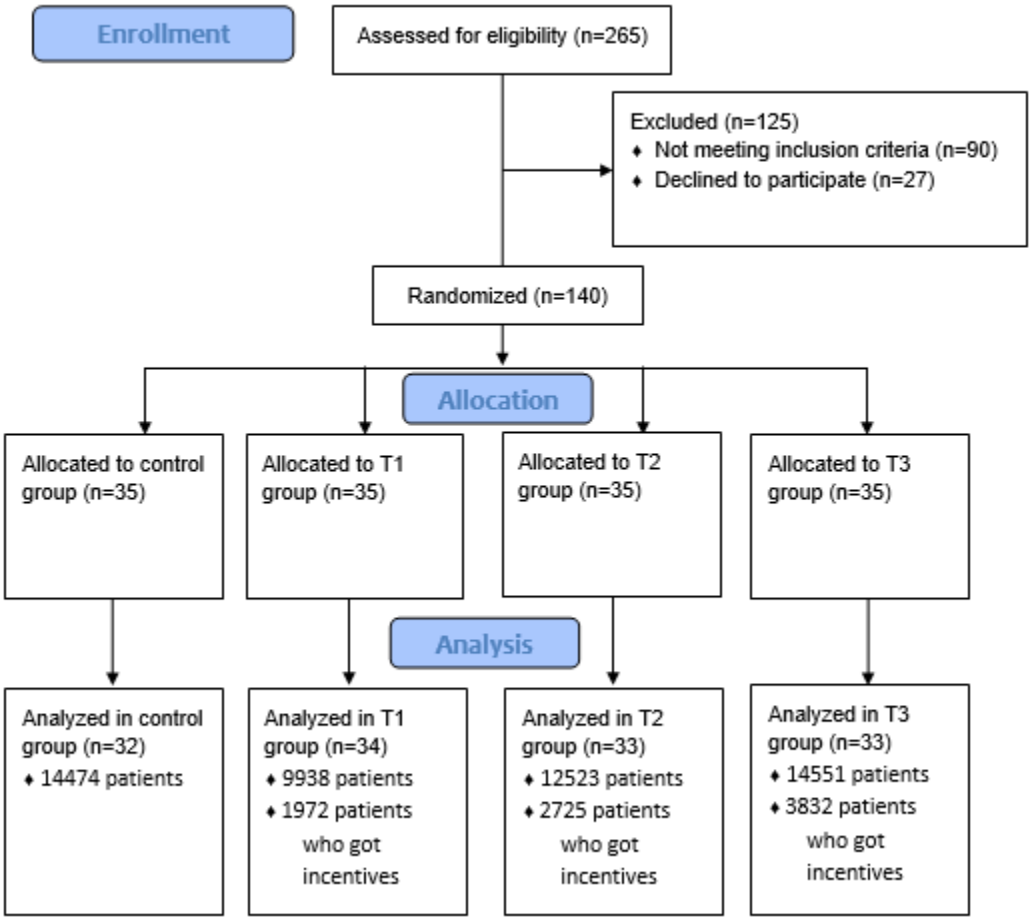


Figure 3.2: Study flow diagram (Back: 3.2)

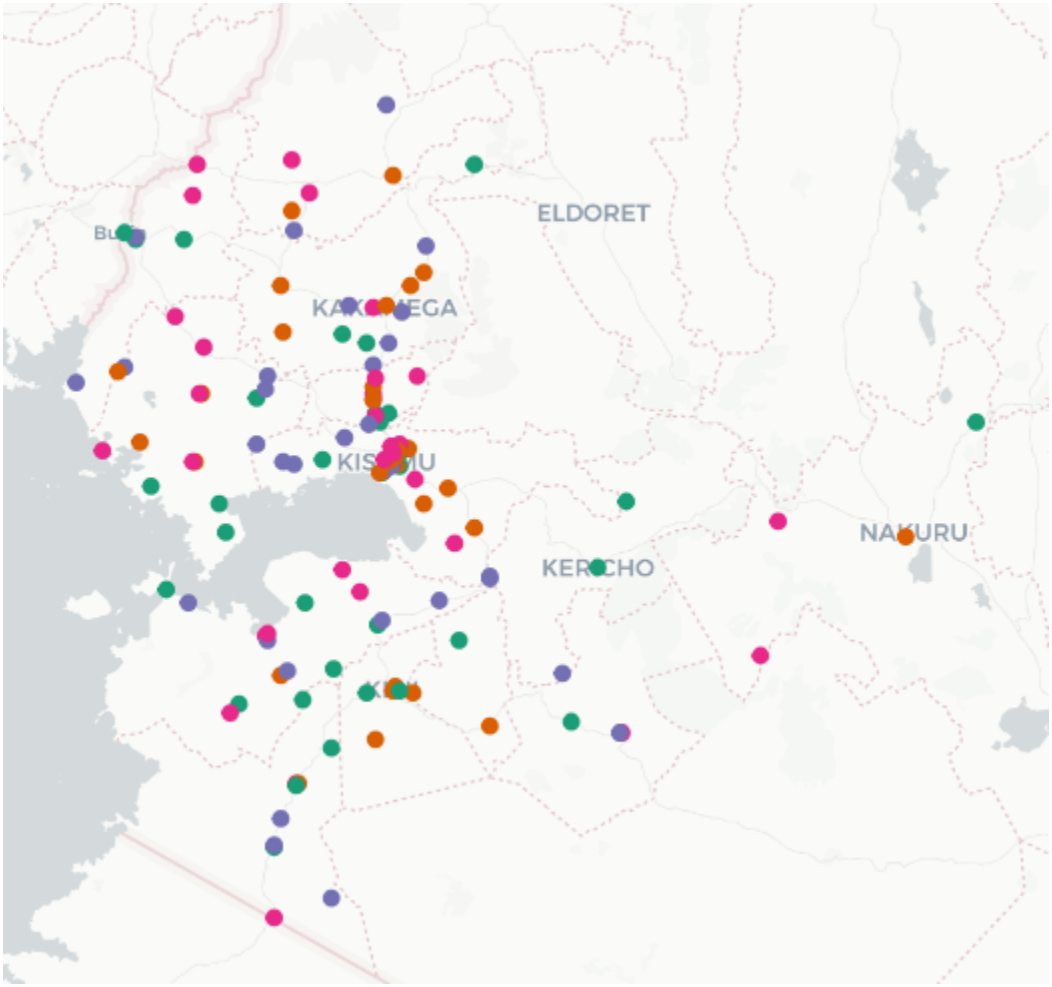


Figure 3.3: Map of study sites (Back: 3.2)

3.8 Tables

Table 3.1: Comparison group means for power calculations (Back: 3.4)

	RDT uptake	ACT targeting
Control group	0.088	0.072
Lower bound comparison	0.209	0.132
Midrange comparison	0.234	0.18
Upper bound comparison	0.343	0.22

Table 3.2: MDEs detectable at 85% power for 140 sites (35/arm), 100 people/site (75 ACT sales/site) (Back: 3.4)

Table 2. Minimum Detectable Effect Sizes at 85% power

	RDT uptake	ACT targeting
C	0.13	0.12
LB	0.17	0.14
MB	0.18	0.16
UB	0.19	0.16

	Control (status quo)	T1 Patient subsidies	T2 Provider incentives	T3 Hybrid
	PARAMETER INPUTS			
P(tested)	0.082	0.344	0.282	0.29
P(untested)	0.918	0.656	0.718	0.71
P(malaria positive | tested)	0.237	0.354	0.495	0.298
P(malaria positive | untested)	0.106	-0.011	-0.152	0.045
P(malaria positive)	0.343	0.343	0.343	0.343
P(ACT | malaria positive & tested)	0.014	0.996	0.93	0.98
P(ACT | malaria positive & untested)	0.086	-0.01	-0.1	0.03
P(malaria negative)	0.657	0.657	0.657	0.657
Share of patients who purchased ACTs	0.867	0.72	0.7763	0.731
Incentive unit cost (RDT) ($)	$0.00	$0.90	$1.20	$1.10
Share of patients purchasing incentivized RDT	0	0.201	0.279	0.391
Incentive unit cost (ACT) ($)	$0.00	$1.10	$0.80	$0.90
Share of patients purchasing incentivized ACT	0	0.072	0.092	0.082
Average antimalarial treatment unit cost ($), untested	$1.92	$2.12	$1.49	$1.99
Share of untested patients	0.919	0.647	0.723	0.721
P(malaria negative | untested)	-0.106	0.011	0.152	-0.045
Average antimalarial treatment unit cost ($), tested	$4.02	$0.79	$1.98	$1.26
Share of tested patients	0.081	0.353	0.277	0.279
P(malaria negative | tested)	0.763	0.646	0.505	0.702
Time cost of seeking care	$18.04	$17.71	$16.33	$18.39
Hourly wage ($)	$2.00	$2.00	$2.00	$2.00
Number of patients who accessed care	10000	10000	10000	10000

Table 3.3: Cost Effectiveness Analysis Inputs (Back: 3.5)

Chapter 4

Impact evaluation results

This chapter is broken into the following sections. Section 4.1 describes characteristics of the study sample, as well as the results of baseline balance tests between treatment and control groups as created through the randomization of incentive interventions. Section 4.2 describes the main intervention effects on testing and treatment targeting outcomes. Section 4.3 describes the main intervention effects on secondary outcomes, including on malaria patient shares and antimalarial sales. Section 4.4 describes the results of the cost-effectiveness analysis.

4.1 Trial take-up and sample characteristics

Incentives for rapid diagnostic tests and high quality treatments (ACTs) were randomized across 140 pharmacies at baseline, with 35 assigned to the control group (25%), 35 assigned to the patient subsidies group (25%), 35 assigned to the pharmacy incentives group (25%), and 35 assigned to the hybrid group that received both patient subsidies and pharmacy incentives (25%). Tables 4.1 and 4.2 report the experimental balance checks at baseline (for variables from the administrative data and survey data, respectively), and shows that randomization was fairly balanced across a large set of prespecified covariates. Balance checks are done at the pharmacy level, as this is the unit of randomization, and is the level at which baseline data are available. Columns 1-4 report group means for the control group and each of the treatment groups. Significance stars in columns 2-4 indicate that the difference in means when compared to the control group is significant at the 10 percent level or below. Notably, pharmacies in the pharmacy incentive group (T2) had been active for 5.4 months on the digital sales management platform prior to the start of the study, whereas control group (C) pharmacies had been active for 10.1 months ($p < 0.05$). Additionally, pharmacies in the hybrid group (T3) sold fewer malaria products per month, on average, than control group pharmacies (43.6 vs. 64.8, $p < 0.10$). Finally, 36% of control group pharmacies have a female owner, while only 15-17% of pharmacies in the treatment arms are female-owned

($p < 0.10$). Columns 5-7 report mean differences and significance levels from pairwise means comparisons between T1 and T2, T1 and T3, and T2 and T3, respectively. Compared with patient subsidy (T1) facilities, T3 facilities have lower average monthly rapid diagnostic test sales at baseline and T2 facilities were newer adopters of the sales management platform and are less likely to be female-owned. When I conduct a joint test for orthogonality using a multinomial logit model with treatment assignment as the categorical outcome, I find that the χ^2-test produces a p-value of 0.46. This suggests that these covariates are not jointly predictive of group assignment. In my adjusted models, I control for covariates that were unbalanced at baseline from comparisons with the control group, consistent with my pre-specified analysis plan.

I measure fidelity to implementation as whether there were any malaria transactions logged in the digital sales tracking platform for a study pharmacy during the study period (June 2021-February 2022). By this metric, 8 facilities out of 140 were inactive during the study period (1 in the patient subsidy arm, 2 in the pharmacy incentive arm, 2 in the hybrid arm, and 3 in the control group). For the remaining active facilities, the number of active facilities by month can be found in Figure 4.1, and the number of facilities actively selling incentivized malaria tests and treatments can be found in Figure 4.2. The pharmacy onboarding occurred between June-December 2021, so the increase in the number of active facilities over this time period is due to pharmacies being onboarded to the study in a staggered way.

Randomization was done prior to enrolling facilities in the study for sites that met all eligibility criteria, due to operational necessity of conducting in person site visits to introduce the program and the study at the same time. Appendix table B.2 reports balance on baseline variables obtained from the administrative data between facilities that, when offered participation in the program and study, accepted (in sample) and those that declined (refusals). Column 3 reports the differences between the two group means and the significance stars from a t-test comparing the difference in group means. Facilities that declined to participate in the program and study had been using the digital sales platform for longer than facilities in the sample frame. No other meaningful imbalances were found. Appendix tables B.3 and B.4 report descriptive results from regressing the primary and secondary outcomes on the sample baseline characteristics.

4.2 Treatment effects on testing and malaria treatment targeting

This section examines the effect of the three interventions on malaria diagnostic testing and treatment targeting outcomes, and compares demand- and supply-side approaches to each other. My main analyses are intention-to-treat (ITT), since some pharmacies who were assigned the interventions may not have fully administered the incentives throughout the study period and some pharmacy clients may not have been offered the interventions. By

including all pharmacies and all malaria clients in an ITT analysis, rather than only including those pharmacies and malaria clients that received the assigned intervention, I preserve the unbiasedness benefits of randomization. However, this will provide a lower bound estimate of the average treatment on the treated (TOT) effect. To complement the ITT analysis, I run additional analyses in Section 4.3 looking at testing and treatment targeting among malaria patients in the treatment groups that received the interventions. Standard errors are clustered at the pharmacy level for all analyses, and all analyses in Sections 4.2 and 4.3, with the exception of some secondary outcomes that will be noted in that section, were pre-specified in the analysis plan.

My first set of analyses answers the first research question (see Section 3.1): how do targeted incentives impact uptake of malaria diagnostic testing? The first two columns in Tables 4.3 and 4.4 show the marginal effects of unadjusted and adjusted logistic models for the outcome measure, rapid test uptake, among all patients who purchased malaria products. All clients who purchased malaria-related products at a participating study pharmacy between June 2021 and February 2022 are included in this outcome measure. In the full sample, there were 51,486 pharmacy clients who purchased malaria related products (a range of treatments, and diagnostic tests) across the eight month study period, and 13,585 (26%) who purchased a rapid diagnostic test during their pharmacy visit. I present results for both the pooled treatment effect - whether any of the incentive interventions had an impact on this outcome - and for each of the three incentive interventions, in columns 1 and 2 of these tables, respectively. Unadjusted results in Table 4.3, column 1 show that there is a 28 percentage point increase in the likelihood that patients purchase a rapid diagnostic test when seeking malaria-related care at any of the pharmacies with the incentive interventions, compared to the control group ($p < 0.01$, control group mean = 0.08). Looking at each intervention separately, I find that all three incentive approaches yield similar outcomes. Patient subsidies (T1) increases the likelihood that a patient purchases a rapid diagnostic test by 25 percentage points ($p < 0.05$), pharmacy incentives (T2) increases this likelihood by 20 percentage points ($p < 0.01$), and the hybrid approach increases this likelihood by 25 percentage points ($p < 0.01$). The Wald tests that test for equality across the three intervention arms, and T1 compared with T2 are unable to reject the null hypothesis that there are no differences across the interventions (research questions 3-4, for the diagnostic testing outcome). Table 4.4, columns 1 and 2 show results of the adjusted analysis on rapid test uptake. These results are consistent with what was found in the unadjusted models - the pooled intervention effect is 25 percentage points ($p < 0.01$), with each intervention separately increasing the likelihood that a patient purchases a rapid diagnostic test by between 20 and 27 percentage points. Again, I am unable to reject the null hypothesis that all three incentive approaches yield the same effect on testing uptake. All of these effects are significantly larger than the minimum detectable effect sizes that were deemed ex-ante economically meaningful of a 13 percentage point change in rapid test uptake when comparing each intervention arm to the control group. While there are very small differences in effect sizes when comparing intervention arms to each other, they are much smaller than the minimum effects that the study was

powered to detect.

Turning now to look at the intervention effects on treatment targeting, the next set of analyses answer research question 2 (see Section 3.1): how do targeted incentives impact the likelihood that patients purchase ACTs with an accompanying test result. The third and fourth columns of Tables 4.3 and 4.4 show the marginal effects of unadjusted and adjusted logistic models for the outcome measure, ACT uptake with a confirmatory diagnostic test, among patients who purchased malaria products. All clients who purchased malaria-related products at a participating study pharmacy between June 2021 and February 2022 are included in this outcome measure. In the full sample, 5,871 out of 51,486 (11%) clients who purchased malaria products for their symptoms purchased an ACT with an accompanying test. Unadjusted results in Table 4.3, column 3 show that there is a 9 percentage point increase in the likelihood that patients purchase an ACT with an accompanying diagnostic test at any of the pharmacies with the incentive interventions, compared to the control group ($p < 0.05$, control group mean = 0.06). This is more than a two-fold increase in appropriate treatment targeting. Looking at each intervention separately, I find that all three incentive approaches have effect sizes of similar magnitudes for this outcome (the effect on patient subsidies is statistically insignificant). Pharmacy incentives (T2) increase the likelihood that a patient purchases ACTs with a diagnostic test by by 7 percentage points ($p < 0.1$), and the hybrid approach increases this likelihood by 7 percentage points ($p < 0.05$) as well. The Wald tests that test for equality across the three intervention arms, and T1 compared with T2 are unable to reject the null hypothesis that there are no differences across the interventions (research questions 3-4, for the first treatment targeting outcome). Table 4.4, columns 3 and 4 show results of the adjusted analysis on the first treatment targeting outcome (uptake of ACTs with a diagnostic test). These results are consistent with what was found in the unadjusted models - the pooled intervention effect is 7 percentage points ($p < 0.05$), with each intervention separately increasing the likelihood that a patient purchases ACTs with a diagnostic test by between 5 and 7 percentage points, with only the hybrid arm statistically significant at the 10% level. Again, I am unable to reject the null hypothesis that all three incentive approaches, or the supply- compared with the demand-side incentives, yield the same effect on this measure of treatment targeting. These effects are smaller than the minimum detectable effects determined to be economically meaningful ex-ante (12 percentage point change when comparing each intervention to the control group, and between 12-19 percentage point change when comparing intervention arms to each other), but are significant because they represent large increases in treatment targeting relative to a very low baseline in these private sector pharmacy outlets.[1]

Columns 5 and 6 of Tables 4.3 and 4.4 look at the second dimension of treatment targeting

[1]Additionally, the benchmarks used to determine the MDEs were based on pilot data and a review of prior literature, which focused on both the public and private sector. Public sector testing and treatment targeting rates tend to be higher than in private sector pharmacies in this setting, so the ex-ante MDEs reflect this as well.

associated with research question 2: how do targeted incentives impact the likelihood that patients purchase antimalarials without an accompanying test. Out of the 51,486 pharmacy clients who purchased malaria products during the study period, 33,390 (67%) purchased ACTs without a diagnosis. Unadjusted results in Table 4.3, column 5 show that there is a 17 percentage point decrease in the likelihood that patients purchase an ACT without any diagnostic test at any of the pharmacies with the incentive interventions, compared to the control group ($p < 0.05$, control group mean = 0.81). Looking at each intervention separately, I find that all three incentive approaches have effect sizes of similar magnitudes for this outcome of between 13 and 19 percentage points, with only the hybrid arm statistically significant at the 5% level. The Wald tests that test for equality across the three intervention arms, and T1 compared with T2 are unable to reject the null hypothesis that there are no differences across the interventions (research questions 3-4, for the second treatment targeting outcome). Table 4.4, columns 5 and 6 show results of the adjusted analysis on the second treatment targeting outcome (uptake of ACTs without any diagnosis). These results are consistent with what was found in the unadjusted models - the pooled intervention effect is a decrease of 20 percentage points ($p < 0.01$), with each intervention separately decreasing the likelihood that a patient purchases ACTs without a diagnosis by between 16 and 22 percentage points. Again, I am unable to reject the null hypothesis that all three incentive approaches, or the supply- compared with the demand-side incentives, yield the same effect on this measure of treatment targeting.

The results from this analysis suggest that all three interventions have strong effects on encouraging patients who seek malaria-related care to get diagnosed in the pharmacy prior to purchasing treatment. Additionally, the results on the two dimensions of treatment targeting - ACT uptake with a test, and ACT uptake without a test - suggest that the incentive interventions encourage malaria positive patients to get the highest quality antimalarials available to them, and discourage malaria negative patients from unnecessarily purchasing antimalarials. The fact that I do not find any significant differences across intervention arms shows that targeting the incentive to either the patient or provider yields similar effects in this setting.

The main tables report unadjusted p-values for my primary comparisons. These were adjusted for multiple hypothesis testing using the approach outlined in Anderson (2008) [52], and the unadjused and FDR-adjusted q-values for the unadjusted and adjusted models in Tables 4.3 and 4.4 are reported in Appendix table B.5. After the p-value adjustments, there are no longer significant differences in the unadjusted models between the patient discount arm compared to the control group for rapid test uptake, between the pharmacy incentive and hybrid arms compared to the control group for the ACT uptake with a diagnostic test outcome, and for the hybrid arm for the ACT uptake without a diagnostic test outcome. For my preferred specification, the models adjusted for baseline covariates, there are no longer significant differences between the patient discount and pharmacy incentive arms compared to the control group for the ACT uptake without a test outcome. Since these comparisons

are likely correlated with each other, this method of adjusting for multiple inference is likely too conservative because it does not take into account this dependence structure. Future publications will use an approach that does consider this, such as that developed by Clarke, Romano and Wolf [53]. Appendix tables B.6 and B.7 show that results are robust to using linear probability models. Appendix table B.7 includes coefficients for covariates used in the adjusted regressions, for reference. Appendix table B.10 shows an alternative specification which includes all baseline covariates from Table 1.

4.3 Treatment effects on secondary outcomes

Tables 4.5 and 4.6 present results from analyses looking at intervention effects on overall malaria patient shares, ACT uptake, and an alternative measure of treatment targeting - ACT uptake with an accompanying test, restricted to patients who purchase ACTs. The first outcome - malaria patient shares - was not pre-specified, but provides an important insight into whether there was meaningful sorting of patients with suspected malaria across intervention pharmacies. The second and third outcomes were pre-specified. Table 4.7 looks at the effects of pharmacy incentives and the hybrid approach compared to the patient subsidies on malaria positivity rate and alternate measures of treatment targeting that link patient purchase behavior to diagnostic test outcome from malaria tests conducted as part of the intervention.

Columns 1 and 2 of table 4.5 shows marginal effects from unadjusted logistic regression analysis on the outcome of share of pharmacy patients who purchased malaria products. During the study period, there were 265,610 pharmacy client interactions overall and 51,486 of these resulted in the sale of a malaria test or treatment (19%). I do not find any intervention effects on malaria patient share looking at the pooled intervention effect (column 1) or at each intervention arm separately (column 2). The adjusted models presented in columns 1 and 2 of table 4.6 yield the same results. This suggests that the interventions did not lead to patient sorting by likelihood of purchasing a malaria test or treatment, and instead the effects found on testing and treatment targeting described in the section above are due to behavior changes in a relatively stable patient pool, at least on this dimension. Chapter 5 presents results from a descriptive analysis of patient characteristics from the random subset of patients surveyed as part of the exit survey activity, to test whether sorting along different dimensions occurred.

Columns 3 and 4 of tables 4.5 and 4.6 shows the marginal effects from unadjusted and adjusted logistic regression models on the intervention effects on overall take up of ACTs, respectively. In the entire sample, 40,261 malaria patients purchased ACTs (78%). Notably, in the control group, 87% of all malaria product sales are ACTs - this is consistent with the availability of low cost, high quality treatment in the private sector due to the AMFm manufacturer level subsidies between 2011-2016, that was described in Chapter 1. Looking

at the intervention effects on this outcome, I find a pooled effect of -14 percentage points ($p < 0.01$), and individual treatment arm impacts of between -9 and -15 percentage points in the adjusted models ($T1 : -15pp, p < 0.05$, $T2 : -9pp, p < 0.1$, $T3 : -14pp, p < 0.01$), but no significant effects in the unadjusted model and no statistically significant differences across the treatment arms, or between supply- and demand-side interventions. The fact that interventions reduced the likelihood that patients purchased ACTs is a result of the increase in diagnostic testing, and in particular, the confirmed malaria-negative patients electing to not purchase medication.

Finally, columns 5 and 6 of tables 4.5 and 4.6 show an alternative measure of treatment targeting, which restricts the sample to pharmacy clients that purchase ACTs and looks at the likelihood that among that group, patients purchase an ACT with an accompanying test. Overall, 40,256 clients purchase ACTs and 15% of them do so with an accompanying diagnostic test ($N = 5,871$). In the control group, only 7% of patients who purchase ACTs do so with a diagnostic test. I find a 13 percentage point increase in the pooled intervention effect, and between a 9-13 percentage point increase when looking at each intervention arm separately. Appendix tables B.8 and B.9 show that results are robust to linear probability models. Appendix table B.9 includes coefficients for covariates used in the adjusted regressions, for reference. Appendix table B.11 shows an alternative specification which includes all baseline covariates from Table 1.

All of the analyses presented above are ITT estimates that look at overall intervention effects across all treatment arms and compare them to the status quo control group, and to each other. All patients who purchased malaria products were included in these ITT estimates, regardless of whether or not they purchased incentivized products. The next set of analyses, in Table 4.7 restricts the sample to only malaria patients who received diagnostic testing through the incentivized interventions (opted in to the incentive interventions). This can be thought of as a treatment-on-the-treated analysis, and I am only able to examine differences across treatment arms (no control group comparisons).[2] I present the marginal effects from unadjusted logistic regressions in this table. There are 8,478 malaria patients that make up this analysis sample - these are patients who sought care at treatment group pharmacies and elected to take a rapid diagnostic test through one of the incentivized channels. I first look at whether there are differences across intervention arms in terms of test positivity rate - the likelihood that a patient who is tested with malaria tests positive. Overall, 2,967 patients tested positive for malaria (35%). This positivity rate is consistent across patients who sought care in T1 pharmacies - those where tests and appropriate treatment were heavily discounted - and T3 pharmacies - those with the hybrid incentive of both patient subsidies and pharmacy incentives. However, the positivity rate in the pharmacy incentive arm (T2) is significantly higher at the 10% level by 14 percentage points, at 49%. This difference in test

[2]The treatment targeting outcomes shown here were pre-specified, but the analysis is more limited than what was pre-specified due to data limitations in the administrative data. I do not observe test result for all tested patients, just those who are tested as part of the incentivized interventions (in T1, T2, and T3).

positivity rate in T2 could mean pharmacists in these sites were more motivated to encourage patients who they perceived as having malaria (perhaps based on a clinical assessment of symptoms) to get tested through the incentive program. Linking this back to the theoretical framework in Chapter 2, this could be evidence of providers being incentivized to increase supplier induced demand for patients who they suspected were malaria positive based on clinical presentation.

Looking now at the treatment targeting outcomes for this selected sample, I turn to columns 2 and 3 of table 4.7. Among those patients who tested for malaria through the incentive programs, 3,025 tested positive across all three arms (35%). In the patient subsidy group, 99% of the malaria positive patients elected to purchase an ACT after receiving their test result, which is comparable to the hybrid group (98%). In the pharmacy incentive arm, this reduces to 92% (marginal effect of -7 percentage points, $p < 0.05$). Across all three groups, the vast majority of malaria positive patients then go on to take ACTs, but there does appear to be some reduced effect when looking at the pharmacy incentive only arm. In economic terms, this effect is not particularly meaningful, and should be considered as part of the results as a whole. Linking this back to the theory section and hypotheses in Chapter 2, this reduction in ACT uptake when positive in the pharmacy incentive only arm could be due to lack of incentive pass-through to patients (patients face a comparatively high cost of ACTs, so elect to purchase alternative medications or go to the public sector to seek care).

Next, I look at the differences across intervention arms of malaria negative patients purchasing antimalarials unnecessarily. Among those patients who tested for malaria through the incentive programs, 5,504 tested negative across all three arms (65%). In the patient subsidy group, 1% of the malaria negative patients elected to purchase antimalarials after receiving their test results. I find no difference in the pharmacy incentive only arm, but in the hybrid arm I find that this likelihood increases to 11% (increase of 10 percentage points, $p < 0.1$). In Chapter 5, I explore whether this result is also reflected in the data from the standardized patient visits, and propose possible hypotheses for why I am finding this effect. However, since the analyses in this table are from a selected sample of patients who chose to get tested in the treatment group sites, these should be thought of as mostly descriptive.

Summary of results and limitations

The interventions have a meaningful impact on both rapid test uptake and treatment targeting. For rapid test uptake, all three approaches to targeting financial incentives (supply side, demand side, or both) yield large increases in the likelihood that pharmacy patients with suspected malaria get diagnosed. This finding provides supporting evidence for Hypothesis 1, from Chapter 3. For treatment targeting, I find evidence that incentives are effective at increasing the likelihood that high quality treatments are appropriately targeted to patients who have a confirmed malaria diagnosis, when compared to the control group. I also find that all interventions reduce the likelihood that antimalarials are sold without an accompa-

nying test, though it is worth acknowledging that there are still high levels of uptake without a test. So, these types of incentives alone do not seem to completely address the problem of treatment without diagnosis. However, both of these results provide evidence in support for Hypothesis 2 in Chapter 3. I do not find any evidence to support Hypotheses 3a-3d, which hypothesized why I might have seen differences between supply- and demand-side incentives. The fact that I find no differences across the arms suggests that supply- and demand-side incentives lead to comparable effects on diagnostic testing and treatment targeting in this setting. I explore one possible mechanism for this result in Chapter 5.

Incentivizing providers, as is done in the provider only arm and the hybrid arm, may be important for improving treatment targeting outcomes in particular. I find no effects on overall malaria patient shares, suggesting that the interventions operate by changing the nature of the provider-patient interaction with a stable patient pool, rather than significant sorting across sites of patients who are more or less likely to seek malaria-related care. This suggests that the intervention effects that I see are a result of changing the nature of the patient-pharmacist interaction and affecting test-taking and treatment targeting behavior. Taken together, these results suggest that appropriately calibrated and targeted financial incentives are promising for improving malaria case management in a pharmacy setting.

This analysis has several limitations, which I discuss here. The first limitation is the reliance on administrative pharmacy transaction data for the main outcomes. Pharmacists log all sales transactions in a sales tracking and inventory management application, and this database is the most comprehensive source of micro-level pharmacy transaction data to my knowledge. However, these data may be incomplete if pharmacists choose to not log certain transactions in the app, and instead record them on paper or not at all. One of the eligibility criteria to participate in the study was commitment to use the digital sales management platform to record all transactions, and on average pharmacies were active on this platform for 8 months prior to the study start. However, it is not possible to tell if these records capture the full universe of all transactions, and the transaction data do not contain information on malaria test result for tests administered outside of the incentives program. The patient exit survey data that was collected for 1654 malaria clients is one source for validating the transaction records and test results for a subset of the study sample. The findings from this survey are discussed in Chapter 5 in more detail, but are again limited to a randomly selected subset of pharmacy patients who were willing to be surveyed. Future studies should consider supplementing administrative data collection with a more robust way of measuring key outcomes, including test result, for all relevant patient encounters.

Second, this study can only identify the impacts of the interventions on testing decisions and treatment purchase behavior. Given the duration of the treatment course, most malaria treatments are taken at home, without any supervision by a trained healthcare provider. It is unclear whether these interventions have an impact on treatment adherence, or on subsequent care-seeking behavior for future illness episodes for patients who were exposed to the

interventions. It is unclear whether removing the incentives would result in sustained behavior change over time, or if patients in this setting would respond quickly by reverting back to low levels of testing and over-use of antimalarials. Future work should consider examining the results on treatment adherence, and medium and long-term impacts of continued or phased out incentive interventions.

Third, this sample is not representative of the rest of Kenya. All pharmacies in the sample were drawn from eleven malaria-prone counties, and had to be active members of the Maisha Meds pharmacy network and be licensed and registered. Therefore, these pharmacies operate in areas of high malaria burden, represent a sample of sites that are more formalized than other chemists and drug shops that are present throughout the country, and may be more predisposed to adopt new technology or try new innovations given that they adopted the digital sales and inventory management platform. The pharmacies are located in mostly rural areas (80% of the sample is in rural areas) in regions of Kenya serving lower income populations. Therefore, it is unclear what these effects would look like if this program were scaled up nation-wide, or introduced to pharmacies that were not already using a digital tool to manage their businesses. Demographic characteristics of pharmacy patients from the exit surveys are discussed in the next chapter.

Finally, because the incentive amounts are held fixed across all three intervention arms (at $2 USD), the patient and provider incentive amounts are not directly comparable between the hybrid group and the other two intervention arms. Therefore, I am not able to look at the additive effect of the patient subsidy and provider incentive in the hybrid group. Any differential effects I may see in the hybrid group are due to the combination of a modest patient subsidy and a modest pharmacy incentive, and I am not able to disentangle which component is driving any differences.

4.4 Cost-effectiveness

In order to compare the efficiency of each intervention, I conducted a cost-effectiveness analysis from the perspective of the program implementer (including only Maisha Meds's program costs) and from a societal perspective (including Maisha Meds's cost and costs incurred by the care-seeking patient). I estimated incremental cost-effectiveness ratios (ICERs) in terms of cost per patient obtaining ACTs appropriately, defined as being malaria positive. I used a time horizon equal to the duration of the intervention period (8 months) and included all malaria patients who sought care at study pharmacies for this analysis. All methods, parameter inputs and assumptions are described in detail in Chapter 3.

Benefits

In the control group, the probability of taking an ACT conditional on being malaria positive is $< 1\%$, in each intervention arm this probability is 12%, 14% and 8% in the patient subsidy

group (T1), pharmacy incentives group (T2), and the hybrid group (T3), respectively. The total number of beneficiaries in each arm are 75, 874, 1093, and 626 in the control group, T1, T2, and T3, respectively (assuming a hypothetical cohort of 10000 suspected malaria patients who sought care in each arm). These estimates can be found in the top panel of Table 4.8.

Costs

In the control group, the total implementation cost is $0, because there is no programmatic cost of administering any incentive interventions. The costs for the intervention arms are $2,601.00, $4,084.00 and $5,039.00 in T1, T2, and T3 respectively. These cost differences are due to the differential take up of incentivized rapid tests and ACTs in each intervention arm, with the hybrid arm having the largest share of patients purchasing incentivized rapid tests driving most of this difference. These cost estimates can be found in the bottom panel of Table 4.8.

For the societal perspective, I also include the direct medication costs of over-treating malaria negative patients in each of the intervention arms, and the time costs to patients for seeking malaria care at pharmacies in each of the intervention arms in addition to the program implementation costs. In the control group, the total social costs are $361,459, and the societal costs for the intervention arms are $358,805, $335,149, and $374,615 in T1, T2 and T3 respectively. The cost differences are due to differential take up of incentivized rapid tests and ACTs in each intervention arm and the arm-specific malaria test negativity rate, which is highest in the hybrid arm. These cost estimates can be found in the bottom panel of Table 4.8.

Results

Table 4.9 presents the incremental cost of each intervention relative to the next less expensive alternative. From the implementer perspective (Maisha Meds's perspective), the incremental costs are relatively small, since the incentive amounts are modest. The control group (status quo) is the cheapest alternative, and the hybrid arm is the most expensive. From a societal perspective, both patient subsidies and pharmacy incentives are cost-saving interventions relative to the control group because of the lower costs incurred from fewer malaria negative patients being treated unnecessarily and lower time costs of care-seeking due to lower patient volumes. The hybrid arm is the most expensive from a societal perspective, because of the larger time cost to patients seeking care, relative to the control group.

Table 4.10 presents the incremental benefits and ICERs from the implementer perspective (top panel) and from the societal perspective (bottom panel). Within each panel, I present incremental gains and ICERs relative to the next best alternative and the incremental gains and ICERs for each intervention (patient subsidies, pharmacy incentives, or hybrid) relative

to the control group. I highlight the results with respect to the control group here, as that is the most policy-relevant benchmark when deciding amongst these possible intervention approaches. The control group resulted in 75 appropriately targeted ACTs. Patient subsidies resulted in 799 additional appropriately targeted ACTs at a cost of $3.26/patient, pharmacy incentives resulted in an additional 1018 patients treated appropriately at a cost of $4.01/patient, and the hybrid approach resulted in an additional 551 patients treated appropriately at a cost of $9.15/patient (all from Panel A, Table 4.10).

From a societal perspective, I find that patient subsidies result in an additional 799 patients treated appropriately with ACTs at a cost of -$32.93/patient compared to the control group, which is cost-saving. I find that pharmacy incentives are also cost saving: compared to the control group, this intervention leads to 1018 additional patients treated appropriately with ACTs at a cost of -$2.61/patient. And finally, the hybrid intervention leads to an additional 551 ACTs targeted appropriately compared to the control group, at a cost of $23.88/patient.

Summary of results and limitations

From the implementer perspective, all interventions are relatively low cost in terms of getting appropriate high quality treatment to malaria positive patients. From a societal perspective, the patient subsidy and the pharmacy incentive approaches were cost-saving compared to the control group, while the hybrid arm cost $24/patient for an additional 551 patients being appropriately treated, so is also relatively low cost. It is difficult to compare these cost-effectiveness estimates to other benchmarks, because the underlying assumptions and measures of costs and benefits vary widely across studies. A multi-country analysis from 2006 suggests that encouraging ACT use costs $150/DALY averted [55], and a study in Myanmar found that various subsidy programs for RDTs cost $639-$1169/DALY averted [56]. The difference between the 2006 and 2015 studies could be due to the fact that in that time frame, ACTs became much more common and the threat of drug resistant strains has increased. In order to make my results directly comparable to other studies, I will need to estimate costs/DALY. However, the pharmacy incentive arm was the most effective - yielding the largest increase ACTs reaching malaria positive patients relative to the control group. It also had an ICER below any potential budget or cost-effectiveness threshold, indicating that it may be the preferred strategy from both the implementer perspective and the societal perspective.

This analysis is limited in several ways, which I discuss here. First, without estimating the long-term costs and benefits, these estimates could be misleading. Second, I do not include the costs of rapid tests and treatments for malaria positive patients in these calculations. Future versions of this analysis should consider these costs as well.

Third, my estimates for calculating the probabilities of being malaria positive and malaria negative in the tested and untested samples are based on data collected from a subset of

the sample. In the control group, this is from the lab tech activity where testing was administered on a random subset of patients. In the treatment groups, the test positivity rates were obtained from the administrative data of patients who were tested through the incentive interventions. The overall (unconditional) malaria positivity rate was estimated in the control group using the testing data from the random subset of patients tested by lab techs associated with the research study, and aggregate test positivity data reported by pharmacies, and assumed to be the same in each intervention arm. While this assumption is not unreasonable, given that the geography and malaria prevalence in the study sites is similar across arms and the interventions were randomly assigned, it is possible that there are differences in overall malaria positivity rates for the set of patients who seek care across intervention arms. Future studies should consider obtaining estimates of test positivity from untested and tested samples of patients at multiple points during the year, to measure malaria prevalence rates throughout the year, and in each intervention arm (particularly to obtain measures of unconditional malaria positivity rates). Future versions of this analysis should include a sensitivity analysis to some of these key parameters.

Fourth, I do not include pharmacist time spent counseling patients as part of the costs for several reasons. First, since Maisha Meds does not compensate pharmacists beyond the incentives administered for selling tests and appropriate treatment, this is not part of the program costs. Second, since the share of patients who sought care at participating pharmacies that were malaria patients is consistent across all arms, including the control group, it is unclear how much additional time is spent by pharmacists in the treatment groups. I also do not have a good measure of pharmacist time spent carrying out the interventions. Future studies should examine the impact on provider workload and time allocation of incentivized programs such as this one.

Fifth, I do not include the costs or benefits to patients who test negative for malaria in obtaining further counseling and appropriate care, nor do I include the broader societal costs of over-treating malaria negative patients (quantifying drug resistance potential, for example). This is because I am not able to track patients who may seek additional care for their symptoms outside of the pharmacy setting, and therefore confirm the appropriateness or inappropriateness of any additional treatments they may receive. I am also not able to obtain convincing estimates of the broader costs of drug resistance in this setting. These are both important areas of future work when thinking about overall management of febrile illness, and future studies should consider measuring this aspect and incorporating it formally into a cost effectiveness framework.

4.5 Figures

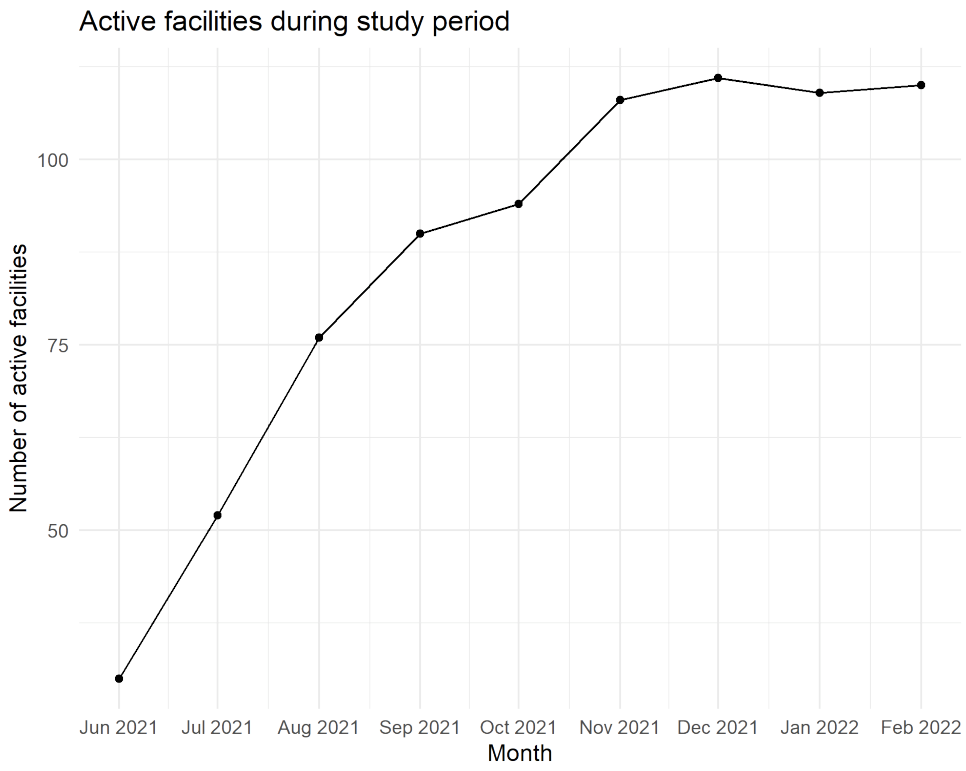


Figure 4.1: Number of active facilities during study period 4.1)

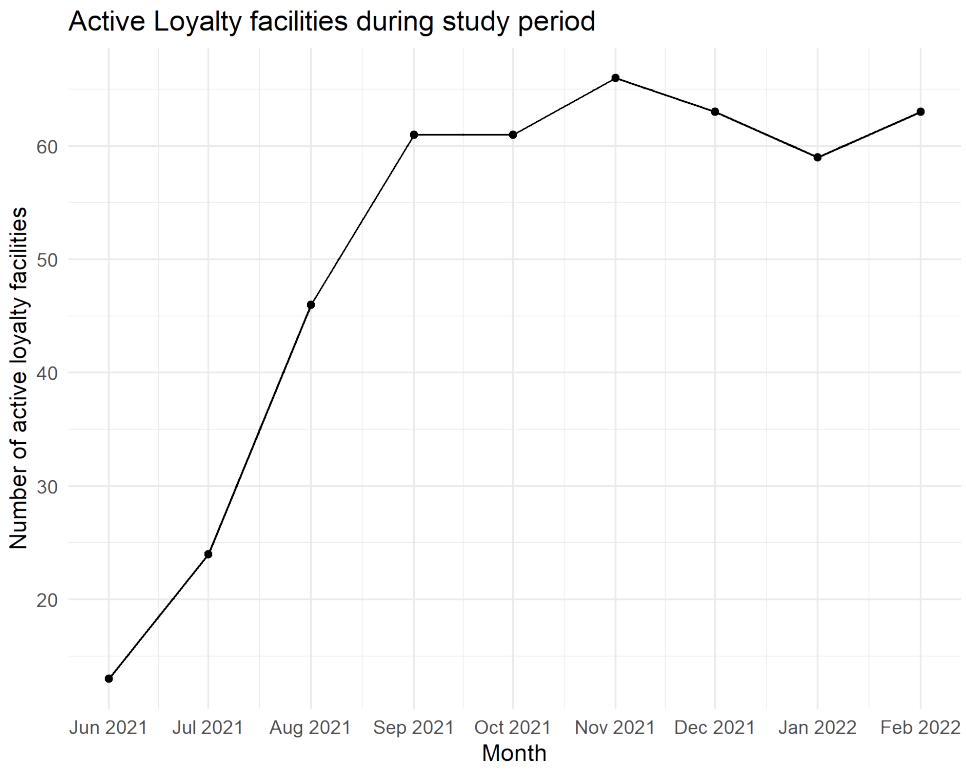


Figure 4.2: Number of active loyalty facilities during study period 4.1)

4.6 Tables

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
Number of months active on digital sales management tool	10.14	9.06	5.37*	8.11	3.69+	-0.94	-2.74
	(10.48)	(9.42)	(8.02)	(10.02)	(0.08)	(0.69)	(0.21)
Below median baseline malaria sales	0.37	0.46	0.37	0.46	0.09	-0.00	-0.09
	(0.49)	(0.51)	(0.49)	(0.51)	(0.47)	(1.00)	(0.47)
Average monthly malaria sales, 2019-2020	64.80	54.97	53.12	43.58+	1.85	-11.39	9.55
	(60.34)	(53.99)	(50.49)	(39.98)	(0.88)	(0.32)	(0.38)
Average monthly quality treatment sales, 2019-2020	52.84	46.89	47.64	38.60	-0.75	-8.29	9.04
	(39.27)	(50.21)	(48.16)	(38.46)	(0.95)	(0.44)	(0.39)
Average monthly rapid test sales, 2019-2020	4.30	7.56	4.61	3.53	2.95	-4.03+	1.08
	(5.34)	(11.35)	(9.17)	(4.06)	(0.24)	(0.05)	(0.53)
Site was in earlier pilot study phase	0.17	0.20	0.17	0.09	0.03	-0.11	0.09
	(0.38)	(0.41)	(0.38)	(0.28)	(0.76)	(0.18)	(0.29)
Site is in an urban area	0.20	0.29	0.40+	0.34	-0.11	0.06	0.06
	(0.41)	(0.46)	(0.50)	(0.48)	(0.32)	(0.61)	(0.63)
Site is in a malaria endemic county	0.89	0.77	0.80	0.91	-0.03	0.14	-0.11
	(0.32)	(0.43)	(0.41)	(0.28)	(0.77)	(0.10)	(0.18)
Site does not have clinical capabilities	0.71	0.83	0.83	0.83	0.00	-0.00	0.00
	(0.46)	(0.38)	(0.38)	(0.38)	(1.00)	(1.00)	(1.00)
Observations	35	35	35	35	70	70	70

Significance stars are from pairwise comparisons with the control group: + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
Multinomial logit test for joint orthogonality produces p-value from χ^2-test of 0.46

Table 4.1: Baseline Balance Between Treatment Arms, Administrative Data (Back: 4.1)

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
Percentage of pharmacy staff who are female	0.44	0.35	0.51	0.44	-0.16+	0.09	0.07
	(0.42)	(0.41)	(0.39)	(0.42)	(0.10)	(0.36)	(0.46)
Age of pharmacy owner	37.43	35.37	36.40	35.71	-1.03	0.34	0.69
	(5.28)	(7.74)	(7.14)	(8.08)	(0.57)	(0.86)	(0.71)
Average age of pharmacy staff	29.37	29.37	28.94	29.29	0.43	-0.09	-0.34
	(5.32)	(4.15)	(5.80)	(5.11)	(0.72)	(0.94)	(0.79)
Owner is female	0.36	0.16*	0.17+	0.17+	-0.01	0.01	0.00
	(0.48)	(0.36)	(0.38)	(0.36)	(0.87)	(0.87)	(1.00)
Number of pharmacy staff	1.54	1.54	1.49	1.40	0.06	-0.14	0.09
	(0.51)	(0.56)	(0.51)	(0.50)	(0.66)	(0.26)	(0.48)
Observations	35	35	35	35	70	70	70

Significance stars are from pairwise comparisons with the control group: + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
Multinomial logit test for joint orthogonality produces p-value from χ^2-test of 0.46

Table 4.2: Baseline Balance Between Treatment Arms, Pharmacy Survey Data (Back: 4.1)

Table 4.3: Impact on Primary Outcomes, Unadjusted Models (Back: 4.2)

	Rapid test uptake		ACT uptake with test		ACT uptake without test	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	.275**		.0854*		-.173*	
	(0.065)		(0.039)		(0.085)	
T1		.246*		.0703		-.171
		(0.123)		(0.060)		(0.135)
T2		.198**		.0729+		-.128
		(0.075)		(0.043)		(0.095)
T3		.249**		.0726*		-.191*
		(0.059)		(0.034)		(0.085)
Control mean	0.081	0.081	0.057	0.057	0.809	0.809
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2} \neq \gamma_{T3}$)		0.84		0.99		0.81
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2}$)		0.72		0.97		0.75
N	51441	51441	51486	51486	51441	51441

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: Strata FE
Wald test comparisons of difference in marginal effects (γ) between treatment arms
Denominator for all three outcomes is all patients that purchased malaria product during study period
Outcome 1 & 3: 45 obs dropped b/c multicollinearity (strata 11)
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 4.4: Impact on Primary Outcomes, Adjusted Models (Back: 4.2)

	Rapid test uptake		ACT uptake with test		ACT uptake without test	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	.252**		.0738*		-.197**	
	(0.052)		(0.035)		(0.061)	
T1		.272*		.0742		-.22+
		(0.113)		(0.055)		(0.117)
T2		.196**		.0752		-.16*
		(0.068)		(0.046)		(0.079)
T3		.198**		.0492+		-.181**
		(0.055)		(0.029)		(0.069)
Control mean	0.081	0.081	0.057	0.057	0.809	0.809
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2} \neq \gamma_{T3}$)		0.83		0.83		0.89
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2}$)		0.54		0.99		0.63
N	51441	51441	51486	51486	51441	51441

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: months active on platform, baseline malaria sales, female owner, strata FE
Wald test comparisons of difference in marginal effects (γ) between treatment arms
Denominator for all three outcomes is all patients that purchased malaria product during study period
Outcome 1 & 3: 45 obs dropped b/c multicollinearity (strata 11)
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 4.5: Impact on Secondary Outcomes, Unadjusted Models (Back: 4.3)

	Antimalarial sales overall		ACT uptake, all malaria product purchases		ACT uptake with test, ACT purchases	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	-.0139		-.102		.134*	
	(0.029)		(0.084)		(0.053)	
T1		.0279		-.104		.0996
		(0.032)		(0.092)		(0.091)
T2		-.0167		-.0568		.103+
		(0.034)		(0.080)		(0.059)
T3		-.0304		-.119		.128*
		(0.034)		(0.076)		(0.050)
Control mean	0.197	0.197	0.867	0.867	0.066	0.066
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2} \neq \gamma_{T3}$)		0.14		0.27		0.57
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2}$)		0.12		0.19		0.54
N	265610	265610	51486	51486	40256	40256

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: Strata FE
Wald test comparisons of difference in marginal effects (γ) between treatment arms
Outcome 3: 50 obs dropped b/c multicollinearity (strata 11)
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 4.6: Impact on Secondary Outcomes, Adjusted Models (Back: 4.3)

	Antimalarial sales overall		ACT uptake, all malaria product purchases		ACT uptake with test, ACT purchases	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	-.000792		-.139**		.125**	
	(0.024)		(0.050)		(0.046)	
T1		.0243		-.147*		.123
		(0.025)		(0.073)		(0.100)
T2		-.00859		-.0907^{+}		.112^{+}
		(0.033)		(0.052)		(0.062)
T3		-.00829		-.136**		.093*
		(0.027)		(0.047)		(0.043)
Control mean	0.197	0.197	0.867	0.867	0.066	0.066
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2} \neq \gamma_{T3}$)		0.27		0.64		0.95
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2}$)		0.19		0.46		0.92
N	265610	265610	51486	51486	40256	40256

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: months active on platform, baseline malaria sales, female owner, strata FE
Wald test comparisons of difference in marginal effects (γ) between treatment arms
Outcome 3: 50 obs dropped b/c multicollinearity (strata 11)
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 4.7: Within intervention arm impacts on test positivity outcomes, Unadjusted Models (Back: 4.3)

	Rapid test positivity rate	ACT uptake with positive test	Antimalarial uptake with negative test
	(1)	(2)	(3)
T2	.141^{+}	-.0655*	.00278
	(0.077)	(0.028)	(0.006)
T3	-.0563	-.016	.104^{+}
	(0.074)	(0.010)	(0.054)
T1 mean	0.354	0.996	0.014
Wald test p-val ($\gamma_{T2} \neq \gamma_{T3}$)	<0.001	0.119	0.054
N	8478	2967	5213

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: strata FE
Wald test comparisons of difference in marginal effects (γ) between T2 and T3
Outcome 1 denominator: all patients were tested through incentive interventions
Outcome 2 denominator: all patients who tested positive through incentive interventions
Outcome 3 denominator: all patients who tested negative through incentive interventions
Outcome 1: 51 observations dropped from strata 11, 12 & 15 for multicollinearity
Outcome 2: 58 observations dropped from strata 1, 4, 7 & 8 for multicollinearity
Outcome 3: 291 observations dropped from strata 1, 4, 8, 11, 14 & 15 for multicollinearity
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 4.8: Benefits and Costs Estimates (Back: 4.4)

	Control (status quo)	Patient subsidies	Provider incentives	Hybrid
		BENEFITS		
P(ACT | malaria positive)	0.009	0.121	0.141	0.086
Number of patients taking ACTs	8670	7200	7763	7310
Number of beneficiaries	75	874	1093	626
		COSTS		
Total cost of incentives	$0.00	$2,601.00	$4,084.00	$5,039.00
Cost of over-treating malaria negative patients	$618.68	$1,943.89	$4,405.45	$1,815.58
Total time cost to patients seeking care	$360,840.00	$354,260.00	$326,660.00	$367,760.00
Total costs - societal perspective	$361,458.68	$358,804.89	$335,149.45	$374,614.58
Total costs - implementer perspective	$0.00	$2,601.00	$4,084.00	$5,039.00

Table 4.9: Incremental Costs (Back: 4.4)

	Implementer perspective	
	Costs	Inc. cost
Control (status quo)	$0.00	-
TI - Patient subsidies	$2,601.00	$2,601.00
T2 - Provider incentives	$4,084.00	$1,483.00
T3 - Hybrid	$5,039.00	$955.00
	Societal perspective	
	Costs	Inc. cost
T2 - Provider incentives	$335,149.45	-
T1 - Patient subsidies	$358,804.89	$23,655.44
Control (status quo)	$361,458.68	$2,653.79
T3 - Hybrid	$374,614.58	$13,155.90

Implementer perspective includes only incentive costs. Societal perspective includes incentive costs, costs of overtreating malaria negative patients, and time costs. Incremental cost = incremental cost relative to next most expensive alternative.

Table 4.10: Incremental Benefits and ICERs (Back: 4.4)

Incremental benefits and ICERs - Implementer perspective

	Number of malaria positive patients treated with ACTs	Incremental appropriately treated	ICER Cost / patient appropriately treated
		Compared to next best alternative	
Control (status quo)	75	-	-
T3 - Hybrid	626	551	$9.15
T1 - Patient subsidies	874	248	-$9.83
T2 - Provider incentives	1093	219	$6.77
		Each intervention compared to the control group	
T1 vs. C	-	799	$3.26
T2 vs. C	-	1018	$4.01
T3 vs. C	-	551	$9.15

Incremental benefits and ICERs - Societal perspective

	Number of malaria positive patients treated with ACTs	Incremental appropriately treated	ICER Cost / patient appropriately treated
		Compared to next best alternative	
Control (status quo)	75	-	-
T3 - Hybrid	626	551	$71.62
T1 - Patient subsidies	874	248	-$63.75
T2 - Provider incentives	1093	219	-$108.02
		Each intervention compared to the control group	
T1 vs. C	-	799	-$32.93
T2 vs. C	-	1018	-$2.61
T3 vs. C	-	551	$23.88

Chapter 5

Analysis of tertiary outcomes

This chapter is broken up into two sections: the first (Section 5.1) is a descriptive analysis of the patient population who sought malaria care at study pharmacies, and was surveyed as part of the exit surveys. It contextualizes the findings from the administrative data presented in Chapter 4, by providing details about what types of patients are served by study pharmacies, and whether there are any key differences in the patient population by intervention arm. The second section of this chapter (5.2) analyzes the extent to which pass-through of incentives occurred in the intervention arms, using data collected as part of the Standardized Patient exit surveys. This analysis tests one key hypothesis about a possible mechanism through which the interventions operate.

5.1 Descriptive patient characteristics

Patients who completed the exit surveys were all adults, the majority completed secondary school (between 59-63%, depending on the intervention arm), over 70% were married, and over half were head of their households. Households tended to have between 4-5 members, with between 2-3 dependents under the age of 18, and an average of 0.89 children under the age of 5. Demographic differences in the patient population surveyed across treatment arms are minimal, and are found in Table 5.1. In Kenya as a whole, 25% of the adult population has completed secondary school, the average household size is 3.9, and the fertility rate is 3.4 [57]. Therefore, compared to recent national averages, the patients in the study sample are more educated, have larger households, and have fewer children. This is not seemingly driven by differences in the age distribution between my sample and the Kenyan national average. Nationally, more than half of adults (53%) are below the age of 35, 20% are between 35-44, and the remaining 27% are above the age of 45 [58]. In my sample, 53% of respondents are below the age of 35, 24% are between 35-44, and the remaining 23% are above the age of 45. It is possible that the distribution of adults in the 45 years and older category in my sample is much younger than the national average, which would explain the smaller household sizes

and lower fertility rates, but I do not have the age distribution at a more granular level for this group.

Table 5.2 provides details about the pharmacy visit. Over half of survey respondents went to the pharmacy to seek care for malaria related symptoms for themselves, and one third went to the pharmacy with one of their children, who was sick with malaria symptoms. The pharmacies that patients visited were familiar to them, with more than half of all respondents stating that they visited the pharmacy often and over 80% stating that they visited at least sometimes. This suggests that patients generally sought care at a pharmacy that was familiar to them, rather than seeking out an unfamiliar pharmacy. Most patients walked or took a boda boda to the pharmacy, with the average travel time being between 15-18 minutes, depending on the intervention arm. Almost all patients who were surveyed reported that they suspected that they had malaria prior to visiting the pharmacy, which is consistent with the idea that patients have strong initial beliefs about their malaria status. However, despite most suspecting malaria, only 65% of respondents reported being very confident in their malaria status prior to visiting the pharmacy. While this is still quite high, it is not the case that all patients who suspect malaria are confident in their illness status. Interestingly, there are differences across intervention arms in terms of whether the patient went to the pharmacy with the intention of being tested for malaria. Only 32% of control group patients wanted a malaria test prior to their visit, while between 46-54% of patients in the treatment arms wanted to be tested. These differences are statistically significant, suggesting that the incentive interventions may have changed the way that patients were thinking about diagnostic testing prior to seeking care, or changed the way that they remembered their preferences for testing before seeking care.

Table 5.3 provides details about the purchases made by patients during their pharmacy visits. First, 27% of control group patients reported purchasing a rapid test at the time of their pharmacy visit. This is significantly higher than the average rapid test uptake from the administrative data in this group of 8%. This difference could be due to social desirability bias of respondents, or perhaps the presence of field staff at the pharmacies on survey days nudged providers to be more proactive in recommending rapid tests for malaria. Looking at the rapid test uptake in the intervention arms, between 37-41% of patients purchased rapid tests, which is significantly different from the control group mean. Among patients who were tested for malaria, I find significant differences in self-reported test positivity. In the control group, 68% of patients tested reported receiving a positive test. In the patient subsidy arm, 33% of patients tested positive, in the pharmacy incentive arm, 52% of patients tested positive, and in the hybrid arm 43% tested positive. These differences in test positivity rates across treatment arms could be due to providers in the intervention testing more people who had a lower chance of positivity, or alternatively, providers in the control group inaccurately reporting test results. Future analysis of the Standardized Patient encounters will test these hypotheses. These rapid tests include both those that were incentivized as part of the interventions, but also any rapid tests that were purchased at study pharmacies outside of

the interventions (not incentivized). Looking at treatment purchases, in the control group 27% of patients report purchasing a WHO pre-qualified ACT, while in the patient subsidy group this reduces to 21% ($p < 0.05$), perhaps as a result of the lower test positivity rate in this sample. In the incentive and hybrid arms, between 32-33% of patients purchase WHO-prequalified ACTs. Looking at purchase of other ACTs and other antimalarials, I find that between 33-47% of patients across intervention arms purchase other ACTs and less than 20% purchase other antimalarials. Very few respondents reported receiving a discount on either a test or treatment: in the control group this was 10%, suggesting that pharmacists may discount products to certain patients on an ad hoc basis. The only intervention arm that was significantly higher than this was the pharmacy incentive arm - 20% of patients in this group reported receiving discounted products.

These descriptive results from the patient exit survey add nuance to the main impact evaluation results. The fact that I find differential intervention impact on testing uptake compared to the control group gives confidence to the results found in the administrative data of the interventions leading to higher testing uptake. The differences in test positivity reported through the exit surveys are directionally consistent with the differences in test positivity reported in the administrative data (group means from Table 4.7, column 1), with the exception of patients in the hybrid arm reporting a 29% test positivity rate in the administrative data but a 43% positivity rate in the patient exit survey data. The differential test positivity rates in the intervention arms suggest either that tests are being given to different types of patients (in terms of malaria positivity) or that providers who administer tests outside of the incentives program (such as in the control group) may report inaccurate results. Understanding these mechanisms will be important to contextualize the overall impact evaluation effects on treatment targeting in each intervention arm. Finally, the lack of awareness of discounts in the intervention arms may be due to pharmacy patients not being aware that they are receiving a product as part of a special promotion (so, the discounted prices that they face could be the price effect, rather than a promotion effect), and therefore may have implications for the durability of the effects. Alternatively, lack of awareness of discounts could be due to pharmacies imperfectly complying with the intervention protocols of offering the incentivized program to all eligible malaria patients. If this is the case, then any differences across intervention arms in terms of which patients are offered the program may have distributional implications if pharmacists are making decisions based on observable patient characteristics like wealth or visible symptoms.[1]

[1]In the control group, 1168 patients purchased rapid tests. In the patient discount group, 3616 patients purchased rapid tests, with 55% of those being through the incentives program. In the pharmacy incentive group, 3709 patients purchased rapid tests, with 73% of those being through the incentives program. In the hybrid arm, 5092 patients purchased rapid tests, with 75% of those being through the incentives program. The fact that not all patients purchase incentivized tests in the intervention groups could either be due to imperfect compliance with intervention protocols on the part of pharmacists, or due to patients choosing to not purchase incentivized products because of the time cost of registering the test result with the digital tool. I am not able to disentangle these mechanisms using the exit survey data, but can explore this in the Standardized Patient data.

Finally, Table 5.4 provides details on malaria prevention behaviors, to measure the extent of knowledge of proper prevention and use of preventative measures outside of the pharmacy setting. Nearly all households in the sample report owning insecticide-treated bednets (between 92-96% of all respondents), and households own an average of 2.81 nets, which they report to be in mostly good condition. Respondents also report that they and their children sleep nearly every night underneath a treated bednet. There are some small differences among treatment arms in these two measures, but the magnitudes are small and not practically meaningful. At best, these data points reflect true prevention behavior for the patient population surveyed. However, due to social desirability bias and the limitations of self-reported data, these data should be interpreted cautiously. These descriptive statistics do demonstrate that the patient population is knowledgeable about how to prevent malaria, and best practices for how to mitigate its spread in their homes. This reflects the fact that malaria is endemic in the study areas, and the patient population targeted by these interventions and this study is very familiar with the illness.

5.2 Evidence of incentive pass-through

In order to test the hypotheses from Chapter 3 about incentive pass-through, I use exit survey data from the SP visits. SPs conducted a total of 411 visits across 137 facilities in the study sample, with three different SPs visiting each facility. Half of the SP visits were conducted by women. SPs followed a uniform script for how to present a suspected malaria case in a pharmacy setting: SPs were instructed to complain of fever, headache and joint pains in their opening statement and then provided additional information about their illness episode and health history if the pharmacist followed up with additional questions. The script can be found in Appendix 3.

SP visits provided a unique opportunity to assess the implementation fidelity and quality of care of the patient-provider interaction at study pharmacies, and Table 5.5 presents the results described below that characterize the visits. Very few SPs reported that there were signs present at the pharmacies they visited with details about discounted rapid tests or treatment - pharmacies were encouraged to put up posters with price information and were provided with program-specific materials to display at their sites. So, the fact that almost none of the pharmacies had these materials visible indicates that information was not readily available to patients when they came in for care. Over half of SPs reported that they were offered a rapid test for malaria, with no differences across intervention arms (C: 53%; T1: 55%, 95% CI: (40%,70%); T2: 54%, 95% CI: (37%,71%); T3: 59%, 95% CI: (42%, 76%)). Interestingly, this is higher than the rapid test uptake seen in the administrative data and patient exit survey data. This could reflect the fact that SPs were instructed to not state that they suspected malaria, but rather presented malaria symptoms and waited for the provider to give a clinical assessment and diagnosis. By contrast, pharmacy patients and patients surveyed by the exit surveys may have been more direct in stating that they suspected

malaria in their initial interaction with the pharmacist, and pharmacists may have been more likely to bypass the step of diagnostic testing if a patient demanded medication or indicated confidence in their malaria status.

Of the SPs that took a rapid test at the pharmacy (N=206), I find significant differences in test positivity rates across intervention arms. In the control group, 39% of SPs reported that the pharmacist told them that their test was positive, with significantly lower test positivity rates in each of the intervention arms (between 16-24%). All SPs were confirmed to be malaria-negative prior to beginning field work, and were tested regularly at high quality labs if any symptoms arose during the data collection period. So, all tests should have come back negative given that the SPs did not have malaria by design. The false positive rate for rapid diagnostic tests is 22%, and the positivity rates found in the intervention arms are consistent with this [36, 37]. The implications of this finding of differential test positivity from the SP data suggests that the interventions allow pharmacists to truthfully report malaria test results to patients without losing potential profits from not selling malaria treatment. In the control group, where pharmacists are not incentivized, they seemingly over-report positive test results in order to sell additional malaria medication.

The fact that test positivity rates were lower in intervention group pharmacies suggests that the interventions may have increased transparency in test result reporting because pharmacists had to upload the result to the digital platform and discuss the result with the patient. Half of SPs who tested in the control group saw their results on the rapid test cassette, while between 63-75% of SPs in intervention arms saw their results on the rapid test cassette.

Consistent with the patient exit survey findings, very few SPs reported that the pharmacist explicitly offered them a discount on rapid tests and/or treatments - suggesting that the incentivized products may not have been framed as discounted. In the control group sites, SPs paid an average of 100.78 KES ($0.87 USD) for rapid tests, which is consistent with the average market price benchmarks found at baseline. In the patient subsidy arm, SPs paid an average of 85.71 KES ($0.76 USD), and in the hybrid arm, SPs paid an average of 91.3 KES ($0.79 USD), both of which are significantly different from the amount paid in the control group. By contrast, patients in the pharmacy incentive arm paid an average of 100.77 KES ($0.87 USD) for the rapid test, which is statistically indistinguishable from the amount paid in the control group. On average, SPs paid 136.84 KES ($1.18 USD) for medications with no significant differences across intervention arms. Besides rapid tests and medications, SPs also may have incurred consultation fees or other fees as part of their pharmacy visit. On average, SPs paid 266.67 KES ($2.30 USD) for medications and other fees, and there are no meaningful differences across intervention arms except for when comparing the pharmacy incentives group to the hybrid group and to the control group. SPs in the pharmacy incentives group had higher total payments than either the control group or the hybrid group, but not when compared to the patient subsidy group. Across

all intervention arms, SPs waited 7.68 minutes for their test result to be ready, which is less than the recommended guidance of 15 minutes. A full analysis of quality of care is beyond the scope of this dissertation, but will be analyzed in future work.

The key comparisons that I test to measure incentive pass-through are whether the effects from the pharmacy incentive group (T2) are different from those in the patient subsidy group (T1), and whether the effects from the hybrid group (T2) are different from those in the patient subsidy group (T1). Table 5.6 shows results from regression analysis of these key comparisons. Column 2 of Table 5.6 shows that the SPs who purchased malaria products from intervention pharmacies were not informed about any discounts at any higher or lower rate than in the control group. However, I find that SPs who sought care in the patient subsidy arm were marginally more likely to have been informed of a discount than those who sought care in the pharmacy incentive arm (7.93% compared to 2.1%, where the Wald test p-value comparing these two coefficients = 0.086[2]) or than those who sought care in the hybrid arm (7.93% compared to 1.9%, where the Wald test p-value comparing these two coefficients = 0.074). Column 4 of table 5.6 looks at whether there were any differences in the $log(price)$ that SPs paid for rapid tests at participating study pharmacies. Overall, I find significant differences in purchase prices when comparing each intervention to the control group. SPs report paying 40% less for rapid tests in the patient subsidy arm, compared to in the control group ($p < 0.05$, 95% CI: (8%, 72%)), and SPs in the hybrid group report paying 17% less for rapid tests compared to SPs in the control group ($p < 0.1$, 95% CI: (0%, 35%)). The magnitude of the discount offered in the patient subsidy group is 90% for rapid tests, so there is suggestive evidence here that not all of this is getting passed through to patients. However, because it is possible that patients purchased rapid tests at an intervention pharmacy that were not subsidized, this may reflect that pharmacists did not always enroll patients in the incentive program. Additionally, I find significant differences in purchase price for rapid tests when comparing the pharmacy incentive group to the patient subsidy group - SPs in the patient subsidy group pay significantly less for rapid tests than they do in the pharmacy incentive group. This suggests that incentive pass through is not occurring in the pharmacy incentive arm, but is at least partially in the patient subsidy arm. I do not find any evidence of differential pricing when looking at medication sales.

Summary of results and limitations

There is suggestive evidence that providers in the pharmacy incentive arm did not pass through any incentives to patients in the form of reduced prices on tests or treatments. SPs who purchased products in the patient subsidy arm were more likely to have reported receiving a discount, and paid less for rapid tests than when they sought care in the pharmacy incentive group. This analysis suggests that the mechanism of incentive pass through in the

[2]For example, 7.98% is calculated by adding the coefficient on T1 0.0313 to the control group mean of 0.048. The rest of these group means are calculated using the same approach, from estimates in column 2 of Table 5.6.

pharmacy incentive and hybrid arms is unlikely to be what drives the results seen in the administrative data of very similar levels of uptake in rapid testing and treatment targeting across all three intervention arms. Other mechanisms, such as improved provider counseling and proactively nudging patients toward testing, should be explored in future work.

This analysis of incentive pass-through suffers from several limitations. First, it is based on a relatively small sample of SP visits. This analysis should be complemented by analysis of price data from a larger sample of patients, which can be obtained from the exit survey data. Second, the SP case presentation was meant to mirror what a typical suspected-malaria patient interaction looked like, and the SPs were instructed to present generalized symptoms but not state that they suspected malaria in the opening statement. This was done in order to assess the diagnostic ability of pharmacists and obtain information on testing recommendations and proper adherence to protocols. If the majority of patients who seek care at these pharmacies are confident in their malaria status and demand antimalarials without a test, then the SP sample could look different from the average patient population. Future work will look at a variant on this standard case where SPs were more certain in their malaria presentation and demanded antimalarials at the onset of the patient-pharmacist interaction.

5.3 Tables

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
Respondent completed secondary school	0.63	0.61	0.59	0.59	0.03	-0.02	-0.01
	(0.48)	(0.49)	(0.49)	(0.49)	(0.48)	(0.58)	(0.85)
Respondent is married	0.71	0.71	0.72	0.71	-0.01	-0.00	0.02
	(0.46)	(0.45)	(0.45)	(0.46)	(0.65)	(0.90)	(0.57)
Respondent is head of household	0.58	0.58	0.51+	0.55	0.07*	-0.03	-0.04
	(0.49)	(0.49)	(0.50)	(0.50)	(0.05)	(0.38)	(0.26)
Number of household members	4.76	4.41*	4.72	4.69	-0.31+	0.28+	0.03
	(2.22)	(2.12)	(2.32)	(2.35)	(0.05)	(0.07)	(0.87)
Number of children in household	2.33	2.26	2.55	2.42	-0.29*	0.16	0.13
	(1.77)	(1.82)	(2.07)	(1.75)	(0.04)	(0.19)	(0.36)
Number of under-5 children in household	0.89	0.94	0.81	0.90	0.13*	-0.04	-0.09
	(0.80)	(0.80)	(0.74)	(0.82)	(0.04)	(0.52)	(0.15)
Observations	422	416	360	432	794	866	810

Significance stars in columns 1-4 are from pairwise comparisons with the control group
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 5.1: Patient demographics, exit surveys (Back: 5.1)

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
Respondent was sick: reason for visit	0.58	0.57	0.60	0.60	-0.03	0.03	-0.00
	(0.49)	(0.50)	(0.49)	(0.49)	(0.47)	(0.39)	(0.93)
Child was sick: reason for visit	0.31	0.35	0.31	0.30	0.04	-0.05+	0.02
	(0.46)	(0.48)	(0.47)	(0.46)	(0.30)	(0.09)	(0.57)
Adult family member was sick: reason for visit	0.08	0.07	0.08	0.07	-0.01	0.00	0.01
	(0.27)	(0.25)	(0.27)	(0.25)	(0.47)	(0.89)	(0.55)
Respondent visits facility often	0.52	0.49	0.55	0.53	-0.06+	0.04	0.02
	(0.50)	(0.50)	(0.50)	(0.50)	(0.09)	(0.29)	(0.50)
Female pharmacy patient	0.53	0.54	0.55	0.51	-0.01	-0.02	0.04
	(0.50)	(0.50)	(0.50)	(0.50)	(0.69)	(0.50)	(0.29)
Patient was referred to pharmacy	0.07	0.05	0.05	0.02	0.01**	-0.04**	0.03*
	(0.26)	(0.22)	(0.21)	(0.13)	(0.61)	(0.00)	(0.02)
Travel time to pharmacy	18.90	15.70**	18.39	16.64*	-2.68**	0.94	1.74+
	(16.82)	(12.81)	(14.64)	(14.62)	(0.01)	(0.32)	(0.10)
Provider gender	0.48	0.42+	0.55+	0.48	-0.13**	0.06+	0.06+
	(0.50)	(0.49)	(0.50)	(0.50)	(0.00)	(0.06)	(0.07)
Patient wanted a malaria test prior to visit	0.32	0.47**	0.46**	0.54**	0.01	0.07+	-0.08*
	(0.47)	(0.50)	(0.50)	(0.50)	(0.86)	(0.05)	(0.04)
Patient suspected to have malaria	0.92	0.95	0.90	0.94	0.05*	-0.00	-0.04*
	(0.28)	(0.23)	(0.30)	(0.23)	(0.02)	(0.92)	(0.02)
Patient was very confident in malaria status	0.61	0.58	0.63	0.68*	-0.05	0.09**	-0.04
	(0.49)	(0.49)	(0.48)	(0.47)	(0.16)	(0.00)	(0.19)
Minutes spent with provider during visit	18.04	17.71	16.81	18.39	0.90	0.67	-1.58
	(29.05)	(22.28)	(20.20)	(20.58)	(0.56)	(0.65)	(0.28)
Observations	422	416	360	432	794	866	810

Significance stars in columns 1-4 are from pairwise comparisons with the control group
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 5.2: Pharmacy visit details, exit surveys (Back: 5.1)

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
Patient purchased rapid test	0.27	0.37**	0.37**	0.41**	0.00	0.03	-0.03
	(0.45)	(0.48)	(0.48)	(0.49)	(0.97)	(0.32)	(0.32)
Patient purchased microscopy	0.09	0.09	0.08	0.06	0.01	-0.03	0.02
	(0.28)	(0.28)	(0.27)	(0.23)	(0.58)	(0.11)	(0.33)
Patient purchased WHO-prequalified ACT	0.27	0.21*	0.32	0.33*	-0.11**	0.13**	-0.01
	(0.44)	(0.40)	(0.47)	(0.47)	(0.00)	(0.00)	(0.70)
Patient purchased other ACT	0.42	0.37	0.33*	0.35*	0.04	-0.02	-0.02
	(0.49)	(0.48)	(0.47)	(0.48)	(0.31)	(0.65)	(0.56)
Patient purchased other antimalarial	0.18	0.14	0.17	0.07**	-0.02	-0.07**	0.10**
	(0.39)	(0.35)	(0.37)	(0.26)	(0.37)	(0.00)	(0.00)
Patient purchased antibiotics	0.29	0.28	0.25	0.24+	0.03	-0.04	0.01
	(0.46)	(0.45)	(0.43)	(0.42)	(0.30)	(0.14)	(0.70)
Patient paid cash for purchase	0.87	0.92*	0.82*	0.92*	0.11**	-0.01	-0.10**
	(0.33)	(0.26)	(0.39)	(0.27)	(0.00)	(0.73)	(0.00)
Patient received discount for purchase	0.10	0.12	0.20**	0.10	-0.08**	-0.02	0.09**
	(0.30)	(0.33)	(0.40)	(0.31)	(0.00)	(0.46)	(0.00)
Patient used rapid test at pharmacy	0.29	0.46**	0.38**	0.43**	0.08*	-0.02	-0.06+
	(0.45)	(0.50)	(0.48)	(0.50)	(0.02)	(0.49)	(0.10)
Patient tested positive for malaria (among rapid test users)	0.68	0.33**	0.52**	0.43**	-0.19**	0.10*	0.09
	(0.47)	(0.47)	(0.50)	(0.50)	(0.00)	(0.04)	(0.13)
Observations	422	416	360	432	794	866	810

Significance stars in columns 1-4 are from pairwise comparisons with the control group
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 5.3: Purchase details, exit surveys (Back: 5.1)

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
Household owns mosquito nets	0.94	0.92	0.95	0.96	-0.03+	0.03*	-0.00
	(0.24)	(0.27)	(0.21)	(0.21)	(0.09)	(0.04)	(0.82)
Number of bednets owned	2.85	2.81	2.74	2.85	0.07	0.03	-0.11
	(1.34)	(1.48)	(1.45)	(1.59)	(0.52)	(0.75)	(0.35)
Bednets are in good condition	0.86	0.87	0.78**	0.87	0.09**	-0.00	-0.09**
	(0.35)	(0.33)	(0.41)	(0.34)	(0.00)	(0.88)	(0.00)
Days in last week patient slept under bednet	6.84	6.69*	6.80	6.66*	-0.11	-0.03	0.14
	(0.84)	(1.28)	(1.09)	(1.32)	(0.21)	(0.73)	(0.11)
Days in last week youngest child slept under bednet	6.63	6.61	6.38+	6.39+	0.23	-0.22+	-0.01
	(1.53)	(1.55)	(1.96)	(1.93)	(0.11)	(0.10)	(0.96)
Observations	422	416	360	432	794	866	810

Significance stars in columns 1-4 are from pairwise comparisons with the control group
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 5.4: Malaria prevention behaviors, exit surveys (Back: 5.1)

Variable	(1) Control	(2) T1	(3) T2	(4) T3	(5) T1-T2	(6) T3-T1	(7) T2-T3
SP is female	0.53	0.52	0.51	0.48	0.01	-0.04	0.03
	(0.50)	(0.50)	(0.50)	(0.50)	(0.89)	(0.58)	(0.68)
Sign visible at pharmacy about rapid test availability	0.03	0.03	0.03	0.01	-0.00	-0.02	0.02
	(0.17)	(0.17)	(0.17)	(0.10)	(1.00)	(0.31)	(0.31)
Sign visible at pharmacy at malaria treatment availability	0.03	0.01	0.03	0.08	-0.02	0.07*	-0.05
	(0.17)	(0.10)	(0.17)	(0.27)	(0.31)	(0.02)	(0.12)
SP was offered rapid test	0.53	0.55	0.54	0.59	0.01	0.04	-0.05
	(0.50)	(0.50)	(0.50)	(0.49)	(0.89)	(0.57)	(0.48)
SP was offered microscopy	0.09	0.17+	0.12	0.06	0.05	-0.11*	0.06
	(0.28)	(0.37)	(0.32)	(0.24)	(0.32)	(0.01)	(0.14)
Length of time (minutes) SP waited for test results	7.98	7.43	7.10	8.19	0.33	0.76	-1.09
	(4.89)	(3.38)	(3.55)	(3.97)	(0.63)	(0.30)	(0.14)
Rapid test result was positive	0.39	0.16**	0.21**	0.24*	-0.05	0.08	-0.03
	(0.49)	(0.37)	(0.41)	(0.43)	(0.54)	(0.33)	(0.72)
SP saw test results on rapid test cassette	0.51	0.63	0.75*	0.67	-0.12	0.03	0.08
	(0.50)	(0.49)	(0.44)	(0.48)	(0.21)	(0.72)	(0.35)
SP was explicitly offered a discount at time of purchase	0.05	0.08	0.03	0.03	0.05	-0.05	-0.00
	(0.21)	(0.27)	(0.17)	(0.17)	(0.12)	(0.12)	(1.00)
Number of medications purchased by SP	1.68	1.59	1.84	1.56	-0.25+	-0.03	0.28*
	(0.91)	(1.01)	(0.88)	(1.00)	(0.06)	(0.83)	(0.03)
Amount paid for rapid test (KES)	100.78	85.71*	100.77	91.30+	-15.05*	5.58	9.47*
	(27.70)	(42.57)	(20.37)	(27.06)	(0.02)	(0.42)	(0.04)
Amount paid for medication (KES)	143.52	163.64	100.69	139.31	62.95+	-24.32	-38.63
	(249.13)	(265.40)	(205.12)	(210.78)	(0.06)	(0.47)	(0.19)
Observations	105	102	102	102	204	204	204

Significance stars in columns 1-4 are from pairwise comparisons with the control group
Differences between treatment arms are in columns 5-7 (p-values in parentheses)
+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table 5.5: SP visit details, descriptive statistics (Back: 5.2)

Table 5.6: Evidence of incentive pass-through, SP visits (Back: 5.2)

	Discount offered		Price of rapid test (log)		Price of medication (log)	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	-.00817		-.183*		-.15	
	(0.027)		(0.082)		(0.160)	
T1		.0313		-.396*		-.169
		(0.037)		(0.162)		(0.228)
T2		-.0269		-.0161		-.272
		(0.033)		(0.071)		(0.167)
T3		-.029		-.17^{+}		-.0121
		(0.031)		(0.093)		(0.193)
Control mean	0.048	0.048	4.600	4.600	5.040	5.040
Test p-val ($T1 \neq T2$)		0.086		0.015		0.606
N	338	338	205	205	251	251

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Wald test comparisons of difference in marginal effects (γ) between treatment arms in Column 2
F test comparisons of difference in marginal effects (β) between treatment arms in Columns 4 & 6
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Discussion and Conclusion

Discussion

This study analyzes the effect of demand- and supply-side incentives on malaria testing and treatment targeting, examining how patients and pharmacists make decisions for malaria case management in Kenya. It provides evidence that both patient subsidies and pharmacy incentives for rapid diagnostic testing and quality-assured malaria treatment (ACT) conditional on being malaria positive have a meaningful impact on rapid test uptake and treatment targeting. I show that the share of suspected malaria patients who get diagnosed with a rapid test prior to receiving treatment increases significantly as a result of all three intervention arms relative to the control group: the patient subsidies, pharmacy incentives, and the hybrid approach. Additionally, I find evidence that incentives are effective at increasing the likelihood that quality-assured treatments are appropriately targeted to patients who have a confirmed malaria diagnosis, when compared to the control group. Again, I find no significant difference in impact when comparing demand- to supply-side incentives. All intervention groups lead to a significant decrease in the share of patients who purchase antimalarials without being diagnosed first, which suggests that the interventions are effective at encouraging testing and that patients adhere to their test result when making subsequent treatment decisions. Despite this, there is still a large share of suspected malaria patients who purchase antimalarials without a test across all intervention arms, suggesting that these incentive interventions are not sufficient to change behavior in all patients.

To understand why there was no significant difference in main intervention effects between demand- and supply-side incentives, I test for incentive pass-through using data from Standardized Patient encounters at all study pharmacies. I find that discounts on rapid tests were passed through to patients in the patient discount and hybrid arms. I find suggestive evidence that providers in the pharmacy incentive arm did not pass through any incentives to patients in the form of reduced prices on rapid tests or quality-assured treatments. This analysis suggests that the mechanism of price pass-through in the pharmacy incentive arm is unlikely to be what drives the main impact evaluation results.

From the implementer perspective, all interventions are relatively low cost in terms of getting

appropriate high quality treatment to malaria positive patients. The control group resulted in 75 appropriately targeted ACTs. Patient subsidies resulted in 799 additional appropriately targeted ACTs at a cost of $3.26/patient, pharmacy incentives resulted in an additional 1018 patients treated appropriately at a cost of $4.01/patient, and the hybrid approach resulted in an additional 551 patients treated appropriately at a cost of $9.15/patient. From a societal perspective, the patient subsidy and the pharmacy incentive approaches were cost-saving compared to the control group, while the hybrid arm cost $24/patient for an additional 551 patients being appropriately treated, so is also relatively low cost. The cost-effectiveness analysis can be extended to be directly comparable with other studies by looking at costs/DALY. The framework that I develop for assessing cost-effectiveness can be extended to other settings that are characterized by diagnostic testing availability and over-treatment that can have negative social consequences.

Contributions to research and policy

This study adds to the literature on how individualized health information and financial incentives can be combined to change health behavior. Critically, this study builds on several other field experiments that look at the effectiveness of incentives in improving uptake of malaria diagnostic testing and treatment targeting. Cohen, Dupas and Schaner [6] find that subsidizing ACTs leads to significant increases in ACT uptake, but that the increased demand comes at a cost: only about half of the subsidized ACTs are taken by malaria-positive individuals. They find that a rapid test subsidy also increases uptake of testing, but does little to improve testing targeting. Subsequent work by Prudhomme O'Meara and colleagues [12] in Kenya finds that providing free rapid tests increases testing uptake, and providing subsidies for ACTs when patients test positive increases the use of ACTs when malaria positive. I find results on testing uptake that are consistent with both of these studies: incentivizing rapid tests does increase usage significantly. However, I additionally find that diagnosis conditional incentives for ACTs improve treatment targeting. One key difference between this study and those that came before it is that these incentive interventions are entirely administered in pharmacies and by pharamcists, rather than using community health workers to test and refer patients to retail outlets to purchase treatments.

This study contributes to our understanding of how incentives targeted at the demand-side or the supply-side can affect health decision-making by comparing demand- and supply-side incentive approaches directly to each other. The fact that I find no significant differences in testing uptake or treatment targeting between demand- and supply-side approaches suggests that targeting incentives to either patients or providers is effective at improving malaria case management in a pharmacy setting. This is interesting from a policy and implementation perspective, because it provides some evidence that incentive programs can be applied at the level that is most operationally-feasible, with limited impact on overall effectiveness in terms of end-user outcomes. However, the fact that I do not find evidence of incentive pass-

through in the pharmacy incentive arm suggests that incentivizing pharmacists may not lead to patients getting discounted tests and treatment, and instead the increased demand may be due to other mechanisms.

Finally, this study provides evidence on how pharmacists make decisions, and provides cost-effectiveness estimates that can be used by decision-makers to compare this intervention to others aimed at improving malaria care. Given that pharmacies play an important role in health care provision in many low- and middle-income countries, understanding pharmacist motivations and how interventions aimed at improving quality of care work in a pharmacy context is a crucial policy relevant question. The fact that I find improvements in malaria case management suggests that pharmacy-level interventions is one promising avenue to improve quality of care.

Future research directions

There are several promising avenues for future research that have come out of this study. The first is to explore other possible mechansisms besides incentive pass through that could explain the fact that there are no differences between patient subsidies and pharmacy incentives in improving malaria case management. One possible mechanism that I plan to explore using the Standardized Patient data is whether there were differences in pharmacist counseling around the importance of diagnostic testing across treatment arms. This could be one possible mechanism underlying supplier induced demand, and should be examined in future work. Additionally, I am interested in exploring whether there are differential impacts for adults who sought care for themselves compared to people who sought care for a child in their care, as well as by pharmacy and provider characteristics. This study is limited in that it only looks at the decision to test and the decision to purchase treatment - future work that explores whether incentive interventions are effective at encouraging adherence to treatment regimen, or whether patients who pay more for treatment are more likely to adhere to the treatment regimen is an interesting future avenue. This links to the broader literature on learning and valuation of health goods under subsidized prices [19, 24]. Finally, these findings raise more questions about pharmacist and provider motivations, and how they influence quality of care. Future work could explore these questions in a wider set of health decisions and settings.

Conclusion

Demand- and supply-side incentives are both effective at encouraging uptake of rapid diagnostic testing for malaria and improving treatment targeting in a pharmacy setting, which is characterized by low levels of diagnostic testing and high levels of antimalarial overuse. This research demonstrates that incentives targeted to either patients or pharmacists can lead to

improvements in malaria case management, and that having pharmacy-level programs aimed at improving malaria care has the potential to improve outcomes.

Research team

This research was led by Maria Dieci under the supervision of Paul Gertler and Jonathan Kolstad. Fieldwork was coordinated by Maria Dieci (UC Berkeley), Nettah Kisavwa and Carolyne Nekesa (REMIT Kenya), and supported by Rita Cuckovich (UC Berkeley).

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Appendix A

Model of patient decision-making

A.1 Patient decision: Private costs only

The patient's decision to buy high quality treatment and rapid test can be modeled as a two-stage discrete choice experiment. I solve for stage 2, and then recursively for stage 1:

Stage 2: Decision to buy high quality treatment. There are two versions of the patient decision at this stage: (1) the decision of whether or not to buy treatment given some true information about the patient's illness status (from test result), and (2) the decision of whether or not to buy treatment given the patient's own beliefs about the illness status, but no improved information from testing.

In (1) a patient's utility in stage 2 (U_2) is:

$$U_{2,1}(z_i) = \left\{ \begin{array}{ll} B(z_i) - p, & \text{for } z_i = 1 \\ 0, & \text{for } z_i = 0 \end{array} \right\}$$

where she has full information about her illness status and therefore only purchases high quality treatment if she is positive ($z_i = 1$). $B(z_i)$ is the benefit of taking treatment when sick and p is the price (cost) of the medication. In this simplified model, the negative social costs of over use of antimalarials is not incorporated into private decision-making, making the only cost the price of the drug.

In (2), a patient's utility in stage 2 is:

$$U_{2,2}(z_i) = \left\{ \begin{array}{ll} (z_i) - p, & \text{for } z_i = 1 \\ -p, & \text{for } z_i = 0 \end{array} \right\}$$

where in this case, the utility of taking Artefan when positive is the same as in the first scenario, but the utility when malaria-negative is different. Here, the benefit of taking

malaria medication is only realized when the patient truly has malaria (when $z_i = 1$). Because there is uncertainty in illness status, the patient will purchase the medication even when she is malaria negative ($z_i = 0$) if she is sufficiently confident in her ability to diagnose malaria. Therefore, the patient will incur the cost (p) every time, but the benefit only when malaria-positive.

In each scenario of the second stage, the expected utility is as follows, over the true probability of being malaria positive, denoted by π_i:

$$E[U_{2,1}(z_i)] = \pi_i(B(z_i) - p) + (1 - \pi_i)0 = \pi_i(B(z_i) - p) \tag{A.1}$$

$$E[U_{2,2}(z_i)] = \pi_i(B(z_i) - p) + (1 - \pi_i)(-p) = \pi_i B(z_i) - p \tag{A.2}$$

Stage 1: Patient's decision to get tested. The patient's decision to purchase a rapid test is driven by her perception of its value. The value of the rapid test is the value of the information obtained from a diagnosis. So, individuals will purchase the rapid test if the expected utility in Stage 2 with the addition of that information (minus the cost of the test) is greater than the expected utility in Stage 2 without that information.

Utility in Stage 1 (U_1) can be broken down into utility from purchasing the rapid test or not (denoted here as r):

$$U_1(\pi_i | r = 1) = E[U_{2,1}(z_i)] - p_r \tag{A.3}$$

$$U_1(\pi_i | r = 0) = E[U_{2,2}(z_i)] \tag{A.4}$$

Patient i will buy the rapid test if $U_1(\pi_i|r = 1) \geq U_1(\pi_i|r = 0)$. We now solve for the marginal case:

$$\begin{aligned} U_1(\pi_i|r = 1) - U_1(\pi_i|r = 0) &= 0 \\ E[U_{2,1}(z_i)] - p_r - E[U_{2,2}(z_i)] &= 0 \\ \pi_i(B(z_i) - p) - p_r - \pi_i B(z_i) + p &= 0 \\ (1 - \pi_i)p - p_r &= 0 \end{aligned}$$

Rearranging, we arrive at our desired equilibrium condition:

$$(1 - \pi_i) = \frac{p_r}{p} \tag{A.5}$$

On the right hand side of this final result, we have the probability of not having malaria (malaria negative). This is the individual's risk in being malaria positive. On the left hand side, we have the ratio between the cost of the rapid test and the cost of the treatment.

This suggests that for patients, the value of the rapid test is greater when the price of Antimalarials is high (relative to test price) and there is uncertainty about one's malaria status.

Implications of this result. There are a few implications of this threshold equilibrium condition by looking at the price ratio:

1. The share of patients who will choose to purchase rapid tests at a given price is decreasing in the malaria prevalence rate (test is less valuable for high π)
2. As the ratio of prices increases (relative price of treatment is smaller than test price), the share of patients purchasing a rapid test will be smaller
3. As the price ratio decreases (relative price of treatment is larger than test price), the share of patients purchasing a rapid test will be larger

It is important to note that this exercise has assumed that the only relevant cost is the price of the rapid test and the treatment option. This simplification excludes two important costs: the first is the time cost of getting a rapid test. Tests take about 15 minutes from when they are administered to when results are ready, so this is an additional non-pecuniary cost that patients may face when making this decision. The second cost is the social cost of not getting the treatment most appropriate to the illness status (i.e. not getting malaria medication in the case of a negative malaria status). This social cost is not factored into private decision-making, but should be considered when setting prices for these products.

Appendix B

Supplementary Tables and Figures

B.1 Appendix Figures

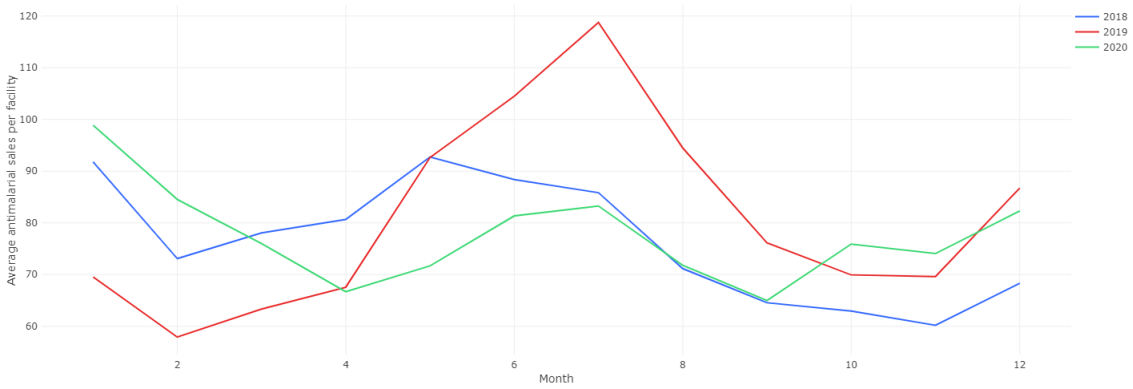


Figure B.1: Malaria sales, seasonal trends (Back: 1.1)

B.2 Appendix Tables

Subsidy and incentive amounts

	Control (C)	**Patient discount (T1)**	**Pharmacy incentive (T2)**	**Both (T3)**
Patient discounts? (USD)				
Rapid test	-	$0.90	-	$0.60
ACT (malaria +)	-	$1.10	-	$0.80
ACT (malaria -)	-	$0.00	-	$0.00
Provider incentives (USD)				
Rapid test	-	-	$0.90	$0.20
ACT (malaria +)	-	-	$0.80	$0.10
ACT (malaria -)	-	-	$0.00	$0.00
Transaction completion	-	-	$0.30	$0.30
Total incentive amount (USD)	$0.00	$2.00	$2.00	$2.00

Table B.1: Incentive amount details, by treatment arm (Back: 3.2)

Variable	(1) In sample	(2) Declined	(3) (2)-(1)
Number of months active on digital sales management tool	12.04	16.81	4.76**
	(9.43)	(8.52)	(0.01)
Average monthly malaria sales, 2019-2020	63.39	66.47	3.08
	(63.56)	(75.45)	(0.83)
Average monthly quality treatment sales, 2019-2020	54.41	61.25	6.84
	(54.26)	(72.61)	(0.59)
Average monthly rapid test sales, 2019-2020	6.48	5.39	-1.08
	(9.93)	(11.26)	(0.62)
Site was in earlier pilot study phase	0.16	0.23	0.07
	(0.37)	(0.43)	(0.32)
Site is in an urban area	0.31	0.34	0.04
	(0.46)	(0.48)	(0.69)
Site is in a malaria endemic county	0.84	0.86	0.01
	(0.37)	(0.36)	(0.84)
Site is a pharmacy	0.56	0.56	-0.00
	(0.50)	(0.51)	(1.00)
Observations	140	35	175

In sample facilities include those that were randomized to one of the study arms and were on-boarded successfully.

Table B.2: Baseline balance between facilities in sample and refusals (Back: 4.1)

Table B.3: Primary outcomes regressed on baseline characteristics (Back: 4.1)

	(1) Rapid test uptake	(2) ACT uptake with test	(3) ACT uptake without test
Months on sales management tool	.00143	.00197^{+}	-.00104
	(0.002)	(0.001)	(0.002)
Below median baseline malaria sales	.194**	.0369	-.155*
	(0.066)	(0.034)	(0.064)
Average monthly malaria sales, 2019-2020	-.000374	-.000687	-.00552*
	(0.001)	(0.000)	(0.002)
Average monthly ACT sales, 2019-2020	-.00211^{+}	-.000573	.00812**
	(0.001)	(0.001)	(0.003)
Average monthly rapid test sales, 2019-2020	.0157**	.0095**	-.0119**
	(0.003)	(0.002)	(0.003)
Site was in earlier pilot study phase	-.00984	-.00811	.0372
	(0.052)	(0.036)	(0.055)
Site is in an urban area	.0183	.0105	.0195
	(0.054)	(0.026)	(0.054)
Site is in a malaria endemic county	.0729	.0648**	-.105
	(0.073)	(0.024)	(0.073)
Site does not have clinical capabilities	.673**	.224**	-.652**
	(0.050)	(0.071)	(0.046)
% of staff who are female	.147^{+}	.0561	-.154*
	(0.078)	(0.039)	(0.075)
Age of pharmacy owner	.00767**	.00261*	-.00754*
	(0.003)	(0.001)	(0.003)
Average age of pharmacy staff	.000397	.000689	-.00217
	(0.006)	(0.002)	(0.005)
Female owner	-.202**	-.0851*	.182**
	(0.063)	(0.033)	(0.063)
Number of staff	.053	.0383	-.0728
	(0.060)	(0.029)	(0.059)
N	51486	51486	51486

Linear probability models for primary outcomes on baseline characteristics
Standard errors are clustered at the facility level
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.4: Secondary outcomes regressed on baseline characteristics (Back: 4.1)

	(1) Antimalarial uptake overall	(2) ACT uptake overall	(3) ACT uptake w/ test, ACT sales
Months active on sales management tool	.000965	.000923	.00249
	(0.002)	(0.002)	(0.002)
Below median baseline malaria sales	-.00931	-.118*	.112*
	(0.029)	(0.051)	(0.053)
Average monthly malaria sales, 2019-2020	-.00297**	-.00621**	.0000527
	(0.001)	(0.002)	(0.001)
Average monthly ACT sales, 2019-2020	.00373**	.00754**	-.00211^{+}
	(0.001)	(0.002)	(0.001)
Average monthly rapid test sales, 2019-2020	.00364*	-.00239	.014**
	(0.002)	(0.002)	(0.003)
Site was in earlier pilot study phase	-.0218	.0291	.000996
	(0.039)	(0.045)	(0.050)
Site is in an urban area	-.0373^{+}	.03	.0146
	(0.022)	(0.040)	(0.043)
Site is in a malaria endemic county	.114**	-.0399	.111*
	(0.025)	(0.064)	(0.049)
Site is does not have clinical capabilities	.177*	-.428**	.738**
	(0.080)	(0.088)	(0.044)
% of staff who are female	-.0224	-.0976^{+}	.115^{+}
	(0.025)	(0.050)	(0.061)
Age of pharmacy owner	.00336**	-.00493^{+}	.00765*
	(0.001)	(0.003)	(0.003)
Average age of pharmacy staff	-.00212	-.00148	-.000143
	(0.002)	(0.004)	(0.004)
Female owner	.0884*	.0974*	-.164**
	(0.034)	(0.042)	(0.054)
Number of staff	.038^{+}	-.0345	.038
	(0.022)	(0.041)	(0.047)
N	265610	51486	40261

Linear probability models for secondary outcomes on baseline characteristics
Standard errors are clustered at the facility level
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

		T1 vs. C	T2 vs. C	T3 vs. C	T1 vs. T2 vs. T3	T1 vs. T2
Table 4.2 - Unadjusted Primary Outcomes						
RDT uptake	p-value	0.046	0.008	0	0.84	0.718
	q-value	0.136	0.06	0.001	0.686	0.686
ACT with test	p-value	0.244	0.088	0.032	0.999	0.971
	q-value	0.323	0.172	0.117	0.686	0.686
ACT without test	p-value	0.203	0.18	0.025	0.792	0.747
	q-value	0.296	0.296	0.117	0.686	0.686
Table 4.3 - Adjusted Primary Outcomes						
RDT uptake	p-value	0.016	0.004	0	0.827	0.543
	q-value	0.051	0.029	0.001	0.613	0.484
ACT with test	p-value	0.178	0.101	0.988	0.095	0.832
	q-value	0.217	0.145	0.654	0.145	0.613
ACT without test	p-value	0.061	0.042	0.009	0.886	0.635
	q-value	0.126	0.102	0.041	0.613	0.53

Table B.5: P-values and FDR-adjusted q-values for primary comparisons (Back: 4.2)

Table B.6: Impact on Primary Outcomes, Unadjusted LPM Models (Back: 4.2)

	Rapid test uptake		ACT uptake with test		ACT uptake without test	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	.216**		.0691*		-.16*	
	(.052)		(.029)		(.075)	
T1		.234^{+}		.0687		-.167
		(.128)		(.065)		(.136)
T2		.185*		.0703		-.122
		(.082)		(.044)		(.098)
T3		.229**		.0685*		-.182*
		(.060)		(.034)		(.081)
Control mean	.081	.081	.057	.057	.809	.809
F test p-val ($\beta_{T1} \neq \beta_{T2} \neq \beta_{T3}$)		0.882		0.999		0.820
F test p-val ($\beta_{T1} \neq \beta_{T2}$)		0.727		0.982		0.749
N	51486	51486	51486	51486	51486	51486

Standard errors are clustered at the facility level

Controls: Strata FE

F test comparisons of difference (β) between treatment arms

Denominators: all patients that purchased malaria product during study period

$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.7: Impact on Primary Outcomes, Adjusted LPM Models (Back: 4.2)

	Rapid test uptake		ACT uptake with test		ACT uptake without test	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	.212**		.0583+		-.188**	
	(.048)		(.030)		(.060)	
Total months active	.00455	.00485	.00182	.00221	-.00642+	-.00642+
	(.003)	(.003)	(.002)	(.002)	(.004)	(.004)
Baseline monthly malaria sales	-.00102*	-.00108+	-.00047	-.000503	.000774	.000808
	(.000)	(.001)	(.000)	(.000)	(.001)	(.001)
Female pharmacy owner	-.0649	-.0692	-.0677*	-.0713*	-.0151	-.0139
	(.058)	(.063)	(.031)	(.034)	(.097)	(.100)
T1		.264*		.0725		-.222+
		(.113)		(.059)		(.119)
T2		.189*		.0673		-.163+
		(.079)		(.048)		(.084)
T3		.194**		.0439		-.182*
		(.061)		(.036)		(.070)
Control mean	.081	.081	.057	.057	.809	.809
F test p-val ($\beta_{T1} \neq \beta_{T2} \neq \beta_{T3}$)		0.824		0.880		0.888
F test p-val ($\beta_{T1} \neq \beta_{T2}$)		0.544		0.937		0.629
N	51486	51486	51486	51486	51486	51486

Standard errors are clustered at the facility level

Controls: months active on platform, baseline malaria sales, female owner, strata FE

F test comparisons of difference (β) between treatment arms

Denominators: all patients that purchased malaria product during study period

$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.8: Impact on Secondary Outcomes, Unadjusted LPM Models (Back: 4.3)

	Antimalarial sales overall		ACT uptake, all malaria product purchases		ACT uptake with test, ACT purchases	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	-.0157		-.0905		.108**	
	(.033)		(.068)		(.040)	
T1		.0262		-.0985		.0972
		(.036)		(.090)		(.100)
T2		-.0179		-.0521		.0989
		(.036)		(.079)		(.064)
T3		-.0315		-.114		.12*
		(.037)		(.072)		(.050)
Control mean	.197	.197	.867	.867	.066	.066
F test p-val ($\beta_{T1} \neq \beta_{T2} \neq \beta_{T3}$)		0.160		0.588		0.960
F test p-val ($\beta_{T1} \neq \beta_{T2}$)		0.122		0.550		0.988
N	265610	265610	51486	51486	40261	40261

Standard errors are clustered at the facility level

Controls: Strata FE

F test comparisons of differences (β) between treatment arms

$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.9: Impact on Secondary Outcomes, Adjusted LPM Models (Back: 4.3)

	Antimalarial sales overall		ACT uptake, all malaria product purchases		ACT uptake with test, ACT purchases	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	-.000747		-.129**		.107**	
	(.026)		(.043)		(.041)	
Total months active	-.000459	-.000606	-.0046	-.00421	.00274	.00299
	(.002)	(.002)	(.003)	(.003)	(.002)	(.003)
Baseline monthly malaria sales	.000443^{+}	.000418^{+}	.000304	.000305	-.000771^{+}	-.000815
	(.000)	(.000)	(.001)	(.001)	(.000)	(.001)
Female pharmacy owner	.0297	.0272	-.0828	-.0853	-.0855	-.0896
	(.046)	(.045)	(.088)	(.089)	(.052)	(.059)
T1		.0263		-.15*		.127
		(.027)		(.071)		(.101)
T2		-.0105		-.0957^{+}		.108
		(.035)		(.054)		(.068)
T3		-.00821		-.138**		.0952^{+}
		(.029)		(.050)		(.050)
Control mean	.197	.197	.867	.867	.066	.066
F test p-val ($\beta_{T1} \neq \beta_{T2} \neq \beta_{T3}$)		0.205		0.653		0.964
F test p-val ($\beta_{T1} \neq \beta_{T2}$)		0.157		0.433		0.856
N	265610	265610	51486	51486	40261	40261

Standard errors are clustered at the facility level

Controls: months active on platform, baseline malaria sales, female owner, strata FE

F test comparisons of difference (β) between treatment arms

$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.10: Impact on Primary Outcomes, All baseline covariates (Back: 4.2)

	Rapid test uptake		ACT uptake with test		ACT uptake without test	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	.168**		.0231		-.166**	
	(0.039)		(0.026)		(0.040)	
T1		.1*		-.0169		-.0956*
		(0.042)		(0.022)		(0.047)
T2		.184**		.0659+		-.171**
		(0.047)		(0.035)		(0.049)
T3		.184**		.0282		-.191**
		(0.058)		(0.033)		(0.053)
Control mean	0.081	0.081	0.057	0.057	0.809	0.809
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2} \neq \gamma_{T3}$)		0.186		0.018		0.265
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2}$)		0.103		0.006		0.185
N	51441	51441	51486	51486	51441	51441

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: all baseline covariates from Table 1
Wald test comparisons of difference in marginal effects (γ) between treatment arms
Denominator: all patients that purchased malaria product during study period
Outcome 1 & 3: 45 obs dropped b/c multicollinearity (strata 11)
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.11: Impact on Secondary Outcomes, All baseline covariates (Back: 4.3)

	Antimalarial sales overall		ACT uptake, all malaria product purchases		ACT uptake with test, ACT purchases	
	(1)	(2)	(3)	(4)	(5)	(6)
Pooled treatment	-.016		-.0171		.0541^{+}	
	(0.023)		(0.022)		(0.031)	
T1		-.0201		-.0179		-.00815
		(0.026)		(0.025)		(0.026)
T2		-.0147		-.0142		.105*
		(0.035)		(0.034)		(0.043)
T3		-.0146		-.019		.0722
		(0.028)		(0.028)		(0.044)
Control mean	0.197	0.197	0.175	0.175	0.066	0.066
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2} \neq \gamma_{T3}$)		0.981		0.988		0.010
Wald test p-val ($\gamma_{T1} \neq \gamma_{T2}$)		0.876		0.911		0.007
N	265610	265610	265610	265610	40256	40256

Reporting marginal effects from the control group, from logistic regressions
Standard errors are clustered at the facility level
Controls: all baseline controls from Table 1
Wald test comparisons of difference in marginal effects (γ) between treatment arms
Outcome 3: 50 obs dropped b/c multicollinearity (strata 11)
$^{+}$ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$

Table B.12: CEA Probability Inputs - sources (Back: 3.5)

	SOURCES
P(tested)	Intervention group means from Table 4.3, column 2 for all 4 arms
P(untested)	1 - P(tested)
P(malaria positive | tested)	Control group: administrative data from pharmacies on positivity rates; Treatment group means from Table 4.6, column 1
P(malaria positive | untested)	Control group: lab tech testing random subset of control group patients; Treatment groups: P(malaria positive) from control group (unselected positivity rate); P(positive | tested) from Table 4.6, column 1 P(malaria positive | untested) = P(malaria positive) - P(malaria positive | tested)
P(malaria positive)	P(malaria positive | tested) + P(malaria positive | untested) obtained from lab tech activity in control group
P(ACT | malaria positive & tested)	Control group mean from Table 4.3 column 6 * P(malaria positive | tested); Treatment group means from Table 4.6 column 2
P(ACT | malaria positive & untested)	Intervention group means from Table 4.3 column 6 * P(malaria positive | untested), for all 4 arms
P(malaria negative | untested)	Control group: lab tech testing random subset of control group patients; Treatment groups: P(malaria negative) from control group (unselected positivity rate); P(negative|tested) from Table 4.6, column 1 P(malaria negative | untested) = P(malaria negative) - P(malaria negative | tested)
P(malaria negative | tested)	Control group: administrative data from pharmacies on positivity rates; Treatment group means from Table 4.6 column 1

Table B.13: CEA Additional Inputs - sources (Back: 3.5)

	SOURCES
Num. patients who purchased ACTs	Intervention group means from Table 4.5 column 4; multiplied by 10000 hypothetical cohort
Incentive unit cost (RDT) ($)	Table B1; transaction completion incentives in T2 & T3 are included
Patients getting incentivized RDTs	Share from Administrative data (positive_rdt); multiplied by 10000 hypothetical cohort
Incentive unit cost (ACT) ($)	Table B1
Patients getting incentivized ACTs	Share from Administrative data (act_purchased); multiplied by 10000 hypothetical cohort
Avg. treatment cost ($), untested	Administrative data (cost_malaria_products if rest_rdt_sales==0)
Num. untested patients	Intervention group means from Table 4.3, column 2; multiplied by 10000 hypothetical cohort
Avg. treatment unit cost ($), tested	Administrative data (cost_malaria_products if rest_rdt_sales==1)
Num. tested patients	Intervention group means from Table 4.3, column 2; multiplied by 10000 hypothetical cohort
Time cost of seeking care	Mean time (mins) spent with provider by treatment arm, from patient survey (s4_a7_prov_treat_min)
Hourly wage ($)	Kenya Continuous Household Survey Program 2020
Num. patients who accessed care	Fixed at 10000 hypothetical cohort across all arms

Table B.14: CEA Benefits and Cost Estimates - formulas (Back: 3.5)

	FORMULAS
P(ACT | malaria positive)	P(ACT | malaria positive) = P(ACT | malaria positive & tested)P(malaria positive | tested)P(tested) + P(ACT | malaria positive & untested)P(malaria positive | untested)P(untested)
Number of patients taking ACTs	Administrative data (act_sales)
Number of beneficiaries	P(ACT | malaria positive)*Number of beneficiaries
	FORMULAS
Total cost of incentives	(RDT incentive*number of patients getting RDT) +(ACT incentive*number of patients getting incentivized ACT)
Total cost of over-treating malaria negative patients	P(malaria negative | untested)*number of untested patients purchasing antimalarials*cost of antimalarial treatment for untested patients + P(malaria negative | tested)*number of tested patients purchasing antimalarials*cost of antimalarial treatment for tested patients
Total time cost to patients seeking care	Number of malaria patients*average time spent with provider*average hourly wage
Total costs - societal perspective	Total cost of incentives + Total cost of over-treating malaria negative patients + Total time cost to patients seeking care
Total costs - implementer perspective	Total cost of incentives

Appendix C

Other supplementary materials

Standardized Case: Malaria (Standard Case, No Previous Treatment)

JAMES (Male Living in Nearest Town)

For endemic lake regions

James is 28 years old and has been living in (either Kisumu, Kakamega, Busia, Siaya, Bungoma, Migori or Homabay) for the last 5 years. He has been using a treated mosquito net but stopped using it a few weeks ago because it was so old and full of holes. A few days ago, James started having headaches and a fever took some pain killers (Panadol/Maramoja) but the headache did not go away. He hasn't had much of an appetite lately.

For non-endemic lake regions

James is 28 years old and has been living in [either Kisii, Kericho, Nakuru, Bomet or Nyamira] for the last 5 years. Two weeks ago, he traveled to Kisumu to visit his friend.

During his stay in Kisumu, he did not use a treated mosquito net, because it was so old and full of holes. A few days ago, James started having headaches and a fever took some pain killers (Panadol/Maramoja) but the headache did not go away. He hasn't had much of an appetite lately.

Continuation for both

Yesterday James woke up with a headache and was feeling cold, despite it being sunny outside. He was also feeling fatigued, had joint pains. A few times overnight he experienced a bit fever and chills, and he thinks that these symptoms remind him of when he had malaria four to six months ago.

John comes from a family of 4: two brothers and one sister whereby he is the second born. He is currently living with the younger brother who he is supporting in a one-bedroom house in [Nearest Town where the SP is working]. No one else from his family seems to be showing symptoms like his. Today, he visits a pharmacy to see if his condition can be helped.

Maeneo yenye athari kubwa

James ana miaka 28 na amekuwa akiishi [aidha Kisumu, Kakamega, Busia, Siaya, Bungoma, Migori ama Homabay] kwa muda wa miaka 5 sasa. Amekuwa akitumia neti ya mbu iliyotibiwa ila aliacha kuitumia wiki chache zilizopita kwa sababu ilikuwa imezeeka na yenye mashimo. Siku chache zilizopita, James alianza kuhisi maumivu ya kichwa, kuhisi joto mwilini na akakunywa dawa za kupunguza maumivu (Panadol/maramoja) lakini maumivu ya kichwa haikuisha. Hajakuwa na hamu ya chakula hivi karibuni.

Maeneo yasiyo na athari kubwa

James ana miaka 28 na amekuwa akiishi [aidha Kisii, Kericho, Nakuru, Bomet or Nyamira] kwa muda wa miaka 5 sasa. Wiki mbili zilizopita, alisafiri kuelekea Kisumu kutembelea rafiki yake. Alipokuwa akiishi Kisumu, hakutumia neti ya mbu iliyotibiwa kwa sababu ilikuwa imezeeka na yenye mashimo. Siku chache zilizopita, James alianza kuhisi maumivu ya kichwa, kuhisi joto mwilini na akakunywa dawa za kupunguza maumivu (Panadol/maramoja) lakini maumivu ya kichwa haikuisha. Hajakuwa na hamu ya chakula hivi karibuni.

Jana James aliamka kichwa kikimuuma na akihisi baridi ilhali jua lilikuwa limewaka nje. Pia alikuwa anajihisi kuchoka na kuumwa na viungo vya mwili. Mara chache usiku kucha alihisi joto kiasi na kutetemeka, na akafikiria kwamba dalili hizo zilimkumbusha wakati mwingine alikuwa na malaria miezi 4 iliyopita.

James anatoka katika familia ya watoto wanne, ndugu wawili na dada mmoja ambapo yeye ndiye wa pili. Kwa sasa anaishi na ndugu yake mdogo kwa nyumba iliyo na chumba kimoja cha kulala (mji ulio karibu na pahali SP anafanya kazi). Hakuna mtu mwingine kwa familia yake anayeonyesha dalili kama zake. Leo, anatembelea duka la kuuzia dawa ili kupata usaidizi.

JANE (Female Living in Nearest Town)

For endemic lake regions
Jane is 28 years old and has been living in (either Kisumu, Kakamega, Busia, Siaya, Bungoma, Migori or Homabay) for the last 5 years. She has been using a treated mosquito net but stopped using it a few weeks ago because it was so old and full of holes. A few days ago, Jane started having headaches and a fever took some pain killers (Panadol/Maramoja) but the headache did not go away. She hasn't had much of an appetite lately.

For non-endemic lake regions
Jane is 28 years old and has been living in [either Kisii, Kericho, Nakuru, Bomet or Nyamira] for the last 5 years. Two weeks ago, she traveled to Kisumu to visit her friend.

During her stay in Kisumu, she did not use a treated mosquito net, because it was so old and full of holes. A few days ago, Jane started having headaches and a fever, took some pain killers (Panadol/Maramoja) but the headache did not go away. She hasn't had much of an appetite lately.

Continuation for both
Yesterday Jane woke up with a headache and was feeling cold, despite how it was sunny outside. She was also feeling fatigued, had joint pains. A few times during last night she experienced a bit fever and chills, and she thinks that these symptoms remind her of when she had malaria four to six months ago.

Jane comes from a family of 4: two brothers and one sister whereby she is the second born. She is currently living with the younger brother who she is supporting in a one-bedroom house in [Nearest Town the SP is working]. No one else from her family seems to be showing symptoms like hers. Today, she visits a pharmacy to see if her condition can be helped.

Maeneo yenye athari kubwa
Jane ana miaka 28 na amekuwa akiishi [aidha Kisumu, Kakamega, Busia, Siaya, Bungoma, Migori ama Homabay] kwa muda wa miaka 5 sasa. Amekuwa akitumia neti ya mbu iliyotibiwa ila aliacha kuitumia wiki chache zilizopita kwa sababu ilikuwa imezeeka na yenye mashimo. Siku chache zilizopita, Jane alianza kuhisi maumivu ya kichwa, kuhisi joto mwilini na akakunywa dawa za kupunguza maumivu (Panadol/maramoja) lakini maumivu ya kichwa haikuisha. Hajakuwa na hamu ya chakula hivi karibuni.

Maeneo yasiyo na athari kubwa
Jane ana miaka 28 na amekuwa akiishi [aidha Kisii, Kericho, Nakuru, Bomet or Nyamira] kwa muda wa miaka 5 sasa. Wiki mbili zilizopita, alisafiri kuelekea Kisumu kutembelea rafiki yake. Alipokuwa akiishi Kisumu, hakutumia neti ya mbu iliyotibiwa kwa sababu ilikuwa imezeeka na yenye mashimo. Siku chache zilizopita, Jane alianza kuhisi maumivu ya kichwa, kuhisi joto mwilini na akakunywa dawa za kupunguza maumivu (Panadol/maramoja) lakini maumivu ya kichwa haikuisha. Hajakuwa na hamu ya chakula hivi karibuni.

Jana Jane aliamka kichwa kikimuuma na akihisi baridi ilhali jua lilikuwa limewaka nje. Pia alikuwa anajihisi kuchoka na kuumwa na viungo vya mwili. Mara chache usiku kucha alihisi joto kiasi na kutetemeka, na akafikiria kwamba dalili hizo zilimkumbusha wakati mwingine alikuwa na malaria miezi 4 iliyopita.

Jane anatoka katika familia ya watoto wanne, ndugu wawili na dada mmoja ambapo yeye ndiye wa pili. Kwa sasa anaishi na ndugu yake mdogo kwa nyumba iliyo na chumba kimoja cha kulala (mji ulio karibu na pahali SP anafanya kazi). Hakuna mtu mwingine kwa familia yake anayeonyesha dalili kama zake. Leo, anatembelea duka la kuuzia dawa ili kupata usaidizi.

James's Dress:

Rift valley: Track suits, cap and sports shoes. Jeans, t-shirts and brown jackets. Maasai region especially in the rural, wear a t-shirt and jeans and carry a small stick.
Nyanza/Western: T-shirt, jeans, normal official trousers and some light warm clothing e.g. pullover. Have closed shoes

Jane's Dress:

Rift valley: No trouser, skirts are advised but not the mitumba. No make-up especially in the rural areas.
Nyanza/Western: In town, wear jeans and t-shirts but no expensive handbags or jewelry. In the rural areas, wear skirts and plastic shoes.

Opening statement:

Hello, I have been having a fever, headache and joint pains for a few days. I have come to seek help.

Kiswahli: Habari, nimekuwa na joto mwilini, kuumwa na kichwa na maumivu ya viungo kwa siku chache. Nimekuja kupata usaidizi.
Luhya: Milembe, nakhamala amatukhu, mubili kuhitsa, murwi kulumanga nende makumba khuluma. Nitsi khunyola bukhonyi.
Kalenjin: Chamge, ko koetyon maat ak koaman metit ak kong'wonen rotionisiek ab borto en betusiek tuten che kokosirto. Konyon acheng' toretet.
Luo: Amosi/Ber, asebedo ka awinjo kadenda chwakore, wiya bara gi fuondena be rama kuom ndalo matin mosekalo. Abiro dwaro kony
Kisii: Mbuyore, nabeire nkoigu riberera, omutu okong'ati nechinengo chikondoma ase amatuko amake aetire. Enachire korigi obokonyi.

History questions that could be asked by the pharmacist/attendant and their answers:

1. Q: Have you been vaccinated for Corona virus?
   Je, umepokea chanjo dhidi ya virusi vya Corona?
   A: Yes
   Ndiyo

2. Q: Have you had Corona virus before?
   Umewahi ugua virusi vya Corona hapo awali?
   A: No
   La

3. Q: Have you been in close contact with someone who has had/tested positive for Corona Virus?
   Umeweza kutangamana na mtu yeyote aliyeugua ama kupima kapatikana na virusi vya Corona?
   A: No
   La

4. Q: Have you at any point lost your sense of smell or taste?
   Je, umewahi kutoweza kunusa au kuonja?
   A: No
   La

5. Q: What are your symptoms?
   Ni dalili gani uko nazo?
   A: I have a fever, headache and joint pains.
   Niko na joto mwilini, kuumwa na kichwa na misuli.

6. Q: Which symptom started first?
   Ni dalili gani iliyoanza kwanza?
   A: They started at the same time
   Zote zilianza wakati mmoja

7. Q: How long have you had these symptoms for?
   Umekuwa na dalili hizi kwa muda gani?
   A: For three days.
   Siku tatu

8. Q: Is the fever constant or does it come and go?
   Joto huwa kila wakati ama inakuja ikipotea?
   A: It comes and goes.
   Inakuja ikipotea

9. Q: Does the fever go up and down?
   Je, joto hupanda na kushuka?
   A: Yes.
   Ndio

10. Q: When you have a fever is it very high?
    Wakati unahisi joto, inakuwanga juu sana?
    A: Sometimes high, sometimes low.
    Saa zingine iko juu, saa zingine iko chini

11. Q: Have you been able to eat and drink?

Umekuwa ukiweza kula ama kunywa?
A: Yes, I ate a small breakfast and drank some water.
Ndio, nilikula kiamsha kinywa kiasi na nikakunywa maji kiasi

12. Q: Have you had any vomiting or diarrhoea?
Umekuwa ukitapika ama kuendesha?
A: Yes, only vomiting.
Ndio, kutapika tu

13. Q: Have you taken any medicines?
Umekunywa dawa zozote?
A Yes, Panadol but it has not helped.
Ndio, Panadol lakini haijanisaidia.

14. Q: For how long?
Kwa muda upi?
A: one day.
Siku moja

15. Q: Have you taken a malaria test?
Umeweza kupimwa malaria?
A: No
La

16. Q: When was the last time you had malaria?
Ni mara gani yako ya mwisho kuwa na malaria?
A: About four months ago.
Miezi nne zilizopita

17. Q: Have you travelled recently?
Umesafiri hivi karibuni?
A:
Yes, I've been to (Either Kisumu, Kakamega, Busia, Siaya, Bungoma, Migori, Homabay) (For the non-endemic regions)
Ndio, nimekuwa (aidha Kisumu, Kakamega, Busia, Siaya, Bungoma, Migori, Homabay)

18. Q: Have you had difficulty breathing?
Umekuwa na ugumu wa kupumua?
A: No
La

19. Q: Have you had any wheezing?
Unahisi kupiga chafya?
A: No.
La

20. Q: Have you had any muscle or joint pain?
Unaumwa na misuli ama viungo zozote?

A: Yes, my muscles and joints ache.
Ndio, misuli na viungo zangu zinauma

21. Q: Do you have chest pain?
    Uko na maumivu ya kifua?
    A: No.
    La

22. Q: Do you have a cough?
    Unakohoa?
    A: No
    La

23. Q: Have you had a cold, sneezing, sore throat or stuffiness in the last few days?
    Umekuwa na homa, chemua ama kuumwa na koo kwa siku kadhaa zilizopita?
    A: No.
    La

24. Q: Have you had any fainting or convulsions?
    Umewahi kuzirai ama kushikwa na kifafa?
    A: No.
    La

25. Q: Do you feel dizzy?
    Unahisi kizunguzungu?
    A: Yes
    Ndio

26. Q: Are you allergic to any medicines?
    Unapata madhara yoyote kutokana na matumizi ya dawa?
    A: No.
    La

27. Q: Do you have any other problems?
    Uko na shida zingine zozote?
    A: No.
    La

28. Q: [For female case] When was your last period?
    [Kwa wanawake] Ni lini mwisho ulipata hedhi?
    A: *About four days ago.*
    Siku nne zilizopita

29. Q: [For female case] Are you/could you be pregnant?
    [Kwa wanawake] Unaeza kuwa mja mzito?
    A: *No*
    La

30. *Do you feel pain while swallowing?*
    Unahisi maumivu wakati unameza?
    A: No
    La

31. *Where is the location of the headache?*
    Kichwa kinauma upande upi?
    A: Front upper part of the head.
    sehemu ya mbele juu ya kichwa

32. *What is the severity/intensity of the headache?*
    Kichwa kinauma kiasi gani?
    A: Moderate
    kiasi