# Antiretroviral drug access and behavior change

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**Abstract**

The rapid expansion in access to antiretroviral (ARV) drugs in sub-Saharan Africa - from under 10,000 in 2000 to more than 8 million in 2011 - has been enormous. Anticipating the impact of this expansion and future policy options requires identification of the impacts of access on individual behaviors that determine the course of the epidemic. This paper combines geocoded information about the timing of introduction of ARVs in each health facility in Kenya with two repeated cross sections of geocoded population surveys to estimate the impact of ARV access on risky sexual behavior. Using difference in differences estimates, I find evidence of a relative increase in risky behavior reflected in pregnancy rates among young people in areas with ARV access. These results are consistent across different definitions of treatment and different sets of fixed effects. Using a simple model, I show that the reduction in transmission from ARVs can outweigh the increase in risk-taking in determining new infections.

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Health - Health Outcomes

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# 1 Introduction

The distribution of antiretroviral (ARV) drugs in sub-Saharan Africa has expanded enormously in the last decade. This expansion has delayed the onset of AIDS symptoms for millions of individuals, leaving children with parents for longer, revitalizing the workforces in many developing countries, and providing the individuals who take them with many additional years of life. There is substantial reason to expect that beyond these direct impacts, ARVs can change welfare of many more individuals through changing the decisions that individuals make. The availability of treatment reduces the cost of becoming infected with HIV, increases the proportion of the population that is infected (by delaying the onset of AIDS symptoms), and decreases the transmission rates between those who are uninfected and those who are infected and on treatment.

Previous models of behavioral disinhibition predict that when faced with an exogenous decrease in the riskiness associated with an activity over which they have some control, individuals may compensate, by increasing the riskiness due to their behavior. These models have been applied in the case of seat belts and airbags generating faster driving. In this case, individuals who believe that treatment will be available, and thus that the cost of becoming infected is not as great, may engage in more risky behavior. This is a specific case of moral hazard associated with access to treatment. This type of model would predict that individuals with greater expected access to ARVs would be more likely to have multiple partners, have sex without a condom, and generally risk HIV infection, than would those who do not anticipate that treatment would be available.

This paper proposes a simple model of the channels through which ARV provision can change infection rates, allowing individual behavior to respond to the change in the cost of infection and the likelihood of transmission conditional on engaging in unprotected sex. In this model, individuals of different types (HIV negative, HIV positive and on treatment and HIV negative not on treatment) decide whether to have unprotected sex based on the cost of being infected, the risk of infection, and an individual specific measure of utility from

unprotected sex. This model demonstrates that whether the introduction of ARVs increases or decreases the rate of new infections depends on behavioral responses, and the models' prediction about the sign of the behavioral response is ambiguous. This model also provides a framework for anticipating the impact on new infections as a function of the measured estimates of behavioral responses.

The behavioral response is estimated using a unique dataset that combines administrative records with population surveys from Kenya. The first data source is a record of ARV distribution for all health facilities in Kenya from the National AIDS and STI Control Program (NASCOP), and Kenyapharma, a procurement agency.This data includes the year in which ARVs began being distributed in each facility. This was matched, by hand, with the GPS coordinates of each health facility provided by the Kenya Open Data Initiative. Measures of individual behavior come from two waves of Demographic and Health Surveys from MeasureDHS. These surveys contain detailed records of fertility and a large set of individual controls along with geographic identifiers, which allow this dataset to be linked with the health facility records. In total, this cover 16,639 women in 798 clusters. Linked with the administrative data on drug roll-out, this allows me to construct historical measures of individual access to ARV drugs to test how this access changes individual behavior.

To estimate the impact of treatment access on risk-taking, I estimate multiple forms of difference in difference specifications, each with a different definitions of treatment and a different set of geographic fixed effects. Access is determined by location, which means that any designation will result in some misclassification as individuals who live near a clinic may not know about it while some who live farther away will be aware and will respond to it. This introduces some inevitable measurement error as well as some concerns about specifications. The detail of the geographic data allows me to test multiple specifications, across which I find consistent results.

The primary vector of HIV infection in Sub-Saharan Africa is unprotected heterosexual sex. Although self-reported sexual behavior is notoriously misreported, fertility is a conve-

nient and commonly used proxy for unprotected sex. This is particularly useful for those who are unmarried and do not yet have children, since this is the group that may be most likely to choose whether or not to engage in unprotected sex based on a perceived risk of HIV infection. In this sample, I find a decrease in pregnancy rates between waves in areas that did not gain access to ARVs, but I find no change in pregnancy rates in areas with treatment available. This demonstrates a relative increase in risk-taking as a result of the introduction of ARVs.

I acknowledge and address two types of measurement error. The first is from inevitable misclassification from choosing an arbitrary boundary. This necessarily attenuates the estimates. The second type of measurement error is introduced because of jittered locations of survey respondents. For the sake of maintaining confidentiality, the DHS data reports the locations of survey clusters with explicit error added to the location. The addition of measurement error due to this jittering is likely to attenuate my estimates, but because the precise formula for the jittering is known, the magnitude of this attenuation can be estimated, as will be done in section 9.

Finally, I conclude the paper by returning to the model to demonstrate the likely impacts of the behavioral response to ARVs on HIV infection rates, arguing that despite an increase in risk-taking, new infection rates are not likely to increase substantially because of the reduction in transmission rates, and with sufficient coverage of ARVs, would be expected to fall.

This paper proceeds as follows. The next section places this project in the context of the larger literatures on risky behavior in the face of changing costs and the impacts of HIV policy on individual behavior. Section 3 outlines the model of individual behavior. Section 4 describes the context and the data. Section 5 describes the data and section 6 outlines the primary empirical methods to be used. Section 7 discusses the main results, and section 8 addresses threats to the identification. Section 9 calculates the influence of measurement error in attenuating the estimates. Section 10 returns to the model to show the larger impacts

and concludes.

# 2 Literature review

## 2.1 Changes to threats and risk-taking

Peltzman (1975) was the first to formalize a theory of risk homeostasis whereby individuals responded to a decrease in the riskiness of an action by increasing their choice of the optimal quantity of that action. This risk offset hypothesis is similar to theories of behavioral disinhibition due to changes in risk, to theories of risk compensation mentioned in the public health literature[1], and moral hazard associated with treatment access. Previous empirical work has found strong evidence for risk homeostasis in the context of driving and car safety innovations (Winston, Maheshri, and Mannering, 2006).

In the context of HIV risk-taking, theories of risk homeostasis have been tested in two papers that look at whether individuals who know that circumcision reduces HIV transmission risk change their risk-taking behavior in response to their own circumcision status (Godlonton, Munthali, and Thornton, 2011; Wilson, Ziong, and Mattson, 2011).

## 2.2 HIV prevalence and risk-taking

If innovations that reduce costs associated with HIV infection are believed to increase in risky-taking through behavioral disinhibition, one would also believe that HIV should have generated behavioral inhibition. This idea has been regularly tested, with mixed findings. Oster (2009) finds very little responsiveness in sexual behaviors in Sub-Saharan Africa to regional variations in HIV rates, but larger responsiveness among those who are richer and likely to live longer. She argues that this is because with low base life-expectancy, the cost of HIV infection is not as high relative to what it is for those who expect to live longer.

[1]This term is commonly used but should not be confused with risk compensation in the labor economics literature referring to increased wages paid to employees asked to undertake greater risks.

A subset of this literature on behavioral inhibition focuses on fertility as an outcome, both because this is easier to reliably measure for large populations than is self-reported sexual behavior and because it has important implications beyond health for population change and welfare. Young (2005) estimated a macroeconomic model of the implications for growth of a high HIV infection rates for an entire society. This included a simple estimate of the impact of HIV on fertility with a large reduction in fertility using DHS data in South Africa. This was followed by a more detailed study of this specific parameter (Young, 2007). This research has been challenged by others who have not found that the correlation between fertility and HIV rate is causal in other contexts. For example, Kalemli-Ozcan and Turan (2010) use newer DHS data, also from South Africa and find no evidence for a relationship between HIV risk and fertility. Juhn, Kalemli-Ozcan, and Turan (2010) find no causal relationship between HIV rates and fertility using DHS data from multiple countries.

It is important to note that the resulting behavior changes are a function not of actual changes in the cost and risks associated with contracting HIV, but of perceived changes in risk. There is evidence that people may overestimate their risk of contracting HIV (Kaler, 2003). In surveys with young women in Western Kenya (Miguel et al, 2005-2007), girls were asked to guess how many people have HIV out of 100 in their area. The median response was 50, even though the overall prevalence is 6% in the country and not more than 10 or 15% in the region. Unfortunately, local variation in beliefs about prevalence is sufficiently difficult to measure that - to my knowledge - there is not a study linking perceived prevalence to fertility or other proxies for risk-taking.

## 2.3 Antiretroviral drugs and risk-taking

Antiretroviral drugs are still a relatively recent innovation and their large-scale availability is even more recent. While there is a great deal of ongoing research about the implications of antiretroviral drugs, the published literature is still very limited.

There are some studies of behavior change as a result of treatment access from the US,

where drugs were widely available earlier. One study in the US (Lakdawalla et al, 2006) uses variation in state level Medicaid eligibility rules in the US to estimate the impact of being on ARV medicine on number of sexual partners. They argue that simply because people are kept alive and healthy longer through the medicine, the average person on ARVs has two additional sexual partners beyond what they would have had without the drugs. Another study finds evidence of an increase in risky sex among gay men in San Francisco just after the introduction of ARVs (Mechoulan, 2007). Papageorge (2012) also finds an increase in risky behavior among gay men after the introduction of HAART in the US. Still, the context of East Africa, where the overall HIV rate is higher, life-expectancy is lower, and access is still limited, there is reason to believe the outcomes could be very different.

In sub-Saharan Africa, research interest in this field is exploding in part because of the massive policy implications of the introduction of ARVs in areas with very high rates of HIV infection. The first well-known paper in this area looked at changes in productivity after ARV treatment began among individual workers on a tea plantation in Kenya (Thirumurthy, Graff Zivin, and Goldstein; 2008). This paper found large impacts on productivity among those on treatment and in well-being for their families.

More recent work has begun to look at the impacts of ARVs on a range of outcomes in sub-Saharan Africa. McLaren (2012) finds an increase in employment in areas between 3 and 15kms from newly opened ARV clinics in South Africa. Baranov, Bennett, and Kohler (2012) find a reduction in mortality risk perceptions - because of a perceived decrease in likelihood of infection - among those exposed to ARVs in one region in Malawi. These authors also find no evidence of an increase in risk-taking in their sample. In Zambia, Wilson finds an reduction in fertility in areas with greater access to PMTCT (2012) and higher demand for HIV testing in areas with ARVs (2010). This paper uses similar methods - exploiting variation in timing of a national roll-out across health facilities - to these papers, with data from all of three new countries and looking at the impact of treatment on risk-taking, which is an extremely important question for policy-makers.

A few other papers have used a variety of methods to look at the impact of ARV access on risk-taking. Using data from Mozambique, de Walque, Kazianga, and Over (2012) focus on those who are HIV negative who are in the same household as an HIV positive individual and compare those in which the HIV positive household member is on ARVs with those in which he or she is not. They find significantly more self-reported sexual risk-taking in households with exposure to ART. This paper has the benefit of a broader sample that includes the general population and can perhaps be more broadly applicable.

There is still an enormous amount that is missing from this literature largely because data collection in this area is limited, but also because the context is rapidly changing. Widespread availability of Antiretroviral medicine only became a reasonable consideration in Sub-Saharan Africa in the last few years and HIV testing was not widely available until only a decade ago. Thus this project addresses a new and very important question of how to shape policy in the current setting to reduce the spread of HIV and increase well-being.

# 3 Model and Conceptual Framework

The following model serves two purposes. First it demonstrates the intuition behind the empirical tests by demonstrating that the sign of the impact of treatment access on the proportion of the population that chooses to engage in unprotected sex is theoretically ambiguous. Second, it demonstrates precisely how this behavioral response determines the impact of the distribution of drugs on the rate of new infections, and it serves as a framework to be used to show how different policy options could generate different outcomes.

## 3.1 Presence of ARVs and beliefs about access

For the sake of clarity, it is first necessary to explain how the presence of antiretroviral drugs in a clinic can change individual perceptions about their own likelihood of receiving treatment if they were to become infected and the likelihood of others to be taking the drugs.

As ARVs are introduced in a health facility, three things that are visible to the community happen quickly. First, many health facilities organize publicity campaigns to announce to the population that drugs are available. Second, some people will begin treatment very quickly. These are the individuals who had previously tested positive and who are eligible for treatment based on having a low CD4 count, meaning that the disease has progressed to the point that they are likely to experience opportunistic infections and become ill or worse. Third, rumors begin to spread through a community.

These signs can change people's beliefs about the perceived likelihood of receiving treatment in three ways. First, some people may hear about the existence of antiretroviral drugs for the first time. Second, others see sick people become healthy. Even if they do not know why these people have become healthy, it may change their beliefs about what happens to a person who becomes infected with HIV. Third, for those who previously knew about the existence of the drugs, their appearance in a local health facility reduces the anticipated cost of acquiring them. Together, all of these channels increase the perceived likelihood of treatment conditional on becoming HIV positive. This pathway is illustrated in figure 1. It is important to note that some may believe that the treatment is a cure, and thus an estimated response may be an estimated response to incorrect information. Although the DHS questionnaire in Kenya does not ask about ARVs explicitly, the Uganda DHS questionnaire used in 2006 did. When asked if they thought that ART was a cure for AIDS, 32% of women who had heard of ARVs said that it was, while 62% said that it was not.

The model presented below is a simple exposition of causal channels through which the introduction of antiretroviral drugs can change individual behavior in significant ways. Previous models of changes in behavior in response to different circumstances have been presented to show the impacts of potential policy options. The first of these was Kremer (1996) who argues that high HIV prevalence may dissuade those who are cautious and least-likely to be infected from participating in sexual activity while causing those who are less cautious to take more risks because of the low probability of their staying negative. He

argues that this creates a cycle increasing the risk of infection among the pool of potential sexual partners generating high-risk equilibria. Gong (2012) adapts earlier models to show how HIV testing can change behavior differently for those who had high and low priors about their own status, and finds support for this using data from an early randomized roll-out of HIV testing in East Africa. Kerwin (2012) constructs a new model that rationally explains a type of fatalism based on previous risk-taking that can generate non-monotonic responses to changes in risk. This model could explain a finding often found in Malawi in which individuals sufficiently overestimate their risk of infection that they stop taking precautions in response to information about higher risks (e.g.: Kaler, 2003).

This new model is designed in particular to show the channels through which the availability of antiretroviral drugs can change behavior, resulting in ambiguous theoretical predictions regarding the impact on overall risk-taking.

## 3.2 Model

In this model, individuals choose whether or not to have unprotected sex based on an individual-specific utility from unprotected sex and the cost of the possible risk of infection. This cost incorporates both the likelihood of becoming infected conditional on having unprotected sex and the utility cost of becoming infected. Access to treatment can change perceptions about both of these.

The rate of new infections among those previously uninfected, referred to as $I$ is equal to the probability of infection conditional on engaging in unprotected sex, $p$, and the proportion of the uninfected population that chooses to do so $A_1$. Treatment availability directly changes $p$ by changing the pool of potential partners and their infectivity, and it can change both $p$ and $A_1$ through the impact on behavior outlined below.

The following **assumptions** will be made throughout:

1. Each individual has full information about his or her own status.[2]

[2]While the opposite was a necessary assumption in earlier models (e.g.: Kremer, 1996), the widespread

2. Each individual has full information about the distribution of other types among potential sexual partners, but no information about the status of a particular potential partner.

There are three types of individuals:

1. HIV negative
2. HIV positive, without treatment
3. HIV positive, with treatment

There are three types because each type makes decisions based on different factors and contributes differently to the risk of infection. Changes in outcomes depend on the relative sizes and behaviors of each type, and antiretroviral drugs change both.

Each individual chooses **whether to have unprotected sex** based on:

1. An individual-specific utility from unprotected sex (incorporating everything including social pressure and desire for children, etc.), and
2. For those who are uninfected, the likelihood of becoming infected and the associated utility cost.

This is outlined for each group:

- Group 1: An individual who is uninfected will choose to have unprotected sex if:

$$\begin{aligned} U(UnprotectedSex) &> U(NoUnprotectedSex) \\ \theta_i + (1-p)(V_{neg}) + p(V_{pos}) &> V_{neg} \\ \theta_i &> p(V_{neg} - V_{pos}) \end{aligned}$$

availability of free HIV testing makes this assumption more plausible.

where $V_{neg}$ represents the continuation value of staying negative in this period and $V_{pos}$ represents the continuation value of being positive. The individual specific parameter, $\theta_i$ follows some distribution with CDF, $F_\theta$. This parameter can represent any utility benefit to unprotected sex relative to the alternative, including social pressure, desire for conception, or anything else.

Therefore, the proportion of the population that is negative (Type 1) that chooses to have sex can be represented as:

$$A_1 \quad = \quad 1 - F_\theta(p * (V_{neg} - V_{pos}))$$

Note that there are two ways that the availability of treatment can change this decision. First, it can change $p$, the probability of becoming infected conditional on having unprotected sex because it changes the population of potential sexual partners. The sign of this is ambiguous, which will be shown below. Second, it changes the cost of becoming infected. As treatment availability increases, this cost clearly is reduced, which would lead to an increase in participation.

- Groups 2 and 3: Those who are positive do not risk changing their HIV status,[3] and thus the only parameter in their utility optimization is the individual-specific utility from unprotected sex. Allowing for altruism, morbidity, fatalism, or any other factor that changes the utility from unprotected sex for those who are positive can be done by allowing this parameter to be drawn from a different distribution for Group 2 and for Group 3.

[3]Those who are HIV positive do risk re-infection from having sex with another person who is HIV positive. This can moderately increase the speed of the progression of HIV into full-blown AIDS. However, this can credibly be assumed to be negligible with no loss to the applicability of the model.

Thus, an individual in Group 2 will choose to have unprotected sex if:

$$\begin{aligned} \gamma_i + V_{pos} &> V_{pos} \\ \gamma_i &> 0 \end{aligned}$$

and an individual from group 3 will choose to have unprotected sex if:

$$\begin{aligned} \omega_i + V_{pos} &> V_{pos} \\ \omega_i &> 0 \end{aligned}$$

These parameters, $\gamma_i$ and $\omega_i$, are drawn from distributions with CDFs, $F_\gamma$ and $F_\omega$, respectively.

Therefore, the proportion of the population that is positive and not on ARVs (Type 2) that chooses to have unprotected sex can be represented as:

$$A_2 = 1 - F_\gamma(0)$$

and the proportion of the population that is positive and on ARVs (Type 3) that chooses to have unprotected sex can be represented as:

$$A_3 = 1 - F_\omega(0)$$

Therefore, a fixed proportion of those in Group 2 and Group 3 will choose to have

unprotected sex, and this does not depend on ARV access. Drugs do, however, change the size of each of these groups. On the other hand, the proportion of those in Group 1 who engage in unprotected sex is a function of treatment access.

The probability of becoming infected from unprotected sex $p$ depends on the proportion of each group among potential sexual partners and the likelihood of transmission from each group. $N_j$ represents the size of each group and $A_j$ represents the proportion that chooses to have sex. The likelihood of transmission if the partner is from Group 2 is $r$ and $r * q$ is the likelihood of transmission of the partner is from Group 3.

The reduction in infectivity as a result of antiretroviral drugs is represented by $q$. Based on the medical literature, $q$ is 0.04 so the reduction in infectivity from treatment is very large. However, individuals respond to beliefs about $q$ and $\hat{q}$ could be anywhere between 0 and 1. If individuals believe that ARVs are a cure for HIV - which is a common belief - then an individual will believe that $\hat{q}$ is 0.[4] On the other hand, if individuals know that it is not a cure, they are still not likely to know about the reduction in infectivity, and this would mean that $\hat{q}$ is equal to 1.[5] This result is new to the medical literature and based on the slow progression of the disease, it would be hard for individuals to perceive this based on observation.

The likelihood of infection from unprotected sex can therefore be represented by:

$$p = r * \frac{A_2N_2 + A_3N_3q}{A_1N_1 + A_2N_2 + A_3N_3}$$

and the perceived likelihood of infection is:

$$\hat{p} = r * \frac{A_2N_2 + A_3N_3\hat{q}}{A_1N_1 + A_2N_2 + A_3N_3}$$

[4]This belief will also overestimate the reduction in the cost of infection.

[5]In informal conversations with HIV clinic employees, this was a commonly held belief. Many expressed concern that people who were HIV positive had become healthy and fat and were at risk of infecting others.

Treatment access changes the $p$ by changing 1) the relative sizes of the population in each group and 2) the proportion of those who are negative who engage, $A_1$.

The fraction of those who are positive who receive treatment is referred to as $D$. First we will outline how this changes the size of the population in each group. $N_1$ is the size of the population that is negative at the beginning of the period. Call $M$ the size of the population that was infected as of the beginning of the current period. Besides having different behavior as outlined above, individuals in type 2 and type 3 have different death rates. The primary thing that provision of ARVs does is to keep people with HIV alive.[6] Therefore the size of each population will be determined by:

$$\begin{aligned} N_1 &\quad \text{is fixed from the previous period.} \\ N_2 &= M * (1 - D) * (1 - d_2) \\ N_3 &= M * D * (1 - d_3) \end{aligned}$$

where $d_2$ and $d_3$ are the death rates for those in Groups 2 and 3 respectively, and we know that:

$$d_2 > d_3$$

If there is no treatment available, then $D = 0$ and $N_3 = 0$. If everybody who is positive is on treatment, then $N_2 = 0$.

To summarize, $D$ changes two components of the individual decision and thus the size of the uninfected population that chooses to have unprotected sex, $A_1$: 1) It decreases the cost of becoming infected ($V_{neg} - V_{pos}$), and 2) it changes $\hat{p}$, the perceived likelihood of becoming

[6]This model serves to show how ARVs change new infection rates and it does not address overall welfare. Still, it is important to mention that regardless of the impact on new infection rates, treatment provides many additional years of healthy life for those treated. This can also have enormous benefits for families of those who are infected who get the additional time with their loved ones.

infected. The sign of this is ambiguous and depends on other parameters.

In particular, if $\hat{q} = 1$, then:

$$\frac{d\hat{p}}{dD} > 0$$

This is intuitive because if there is no reduction in infectivity, then the drugs keep more infected people alive and in the pool of potential sexual partners. If others who are negative respond by not choosing to have sex and therefore not adding themselves to the pool of potential partners (i.e.: if $A_1$ decreases), then $p$ will increase even more.

On the other hand, if $\hat{q} = 0$, then:

$$\frac{d\hat{p}}{dD} < 0$$

If some of those who are positive are put on treatment and these individuals cannot transmit the virus, then the possibility of matching with someone who can transmit HIV is reduced. As above, if others who are HIV negative respond to this, the effect is exaggerated.

dp/dD is ambiguous so dA/dD is ambiguous and needs to be measured.

The model was set up in part to show how drugs change new infections directly and through changes in behavior. As previously stated,

$$I = A_1 * p$$

All parameters that contribute to the above equation can be taken from the existing medical literature, with the exception of the behavioral response to treatment, which determines $A_1$, and indirectly, $p$.

This response will be measured in the empirical analysis of this paper, and then this estimated parameter can be used to predict the impact of drugs on new infections.

# 4 Context

Antiretroviral drugs were developed beginning in the 1980s and became widely available in developed countries in the 1990s. Because of prohibitively high prices, they were almost completely unavailable to residents until the last decade. In the early 2000s, a number of agreements between developing countries and pharmaceutical companies, organized by the Clinton HIV/AIDS Initiative (CHAI), the World Health Organization (WHO), the Global Fund to Fight AIDS, Tuberculosis, and Malaria, and other organizations, introduced reduced prices for ARVs for governments of developing countries. Since then, the price has fallen from more than $10,000 per person per year to under $100 per person per year.

Kenya has a relatively high rate of HIV infection, reported in table 1, and it has seen a large expansion in access to antiretroviral drugs in the last decade. ARVs are distributed through pre-existing health facilities. In the early stages of the roll-out, the Ministry of Health and other associated government organizations charged with fighting HIV outlined plans to provide geographically disbursed access through capable facilities. Although initially only large hospitals were considered to have all the necessary staff and equipment to provide treatment, requirements for facilities to be designated as capable have been dramatically reduced. In 2004, only 7 facilities distributed ARVs in Kenya but this increased substantially, shown in Figures 2 and 3. Treatment is provided for free to those who are HIV positive and eligible for treatment[7], paid for by the governments through donor funds or by international organizations directly.

While the explicit goal of geographic dispersion presented some exogeneity in placement, there are some particular characteristics of locations that made them more likely to have ARV provision, and the empirical analysis will control for these factors. Urban areas and areas with high rates of HIV were more likely to introduce ARV provision. Because distri-

[7]Eligibility was initially based on assessments of whether a person was expected to be able to adhere to the medicine, and the progression of the disease. Now the primary metric for eligibility is the progression of the disease. Initially a person was eligible with a CD4 count below 200, but the WHO has increased the threshold to 350.

bution happened through existing facilities, areas with large hospitals (regional, provincial, or district hospitals) were more likely to distribute ARVs, while areas without nearby health facilities were less likely. The Kenya Open Data Initiative provides a record of the GPS locations of all health facilities currently in Kenya and this information is included in the analysis to deal with non-random placement.

# 5 Data

The data for this project comes from Kenya which has had one of the highest HIV rates on the continent with 9.5% in 2000.[8] It is home to many early research projects about HIV prevention and treatment (e.g.: Bailey et al, 2007), yet it has not seen as much investment in ARVs and health generally and it has not been as successful in combatting stigma as its neighbors.

Information about ARV access comes from a new dataset constructed using administrative records obtained from meetings with government and NGO officials in Kenya. The GPS information comes from the Kenya Open Data Initiative [9] and the timing information comes from reports provided by Kenyapharma, a procurement agency, and the National AIDS and STI Control Program of the Ministry of Health. This combined database of health facilities that currently provide ARVs includes information for each facility on the year ARVs began being distributed, and its latitude and longitude.

There are a number of challenges to linking these data sources. First, each data source has slightly different names for clinics. This is even true across years within records from the same organization. Second, the administrative records from the National AIDS and STI Control Program (NASCOP) are not complete, because not all facilities always fill the required paperwork. In some years, records are missing for some clinics, which means that not every clinic that is represented in one year appears the next year, even though they

[8] 2008 Report on the global AIDS epidemic, UNAIDS/WHO, July 2008.
[9] (opendata.go.ke)

continue to supply drugs.[10] Based on the clinic names, I matched the clinics from the NASCOP to the GPS locations of health facilities. I use the first instance that a health facility appears in the records as the first year in which treatment was available. Table 1 shows the number of health facilities and the number of individuals receiving treatment in each year. The records from before 2007 are from Kenyapharma, a procurement organization that previously handled antiretroviral drugs. These records are incomplete because a few organizations used another procurement agency. However, whether a clinic opened in 2006 or 2007/2008 will not change the analysis because of the timing of the Demographic and Health Surveys data.

The data on individual behaviors comes from two rounds of geocoded Demographic and Health Surveys (DHS) from 2003 and 2008/2009. Kenya expanded treatment availability largely between 2007 and 2009, so these rounds provide information from before and during the middle stages of the expansion. Table 2 shows the number of clusters and the number of individual men and women in each survey. Each cluster has an average of 18 households and 30 individuals. Summary statistics of relevant variables are reported in Tables **??** - **??**.

The DHS data contain responses to questions about risk-taking, number of partners, whether the individual has had sex, as well as questions that reflect conditions which are less likely to be underreported than explicit sexual activity, including childbearing and STD infections. There is extensive evidence of misreporting of sexual activity and risk-taking from direct survey questions (e.g.: Jamison and Karland, 2012; Minnis et al, 2009). In this particular dataset, the documentation for the second wave of DHS data points out that for 609 women surveyed, the age at which they reported that they first had sex was later than the age at which she first gave birth. Of 2096 individuals in both waves who reported that they had never had sex, 24 tested positive for HIV. All individuals in the sample are over age 15 and this rate is well above the error rate of the system of tests used. Because of these

[10] In conversations with officials working on Monitoring and Evaluation of ARV distribution, I was not told of any health facilities that stopped distributing drugs, unless they were replaced by another organization in the same location.

concerns about self-reporting, childbearing is a commonly used measure of HIV risk taking in the literature (e.g.: Dupas, 2011). This paper will follow this convention and use current pregnancy as a proxy for unprotected sex.

For this analysis, the sample includes women, aged 15-49. Nairobi is not included because access to antiretroviral drugs was present there as of 2003. For a large part of the analysis, the sample is restricted to those who do not already have children. This represents a population that is most likely to not know the HIV status of potential sexual partners and to be choosing new partners, and thus more likely to incorporate the risk of HIV into their decisions. An alternative selection would be to use those who are unmarried. However, there is a large amount of discrepancy even within the data regarding who is and is not married. There are multiple steps taken to be considered married in Kenya - including the payment of bride price, cohabitation, a religious ceremony, and a legal contract - and since these do not always happen at the same time, the distinction between those who are and are not married is fuzzy and can depend on how the question is asked and the individual responding. An additional concern with using marriage as a cut-off is that once someone becomes pregnant, there may be a quick wedding or the couple may begin to refer to themselves as married, and so the selection is endogenous. Another cut-off that could be used is age, but many of the youngest respondents are married and so which age to use is a question. I will present results using married and young as the selection criteria as well as robustness checks.

# 6 Estimation Strategy

Because of variation across space and over time with two waves of data, the analysis in this paper will rely on a difference in differences framework. However, unlike a standard difference in difference estimation, the classification between treatment and control is flexible, and the availability of GPS locations with different samples selected in each wave means that the appropriate comparison across rounds is also not obvious. Thus the analysis adapts a

range of similar specifications to narrow in on precise estimates of the impact of ARV access on individual behavior.

In all specifications, the relevant time period for access will be the year before the survey was conducted. This gives some time for information to diffuse, and is appropriate because the outcome of interest reflects action taken in the previous 12 months. All observations are weighted using DHS sampling weights, unless otherwise noted, and all specifications include controls for age, education, district and division HIV rate.[11] Finally, each specification includes controls for urban-rural status, the presence of a large (district, provincial, or regional) hospital within 10 kilometers and the absence of any health facility within 10 kilometers, as well as all three of these interacted with Wave 2.

The basic equation to estimate is:

$$Y_{ijt} = \beta_0 * Treat_{ij} * Wave2_t + \beta_1 * Treat_{ij} + \beta_2 * Wave_{it} + \gamma_j + \delta_t + \sum_{k=1}^{n} \beta_k * X_{kijt} + \epsilon_{ijt}$$

where $Y_{ijt}$ is an indicator for whether the respondent reports that she is currently pregnant. $Treat_{ijt}$ is a binary variable that represents whether individual $i$ in area $j$ at time $t$ is located in a place in which ARVs were available before Wave 2, while $\gamma_j$ is an area fixed effect, and $\delta_t$ is a time fixed effect and $X_{ijt}$ is a vector of individual-specific controls. Each wave surveys different villages, and therefore the definition of an area $j$ cannot be a village. Each specification will define area differently.

In the first specification, $Treat_{ijt}$ is defined as residing within a district in which at least one health facility provided ARVs by 2008. This specification includes district fixed effects and standard errors clustered at the level of the district.[12] One concern with this is that districts in Kenya are different sizes and subsequent specifications will address this

[11] This is constructed using the DHS sample as this is the standard source of information about HIV rates. Each respondent is excluded from the estimate of the HIV prevalence in her area.

[12] During the time between waves, politicians changed district boundaries many times. For consistency, I use current district boundaries and place observations in them using their GPS locations.

issue. This specification introduces inevitable misclassification as some of those within the borders of a district presumably do not know about the treatment available and do not respond, while others outside of the district anticipate access within the district. This type of misclassification is unavoidable in this context, and it is likely to attenuate estimates of the treatment effect.

In the second specification, $Treat_{ijt}$ is defined as being within 10 kilometers of a facility with ARVs by 2008. While this deals with the variation in district size by using a fixed distance to determine access, it still will provide attenuated estimates from misclassification. Because the same villages were not sampled across waves, it's not possible to include fixed effects using the same measure area used to define treatment. Instead this specification includes district fixed effects and standard errors clustered at the district level.

In order to address concerns about variation in district size, the third specification uses evenly-sized squares as the definition of an area. This is done latitude and longitude lines to create squares. Because Kenya lies on the equator, latitude and longitude lines do outline a shape that is very close to a square, although this would not be true in other parts of the globe. In this specification, an observation in a grid-square is defined as treated if that grid contains a health facility that began distributing ARVs by 2008. For this specification, grid-square fixed effects control for time-invariant spatial characteristics and standard errors are clustered at the same level.

## 6.1 Matching observations across rounds

Because of the geographic detail available in this data, it is possible to compare across very similar observations, however identifying the most appropriate comparison group is not obvious because the samples are different across waves. In the initial analysis, district fixed effects were used to compare observations over time that are likely to be in approximately the same area. However, using large districts or provinces means that the comparison is with observations from far away, and using small districts means that some contain only

observations from one wave and thus the fixed effects remove any variation that can be used to identify the impact. In what follows, I make use of the GPS locations to come up with more precise comparisons and construct a fixed effect analysis within pairs of neighboring survey clusters.

For each survey cluster in wave 2, I link this with the five closest survey clusters from wave 1 that are in the sample.[13] This gives five pairs of clusters per survey cluster from wave 2. For the analysis, each respondent from wave two is included five times and each observation from wave 1 is included as many times as it is matched. This means that the dataset is now ten times the size of the wave 2 sample. Any pair that is more than 100kms apart is dropped.

Using this new expanded and matched sample, I estimate the same equation as above, with three changes. First, I include fixed effects for each matched pair of clusters. Second, each observation is weighted by the DHS sampling weights multiplied by 1/100 if the distance between the two is less than 10 kilometers and the sampling weights multiplied by the inverse of the distance if they are more than 10 kilometers apart.[14] Third, the standard errors are clustered at the level of the wave 2 survey cluster to reflect the duplication. The coefficient of interest remains the interaction between $Wave2$ and $Treat_{ijt}$ where treatment is defined as in specification 2.[15]

# 7 Results

The main results are reported in Table 7. Column 1 presents the estimates when treatment is defined as having a facility with ARVs in the same district. The treatment effect is the coefficient on the interaction term, reported in the first row. For clarity, these are pre-

[13]Because the locations of villages is jittered and some villages may be sampled twice, it is possible that some of these matched pairs are truly taken from the same villages at two points in time.

[14]This weighting scheme is used in place of the inverse distance so as not to overweight extremely small distances. Because of the jittered data, these distances are not likely to be precise at this level.

[15]To current readers: the next iteration of this specification will use two-way clustering with wave 2 and wave 1 survey clusters. This should slightly increase the standard errors and reduce the significance, but I do not think the difference will be substantial.

sented in percentage points so the first coefficient implies a treatment effect of 8.3 percentage points. This implies that change in the likelihood of being pregnant increased between the two waves by 8.3 percentage points more in districts with ARVs. The coefficient on $Wave2$ is negative and significant and of a similar magnitude. Therefore, this demonstrates a decline in the likelihood of being pregnant in districts without treatment available and no such decline in districts that did receive treatment.

The second column presents the results defining treatment as being within 10 kilometers of a facility with ARVs by 2008. This coefficient is smaller and insignificant, although still positive. One concern with this specification is that the fixed effects are not at the same level as the treatment assignment and thus they are unable to pick up important geographic variation.

The third column shows results using grid regions. Again, the coefficient of interest is large and positive, but statistically insignificantly different from zero. This specification has the benefit of using uniformly sized areas, but it also introduces noise because the area is arbitrary. In this specification, a large fraction of regions have observations from both rounds and thus the pool of observations that contribute to the identification is small, which may explain the larger standard errors.

The fourth and fifth columns present results using the matched observations. In column 4, this specification is implemented as described above. However, the variable representing $Treat$ noticeably is not dropped. This is because there are some pairs in which the cluster from one wave is designated as treated, but not its pair. In column 5, these unbalanced pairs are dropped from the analysis. This is similar to other spatial analysis in which areas along the borders between regions are removed. In both specifications, the coefficient of interest is positive and significant, representing in increase in the likelihood of being pregnant of approximately five percentage points.

## 7.1 Robustness

This section directly deals with concerns about endogeneity or selection that could arise from the main specification.

Table 8 demonstrates that the impact of the change differentially falls on those who do not yet have kids. The same analysis from table 7 is repeated using the entire sample, and interacting $Treat * Wave2$, $Treat$, and $Wave2$ with whether the individual has children. The coefficients in the first row match those from table 7. Any differences are a result of more precise identification from the use of the entire sample to identify the impact of the control variables, resulting in smaller standard errors on the coefficients of interest. These coefficients are similar in both tables, although the main coefficient in the second column is now significant. The fourth row shows the interaction between the treatment effect and having children, and this is consistently negative across specifications. It is significant in only two specifications, so this is perhaps suggestive evidence of a differential impact. The magnitude of the coefficients in the fourth row is similar to the magnitude of the coefficients in the first row demonstrating that the treatment effect does not exist for those with children, but it does for those without.

Tables 9 and 10 repeat the analysis restricting the sample to those who were not married as of 12 months before being surveyed and under age 25[16]. This presents results very similar to the estimates restricting the sample to those without children.

One concern with using pregnancy rates is that perhaps the results reflect a change in desired fertility rather than a change in willingness to engage in unprotected sex. Table 11 repeats the main analysis using the ideal number of children as the outcome. The treatment effect on this outcome is small and insignificant.

Finally, difference in differences can deal with non-random placement only if we can assume that the treated and untreated areas would have had parallel trends. One way to

[16]Of those who report being not married, 46% of the entire sample already had children, and thus the designation may not reflect un-partnered or single. Restricting the age group to those under 25, only 23 percent previously had children

show that this is plausible is to show previous trends. While there are only two rounds of geocoded data, the detailed birth registry in the DHS data makes this possible. Using information provided by respondents about the timing of previous births, I show that there are not differential pre-trends by replicating the analysis using information from ten years before. In this case, pregnancy is determined by whether the respondent reports having given birth between 10 years and 9 years and 3 months ago. The sample is limited to those who had not previously had children and who are at least 25 years old at the time of the survey to replicate those who would have been age 15 (the lower age cut-off for the DHS surveys) ten years before. These results are reported in Table 12. As can be seen, the coefficients on the placebo treatment effect are small and insignificant, suggesting that the results shown in the main specification represent the impact of ARVs rather than different trends across regions.

# 8 Measurement Error

There are two types of measurement error that are important in this estimation. One is inevitable, and the other can be explicitly addressed to understand how much the estimated parameters may be attenuated.

The first type of measurement error is unavoidable when using distance as a measure of access. At any distance chosen, there are likely to be people who live in the catchment area of a clinic but do not know about it or are unable to obtain its services, while others live outside of the catchment area and are able to access what the clinic offers. Thus defining treatment with distance means that there will necessarily be some misclassification.

The goal in choosing a cut-off distance is to minimize this misclassification. Unfortunately, given the data, eliminating this misclassification is impossible, and therefore, the parameters estimated are likely to be biased towards zero.

The second type of measurement error is deliberately added to the GPS locations of DHS

respondents in order to protect confidentiality. For each rural cluster, a distance is drawn from the uniform distribution between 0 and 5 kilometers. The GPS location is reported to be this distance away from the actual point in a randomly selected direction. For urban clusters, the method of jittering is the same, but the distance is between 0 and 2 kilometers. This means that the distances between the facilities and the respondents are mis-measured by between 0 and 5 kilometers.

Because this measurement error is perfectly known, it is possible to simulate the magnitude of attenuation that it generates in the estimates. IN PROGRESS.

# 9 Implications and Conclusion

The introduction of antiretroviral drugs could influence the spread of HIV both through changing behavior and through biological channels - reducing infectiousness of those on treatment and keeping more people who are HIV positive alive. This is formalized in Section 3, demonstrating how the sign of the impact of ARVs on new infections is ambiguous and depends on behavior.

The empirical analysis above showed a relative increase in risk-taking among those with access to antiretroviral treatment. This directly increases the rate of new infections by increasing those who put themselves at risk. However, it also can indirectly decrease the rate of new infections as the increase in $A_1$ means that a larger fraction of the pool of potential sexual partners is HIV negative, decreasing the risk of infection for those who engage, $p$.

In addition, the reduction in transmission risk from treatment, $q$, can outweigh a substantial change in behavior among those who are negative so that the rate of new infections will decline with treatment. Again, it bears mentioning that beyond the impact on new infections, ARV access has large and important welfare impacts for those who are infected and receive treatment. NOTE: THIS WILL BE FORMALIZED

In practice, the effect of $D$ on behavior is likely to be non-linear with larger effects for

those who are sicker. The benefit to an individual who is HIV positive of being on treatment is high when he or she has a low CD4 count, which means being close to AIDS onset and opportunistic infections. However, especially given the toxicity and unpleasant side-effects regularly supported, treatment early, when the CD4 count is high and the person's immune system is not yet fully compromised, is not likely to be as large a benefit to the individual. Thus promising access to treatment when someone's CD4 count gets to 200 (which was previously the WHO recommended threshold) is likely to generate the largest difference in behavior relative to no treatment. This will extend life by approximately ten years, dramatically changing the cost of infection. Moving this threshold to 350 - in practice - may have an additional benefit, extending life a little bit more and reducing time spent with opportunistic infections. However, increasing this threshold beyond that may not dramatically change perceived costs of infection. However, the reduction in infectiousness from ART will continue with a higher and higher threshold. [17]

WHO changed the recommended CD4 count threshold to determine ARV eligibility from 200 to 350, however most countries in sub-Saharan Africa have not reached full coverage even with the lower threshold due to a lack of supplies. Rwanda is one exception, reporting nearly 100 percent coverage of those eligible, and experimenting with using 500 as a threshold for those in serodiscordant couples to reduce the likelihood of transmission to the uninfected partner.

Based on the reasoning above, a low level of ARV access could change behavior but not see a significant reduction in infectiousness, while a very high level in which treatment is available upon diagnosis of HIV infection would reduce incidence of HIV. This is outlined in Over et al (2006) and Granich et al (2009) who propose beginning treatment immediately after a positive HIV test.

This paper shows an increase in risk-taking associated with the introduction of antiretroviral drugs using new national data from Kenya. While this was a significant fear raised by

[17]This effect also is not quite going to change linearly because viral loads are lower for those whose infection has progressed less.

opponents of widespread provision of ARVs in sub-Saharan Africa, I show that even with a significant increase in risk-taking, new infections are still likely to be reduced, especially if the level of ARV distribution is raised to a sufficiently high level to provide treatment earlier to infected individuals.

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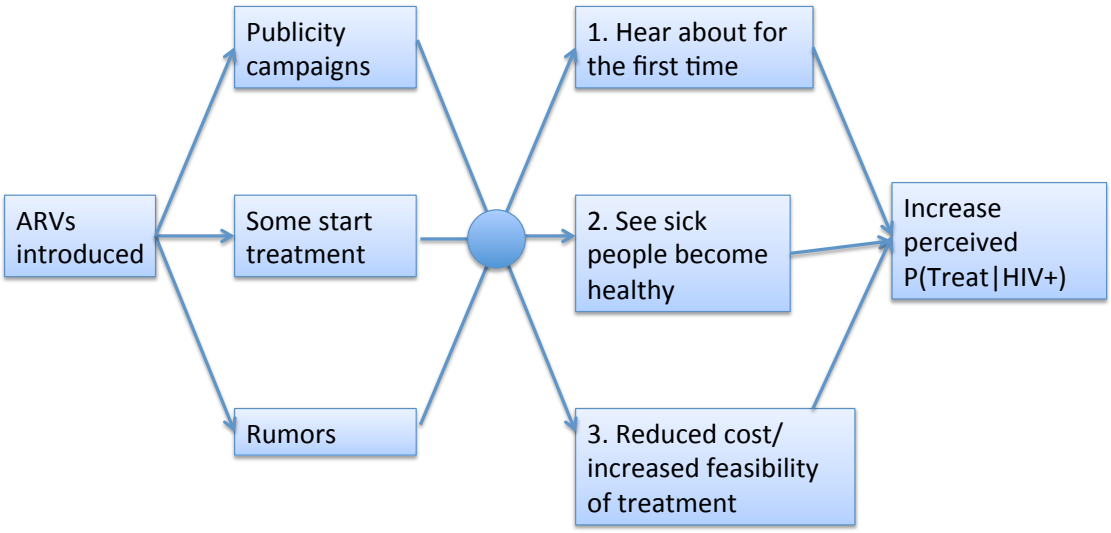
Figure 1: Conceptual Framework Part 1




# 10 Figures

Figure 2: ARV distribution sites in 2004 in Kenya:

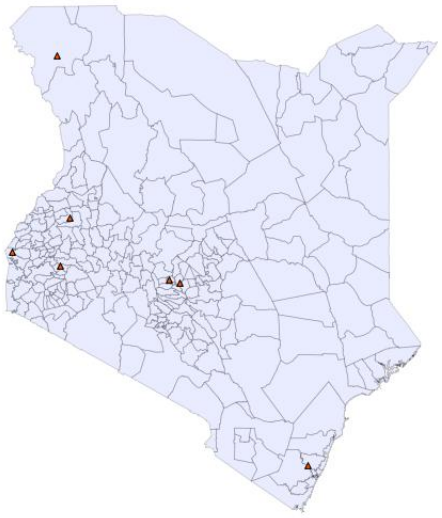


Figure 3: ARV distribution sites in 2009 in Kenya:

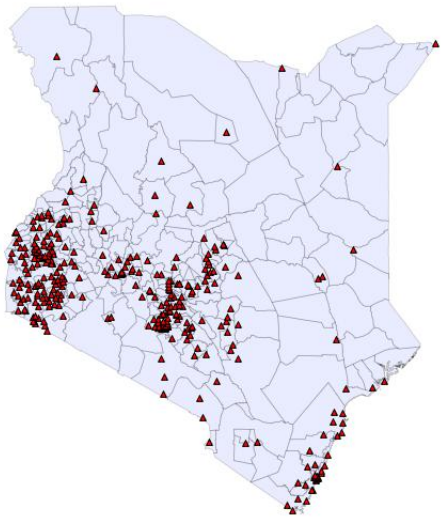
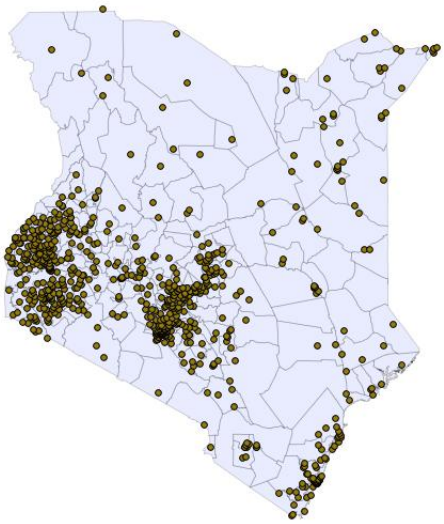


Figure 4: DHS clusters in Kenya, 2003, 2008/2009



# 11 Tables

Table 1: Number of health facilities in Kenya providing ARVs by year

| *Year* | Number Facilities with ARVs | HIV Prevalence (WHO) | DHS Survey |
|---|---|---|---|
| 2003 | 1 | 7.0 | X |
| 2004 | 7 | 6.8 | |
| 2005 | 153 | 7.1 | |
| 2006 | 188 | 7.6 | |
| 2007 | 263 | 7.8 | |
| 2008 | 336 | | X |
| 2009 | 392 | | X |
| 2010 | 610 | | |

Note: Facilities counted as distinct only if in different locations.

Table 2: DHS Data: Number of respondents and clusters in each survey

| *Year* | Men | Women | Clusters |
|---|---|---|---|
| 2003 | 3,578 | 8,195 | 400 |
| 2008/2009 | 3,465 | 8,444 | 398 |
| **TOTAL** | **7,043** | **16,639** | **798** |

Table 3: Summary Statistics 1

| | 2003 | 2008/2009 |
|---|---|---|
| HIV positive | 8.49 | 7.9 |
| | (27.88) | (26.97) |
| Age | 28.14 | 28.56 |
| | (9.47) | (9.64) |
| Years of education | 6.81 | 7.53 |
| | (3.83) | (3.67) |
| Married | 55.41 | 54.87 |
| | (49.71) | (49.77) |
| Knows someone who has or died of AIDS | 75.19 | 79.37 |
| | (43.2) | (40.47) |
| Ever been tested for AIDS | 13.38 | 57.32 |
| | (34.05) | (49.47) |
| Wants to be tested for AIDS | 69.57 | 73.19 |
| | (46.01) | (44.3) |
| Facility with ARVs within 10kms | 0 | 54.51 |
| | (0) | (49.8) |
| Facility with ARVs in district | 0 | 77.14 |
| | (0) | (42) |

Note: Standard deviations in parentheses.

Table 4: Summary Statistics 2

| | 2003 | 2008/2009 |
|---|---|---|
| Ever had sex | 82.39 | 81.95 |
| | (38.09) | (38.46) |
| Ever had sex, under age 16 | 19.78 | 21.71 |
| | (39.86) | (41.25) |
| Has at least two sexual partners | 1.75 | 1.15 |
| | (13.12) | (10.68) |
| Had sex in the last 4 weeks | 47.55 | 48.85 |
| | (49.94) | (49.99) |
| Age at first intercourse | 16.58 | 16.96 |
| | (2.97) | (3.02) |
| Last intercourse used condom | 4.82 | 7.91 |
| | (21.41) | (26.99) |
| Number of partners, last 12 mos. | 68.49 | 69.84 |
| | (46.42) | (45.87) |
| Currently Pregnant | 7.98 | 7.16 |
| | (27.1) | (25.78) |
| Current unwanted pregnancy | 3.96 | 3.41 |
| | (19.49) | (18.14) |
| Ideal number of children | 3.78 | 3.7 |
| | (2.39) | (2.07) |
| Had any STD in last 12 months | 1.36 | 1.58 |
| | (11.57) | (12.47) |
| Had STD symptoms in last 12 mos. | 3.35 | 4.24 |
| | (17.99) | (20.15) |

Note: Standard deviations in parentheses.

Table 5: Comparison of ART in District and ART in 10kms

| | No ART in District | ART in District | **TOTAL** |
|---|---|---|---|
| No ART in 10kms | 121 | 164 | 285 |
| ART in 10kms | 34 | 364 | 398 |
| **TOTAL** | 155 | 528 | 683 |

Notes: Number of clusters reported in each category, across both rounds.

Table 6: Access by round: Full sample

| | Kenya | |
|---|---|---|
| | $> 10kms$ | $< 10kms$ |
| Wave 1 | 349 | 0 |
| Wave 2 | 156 | 189 |

Notes: This counts clusters of observations. The sample excludes Nairobi.

Table 7: Impact of ART on pregnancy (dependent variable= currently pregnant), No previous children

| VARIABLES | (1) ART in District | (2) ART in 10kms | (3) Grid Regions | (4) Matched | (5) Matched No Switchers |
|---|---|---|---|---|---|
| Treat*Wave2 | 8.292*** | 3.025 | 4.529 | 4.335** | 5.135** |
| | (2.814) | (2.416) | (3.788) | (2.024) | (2.018) |
| Treat | 0 | -2.999* | 0 | -3.932*** | 0 |
| | (0) | (1.752) | (0) | (1.357) | (0) |
| Wave2 | -7.812*** | -2.901 | -5.318 | -4.529*** | -5.740*** |
| | (2.521) | (1.898) | (3.520) | (1.560) | (1.668) |
| HIVprev(District) | -8.566 | 43.76 | 39.38* | 16.30 | 19.53 |
| | (35.72) | (29.06) | (20.91) | (18.48) | (23.20) |
| HIVprev(Division) | 11.29 | 10.33 | -12.87 | 16.07 | 16.74* |
| | (16.03) | (10.66) | (13.62) | (9.941) | (9.542) |
| Urban | 0.417 | -1.396 | -3.387* | -1.372 | -2.549 |
| | (1.521) | (1.424) | (1.790) | (1.500) | (1.823) |
| Urban*Wave2 | 4.359 | 6.718** | 8.503** | 6.394* | 6.600* |
| | (3.673) | (3.038) | (3.757) | (3.271) | (3.704) |
| LargeHospIn10kms | -2.304 | 1.571 | -0.479 | -0.251 | 0.201 |
| | (3.903) | (2.346) | (2.461) | (2.370) | (2.539) |
| LargeHospIn10kms*Wave2 | -3.038 | -4.380 | -5.904 | -1.139 | -1.172 |
| | (4.716) | (3.715) | (4.395) | (4.551) | (4.845) |
| NoHCin10kms | 2.839 | 3.742 | 0.548 | 4.492 | 1.007 |
| | (4.742) | (4.456) | (7.472) | (4.234) | (4.803) |
| NoHCin10kms*Wave2 | -7.071 | -6.601 | -17.19* | -8.486* | -7.443 |
| | (5.691) | (5.020) | (9.987) | (5.018) | (5.584) |
| Observations | 3,764 | 3,764 | 3,832 | 17,743 | 12,039 |
| $R^2$ | 0.111 | 0.051 | 0.116 | 0.148 | 0.148 |
| Clusters | 214 | 214 | 220 | 343 | 329 |

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Note: All estimates include controls for age and education. Columns 1 and 2 include district fixed effects and standard errors are clustered at the district level. Column 3 includes grid-square fixed effects with standard errors clustered at the square level. Columns 4 and 5 include pair fixed effects with standard errors clustered at the level of the wave 2 survey cluster. Estimates in columns 1-3 are weighted with DHS sampling weights. Estimates in columns 4 and 5 are weighted by the DHS sampling weight multiplied by the minimum of 1/10 and the inverse of the distance between the pair.

Table 8: Impact of ART on pregnancy (dependent variable= currently pregnant), interacted with previous children

| VARIABLES | (1) ART in District | (2) ART in 10kms | (3) Grid Regions | (4) Matched | (5) Matched No Switchers |
|---|---|---|---|---|---|
| Treat*Wave2 | 5.973** | 4.587* | 3.429 | 3.601* | 4.985** |
| | (2.631) | (2.589) | (3.488) | (2.125) | (2.033) |
| Treat | 0 | -3.393** | 0 | -2.557** | 0 |
| | (0) | (1.556) | (0) | (1.002) | (0) |
| Wave2 | -5.261** | -3.428* | -3.935 | -3.482** | -4.952*** |
| | (2.269) | (1.896) | (3.284) | (1.378) | (1.560) |
| PrevChild*Wave2*Treat | -3.552 | -4.735 | -6.226* | -4.029 | -5.587** |
| | (2.505) | (2.961) | (3.698) | (2.525) | (2.396) |
| PrevChild*Wave2 | 2.680 | 2.556 | 5.136 | 2.980* | 4.199** |
| | (1.907) | (1.933) | (3.338) | (1.526) | (1.793) |
| PrevChild*Treat | 1.389 | 3.593* | 4.813** | 3.128*** | 3.714*** |
| | (1.922) | (1.874) | (2.243) | (0.990) | (1.114) |
| PreviousChildren | 1.360 | 0.697 | -1.512 | 0.585 | 0.175 |
| | (1.950) | (1.885) | (2.207) | (1.020) | (1.189) |
| HIVprev(District) | 17.57 | 35.58* | 31.62*** | 24.39*** | 26.20*** |
| | (22.31) | (18.13) | (11.25) | (8.342) | (9.699) |
| HIVprev(Division) | 4.134 | -0.574 | -1.128 | 1.116 | -0.739 |
| | (7.504) | (4.484) | (6.116) | (4.681) | (5.628) |
| Urban | -1.521 | -1.515 | -2.364* | -1.873** | -2.545** |
| | (1.408) | (1.224) | (1.233) | (0.787) | (0.987) |
| Urban*Wave2 | 1.346 | 1.804 | 3.285** | 2.558** | 3.543** |
| | (1.667) | (1.424) | (1.509) | (1.225) | (1.401) |
| LargeHospIn10kms | 3.409 | 1.519 | 2.607 | 2.963** | 3.959*** |
| | (3.190) | (1.600) | (1.924) | (1.213) | (1.302) |
| LargeHospIn10kms*Wave2 | 0.717 | 1.326 | 0.686 | 0.727 | 0.295 |
| | (2.738) | (2.130) | (2.275) | (1.668) | (1.784) |
| NoHCin10kms | 1.559 | 0.839 | -0.653 | 2.845 | 2.363 |
| | (5.202) | (2.769) | (3.837) | (2.389) | (2.663) |
| NoHCin10kms*Wave2 | 1.665 | 1.130 | -0.706 | -0.0977 | 0.632 |
| | (5.024) | (2.891) | (5.695) | (2.773) | (3.090) |
| Observations | 14,227 | 14,227 | 14,473 | 66,972 | 45,555 |
| $R^2$ | 0.047 | 0.034 | 0.051 | 0.060 | 0.062 |
| Clusters | 215 | 215 | 223 | 343 | 329 |

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Note: All estimates include controls for age and education. Columns 1 and 2 include district fixed effects and standard errors are clustered at the district level. Column 3 includes grid-square fixed effects with standard errors clustered at the square level. Columns 4 and 5 include pair fixed effects with standard errors clustered at the level of the wave 2 survey cluster. Estimates in columns 1-3 are weighted with DHS sampling

Table 9: Impact of ART on pregnancy (dependent variable= currently pregnant), Not married 12 months before

| VARIABLES | (1) ART in District | (2) ART in 10kms | (3) Grid Regions | (4) Matched | (5) Matched No Switchers |
|---|---|---|---|---|---|
| Treat*Wave2 | 3.551 | 5.928** | 5.519* | 2.334* | 2.743* |
| | (4.690) | (2.523) | (2.813) | (1.410) | (1.438) |
| Treat | 0 | -0.865 | 0 | -1.204 | 0 |
| | (0) | (1.850) | (0) | (0.988) | (0) |
| Wave2 | -3.296 | -2.757* | -4.218** | -2.875*** | -3.782*** |
| | (4.417) | (1.461) | (1.877) | (1.095) | (1.221) |
| HIVprev(District) | 65.96 | 78.79*** | 8.995 | 34.83*** | 37.50*** |
| | (48.24) | (29.41) | (26.51) | (12.14) | (14.24) |
| HIVprev(Division) | 18.45 | 9.923 | 10.72 | 3.816 | -1.997 |
| | (17.87) | (10.59) | (20.60) | (5.889) | (6.873) |
| Urban | -1.850 | -0.781 | -1.803 | -2.163* | -3.467** |
| | (2.619) | (1.866) | (2.490) | (1.144) | (1.365) |
| Urban*Wave2 | 7.798*** | 0.750 | 5.574* | 5.187*** | 6.362*** |
| | (2.665) | (3.102) | (3.194) | (1.788) | (2.013) |
| LargeHospIn10kms | 5.946 | 2.396 | 4.544 | 2.427 | 3.195 |
| | (7.325) | (3.858) | (5.401) | (1.890) | (2.060) |
| LargeHospIn10kms*Wave2 | -5.962 | -1.783 | -6.332 | -0.740 | -1.187 |
| | (4.404) | (3.433) | (4.547) | (2.518) | (2.651) |
| NoHCin10kms | 4.112 | -1.887 | -1.458 | 3.970* | 2.763 |
| | (2.668) | (2.499) | (2.451) | (2.290) | (2.760) |
| NoHCin10kms*Wave2 | 4.507 | 9.887*** | 6.212 | 0.116 | 1.632 |
| | (4.051) | (3.546) | (4.301) | (2.882) | (3.381) |
| Observations | 2,235 | 2,235 | 2,275 | 39,122 | 26,590 |
| $R^2$ | 0.150 | 0.059 | 0.125 | 0.078 | 0.081 |
| Clusters | 210 | 210 | 208 | 343 | 329 |

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Note: All estimates include controls for age and education. Columns 1 and 2 include district fixed effects and standard errors are clustered at the district level. Column 3 includes grid-square fixed effects with standard errors clustered at the square level. Columns 4 and 5 include pair fixed effects with standard errors clustered at the level of the wave 2 survey cluster. Estimates in columns 1-3 are weighted with DHS sampling weights. Estimates in columns 4 and 5 are weighted by the DHS sampling weight multiplied by the minimum of 1/10 and the inverse of the distance between the pair.

Table 10: Impact of ART on pregnancy (dependent variable= currently pregnant), Interacted with married

| VARIABLES | (1) ART in District | (2) ART in 10kms | (3) Grid Regions | (4) Matched | (5) Matched No Switchers |
|---|---|---|---|---|---|
| Treat*Wave2 | 6.132* | 5.534** | 5.269* | 5.045*** | 5.192*** |
| | (3.377) | (2.335) | (2.962) | (1.874) | (1.897) |
| Treat | 0 | -1.200 | 0 | -0.935 | 0 |
| | (0) | (1.719) | (0) | (1.041) | (0) |
| Wave2 | -3.576 | -1.526 | -2.776 | -1.524 | -2.006 |
| | (3.007) | (1.417) | (2.493) | (1.112) | (1.244) |
| MarriedLY*Wave2*Treat | -5.793 | -5.267** | -6.697** | -6.663*** | -6.774*** |
| | (4.150) | (2.581) | (2.999) | (2.411) | (2.467) |
| MarriedLY*Wave2 | 2.393 | 0.860 | 3.286 | 2.141 | 2.678 |
| | (3.749) | (1.798) | (2.525) | (1.552) | (1.738) |
| MarriedLY*Treat | 3.433 | 1.307 | -0.473 | 2.308** | 3.242*** |
| | (2.589) | (1.825) | (2.077) | (0.937) | (1.144) |
| MarriedLastYear | 1.298 | 3.250** | 4.512** | 2.095*** | 1.652* |
| | (2.390) | (1.335) | (1.749) | (0.721) | (0.866) |
| HIVprev(District) | 42.54* | 44.12** | 15.25 | 20.56** | 28.63** |
| | (22.55) | (19.23) | (12.60) | (9.765) | (12.56) |
| HIVprev(Division) | -1.901 | -2.688 | 4.219 | -0.192 | -1.227 |
| | (8.754) | (5.771) | (7.552) | (5.782) | (6.823) |
| Urban | -1.134 | -0.107 | -1.484 | -0.805 | -0.574 |
| | (1.624) | (1.298) | (1.343) | (0.926) | (1.168) |
| Urban*Wave2 | 0.798 | -1.482 | 0.908 | -0.178 | 0.231 |
| | (1.587) | (1.622) | (1.774) | (1.518) | (1.716) |
| LargeHospIn10kms | 4.061 | 1.249 | 3.205* | 3.147** | 4.000*** |
| | (3.308) | (1.738) | (1.840) | (1.388) | (1.520) |
| LargeHospIn10kms*Wave2 | 2.114 | 4.429* | 2.571 | 0.789 | 0.672 |
| | (2.808) | (2.617) | (2.970) | (1.987) | (2.059) |
| NoHCin10kms | -0.100 | 1.427 | -2.388 | 1.636 | 1.834 |
| | (6.176) | (2.770) | (3.018) | (2.473) | (2.644) |
| NoHCin10kms*Wave2 | 3.684 | 2.929 | 1.918 | 0.585 | 0.927 |
| | (5.850) | (3.090) | (3.856) | (2.837) | (3.056) |
| Observations | 8,213 | 8,213 | 8,349 | 38,652 | 26,468 |
| $R^2$ | 0.065 | 0.044 | 0.070 | 0.090 | 0.089 |
| Clusters | 215 | 215 | 223 | 343 | 329 |

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Note: All estimates include controls for age and education. Columns 1 and 2 include district fixed effects and standard errors are clustered at the district level. Column 3 includes grid-square fixed effects with standard errors clustered at the square level. Columns 4 and 5 include pair fixed effects with standard errors clustered at the level of the wave 2 survey cluster. Estimates in columns 1-3 are weighted with DHS sampling

Table 11: Impact of ART on ideal number of kids (dependent variable= ideal number of kids), No previous children

| VARIABLES | (1) ART in District | (2) ART in 10kms | (3) Grid Regions | (4) Matched | (5) Matched No Switchers |
|---|---|---|---|---|---|
| Treat*Wave2 | 0.183 | -0.117 | -0.0685 | -0.0176 | -0.0402 |
| | (0.165) | (0.122) | (0.266) | (0.0880) | (0.0919) |
| Treat | 0 | -0.0926 | 0 | -0.169** | 0 |
| | (0) | (0.104) | (0) | (0.0685) | (0) |
| Wave2 | -0.121 | 0.0566 | 0.0775 | 0.0178 | 0.0567 |
| | (0.160) | (0.0940) | (0.255) | (0.0723) | (0.0797) |
| HIVprev(District) | 1.267 | 2.111 | 2.243** | 2.208*** | 2.549** |
| | (2.714) | (2.465) | (0.957) | (0.748) | (0.995) |
| HIVprev(Division) | -1.450* | -1.468** | -1.580** | -1.761*** | -1.588*** |
| | (0.862) | (0.575) | (0.670) | (0.399) | (0.486) |
| Urban | -0.0518 | -0.110 | -0.0201 | -0.00828 | 0.00609 |
| | (0.149) | (0.126) | (0.132) | (0.0702) | (0.0754) |
| Urban*Wave2 | -0.527** | -0.252* | -0.389* | -0.261** | -0.265* |
| | (0.207) | (0.150) | (0.198) | (0.128) | (0.138) |
| LargeHospIn10kms | 0.0647 | -0.0677 | -0.0921 | -0.119 | -0.0819 |
| | (0.278) | (0.150) | (0.171) | (0.107) | (0.121) |
| LargeHospIn10kms*Wave2 | 0.00806 | -0.0821 | -0.0965 | 0.0420 | 0.0192 |
| | (0.245) | (0.200) | (0.202) | (0.140) | (0.148) |
| NoHCin10kms | 1.991** | 1.571** | 2.225*** | 1.692*** | 1.709** |
| | (0.841) | (0.672) | (0.728) | (0.610) | (0.745) |
| NoHCin10kms*Wave2 | -2.085** | -1.589** | -3.124*** | -2.001*** | -2.139*** |
| | (0.824) | (0.642) | (0.677) | (0.574) | (0.619) |
| Observations | 6,547 | 6,547 | 6,652 | 30,980 | 21,037 |
| $R^2$ | 0.262 | 0.213 | 0.265 | 0.216 | 0.220 |
| Clusters | 215 | 215 | 221 | 343 | 329 |

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Note: All estimates include controls for age and education. Columns 1 and 2 include district fixed effects and standard errors are clustered at the district level. Column 3 includes grid-square fixed effects with standard errors clustered at the square level. Columns 4 and 5 include pair fixed effects with standard errors clustered at the level of the wave 2 survey cluster. Estimates in columns 1-3 are weighted with DHS sampling weights. Estimates in columns 4 and 5 are weighted by the DHS sampling weight multiplied by the minimum of 1/10 and the inverse of the distance between the pair.

Table 12: Parallel trends, 10 years earlier, No previous children

| VARIABLES | (1) ART in District | (2) ART in 10kms | (3) Grid Regions | (4) Matched | (5) Matched No Switchers |
|---|---|---|---|---|---|
| Treat*Wave2 | 0.747 | -0.702 | 0.972 | 0.190 | 0.253 |
| | (1.151) | (0.912) | (1.654) | (0.968) | (0.946) |
| Treat | 0 | -0.745 | 0 | -0.631 | 0 |
| | (0) | (0.785) | (0) | (0.696) | (0) |
| Wave2 | -1.996** | -0.587 | -2.216 | -1.593** | -1.686** |
| | (0.971) | (0.673) | (1.593) | (0.718) | (0.799) |
| HIVprev(District) | 33.68** | 27.33** | 8.879 | 12.36** | 14.44** |
| | (13.86) | (11.42) | (7.775) | (6.170) | (7.134) |
| HIVprev(Division) | -14.23*** | -6.801 | -6.724 | -3.107 | -0.485 |
| | (5.039) | (4.244) | (5.516) | (4.285) | (5.547) |
| Urban | -1.305 | -0.792 | -0.338 | -0.249 | 0.611 |
| | (0.926) | (0.820) | (0.922) | (0.771) | (0.935) |
| Urban*Wave2 | 2.722** | 1.912* | 2.240* | 2.766 | 2.694 |
| | (1.356) | (1.137) | (1.279) | (2.172) | (2.753) |
| LargeHospIn10kms | 0.467 | 0.101 | -0.0787 | -1.349 | -1.782 |
| | (1.779) | (1.228) | (1.383) | (1.248) | (1.583) |
| LargeHospIn10kms*Wave2 | 1.856 | 2.396 | 1.342 | 1.208 | 1.155 |
| | (1.867) | (1.646) | (1.784) | (2.150) | (2.477) |
| NoHCin10kms | -5.037 | -0.898 | -2.547 | -1.777 | -0.389 |
| | (3.828) | (1.347) | (4.956) | (1.618) | (1.294) |
| NoHCin10kms*Wave2 | 4.311 | -0.786 | 1.401 | 0.368 | 0.839 |
| | (3.288) | (1.667) | (5.503) | (1.858) | (1.839) |
| Observations | 6,777 | 6,777 | 6,898 | 31,662 | 21,537 |
| $R^2$ | 0.056 | 0.030 | 0.054 | 0.079 | 0.081 |
| Clusters | 215 | 215 | 223 | 343 | 329 |

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Note: All estimates include controls for age and education. Columns 1 and 2 include district fixed effects and standard errors are clustered at the district level. Column 3 includes grid-square fixed effects with standard errors clustered at the square level. Columns 4 and 5 include pair fixed effects with standard errors clustered at the level of the wave 2 survey cluster. Estimates in columns 1-3 are weighted with DHS sampling weights. Estimates in columns 4 and 5 are weighted by the DHS sampling weight multiplied by the minimum of 1/10 and the inverse of the distance between the pair.