The Effect of HIV Infection Risk Beliefs on Risky Sexual Behaviors: Scared Straight or Scared to Death?

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November 2014

Abstract

Economists typically assume that risk compensation is uniformly self-protective – that people become more careful as the health risks of their actions increase. However, risk-seeking, or fatalistic, responses can also be rational: increased risks can lead people to take fewer precautions. I extend the typical model of risk compensation to show that fatalism is a rational response to sufficiently high risks if people do not have perfect control over all possible exposures, and if the condition in question is irreversible. This result holds even for people who do not understand how to add up probabilities. I test this model’s implications by randomizing the provision of information on HIV transmission risks to people in Malawi, a country with a severe HIV epidemic where there is qualitative evidence of fatalistic responses to the virus. Average risk responses are self-protective and statistically significant, but small in magnitude: the mean risk elasticity of sexual behavior is roughly -0.6. To test the model of rational fatalism, I develop a method of decomposing 2SLS estimates of the risk elasticity of sexual behavior by baseline risk beliefs. Consistent with the predictions of my theoretical framework, I find that this elasticity varies sharply by baseline risk beliefs: the risk elasticity varies from -2.3 for the lowest initial beliefs to 2.9 for the highest initial beliefs. 13.8% of the population has a positive elasticity, suggesting they are fatalistic.

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I am grateful to Rebecca Thornton, Jeff Smith, John DiNardo, David Lam, Matías Cattaneo, Bob Willis, Mel Stephens, John Bound, Miles Kimball, Susan Watkins, Pinar Keskin, Daniel Bennett, Nate Young, Sanyu Mojola, Ophira Vishkin, Evan Herrnstadt, Joe Golden, Audrey Dorélien, Laura Derksen, Eric Chyn, Olga Malkova, Justin Ladner, and seminar participants at the University of Michigan, PAA, MIEDC, ABCDE, and NEUDC for their valuable feedback. This project would not have been possible without the excellent fieldwork supervision efforts of Anderson Moyo, Synab Njerenga, and Christopher Nyirenda. Data collection on this project was supported by internal UM grants from the Center for Global Health, the Population Studies Center, the Weinberg International Travel Fund, the Center for Education of Women, and the Michigan Institute for Teaching and Research in Economics as well as the Russell Sage Foundation’s Small Grants in Behavioral Economics program. I acknowledge fellowship support from the Population Studies Center and the Rackham Graduate School. All errors and omissions are my own.
1 Introduction

Risk compensation is central to our understanding of how people make decisions about protecting their health. Beginning with the seminal Peltzman (1975) paper on automobile safety regulation, economists have realized that a decline in the risk associated with a particular behavior is often offset by a rational increase in risk-taking. Empirical research on risk compensation typically assumes that people are uniformly risk-avoiding. When the per-act risk of an activity goes up, people are presumed to take fewer chances, a pattern that can be described as “self-protective.” This paper considers the possibility that for some people rational responses to health risks are instead risk-seeking, or “fatalistic” – that the optimal choice may be to increase one’s risk-taking when the per-act risk rises.

Risk-avoiding behavior is always rational if the expected cost of risk-taking can be approximated as a linear function of the per-act risk.¹ In this paper I show that fatalistic behavior is rational if a) the linear approximation is replaced with any reasonable function that is bounded above by a 100% chance of the negative outcome occurring and b) the per-act risk is sufficiently high.² This happens because an increase in the per-act risk (in my example, the risk of contracting HIV) affects not only the marginal cost of the acts the agent is deciding over, but also a stock of previously-chosen acts over which one no longer has any control. If the per-sex-act risk of contracting HIV rises, this raises the marginal cost of additional sex acts, by increasing the chance that they will lead to HIV infection. But it also increases the probability that the agent already has HIV, which decreases the marginal cost of more risky sex. When the second effect dominates, increases in perceived risks will lead to more risk-taking rather than less. Furthermore, if people cannot perfectly avoid all future exposures to HIV – for example, because condoms sometimes break – then unpreventable future exposures can also drive fatalistic behavior, and HIV testing will not prevent people from becoming fatalistic.³ This mechanism is conceptually similar to the one that drives models of rational habit formation. In Becker and Murphy (1988), for example, past consumption has a large effect on current choices by changing the marginal utility of consumption. In my model, the linkage across periods operates through the marginal cost of consumption instead.

This theory of rationally fatalistic behavior suggests that people with sufficiently high beliefs about the risks of their behaviors, and imperfect control of their entire risk-taking history, will tend to become fatalistic, because they feel that they are doomed irrespective of what they do. I test this implication using data from field experiment that I conducted in Southern Malawi, an area with a severe HIV epidemic. It is also an area where qualitative evidence suggests that some

¹This model is explicit in Oster (2012), but is used implicitly in many empirical analyses which restrict the relationship between risks and behavior to be linear and therefore monotonic.
²This result does not depend on agents using the true expected cost, which is based on the binomial CDF. A number of theoretical papers (Kremer 1996; O’Donoghue and Rabin 2001; Sterck 2013) have used the assumption that agents can compute the true expected cost to make the point that rational responses to increased risks will be fatalistic rather than self-protective for certain individuals.
³This basic idea relies on the condition in question being irreversible: if the disease in question can easily be cured, risks will not continue to aggregate because the probability of infection will reset to zero after it reaches one.
people are responding fatalistically to the virus\textsuperscript{4}, and where HIV prevention education emphasizes that the risk of contracting HIV is extremely high (see Figure 1). The experiment recruited 1292 respondents from 70 villages, and randomly assigned the respondents from 35 of the villages to be taught medically-accurate information about HIV transmission risks. A baseline survey was conducted prior to the information treatment, followed by an endline survey four to twelve weeks later.

The randomized information treatment substantially decreased people’s beliefs about the risks of unprotected sex: at the endline survey, the average person in the treatment group believed the risk of HIV transmission from unprotected sex with an infected partner was 33\% per sex act, as opposed to 74\% in the control group.\textsuperscript{5} Using the experimental treatment as an instrumental variable, I estimate that the risk belief elasticity of sexual activity is small but statistically significant at about -0.6. This elasticity estimate is larger than those found in studies that measure the response of sexual behavior to the actual prevalence of HIV in sub-Saharan Africa, and comparable to estimates for the United States. However, because people do not accurately know the true prevalence of the virus, the implied prevalence elasticity from my results would be smaller, and could be consistent with previous estimates for Africa.

This estimated mean elasticity follows the literature in assuming that the risk term enters linearly into the regression function.\textsuperscript{6} This assumption is consistent with monotonically self-protective responses to risks. A model of rationally fatalistic behavior, however, implies that risk responses are non-monotonic, and so a linear regression is misspecified. I therefore examine whether responses to the information treatment are heterogeneous by people’s baseline (pre-treatment) beliefs. I find that people with initially low risk beliefs respond self-protectively to the new information (which lowers their risk beliefs), while people with initially high risk beliefs respond fatalistically. This is the same non-monotonic pattern of responses predicted by a model of rational fatalism; the results reject the typical model of monotonically self-protective responses to risks. I can rule out that this heterogeneity is due to correlations between beliefs and other respondent attributes, and find no other factors that cause statistically-significant heterogeneity.

Having demonstrated that the estimated mean elasticity from simple 2SLS is misspecified, I develop a method for decomposing instrumental-variables estimates by exogenous covariates and show that this method gives consistent estimates of the underlying conditional parameter. This approach reveals that the risk elasticity of sexual behavior varies substantially across the population, from -2.3 for the lowest initial risk beliefs to 2.9 for the highest initial beliefs; 13.8\% of the population has a positive elasticity. The fatalistic group has higher-than-average risk factors for HIV, such as years of sexual experience and perceived HIV-positive status. This suggests that they may be more important in driving the overall prevalence of HIV. Therefore, the effect of the status quo policy – in

\textsuperscript{4}E.g. Kaler (2003), Kaler and Watkins (2010)
\textsuperscript{5}People in Malawi greatly overestimate how easily HIV is transmitted: the actual rate is just 0.1\%.
\textsuperscript{6}An extreme example is the Viscusi (1990) study of cancer risk perceptions and smoking behavior, which employs one-sided rather than two-sided t-tests. This eliminates any possibility of fatalistic responses, although Viscusi’s estimated standard errors are small enough that this assumption does not affect inference.
which health educators encourage people to greatly overestimate HIV transmission risks, for their
own good – is ambiguous from both an ethical and an epidemiological standpoint. More generally,
these results militate against programs that attempt to “scare people straight” via messages that
emphasize that risks are extremely high – especially when they actually are not.

This paper contributes to four bodies of research in economics. First, it builds on our under-
standing of risk compensation by providing what I believe to be the first experimental evidence
on the elasticity of risk-taking behavior with respect to perceived risks. Moreover, it shows that
that elasticity cannot be meaningfully summarized by a population average, because the subgroup
of the population with the highest baseline risk beliefs may respond positively (fatalistically) to
risks. This implies that future empirical work on risk compensation should take into account the
possibility of non-monotonicity.

Second, it contributes to a growing empirical literature that studies how people’s subjective
expectations affect their behavior. Expectations have long played an important role in economic
models, but recent research has shown that it is possible to collect meaningful information on
people’s subjective expectations both in the developed world (e.g. Lillard and Willis 2001, Manski
2004) as well as in developing countries (e.g. Attanasio 2009; Delavande, Giné and McKenzie 2011;
Delavande 2014). I provide the first experimental evidence that subjective expectations about risks
have a measurable, causal effect on people’s behavior, lending credence to the broader idea that
we should be asking people about their subjective beliefs rather than assuming they know the true
probabilities of events.

Third, it helps reconcile the substantial responses to HIV risks found in America (Ahituv,
Hotz and Philipson 1996) with very small ones in Africa (Oster 2012). Self-protective responses
by the majority of people may be offset by opposite-signed, fatalistic responses by a subset of
the population, yielding an average response that is self-protective but low in magnitude. This
is particularly plausible because gay men in the US perceive the prevalence of HIV to be much
lower than Africans do (White and Stephenson 2014). The same reasoning may also help explain
why recent field experiments in Africa have found large responses to relative HIV risk information
for specific population groups despite the fact that overall risk responses appear to be small in
magnitude.7

Fourth, it also helps explain the small measured responses of sexual behavior to HIV testing.
As Philipson and Posner (1993) point out, the effect of learning one’s HIV status is theoretically
ambiguous, because learning that you are HIV-positive can have two opposite-signed effects on
your behavior. Purely self-interested people should see little or no marginal cost from further risky
sex if they are already infected, while altruistic people would want to take measures to protect
their prospective partners. A parallel logic applies to those who learn they are HIV-negative.
Experimental research on HIV testing has found fairly small responses: Thornton (2008) finds zero
average effects for HIV-negatives and very small average reductions in risk-taking for HIV-positives

7Godlonton, Munthali and Thornton (2014) find that uncircumcised men in Malawi take fewer sexual risks when
they are told that circumcised men face a lower risk of HIV infection. In a study in Kenya, Dupas (2011) finds that
girls in secondary school choose younger partners when they are told that older partners are riskier.
in Malawi. One possible explanation for the small responses she finds for people who test HIV-positive is that HIV-positive people are altruistic, but some of them are fatalistic about transmitting the virus to their sex partners, leading to small effects on average. Gong (2014), studying people in urban Kenya, finds that responses to HIV testing vary by people’s priors about their HIV status; this is conceptually similar to the logic of my paper, except that I focus on heterogeneous responses by people’s perceptions of transmission risks rather than perceptions of their own HIV status.

The remainder of this paper is organized as follows: I begin in Section 2 by laying out a model of responses to risks that extends the typical approach to allow for the possibility of rational fatalism, showing that under very general conditions people may rationally respond to high perceived risks fatalistically (as opposed to self-protectively). In Section 3, I describe a randomized field experiment that I conducted in Southern Malawi to test the implications of this model, as well as the data on risk beliefs and sexual risk-taking that I rely on. Section 4 lays out my empirical strategy and results, and in Section 5 I address the mechanisms behind my results, address some potential limitations of this paper, and discuss the implications of my findings for the design of HIV prevention policies. Section 6 concludes. All appendix material can be found in the Online Appendix to the paper.\(^8\)

2 Theoretical Framework

This section outlines a model of behavioral responses to HIV risks that relaxes a key assumption made by the previous empirical literature. Most empirical work on responses to risks, HIV or otherwise, relies on the assumption that the stochastic cost of risk-taking is linear in the riskiness of each individual act. My model allows that cost to follow a concave shape that asymptotes to a probability of 1, which is consistent with the risks of individual sex acts adding up into a sensible total probability of HIV infection. The core result is that the comparative static in question – the derivative of risk-taking with respect to per-act risks – is not always negative, or self-protective. In general, the sign of the comparative static will flip from negative to positive if an agent’s risk beliefs and stock of unavoidable risks are sufficiently high. This happens because the marginal cost of risk-taking will approach zero as the total chance of HIV infection gets close to 100%.

2.1 Model Basics

In this model, I assume that agents weigh the benefits of choosing a level of risky sex, \(y\), against its costs. These costs include both a fixed per-act cost (which could be a pecuniary cost but also time cost) \(q\), and a stochastic component due to the risk of HIV infection. An agent’s perceived risk per sex act is \(x\). The expected cost of HIV infection is the agent’s subjective belief about the total probability of it occurring, \(P\), times its perceived cost, \(c\). The subjective probability can be written as a continuously differentiable function \(P = P(x, n)\), where \(n = y + m_0 + m_1\) is the total number of sex acts, including both the current choice \(y\), and an immutable stock of acts \(m_0 + m_1\). This stock includes all previous sex acts since one’s most recent HIV test, \(m_0\), and also all future

\(^8\)http://www-personal.umich.edu/~jtkerwin/Papers/JMP/Kerwin_JMPAppendix_Latest.pdf
risky acts that are unavoidable, $m_1$. The latter captures accidental exposures through things like condom breakage, situations where an agent may lack the bargaining power to turn down some future sex acts, imperfect self control, and so forth.

Throughout the model I will treat HIV infection as irreversible, so that all risky acts aggregate into a single probability $P$. This is true of HIV if we consider fatalism to be driven only by inevitable future exposures, or if testing is unavailable. It will only hold for certain other risks, and depends on perceived rather than actual irreversibility of the condition. For example, if people perceive lung cancer to be a binary and irreversible condition, the model results will go through, but if a condition is widely known to be curable, such as Chlamydia, then they will not. It is possible to compute the actual value of $P$ using the binomial distribution, but my results will be robust to agents potentially not understanding how to correctly compute probabilities. The benefit of sex acts is described by a continuously differentiable benefit function, $B(y)$, with positive and diminishing marginal benefits.

To focus the exposition on the mechanism that drives fatalistic risk responses, rather than on mathematical derivations, I model the agent’s choice as a one-shot, static decision. This collapses the future into the expected cost of HIV infection $P(x, y + m_0 + m_1)c$. The results in this section can be generalized to a multi-period setting – see Appendix I-D for details. The single-period optimization problem is:

$$\max_{y \geq 0} \{U(y; x, m_0, m_1, q, c)\} = \max_{y \geq 0} \{B(y) - qy - P(x, y + m_0 + m_1)c\}$$

(1)

By the assumption that $y$ is continuous, the maximand $U(n; m_0, m_1, p, c, x)$ is a sum of continuously differentiable functions and therefore continuously differentiable itself.

I do not assume that agents can correctly convert levels of risk-taking and per-act risks into an aggregate probability of HIV infection. Instead, I simply assume that $P(x, y + m_0 + m_1)$ corresponds to sensible probabilities: it must lie between 0 and 1, and be equal to zero if either sex is risk-free ($x = 0$) or an agent engages in no risky sex ($y + m_0 + m_1 = 0$). I also assume that higher riskiness $x$ is in fact interpreted as leading to a higher subjective probability of HIV infection, and more risk-taking $y + m_0 + m_1$ also increases the chance of contracting HIV.\(^9\) The subjective probability also approaches 1 as riskiness rises toward 1 or as total risk-taking goes to infinity.\(^10\)

The model formulated above is similar in spirit to those used in the literature on rational habit formation and addiction. In Becker and Murphy (1988), consumption choices are linked across periods by the effect of past consumption on the marginal benefit of current-period consumption. In this model, both past and future consumption of the risky act have a large impact on the marginal cost of current risk-taking. This analogy is made even more clear by the multi-period formulation of the model in Appendix I-D.

For most possible functional forms of $B(\cdot)$ and $P(\cdot, \cdot)$ this optimization problem will have no

\(^9\)Formally, $P_1 \geq 0$, with $P_1(0, y + m_0 + m_1) > 0$ if $y + m > 0$ and $P_1(x, 0) = 0$; $P_2 \geq 0$, with $P_2(x, 0) > 0$ if $x > 0$ and $P_2(0, y + m_0 + m_1) = 0$.

\(^10\) $P \to 1$ as $y + m \to \infty$ as long as $x > 0$, and $P = 1$ if $x = 1$ and $y + m_0 + m_1 \neq 0$. 

closed-form solutions for the optimal number of sex acts \( y^* \). However, there must be some interior solution as long as the marginal benefit of risky sex outweighs the costs for at least one act, and approaches zero as \( y \to \infty \). A sufficient condition for the existence of interior optima is that \( q > 0 \), so there is some fixed price or time cost to risky sex.\(^{11}\)

### 2.2 Comparative Statics

Given the existence of an interior solution, we are interested in a specific comparative static: how does risk-taking \( y^* \) respond to a change in the per-act risk \( x \)? I derive the properties of \( \partial y^*/\partial x \) using the implicit function theorem. For an interior solution, the optimal number of sex acts \( y^* \) must satisfy the following first- and second-order conditions:

\[
\begin{align*}
B'(y^*) - q - P_2(x, y^* + m_0 + m_1)c & = 0 \quad (2) \\
B''(y^*) - P_{22}(x, y^* + m_0 + m_1)c & \leq 0 \quad (3)
\end{align*}
\]

The first-order condition in equation 2 is a function \( G(y^*, x, m_0, m_1, q, c) = 0 \). Therefore the implicit function theorem allows us to compute the comparative static for changes in \( y^* \) in response to changes in \( x \):

\[
\frac{\partial y^*}{\partial x} = -\frac{\partial G}{\partial y^*} = \frac{P_{21}(x, y^* + m_0 + m_1)c}{B''(y^*) - P_{22}(x, y^* + m_0 + m_1)c} \quad (4)
\]

The denominator is just the left-hand side of the second-order condition, and is thus negative.\(^{12}\) Since \( c > 0 \), \( \text{sign}(\partial y^*/\partial x) = -\text{sign}(P_{21}(x, y^* + m_0 + m_1)) \). It is typical in the literature to approximate \( P \) by a linear function, \( P(x, y + m_0 + m_1) \approx x(y + m_0 + m_1) \). This is done explicitly in Oster (2012) and implicitly by Viscusi (1990), for example. In that case \( P_{21} = 1 > 0 \) always, so \( \partial y^*/\partial x < 0 \). This is analogous to the Oster (2012) result that sexual activity should fall as the prevalence of HIV rises. More broadly, it says that behavior is uniformly self-protective: people always choose fewer risky acts as the per-act risk of each act rises.

However, the linear approximation does not satisfy the requirements for being a sensible probability laid out in Section 2.1. For low values of \( x \) and \( y + m_0 + m_1 \) this is not an issue, since \( P \) will lie between 0 and 1. In the context of HIV risk beliefs, however, \( x \) is often quite high, since perceived risks are typically large overestimates, and \( m_0 + m_1 \) will reflect a potentially long sexual history and an extensive future of possible condom failures and so forth. These can easily push the linear approximation above 1, which is obviously wrong. One way of imposing sensible probabilities on \( p \) is to use the true probability function \( P = \pi(x, y + m_0 + m_1) = 1 - (1 - x)^{y + m_0 + m_1} \). O’Donoghue and Rabin (2001) point out that for this function, \( \pi_{12} = (1 - x)^{y + m_0 + m_1 - 1}[1 + y + m_0 + m_1 \ln(1 - x)] \), and hence \( \pi_{12} > 0 \) if \( y + m_0 + m_1 < 1/[-\ln(1 - x)] \) and \( \pi_{12} < 0 \) if \( y + m_0 + m_1 > 1/[-\ln(1 - x)] \).

\(^{11}\)See Appendix I-A for a proof.

\(^{12}\)Technically it is only weakly negative since the second-order condition is a weak inequality. The discussion that follows assumes strict negativity, since otherwise \( \partial y^*/\partial x \) is undefined. However, all the results in this section will hold as the second-order condition approaches 0 from above.
In words, $P_{12}$ is not constant in sign, but shifts from positive to negative if $x$ rises above a point defined by the total number of risky acts $y + m_0 + m_1$. This then implies that the sign of $\partial y^*/\partial x$ will shift from negative to positive when it crosses that tipping point.

This result is not specific to relying on the true function $\pi(x, y + m_0 + m_1)$ but is true for any function $P(x, y + m_0 + m_1)$ that satisfies the basic conditions laid out in Section 2.1. I prove this fact formally in Appendix I-B, but can readily be understood from the conceptual illustration in Figure 2. The horizontal axis shows the number of risky acts chosen, while the vertical axis shows the total subjective probability of contracting HIV. The dashed blue line shows the relationship between $P$ and $y + m_0 + m_1$ for a low perceived per-act risk $x$, and the solid red line shows the relationship for a higher value of $x$. Consistent with the basic rules of sensible probabilities, and also with the linear approximation used in most empirical research on risk responses, the slope of the red line is initially higher. When sex is riskier, the total probability of contracting HIV initially rises faster for the same number of sex acts. But the total probability is capped at one, so there must be some point above which the slope of the red line is lower than that of the blue line.\footnote{The results here technically rely on $P(x, y + m_0 + m_1)$ being continuous, but as discussed in Appendix I-C it is possible to reach similar conclusions even if people use heuristic methods for aggregating risks into total probabilities that are not continuous.}

Formally, this can be written as follows:

**Proposition 1 (Tipping point in $P_{21}$)**

$$\exists \tilde{x} = \tilde{x}(y + m_0 + m_1) \text{ s.t. } P_{21}(x, y + m_0 + m_1) > 0 \text{ if } x < \tilde{x} \text{ and } P_{21}(x, y + m_0 + m_1) < 0 \text{ if } x > \tilde{x}$$

Recall that part of the total level of risk-taking is tied up in $m_0 + m_1$, which is out of the agent’s control. It is useful to think about this as including the agent’s sexual history (in a context where HIV testing is unavailable, for example), but it also contains all future risks that the agent cannot avoid. To fix concepts, suppose that everyone thinks that they will experience at least one condom break some time in the future, so $m_1 \geq 1$. For $m_1 = 1$, and using the true function $\pi(x, y + m_0 + m_1)$, the tipping point occurs at $x = 0.63$. This is extremely high compared with the actual per-unprotected-act risk of contracting HIV from a randomly-selected partner, but it is not particularly high compared with the subjective beliefs expressed by people in Malawi. At baseline, 28\% of my sample believed the risk was at least that high.

If we maintain the assumption that sexually active adults cannot eliminate all possible exposures to HIV (so $m_0 + m_1 \geq 1$ in general), this eliminates the possibility of a corner solution where $y + m_0 + m_1 = 0$, and guarantees that the tipping point value $\tilde{x}$ that changes the sign of $P_{12}$ from positive to negative will be somewhere below 1. Proposition 1 then implies that $\partial y^*/\partial x$ will itself have a tipping point:

**Proposition 2 (Tipping point in comparative static $\partial y^*/\partial x$)**

$$\exists \tilde{x} = \tilde{x}(y + m_0 + m_1) \text{ s.t. } \frac{\partial y^*}{\partial x} < 0 \text{ if } x < \tilde{x} \text{ and } \frac{\partial y^*}{\partial x} > 0 \text{ if } x > \tilde{x}$$
Below the threshold value of the per-act HIV infection risk \( \tilde{x} \), rational agents will behave self-protectively (reducing their risk-taking in response to increased risks); above \( \tilde{x} \) they will behave fatalistically (increasing their risk-taking in response to increased risks).

This result is somewhat counterintuitive, but it captures a fairly simple logical conclusion: if the risks are sufficiently high and I can’t totally avoid exposure, there is no value to limiting how much sex I have; I am doomed no matter what. It is a purely rational alternative to the psychologically-driven fatalism derived by Caplin (2003). In his model, agents do not compensate away from extremely high risks because not responding lets them ignore the problem and thereby avoid the stress and fear associated with it. In my model, agents do not compensate away from extremely high risks because the perceived marginal benefit of abatement is nearly zero.

This sort of rationally fatalistic response is a potential issue for a wide range of decisions. Anti-smoking campaigns, to take one example, often feature “Benefit Timelines” that emphasize the health benefits that accrue to ex-smokers 20 minutes after quitting, 24 hours, 3 months, and so forth (e.g. National Health Service 2013). These timelines can be understood as a way to combat the possibility that smokers will think they are doomed to eventual cancer, no matter what they now decide. Similar to the benefit timelines in logic, HIV prevention messaging targeted at HIV-positive people emphasizes the risk of “reinfection” with a different strain of HIV (e.g. Cichocki 2014). Actual cases of reinfection are rare enough that the medical importance of this possibility is unclear (Smith, Richman and Little 2005), but one goal of this kind of messaging is to avoid a rise in risky sex by selfishly rational people who believe they have nothing to lose. Indeed, there is suggestive evidence that fatalistic reasoning about HIV infection is important in sub-Saharan Africa’s HIV epidemic (Barnett and Blaikie 1992; Kaler 2003; Kaler and Watkins 2010; Wilson, Xiong and Mattson 2014).

It is possible to extend Proposition 2 to account for altruistic behavior on the part of people who know they are HIV-positive, and may choose to be careful to protect their sex partners. In this case, there is no stochastic personal cost of risky sex, and \( P(x, y + m_0 + m_1) \) can instead be interpreted as the total subjective probability of infecting one’s partner given a perceived risk \( x \) and total risk-taking \( y + m_0 + m_1 \). \( c \) is then the extent to which agents care about their partners avoiding HIV. All the same results then go through: for relatively low values of perceived risks and low levels of risk-taking, agents will respond to rises in the per-act risk by reducing how much sex they have, but when the risks are sufficiently high they give up, assuming their partner is either already infected or doomed to infection in the future.

One consequence of Proposition 2 is that the linear relationship between \( x \) and \( y^* \) typically estimated in empirical analyses of risk responses may be misspecified, since \( y^* \) is in general a non-monotonic function of \( x \). Estimated average partial effects of \( x \) on \( y^* \) will in general include both positive and negative ranges of \( \partial y^* / \partial x \), which will tend to push the average toward zero. They will also ignore potentially-crucial heterogeneity in the effect of risk beliefs on risk-taking behavior. In my empirical analysis in Section 4, I explicitly examine risk responses for heterogeneity by initial
beliefs.

3 Data and Experimental Design

This section outlines the data and experiment that I use to test the model laid out in Section 2. I begin by describing the randomized field experiment that I conducted in Southern Malawi to collect data on how individuals’ sexual behavior responds to changes in their beliefs about HIV infection risks. I then describe my preferred measures of sexual risk-taking, which come from retrospective sexual diaries collected as part of the survey. Throughout this paper, I use the word “sex” to refer to heterosexual vaginal intercourse. Other forms of sexual activity are extremely uncommon in Malawi and are potentially sensitive topics (cf. Kerwin, Thornton and Foley 2014), so they were not included in the survey. Finally, I discuss my measures of beliefs about HIV infection risks.

3.1 Experimental Design

This paper uses data from a field randomized controlled trial I conducted from August to December 2012. The experiment took place in Traditional Authority (TA) Mwambo, in the Zomba District of Malawi’s Southern Region. I sampled roughly 30 sexually active adults aged 18-49 from each of 70 villages. Each participant was interviewed twice: once for a baseline survey, and again for an endline survey conducted 1-3 months later. At the end of the baseline survey, all participants were provided with basic information about the sexual transmission of HIV and the benefits of condoms. Participants from half of the villages, chosen at random, were assigned to the treatment group. They were read an information script that presented the actual annual risk of HIV transmission in serodiscordant couples that have unprotected sex, based on estimates from Wawer et al. (2005) and also figures from the Malawi National AIDS Commission.

The village sample for the study was constructed from the Malawi National Statistics Office GIS files for the 2008 Census. I began by removing all duplicate village entries from the dataset. Because existing evidence indicates that fatalistic responses to HIV risks and risky sexual activity may be concentrated around major trading centers (Kaler 2003), I then constructed sampling strata based on the distance to the closest major trading center. 24 of the sampled villages (34%) were within 2 km of a trading center; another 24 (34%) were within 2 and 5 km from a trading center; 24 of the sampled villages (34%) were within 2 km of a trading center; another 24 (34%) were within 2 and 5 km from a trading center;

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14Knowledge of the basics of HIV transmission and prevention is already high in this population. In the 2010 DHS, nearly 100% of individuals said that HIV was sexually transmitted and over four fifths knew that condoms were effective prevention (Malawi National Statistical Office and ORC-MACRO 2010).
15Relationships with one HIV-positive and one HIV-negative partner.
16The Population and Housing Census uses Enumeration Areas as its basic sampling unit, rather than villages. The boundaries of these enumeration areas commonly cross through villages, leading to duplicate entries in the GIS datasets.
17Trading centers were identified based on their designation by the 2008 Malawi Population and Housing Census. Since TA Mwambo adjoins the city of Zomba, I also included the main markets in that city as trading center equivalents. In addition, based on conversations with key informants, I included several more trading centers in the local area that were not designated as such by the census.
18In discussions with key informants in TA Mwambo, 2 km was generally agreed to be the maximal distance people will walk for nightlife. These strata thus roughly proxy for how easily people could access the trading centers in order
and 22 (31%) were more than 5 km away from the closest center. This compares with overall proportions of 10%, 40% and 50% of all villages in TA Mwambo. Within each sampling stratum, I randomly assigned half of the villages to the treatment group and half to the control group.

In each village, a team of enumerators first conducted a comprehensive household census. Using this census, 15 men and 15 women aged 18-49 were then sampled from each village, with only one respondent allowed per household. The sample was thus stratified by both gender and distance to the nearest trading center, so the effective sampling strata are formed by combinations of gender and distance indicators. Some villages had too few households for 30 eligible-age adults to be selected, and hence the maximum feasible number was chosen instead. This yielded a total of 2024 sampled individuals. The survey team then attempted to contact all sampled people for a baseline survey. Although refusals were rare (< 1% of respondents refused the baseline survey), 23% of sampled people could not be found at baseline, typically because they were temporarily away from the household. A total of 1543 respondents had a successful baseline survey. Because the survey contained sensitive questions about sexual behavior, and the model of fatalism applies mainly to sexually active adults, the survey used an early screening question to eliminate people who had never had sex from the sample. This removed 2.6% of the respondents, leaving 1503 sexually-active adults in the baseline survey.

After a minimum delay of 30 days, the enumerator team attempted to recontact all 1503 sexually-active respondents from the baseline survey, successfully finding 1292. There is no evidence of differential attrition: an indicator for inclusion in the final sample is not significantly correlated with treatment status, irrespective of whether I control for other baseline covariates. There is also no evidence of differential attrition by baseline covariates, which I examine by interacting the treatment indicator with different baseline variables.

Baseline summary statistics for the overall sample, as well as a comparison of the treatment and control groups, are presented in Table 1. The sample is 43% male and 82% married, with a mean age just below 30. Respondents are fairly poor on average: household cash expenditures average just under $2 (at purchasing-power parity) per person per day. The sample is well-balanced across the treatment and control groups with the exception of household cash income, which is approximately $64/month higher in the control group. However, this discrepancy can be attributed to seasonal variation in income combined with the differential timing of the baseline surveys: for reasons discussed below in Section 3.2, the control group baseline surveys were done first and the

to drink and search for sex partners.

My respondents therefore form a weighted probability sample of TA Mwambo, with oversampling of villages closer to trading centers as well as oversampling of people from smaller villages. I do not adjust any of the results in the paper using sampling weights, but all of my main findings are robust to using such weights.

It is common for people in this area of Malawi to travel during the agricultural off-season to look for casual wage labor.

See Appendix Table II-1 for detailed figures on the number of people in each study arm and sampling stratum.

See Appendix Table II-2.

See Appendix Table II-3.

In this table, and in all the other balance tests in this paper, the p-values are adjusted to account for the clustered design of the study, following Donner and Klar (2000).
treatment group baseline surveys were done second. A comparison of incomes at the endline survey is valid if we make the plausible assumption that the information treatment had no impact on earnings. Monthly household income at the endline survey is still $23 higher in the control group, but this difference is not statistically significant. The summary statistics are consistent with the randomization having successfully generated balanced treatment and control groups.

3.2 Information Treatment

At the end of the baseline survey, all respondents from the treatment villages were read and shown information about the true risk of HIV infection between serodiscordant partners who have unprotected sex, as measured by the Wawer et al. (2005) study of serodiscordant couples in Rakai, Uganda. I used the annual risk for the information treatment because it is simpler to explain than the per-act risk, which is very small, and also because it is the figure available on the Malawi National AIDS Commission’s website. For a discussion of the ethical dimensions of teaching people the true risk of HIV transmission, see Appendix III.

The information treatment was administered by the survey enumerators in a one-on-one setting. It involved both an oral component and an interactive visual component. In the oral component, the basic details of the original Rakai study were explained, with certain aspects simplified for clarity. Respondents were told that the study occurred in Uganda, and that 100 serodiscordant couples were followed for a single year. They were told that all the couples had regular sex without using condoms, about once every three days on average, and asked how many people they thought would contract HIV. They were then informed that in fact only ten of the initially HIV-negative people became HIV-positive. Respondents were asked if they believed the results of the study, and enumerators were trained in how to respond to a number of common questions, such as whether the testing equipment was faulty. The script listed the reasons that HIV transmission sometimes does not happen even when serodiscordant couples have unprotected sex, for example the fact that HIV sometimes cannot penetrate the genitalia. The script then emphasized that HIV transmission is something that happens by chance, comparing it to popular games of chance used by local cell phone companies as marketing tools.

The interactive visual component complemented the oral component and occurred at the same time. It involved showing respondents a diagram with 100 pairs of stick figures representing serodis-
cordant couples, with a black stick figure indicating an HIV-negative partner and white stick figure indicating an HIV-positive partner. The respondent was asked to guess as to the number of people who would contract HIV after a year of regular unprotected sex with an infected partner, and this guess was indicated by circling an appropriate number of these stick figure couples. When the true rate was presented, the enumerator showed a second diagram in which ten of the initially HIV-negative individuals had turned from black to white. Enumerators then counted and circled these transmissions.

To minimize the risk of contaminating the control villages, all the baseline treatment surveys were done after the baseline control surveys were completed. This approach parallels that taken by Godlonton, Munthali and Thornton (2014). The survey enumerators were only taught to administer the information intervention after all the control surveys were completed.

3.3 Measures of sexual behavior

My primary outcome measure is self-reported sexual behavior as recorded using a detailed retrospective sexual diary. The diary walks respondents through the previous seven days beginning with yesterday. On each day, respondents were asked what time they woke up, how much alcohol they had, whether they were menstruating (or for men, whether their sex partner was menstruating), the value of gifts they received from their partner (or for men, gifts they gave to their partner), how many times they had sex, and the time they went to sleep. Then, for each reported sex act, they were asked detailed questions such as the time of day, the length of the act, condom use, and whether the sex act was with their primary sex partner or a different partner. The surveys also contained single-question recall measures of sexual behavior, for example: “In the past 30 days, how many total times did you have sex, including serious and non-serious partners?”

The diary-based approach to measuring sexual behavior was initially developed and refined in previous research on sexual behavior in Southern Malawi (Kerwin et al. 2011). It builds on research that shows that calendar-based methods reduce recall bias compared with single-question recall methods (Belli, Shay and Stafford 2001). Luke, Clark and Zulu (2011) have found that relationship history calendars improve the quality of responses to questions on sexual behavior, showing that apparent biases due to social desirability effects are smaller. The sex diary approach adapts these insights to a much shorter time frame to assist respondents in the recall of all sex acts over the past 7 days. The improved accuracy of the sex diary over other methods is reflected in the data captured by the surveys. Column 1 of Table 2 shows that the two variables record fairly similar levels of sexual activity. The distributions of the two variables are also very different, with substantially more heaping at multiples of 5 in the single-question recall variable. Given the lower quality of the single-question recall variables, and because I used total sex acts as recorded on the diary as my primary outcome in an earlier working paper that I wrote prior to the experiment

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28The culture of gift-giving in sexual relationships in Malawi is strongly gender-driven: with very few exceptions men give gifts to women and not the other way around.

29See Appendix IV for histograms and a discussion of the implications of heaping for regression estimates.
(Kerwin 2012), I will rely primarily on the sex diary variables for my analysis.

Table 2 presents summary statistics for all the available measures of sexual activity in the data. Columns 3 and 4 show the means of my measures of sexual activity for the control and treatment groups respectively, while Column 5 shows the difference between the two. These are generally balanced across the two study arms, with the only statistically significant differences being a lower number of lifetime sex partners ($p < 0.05$). All the differences are fairly small in magnitude, but none of the variables has exactly equal means across the treatment and control groups at baseline. This is one reason my analyses will control for respondents’ baseline values of self-reported sex, as described in Section 4.2.

An additional measure of the demand for safer sex comes from the sale of subsidized condoms to respondents that occurred immediately after the endline survey. All participants were given six coins worth five Malawi Kwacha each (30 Kwacha total, or about ten cents). They were then offered the chance to purchase 3-packs of condoms for five Kwacha apiece, or individual condoms for two kwacha. While the price represents a sizeable subsidy relative to the sale of condoms at local stores, the vast majority of respondents who acquired condoms got them for free. When asked about the nearest place to acquire condoms, respondents commonly named health centers and health extension workers, both of which offer condoms free of charge. This measure was only collected at the endline survey.

It is common in the literature to present results using a constructed combined outcome index, both to reduce concerns about multiple comparisons and to improve the precision of estimates (e.g. Kling, Liebman and Katz 2007). However, the value of such an index is unclear in situations where some outcomes are measured with greater error or where baseline data is not available for particular outcomes (for example, condom sales were only done at endline). I therefore present two versions of the sexual risk index. One uses all outcomes that can be constructed from the retrospective sexual diary, which I argue provides more accurately-measured outcomes than the single-question recall variables. An alternative index includes both the sex diary outcomes as well as all other outcomes that can be constructed from the survey, including the condom sales.

Each index is constructed separately for the baseline and endline waves by normalizing all component variables (subtracting the control-group mean and then dividing by the control-group standard deviation). The normalization is reversed in sign for condom use, condom acquisition, and condoms purchased, for which positive numbers imply less risk-taking. These normalized values are then averaged for each respondent, weighted by the factor loadings for the first principal component of the matrix of the data for the control group. This follows Black and Smith (2006) in assuming that there is a single underlying sexual activity factor, and that the different outcomes measured in the data are noisy signals of that factor; the procedure selects the linear combination of the data that gives the best estimate of the underlying sexual activity factor.\footnote{I also explored unweighted averages; these produce similar results with slightly smaller magnitudes.}
3.4 Measures of risk beliefs

The central prediction of the model I outline in Section 2 is that individuals’ responses to risk information will depend on their initial perceptions of those risks. A key input for my analysis, therefore, is a quantitative measure of risk perceptions. Due to data limitations, one common strategy for this is to utilize some measure of the true risk.\(^{31}\) However, an emerging literature has shown that it is feasible to collect meaningful data on subjective beliefs about probabilities using surveys, not just in the United States (e.g. Lillard and Willis 2001; Manski 2004) but also in the developing world (e.g. Attanasio 2009, Delavande, Giné and McKenzie 2011, Delavande 2014). Delavande and Kohler (2009) have developed a method of eliciting subjective expectations using visual aids that they show performs very well in Malawi.

Rather than following Delavande and Kohler, I rely on measures of subjective risk beliefs collected using concrete questions about proportions out of a fixed number of people. These are questions of the form “If 100 men, who do not have HIV, each sleep with a woman who is HIV-positive tonight and do not use a condom, how many of them do you think will have HIV after the night?” I then divide the reported number by the denominator used to construct a subjective probability. Question E1a in Figure 3 is an example of one of these questions. All the questions were gender-specific: for instance, when men were asked about HIV transmission they were asked about 100 men having sex with an HIV-positive woman, and likewise women were asked about 100 women having sex with an HIV-positive man.\(^{32}\)

I use these concrete expectations questions for two reasons. First, the Delavande and Kohler approach adds considerably to the logistical complexity of surveys, as well as the time needed to conduct them. Second, this concrete style of expectation question has been validated through extensive use in previous research across a variety of contexts in Malawi, including in urban areas as well as in areas of rural southern Malawi near my study site.\(^{33}\) They also appear to be fairly scale-invariant: switching the denominator from 100 to 1000 or 10,000 yields nearly the same subjective probabilities on average, and respondents give the exact same answer roughly 60% of the time.\(^{34}\) The questions also perform well in terms of respecting nested probabilities: if the chance of event B occurring includes all possible instances of event A, then respondents should ideally report a weakly higher probability for B than for A. Delavande and Kohler emphasize this as one of the major strengths of their approach.

My data do not afford many direct comparisons with Delavande and Kohler’s on HIV transmission and HIV prevalence, because their survey instrument did not ask many HIV-related questions.


\(^{32}\)Six HIV risk belief variables were collected: the unprotected transmission rate (both per-act and annual), the condom-protected transmission rate (both per-act and with a condom), and two questions about the prevalence of the virus: the share of all members of the opposite sex that respondents thought were HIV-positive, and the share of members of the opposite sex that they find attractive.

\(^{33}\)Chinkhumba, Godlonton and Thornton (2014)

\(^{34}\)Godlonton, Munthali and Thornton (2014), Kerwin et al. (2011)

\(^{35}\)Author’s calculations based on Chinkhumba, Godlonton and Thornton (2014)
that are necessarily nested within one another. One comparison, however, is the per-unprotected-
sex-act risk of contracting HIV from an infected partner, compared with the annual risk. In my
data, the latter probability was weakly higher 92.2% of the time, whereas this was the case 91.9% of
the time in the Delavande and Kohler data. In addition to performing comparably to the
Delavande and Kohler approach in terms of nesting probabilities, the concrete probability method
also produces similar results in terms of the mean expectation of the risk of HIV transmission: this
is 82.8% per act for the control group at baseline using concrete probabilities, and 85.9% per act
using Delavande and Kohler’s method.

One potential concern with eliciting subjective expectations is the tendency for probabilities to
heap at the “focal” probability of 50%. The typical interpretation, cited by Delavande and Kohler,
is that this heaping reflects a misunderstanding of the question, or simple uncertainty, rather than
a true belief. People commonly use 50% (or in my case, report half of the total denominator), when
they are simply unsure about the answer. To address this issue, respondents who reported beliefs
of 50% were prompted with a followup question about whether they really believed the chance
was 50%, or if they were just not sure, which is an approach taken on the Health and Retirement
Study’s subjective expectations questions (Hudomiet, Kézdi and Willis 2011). Building on that
approach, respondents who said they were just not sure were then prompted for their best guess.
Question E1b in Figure 3 illustrates these followup questions. In my measure of risk beliefs I use
the response to the followup question for people who change their answer.

3.5 Enumerator-knowledge contamination of measured beliefs

As described in Section 3.2, the enumerators were only trained to provide the information
intervention after the baseline interviews for the control group were finished. This was done to
minimize any chance of the information intervention contaminating the control group. However,
it also meant that this was the first time the enumerators themselves were taught the true risk of
HIV transmission. As a result, enumerators brought different beliefs with them into the baseline
treatment and control surveys. This had a relatively small but statistically-significant effect on the
measured beliefs of treatment-group respondents at baseline.

Figure 4 shows the daily average recorded risk belief, separately for treatment and control
surveys, and including both baseline and endline surveys. The lines show linear time trends fit
to the data. One thing that is immediately clear is that the measured difference at baseline is
much smaller than the impact of the information intervention. This can be confirmed numerically
by comparing Panel A of Appendix Table II-4, which shows the enumerator effects on measured
baseline beliefs, to Table 3, which presents the effects of the information intervention people’s beliefs
about the transmission rate of the virus. The treatment effects are at least four times as large as

36The annual question for Delavande and Kohler actually asks about someone who is married to an HIV-positive
person, and does not explicitly specify unprotected sex. However, social norms in Malawi strongly proscribe the use
of condoms within marriages (Tavory and Swidler 2009) and married couples use condoms just 11.2% of the time in
my sample. Repeating this analysis just for people in the MLSFH sample who say there is no chance they would use
condoms with their own spouse yields a similar nesting rate of 94.1%.
the enumerator effects, no matter what specification is used.

There are two potential explanations for this pattern. One is that different knowledge may have led enumerators to prime subjects differently, possibly even subconsciously. Enumerators were trained to follow up with probing questions when respondents answered a question by just saying that they did not know. The phrasing of these probing questions could have been affected by the knowledge enumerators brought to the surveys. A second possibility is enumerator experience with the questions. While the sex diary questions that form my outcome measure use very simple statements that enumerators were already familiar with using, the phrasing used on the subjective expectations questions was fairly complex. This may have lead to some temporal pattern in reported risk beliefs as the phrasing of the probing questions used was refined over time.

There is evidence for both explanations in Figure 4. A downward trend in measured risk beliefs is evident prior to the enumerators being taught the information about HIV transmission, and there is a large drop in beliefs after the first vertical line that marks the training session. Further confirmation of the importance of enumerator knowledge for measured risk beliefs can be seen based on the light blue dots that appear after the first vertical line. These are average beliefs for “cleanup” surveys – a handful of control-group interviews that were done after the treatment surveys had begun, because respondents were not home when surveys were attempted prior to the information treatment training. Excluding the large negative outlier (which is the average for a day when just a single control-group survey was done) these generally match the measured beliefs for the baseline treatment group surveys.

Another way of understanding the importance of enumerator knowledge is to compare the beliefs recorded at baseline for the treatment group to the endline beliefs for the control group; this can be done by comparing the hollow triangles to the solid circles in Figure 4. These are both surveys during which the enumerators’ knowledge is identical (they know the information about HIV) and the respondents in the treatment and control groups have identical information sets (neither has been told the HIV risk information). This is reflected in the recorded values, which look the same in the two groups.\footnote{Panel B of Appendix Table II-4 does formal t-tests for this comparison. The only statistically-significant differences are in annual unprotected transmission risks and the prevalence of HIV among attractive people of the opposite sex.}

To correct for the evident contamination of measured risk beliefs due to differential enumerator knowledge, I adjust reported beliefs based on time trends with a trend break. This involves estimating the following regression:

\[
x_i = \rho_0 + \rho_1 Date_i + \rho_2 After_i + \rho_3 After_i \times Date_i + v_i
\]

\(Date_i\) is the date of the baseline survey for respondent \(i\) and \(After_i\) is an indicator for whether the baseline survey was done after the information treatment training session. I then construct \(x_{i,Adj} = x_{i, resid} + \hat{\rho}_0\), and bound the resulting variable to lie within \([0, 1]\) by replacing values below 0 with 0 and those above 1 with 1. Panel C of Appendix Table II-4 presents the trend-adjusted
risk beliefs for the control and treatment groups. They are unsurprisingly similar across groups. As robustness checks, I also replicate my analysis using the raw (unadjusted) risk belief measures, as well as two other kinds of trend adjustment: using a single trend across the whole baseline period, and using just a level shift in reported beliefs. My results are not sensitive to any of these variations, but my preferred specifications use the adjustments described above. These have a simple interpretation: they are my best estimate of how a respondent’s initial beliefs compare with the rest of the sample, given the known time trend and trend break evident in the data due to enumerator-knowledge contamination.

3.6 Composite belief measures

My analysis focuses on a composite measure of the perceived risk of contracting HIV from unprotected sex with a randomly-chosen potential sex partner. This is the product of the perceived per-act risk of HIV transmission from unprotected sex with an infected partner and the perceived prevalence of HIV among attractive people of the opposite gender. I use this composite variable for two reasons. First, using the perceived HIV prevalence among attractive people of the opposite sex mitigates concerns that people’s self-beliefs about risks may differ from their beliefs about the risks faced by the rest of the population. Recent research on subjective expectations has highlighted that people’s self-beliefs can be very different from what they believe about people in general and that people are more responsive to self-beliefs (e.g. Wiswall and Zafar 2014). For risks, this commonly takes the form of unrealistic optimism about one’s own risk relative to the rest of the population (Weinstein and Klein 1996). In my context, there is also the potential for unrealistic pessimism: people’s stated perceptions of both HIV prevalence and transmission risks are much higher than the truth, and they may feel more at risk personally than they believe to be the case for the broader population. While I cannot totally eliminate the potential for differences between self-beliefs and general beliefs, focusing on the risk from unprotected sex with a random attractive member of the opposite sex (rather than all local people of the opposite sex) is likely to be a superior measure of the level of risk people feel they actually face.

Second, relying on variation in the other beliefs allows me to avoid one of the shortcomings of using perceived per-act HIV risks, which is that they are extremely concentrated in the right tail. At baseline, over four in ten respondents believe that the per-act risk of HIV transmission from unprotected sex is 100% (Figure 5, Panel A). If I use this variable to conduct the heterogeneous treatment effects analyses in Sections 4.5 and 4.6, I find the same basic pattern of heterogeneity as with my preferred risk measure. People with the highest risk beliefs have sharply lower treatment effects that people with the lowest beliefs, and I estimate a zero treatment effect for people with beliefs above 65% per act. However, by clustering 40% of people at the very top of the belief distribution, this approach hides the fact that people in the highest category of per-act risk beliefs actually perceive sharply different risks. Interacting the per-act risk belief variable with the respondent’s perceived prevalence breaks up the mass point of people who think the per-act risk is 100%, and does so according to their perception of how risky they think having unprotected sex
actually is. The resulting product also has a natural interpretation: it is how risky people perceive any given sex act to be if they do not know the HIV status of their partner, given their perceptions about the prevalence of HIV among potential sex partners and the transmission rate of the virus. Panel B of Figure 5 shows the distribution of this combined variable, which has a much smaller mass point at 100%.

In Figure 6 I present the baseline CDFs of the combined risk measure I focus on in this paper, constructed two different ways. Panel A uses unadjusted values of the per-act risk and prevalence belief variables, while Panel B uses values that have been adjusted for a linear time trend with a trend break as described in Section 3.5. In each panel the solid line shows the control group’s beliefs while the dashed line shows the treatment group’s beliefs. The treatment and control group distributions are different using the raw values, and this is largely corrected by the regression adjustment.

4 Empirical Results

This section details the empirical results of the study. I begin by showing that the information treatment has large effects on people’s risk beliefs. I then show that the average effect of the information treatment is to slightly (but statistically significantly) increase the amount of risky sex people have. This is consistent with a small negative risk elasticity of sexual behavior, which I estimate directly using two-stage least squares. I then construct semiparametric decompositions of the treatment effect by people’s initial risk beliefs, and show that the overall average masks substantial heterogeneity by baseline beliefs. I extend this analysis to 2SLS estimates of partial effects as well: I use indirect least squares to develop an estimator of the local average treatment effect (LATE) that allows for heterogeneity by baseline covariates. Using this heterogeneous LATE estimator, I show that the elasticity of sexual behavior with respect to risk beliefs is negative for individuals with low risk beliefs, and becomes positive for individuals at the high end of the risk belief distribution.

4.1 Impact of the information treatment on risk beliefs

The information treatment has large effects on respondents’ risk beliefs. Panel A of Table 3 shows the endline treatment-control differences for all the measures of people’s beliefs about HIV transmission and prevalence. The treatment group believes the annual risk from unprotected sex is 38 percentage points lower than the control group does. Their belief about the per-act risk decreases even further, by 41 percentage points.\(^{38}\) Note that the respondents do not update their beliefs perfectly: the actual annual transmission rate is about 10%; just 2% of the treatment group reports beliefs that low. The alternative specifications in Panels B and C confirm that these results

\(^{38}\)The larger impact on per-act risks is a consequence of the ceiling of 100% on transmission rates; 50% of treatment group respondents who think the annual transmission rate is 100% believe the per-act transmission rate is less than that.
are robust to controlling for baseline values of the outcome variable and running a difference-in-differences respectively.

Respondents also update their beliefs about HIV risk variables other than the transmission rate from unprotected sex. For example, beliefs about the risk of condom-protected sex and about HIV prevalence are both reduced. This suggests that instead of simply memorizing the numbers they were told, respondents learned the information and updated their beliefs accordingly: if they understand that the current prevalence of HIV depends on infected people transmitting the virus to others, then a reduction in the transmission rate implies a reduction in the prevalence of the virus. The information treatment contained no direct information about the prevalence of the virus nor about condom-protected sex, so the effects on these variables can be ascribed purely to this learning process.

4.2 Estimation Strategy

All my regressions control for baseline values of the outcome variable. Frison and Pocock (1992) and McKenzie (2012) show that this generates estimated treatment effects with a lower variance than either a) relying the endline values of the outcome alone or b) using changes in the outcome (i.e. a difference-in-differences). When there are baseline differences in outcomes across study arms, this approach also generates estimates with a lower bias than either alternative. (See Appendix IV for a mathematical derivation). Controlling for the baseline value of the outcome will reduce the bias anytime the outcome variable is not exactly equal across study arms – even if the difference is not statistically significant. Since there are small but non-zero differences in the means of outcome variables across study arms, this is the preferred estimator for my sample. The specifications used in this paper also control for the stratification cells (combinations of distance categories and gender) used to draw the original sample, which improves statistical efficiency (Bruhn and McKenzie 2009).

My regressions have the following form:

$$y^e_i = \alpha + \beta T_i + \gamma y^b_i + Z_i \eta + e_i$$

where $y^e_i$ is the endline value of the outcome variable, $T_i$ is an indicator of whether the respondent was in the treatment group, $y^b_i$ is the baseline value of the outcome variable, $Z_i$ is a vector of categorical dummy variables for the sampling strata, and $e_i$ is an error term.

4.3 Reduced form effects of the information treatment

The results of the reduced-form specifications are shown in Table 4; all continuous outcomes are presented in logs so the coefficient estimates can be interpreted as percentage-point changes. The estimated impact is small in magnitude: it is possible to rule out magnitudes larger than 20 percentage points, or greater than 0.16 standard deviations for the indices. The number of sex acts

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39Because many outcomes contain zeroes, I use the inverse hyperbolic sine transformation of Burbidge, Magee and Robb (1988) rather than logging the variable directly, constructing $\ln_{\text{inh}}(y) = \ln(y + \sqrt{y^2 + 1})$. 

19
in the past week rises by 10 percentage points. Focusing specifically on the margin of abstinence (whether people have any sex at all), this shifts by 5 percentage points, which is roughly 0.1 standard deviations. The risk indices confirm that these results are robust to multiple hypothesis testing: both the overall and sex diary risk indices rise by 6%, significant at the 10% and the 5% level respectively. The treatment has no effect on condom use, nor on condom purchases. This is consistent with the extremely high rates of unprotected sex: at baseline just 1 in 10 sex acts involved a condom, leaving limited room for increases in risk-taking at this margin.

4.4 The risk belief elasticity of sexual behavior

The effect of this specific information treatment on sexual behavior is less generalizable than the marginal effect of HIV risk beliefs on sexual risk-taking, which can be used to design other policy interventions involving responses to HIV infection risks.\(^\text{40}\) Consider the OLS regression

\[
y_e^i = \alpha + \delta x_e^i + \gamma y_b^i + Z_i'\eta + e_i
\]

\(\hat{\delta}\) is an estimate of \(\partial y^*/\partial x\), the partial effect of risk beliefs on risky sex. The results of running this regression with various outcomes are shown in Panel A of Table 5, and discussed below. However, for these estimates to be consistent, \(x_e^i\) must be independent of the error term. This is unlikely to be true. One reason it may fail is that individuals may form their risk beliefs based in part on sexual experience, and sexual experience is highly autocorrelated. Another reason, noted by Oster (2012), is that the subjective risk will probably have some association with the actual prevalence of HIV – and that the prevalence is itself the outcome of local sexual behavior.

I therefore estimate \(\hat{\delta}\) via two-stage least squares, using \(T_i\) as an instrument for \(x_e^i\). \(T_i\) is plausibly excludable from the second-stage regression. Because the treatment was randomized, membership in the treatment group should have no association with sexual behavior other than through the information treatment. Furthermore, the information treatment is very unlikely to affect sexual behavior through any channel other than individuals' risk beliefs: it does not contain any guidance or information about sex. The instrument also easily satisfies the relevance condition. The F-statistic on \(T_i\) in the first-stage regressions is roughly 220 for all specifications.\(^\text{41}\) This allows me to estimate two-stage regressions as follows:

\[
x_e^i = \alpha + \beta T_i + \gamma y_b^i + \rho x_b^i + Z_i'\eta + e_i
\]

\[
y_e^i = \alpha + \delta x_e^i + \gamma y_b^i + \rho x_b^i + Z_i'\eta + e_i
\]

\(x_b^i\) is included as a control in the first stage in order to improve efficiency and reduce bias, for the

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\(^{40}\) As noted above, I use the adjusted versions of the belief variables, which removing time-varying trends in beliefs. All the results are robust to using the original belief variables instead.

\(^{41}\) It is not possible to conduct a formal test for weak instruments unless the number of excluded instruments is at least two more than the number of endogenous regressors (Stock and Yogo 2005). However, the informal “rule of thumb” generally used in applied econometrics is an F-statistic of at least 10; by this standard, my instrument easily passes.
The 2SLS estimates are shown in Panel B of Table 5, with OLS results (estimated on the control group only) shown in Panel A for comparison. The OLS results have a uniform positive bias relative to 2SLS, confirming that OLS is not consistent in this context. This is consistent with Oster (2012), who finds that OLS estimates of the elasticity of sexual behavior with respect to the true prevalence of HIV are biased and wrong-signed. The fact that the omitted variable in the second-stage regression is positively correlated with risk beliefs can be explained in one of two ways. First, people may form their risk beliefs through a process in which sexual activity plays a part. For example, people who have more sex may be exposed to more gossip, which (if the tone is frightening) leads them to raise their risk beliefs. Second, people who have a latent desire for more sex may select into opportunities to learn about HIV risks; since HIV risk messaging tends to overstate transmission risks, this would lead them to have upward-biased beliefs.

The elasticity of sex acts in the past week with respect to HIV risk beliefs is approximately 0.6. The other elasticities are smaller in magnitude: they are mostly around -0.3, which is the estimate yielded by the sexual activity index method. These results are much larger than Oster (2012), which estimates prevalence elasticities of about -0.01 to -0.02 for binary outcomes (compared with -0.3 for my binary outcome in column 1). My estimates are closer to the Ahituv, Hotz and Philipson (1996) estimates for the US: they find elasticities of about -0.2 for binary outcomes. My estimates for continuous outcomes are also close to those found in US studies: focusing on gay men in San Francisco, Auld (2006) estimates a prevalence elasticity of sexual activity of -0.5. However, my results are not directly comparable with this earlier work, which uses the true prevalence as the regressor of interest. People do not accurately know the true prevalence, so changes in the true prevalence are unlikely to show up 1-for-1 as changes in perceived prevalence. This means that the implied prevalence elasticities from my results are likely to be smaller than those for the US, and closer to the Oster (2012) findings.

The population-average reduced form and marginal effects both fit a model of self-protective risk-compensation, which is consistent with the existing literature. However, the specifications in Tables 4 and 5 impose common effects across all respondents, and hence across all levels of risk beliefs. To explore the importance of this restriction, I explore heterogeneity in ITT and marginal effects by baseline covariates, with a focus on baseline risk beliefs.

### 4.5 Heterogeneity in the reduced-form effect of the risk information treatment

The key prediction of the rational fatalism model is that responses to risks will be heterogeneous by individuals’ baseline characteristics. Specifically, it predicts that the magnitude and sign of the comparative static will vary by baseline beliefs about risks. This implies that, provided the first-stage effect of the information treatment on risk beliefs is uniformly negative, the sign of the effect of the information treatment should vary by baseline risk beliefs as well, I test this prediction by
estimating a modified version of the reduced-form regression:

\[ y^e_i = \alpha + \beta T + \delta_1 w^1_i + \sum_{j=2}^{J} \left[ \beta^T w^j T_i w^j_i + \delta_j w^j_i \right] + \gamma y^b_i + Z'_i \eta + \epsilon_i \] \hspace{1cm} (10)

Here \( w^1_i, \ldots, w^J_i \) are a set of \( J \) baseline covariates. My primary focus is on heterogeneity by baseline risk beliefs \( x^b_i \). I also examine other potential sources of heterogeneity in responses, such as gender, baseline sexual activity, and previous HIV exposures.

The results of these heterogeneous treatment effects analyses for the total number of sex acts in the past week are presented in Table 6. Responses to the information treatment are strongly heterogeneous by baseline risk beliefs (Column 1). Using this linear specification, people with baseline risk beliefs of 0% respond to the information treatment by increasing their sex acts per week by 32%. For people with baseline beliefs of 100%, the response is lower by 50%, meaning that weekly sexual activity declines by 18%. I can reject that responses for people with high risk beliefs are the same as for those with low beliefs at the 1% level; the negative response for people with the highest risk beliefs is statistically significant at the 10% level. The positive treatment effect for people who have baseline beliefs of 0% suggests that a linear specification for the treatment effect heterogeneity is misspecified, since their risk beliefs should increase rather than decreasing. This lends further support to the flexible analyses I conduct below.

In Columns 3 through 6 I look for heterogeneous responses by gender, baseline sexual activity, perceived previous exposure to HIV, and whether the respondent believes he or she may currently be HIV-positive. There is also no statistically-significant heterogeneity by any of these factors. Moreover, the results for baseline risk beliefs are also robust to including three-way interactions with gender, as well as the other variables in Table 6.

The specification in Table 6 assumes that the heterogeneity in treatment effects is linear in form. While this is not a concern for binary \( w^j \) such as gender, it is a more substantive restriction for continuous variables like baseline beliefs. As an alternative, I estimate semiparametric regressions of \( dy/dT \) by baseline risk beliefs for the treatment and control groups:

\[ y^e_i = \beta^T + f^T (w_i) + \gamma^T y^b_i + Z'_i \eta^T + \epsilon_i \text{ if Treatment} = 1 \] \hspace{1cm} (11)
\[ y^e_i = \beta^C + f^C (w_i) + \gamma^C y^b_i + Z'_i \eta^C + \nu_i \text{ if Treatment} = 0 \] \hspace{1cm} (12)

\[42\]The effect of gender on responses to the information treatment is theoretically ambiguous. Malawian women commonly have less bargaining power in sexual relationships than men. However, most of my sample comprises matrilocal villages, which grant women more power to divorce their husbands and hence may increase bargaining power within relationships as well (Schatz 2005).

\[43\]Perceived previous exposure to HIV is an indicator that is coded to 1 if the respondent believes any of their past sex partners was HIV-positive and zero if they do not. This ignores the possibility that a condom was used for the sex acts with an HIV-positive partner, but given the low rates of condom use in this population that should not affect the results appreciably.

\[44\]The perceived HIV status variable is an indicator that collapses a Likert scale question in which respondents report how likely they think it is that they are HIV-positive now on a scale from “No Likelihood” up to “High Likelihood.” “No Likelihood” is coded as a zero, while any other response is coded as a one. “Don’t Know” is coded as a missing value.
These regressions give me estimates of $E[y | T = 1]$ and $E[y | T = 0]$ for each value of $w_i$.\(^{45}\) Thus taking the difference gives me estimates of the $w_i$-specific treatment effect $\hat{\tau}_y(w_i) = \hat{f}_T(w_i) - \hat{f}_C(w_i)$.\(^{46}\)

I implement the semiparametric regressions using the Robinson (1988) double residual estimator for partially linear regressions. The basic logic of the Robinson estimator is as follows: consider the regression function for the control group. If we take its conditional expectation given $w_i$, and subtract that from the original equation, the $f(w_i)$ component drops out and we have

$$y_i^e - E[y_i^e | w_i] = \gamma^C(y_i^b - E[y_i^b | w_i]) + (Z_i' - E[Z_i' | w_i])\eta^C + \nu_i$$

The conditional expectations of $y_i^e$ given $w_i$ and of the controls given $w_i$ are estimated by separate nonparametric regressions for each variable. These estimates are plugged in to the equation above, which is estimated by OLS. Finally, the parametric component of $y_i^e$ is removed using the estimates of $\gamma^C$ and $\eta^C$, allowing the function $f^C(w_i)$ to be estimated nonparametrically. I choose data-driven bandwidths to minimize the mean-squared prediction error using the generalized cross-validation (GCV) statistic of Loader (2004). My results are qualitatively robust to halving all the bandwidths as well (see Appendix Figures IV-1 to IV-3). The underlying semiparametric regressions do not have boundary bias problems because they are fit using local linear regressions. However, my estimates (which are the difference of two sets of local linear regressions) show a high degree of variability at the very edges of the distribution, so I truncate the display of my graphs to eliminate points outside $(0.05, 0.95)$.

I apply this approach to heterogeneity in my first-stage regressions of endline risk beliefs $x_i^e$ on the information treatment, and construct a function $\tau_x(x_i^b)$. I also apply it to my reduced-form regressions of treatment effects on sexual activity, estimating a function $\tau_y(x_i^b)$. I then construct confidence intervals via a clustered bootstrap with 1000 repetitions; for each bootstrap repetition, I repeat the procedure of adjusting belief variable to correct my estimated confidence intervals for the fact that it is a generated regressor. In each bootstrap sample, I trim observations with estimated densities lower than the minimum observed in the original dataset. The original sample has no estimated densities that are near zero, so my point estimates do not have trimming issues. Replicating the results while trimming at zero instead does not appreciably change the estimates, suggesting that very few observations have extremely small estimated densities.

Figure 7 shows the results of this semiparametric regression for the first stage, and Figure 8 shows the results for the reduced form. The first-stage results show that the change in risk beliefs is largest for people with the highest beliefs, and drops fairly steadily as baseline beliefs fall.\(^{47}\)

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\(^{45}\)Technically these are $E[y | T = 1, y_i^b, Z_i]$, but the randomization of $T_i$ means I can ignore the expectation over the control variables.

\(^{46}\)A purely nonparametric version of this estimator is used in the Bennear et al. (2013) study of behavioral responses to information about arsenic in drinking water.

\(^{47}\)Near the low end of the scale the estimated $dx/dT$ is larger in magnitude than the baseline beliefs $x_i^b$. This happens because $dx/dT$ is estimated off of endline beliefs, which tend to revert toward the mean for the control group. For example, for people with baseline beliefs below 0.10 the average endline belief was 0.18 in the control group and 0.10 in the treatment group. My randomized treatment is orthogonal to this mean-reverting measurement error, so the consistency of my estimates should not be affected, but they may represent the wrong points on the baseline.
This pattern is reasonable, since people with the highest risk beliefs should update their priors by a larger amount than people with lower beliefs. These results are robust to an alternative semiparametric approach, using brackets of the baseline belief distribution instead of the Robinson estimator. In that approach, I construct indicator variables for eight quantiles of the baseline risk belief variable, and interact those with the treatment indicator; I then regress the outcome on the full set of interactions plus my controls (see Appendix Figure VI-1).

The semiparametric reduced-form estimates are consistent with those from the linear approximation in Table 6: the treatment effect is initially positive, and then becomes negative for people with extremely high baseline risk beliefs. For people with the highest baseline beliefs, I can reject the null that the treatment effect is $\geq 0$ at the 1% level. The pattern of heterogeneity is also confirmed by the bracketed approach described above (see Appendix Figure VI-2). In addition, I try a wide range of alternative specifications, several alternative methods of handling the baseline risk beliefs, and a number of different outcome measures. The results uniformly confirm the same pattern of heterogeneity: people with the highest baseline risk beliefs respond negatively, rather than positively, to the information treatment. By pooling the data for the middle 6 brackets in the bracketed approach, I can also confirm that the point estimates are positive in the middle range of the data. Even though the pointwise CIs include zero, the estimated treatment effects are all similar to one another, and so I can reject the null hypothesis of a zero effect in the middle range of the data.

4.6 Heterogeneity in the risk belief elasticity of sexual behavior

My theoretical framework predicts not just heterogeneity in treatment effects but also heterogeneity in the effect of risk beliefs $x$ on sexual behavior $y^*$. In particular, it implies that the partial effect of $x$ on $y^*$ will be initially negative, and then positive for sufficiently high $x$. I therefore also examine heterogeneity in the instrumental-variables estimate of the effect of $x$ on $y^*$.

To do this, I develop an estimation strategy that can be applied to any baseline covariate $w_i$. I begin by defining subgroup $k$ of the sample as those individuals with $w_i = w_k$. It is possible to construct an estimator of the group $k$-specific marginal effect $\hat{\delta}_k^{IV} = \hat{\delta}_k^{IV}(w_k)$, which will in general be a function of $w_k$. Since $T_i$ and $w_i$ are independent, the treatment remains a valid instrument for this subsample. Selection on right-hand side variables likewise does not affect the consistency of an estimator, so any valid instrumental variables estimator for the whole sample will be valid for this subsample (Heckman 1996). While I could rely on 2SLS estimation, in general I will want to estimate the relationships semiparametrically, so I instead use the indirect least squares (ILS) estimator. I estimate the following separate regressions:

$$x_i^e = \alpha^x + \beta^x T_i + \gamma^x y_i^b + Z_i^x \delta^x + e_i \text{ for } w_i = w_k$$

(13)

$$y_i^e = \alpha^y + \beta^y T_i + \gamma^y y_i^b + Z_i^y \delta^y + v_i \text{ for } w_i = w_k$$

(14)
with \( w_i \) being the baseline belief variable and \( w_k \) represents each of its values. I then construct

\[
\hat{\delta}_{ILS,j}(w^k) = \hat{\beta}_y(w^k) \cdot \frac{\tau_y(w^k)}{\tau_x(w^k)} = \frac{dy}{dT}(w^k),
\]

where convergence in probability comes from Slutsky’s theorem.\(^{48}\) I estimate the \( w_k \)-specific treatment effects \( \hat{\beta}_x(w^k) \) and \( \hat{\beta}_y(w^k) \) using \( \tau_x(w^k) \) and \( \tau_y(w^k) \) as described above. While it is possible to construct analytic standard errors for ILS, I rely instead on cluster-bootstrapped confidence intervals since my preferred underlying estimator is already semiparametric and has standard errors without a known analytical form.

The results of this procedure, using the log of sex acts in the past week as the outcome variable, are shown in Figure 9. These elasticities are consistent with the theoretical framework from Section 2, in which the relationship between risk beliefs and risky sex has an overall U-shape: the slope is initially negative and then becomes positive for people with sufficiently high risk beliefs. My confidence intervals are pointwise, rather than simultaneous; due to the nature of my estimation procedure, constructing simultaneous confidence intervals is difficult. However, using the bracketed version of the results I reject the null that marginal effects are less than zero for the highest risk belief category at beyond the 0.01 level; the Bonferroni-adjusted p-value is below 0.02. The bracketed approach thus suggests that the top octile, or highest 12.5%, of respondents are fatalistic. Looking instead to the results using the Robinson estimator, I find that 13.8% of people have elasticities greater than zero: the risk elasticity of risky sex varies from -2.3 for the lowest risk beliefs to 2.9 for the highest ones. Note that although this evidence suggests a U-shaped relationship, I am unable to recover the underlying function: I can estimate heterogeneity in the marginal effect of endline risk beliefs on risky sex only by baseline risk beliefs, not by endline beliefs.

5 Discussion

In this section I discuss the implications and limitations of the results of this study. I begin by showing that the fatalistic responses I observe are consistent with the mechanisms of rationally fatalistic responses described in Section 2. Then I show that my results are not driven by baseline risk beliefs capturing other observed variation in the baseline data, such as education or sexual activity. I then discuss several potential limitations of this study. Finally, I consider what my results imply for HIV prevention policy.

5.1 Mechanisms for Fatalistic Responses

The theoretical framework in Section 2 predicts fatalistic responses to risks in two different situations. First, people may have an accumulated stock of past risks they have taken whose

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\(^{48}\)The overall LATE can be recovered from these \( w_i \)-specific LATEs by taking a weighted average of them, where the weights are the product of the share of the data that has a given value of \( w^k \) and the strength of the first stage for \( w^k \). See Appendix VII for a derivation.
outcome has not yet been realized. Second, they may not have perfect control over their future risky behavior: condoms may break, they may be tempted into mistakes, and so forth. If the first mechanism alone is driving the fatalism measured in our sample, then people’s responses to the information treatment should be fatalistic if (and only if) they believe they are currently HIV-positive. There is no evidence of this pattern in my sample: Column 6 of Table 6 shows that there is no statistically-significant difference in the treatment effect by people’s baseline beliefs about their HIV status. This result does not differ for people who are in the highest category of risk beliefs (not shown).

Another implication of the model is that the information treatment should shift people’s beliefs about their current HIV status or about whether they will contract HIV in the future. To examine this, I use endline data about respondent’s perceived likelihoods of current or future HIV infection. I run multinomial logits of the endline perceived likelihood variables on a treatment indicator, controlling for sampling strata and categorical indicators for the values of the baseline perceived likelihood variable. These consider the different likelihood values, as well as “Don’t Know,” as discrete choices. I estimate these regressions separately for each quantile of risk beliefs. Figure 10 reports the mean marginal effects on people reporting there is “No Likelihood” that they have HIV from these regressions, multiplied by negative 1. These can be interpreted as the effect of the information treatment on people believing there is any chance that they have HIV now (Panel A) or will get it in the future (Panel B).

I find evidence for both potential mechanisms for fatalism. The information treatment decreases the probability that people with high initial risk beliefs think there is any chance they currently have HIV by 18 percentage points compared to a control-group mean of 38%. The effect on perceiving there is any chance that you will contract HIV in the future is even stronger: it decreases by 19 percentage points. This suggests that the results presented in Figures 8 and 9 can indeed be explained by reductions in fatalism among the highest-risk group. It also implies that HIV testing may not on its own be able to eliminate fatalistic behavior: the response in terms of changes in qualitative beliefs is slightly stronger for contracting HIV in the future, rather than having it at present. The results on the perceived chance of getting HIV in the future are also robust to conditioning on respondents saying there is no likelihood that they currently have HIV. These findings demonstrate that the fatalistic responses I observe are consistent with my model of rational fatalism. They also rule out the possibility that beliefs about current HIV status alone are the sole source of fatalistic responses: perceptions about contracting HIV in the future are also important.

5.2 Is heterogeneity by beliefs driven by correlations with other variables?

The results shown in Figures 7, 8, and 9 show that responses to HIV risks vary by respondents’ baseline beliefs. However, these beliefs are not assigned at random, and therefore may be
correlated with the respondents’ other characteristics. For example, people form their risk beliefs partly through experience with sexual partners, so their sexual behavior may affect their beliefs. Also, qualitative evidence suggests that Malawi’s education system plays an important role in the formation of risk beliefs, hence it is likely that baseline risk beliefs are also capturing variation in education. As a result, it is possible that some of the heterogeneity in risk responses is coming from other factors correlated with risk beliefs, rather than from the beliefs themselves.

To explore this possibility, I run a regression of baseline HIV risk beliefs on an extensive list of demographic, socioeconomic, and sexual behavior variables measured at baseline that could plausibly play a role in shaping respondents’ beliefs. Observable factors can explain only a tiny share of the variation in beliefs: this regression (omitted for space) has an R-squared of 0.067. I also repeat the analysis from Column 2 of Table 6, including interactions between the treatment indicator and the full set of baseline covariates. The results, shown in Column 7 of the table, show no significant heterogeneity by any other baseline factor, and leave the coefficient on the interaction between the information treatment and risk beliefs nearly unchanged. Thus the heterogeneity in risk responses by baseline risk beliefs is not due to those beliefs being correlated with other respondent attributes.

5.3 Potential limitations

The estimates in Section 4 are representative of the local population in the region where the experiment took place. Because my sample was chosen to mirror the overall population, where marriage is nearly universal among sexually-active adults, almost 90% of my respondents are married. The effects I estimate, are therefore mostly for married people, and so represent changes in either marital sex or extra-marital activity. My experimental results confirm this: responses to the information treatment are not statistically different by marital status, but the magnitude of the response is much larger for married individuals (results not shown). This suggests that my results do mostly represent changes in sexual activity by married individuals.

Both changes in sexual activity within marriages and changes in infidelity are reasonable to expect in this setting, because Southern Malawi has high rates of perceived and actual infidelity. 18% of married women and 10% of married men think their spouse is unfaithful (Conroy 2014). My survey did not ask whether reported sex partners were the respondent’s spouse, in order to enhance respondents’ comfort with revealing details of their sex lives, but did instruct enumerators to record this information if the respondent happened to mention it. Nearly a quarter of married respondents volunteered this information; of those, 5% of men and 19% of women said their primary sex partner was not their spouse. As a result, both the perceived and actual risk of contracting

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51 The independent variables in this regression were three sexual behavior variables (lifetime number of sex partners, total sex acts in the past week, any sex in the past week), four measures of cognitive ability (immediate word recall, delayed word recall, numeracy quiz score, and Raven’s Progressive Matrices score) and categorical indicators for gender, marital status, age bracket, ethnic group (collapsing small cells), education level, whether respondent read a newspaper in the past week, whether respondent listened to the radio in the past week, and whether respondent watched television in the past week.
HIV from one’s spouse is high. Longitudinal studies have estimated that up to 70% of all people newly-infected with HIV in Africa are married (Gray et al. 2011). My respondents are aware of this channel of infection: baseline, 36% of married people in my sample think there is some chance their primary sex partner has HIV.

The changes in behavior that I measure should be considered in light of the risk environment my respondents face. The majority of the population, having realized sex is less risky than they thought, is more open to sex with a spouse they might see as high-risk: perhaps a husband who is away a lot, or who is rumored to have another sex partner. Alternatively, they may be more open to sex with high-risk outside partners themselves. The fatalistic group that has the highest initial risk beliefs has realized that previous unprotected sex has not, in fact, doomed them to share the fate of their high-risk sex partner, and they reduce how much sex they are willing to have with that person.

A related issue is that both the theoretical model in Section 2 and the estimates in Section 4 assume that people can independently choose how much sex they have. In reality, sexual activity is a matching market, and people must find willing partners in order to have sex. I can close the model by assuming that people have a number of opportunities for sexual activity, and can choose how many to take advantage of, with their choices ranging from zero to some upper bound. My estimated effects can then be interpreted as the partial equilibrium effect of changing the risk beliefs of a single person, or a small number of people within the community. The general-equilibrium effect of changing everyone’s beliefs would differ, and depend on how people sort into couples by their initial risk beliefs. In an additional set of analyses (not shown), I find no differential responses by village size, suggesting that the sexual markets are broader than individual villages. Thus my results are not affected by these general-equilibrium issues. I also find no differences in treatment effects by respondents’ number of lifetime sex partners nor by the length of time they have been in their current relationship.

A separate potential limitation of this paper is that it relies almost exclusively on self-reported sexual behavior as a measure of sexual risk-taking. This could conceivably bias my results, but in my specific context there is no reason to believe that there would be differential social-desirability bias across study arms: the information treatment provided no direct modeling of “good” behavior nor encouragement to behave in a specific way. While Baird et al. (2012) find that self-reports do not yield accurate estimates of treatment effects, they study a specific treatment that may have led to differential self-report bias. Their intervention was focused on keeping girls in school, and one of the treatment arms conditioned cash transfers on school attendance. It is commonly believed in Malawi that girls who become sexually active automatically drop out of school (Grant 2012). Thus respondents who are being incentivized to stay in school may be reluctant to share that they are having sex. In contrast, treatments where there is no reason to expect differential self-report bias have fewer problems: de Walque, Dow and Gong (2014) find that STI incidence measures and self-reports yield similar estimates of the effect of economic shocks on sexual activity. Beyond concerns about social-desirability bias being minimal, my approach also has the advantage
of capturing changes in behavior among high-risk individuals. This cannot be done when using STIs as outcome measures unless treatable STIs are used and individuals are treated for existing STIs at baseline.

A final potential limitation is that my analyses of heterogeneous treatment effects are potentially subject to the Deaton (2009) critique that subgroup analyses can constitute ex post “fishing expeditions.” However, that concern is mitigated due to the fact that my main theoretical results were laid out in earlier work done prior to the experiment (Kerwin 2012). I also use the same primary outcome variable as well as the same risk belief variable as I employed in the preliminary empirical analysis in that paper, limiting the number of researcher degrees of freedom involved in my analysis.

5.4 Implications for HIV Prevention Policy

The randomized treatment provided by my experiment – information about the true risk of HIV transmission – slightly increases sexual activity for most people, but sharply decreased it for people with the highest risk beliefs. The effect of the information treatment on overall HIV transmissions is therefore ambiguous: HIV transmission depends strongly on high-activity groups, who are responsible for keeping the epidemic alive and spreading it to the rest of the population (Koopman, Simon and Riolo 2005). Determining the overall effect my information treatment on the HIV epidemic would require detailed knowledge of the epidemiological model for the virus in my region, and is beyond the scope of this paper. However, it is informative to look at how risk factors for HIV transmission vary with the baseline beliefs that determine who responds fatalistically to the information treatment.

Figure 11 presents this analysis for four variables that are significant determinants of HIV prevalence and spread: age, total years of sexual activity, total lifetime sex partners, and perceiving that one may be HIV-positive. All four are positively correlated with risk beliefs, and the fatalistic group is significantly higher than the lowest risk belief category at the 0.10 level for all of them and at the 0.05 level for three of them. This suggests that people with extremely high risk beliefs may be crucial for the HIV epidemic, and that even if the information treatment increases the sexual activity of most people, it may decrease the overall spread of the virus by reducing risk-taking in this key group. A targeted information campaign, that restricted access to the information only to fatalistic people, could be even more beneficial; however, it may be difficult to prevent the information from spreading to other groups.

6 Conclusion

Empirical research on behavioral responses to health risks has traditionally assumed that responses are uniformly self-protective, and has therefore focused on mean elasticities as summaries of risk compensation across a population. I use a randomized field experiment in rural Southern Malawi to explore the validity of this assumption in the context of behavioral responses to HIV
infection risks. The experiment provided the treatment group with information on the true risk of HIV transmission from unprotected sex with an infected partner, which is much lower than most respondents thought. I find that the mean elasticity of sexual behavior with respect to HIV risk beliefs is small but statistically significant, with an elasticity of about -0.6. This is similar in magnitude to estimated responses to changes in HIV prevalence in the United States, and larger than previous estimates of prevalence elasticities in sub-Saharan Africa. However, because people do not accurately know the prevalence of HIV, I would expect the corresponding prevalence elasticity for my sample to be smaller, and possibly in line with the small measured responses for Africa. I develop a method to allow for heterogeneity in marginal effects (as opposed to just the reduced form effect of the treatment indicator) and find that the average marginal effect masks significant heterogeneity. The effect of risk beliefs on risky sex is negative (consistent with self-protective responses) for people who initially hold low risk beliefs, and becomes positive (consistent with fatalism) as initial risk beliefs become sufficiently high.

This heterogeneity is consistent with a model of rationally fatalistic behavior in which changes in perceived risks affect agent’s choices not only via the risky sex acts being chosen at present, but also through a stock of previous – or unavoidable future – risky sex acts. A rise in the per-act risk increases the marginal cost of more risky sex due to the first channel, but also raises the chance that HIV is simply unavoidable, which lowers the marginal cost of additional risk-taking. I show that for this population, fatalistic responses appear to be driven not only by people who think they already have HIV, but also by those who believe that they are doomed to contract HIV in the future - for example, because of condom breaks. This suggests that HIV testing alone may not be sufficient to eliminate fatalism.

My results imply that the use of mean marginal effects as a way to summarize the response of health behaviors to health risks may be misleading. In the case of HIV in particular, epidemiologists have found that aggregate HIV transmission is dominated by high-sexual activity individuals. As a result, the effect of an increase in the perceived risk of HIV infection on the prevalence of the virus will depend predominantly on the response of people with high sexual activity. If these individuals are fatalistic, the effect on prevalence may be the opposite of that implied by the mean marginal effect. My data suggests that this may in fact be true for HIV in Malawi: the 13.8% of people who respond fatalistically to the information treatment have an average of 4.4 lifetime sex partners, significantly higher than the rest of the population (p=0.07); they look worse in terms of other HIV risk factors as well. The extent to which mean marginal effects are a useful summary statistic for risk compensation for other health risks will depend on how many people hold extreme risk beliefs, whether the condition in question is incurable, and the dynamics of the broader economic or epidemiological system in which people are interacting.

Further research is needed on explicitly incorporating agents’ perceived risk of HIV infection into rational epidemic models of HIV, rather than just assuming agents understand the true prevalence and transmission rate of the virus. Such models should also allow for responses to perceived risks to be heterogeneous by the level of the perceived risk, rather than imposing that they are the same.
across the whole population. The formation of people’s risk beliefs is another important area for study. While anecdotal evidence suggests that people learn about HIV in school, the exact process by which many people arrive at gross overestimates of the prevalence and transmission rate of the virus is still unknown. Given that overestimating HIV risks seems to scare people to death, rather than scaring them straight, getting at the source of these overestimates may be crucial for understanding the continued spread of the African HIV epidemic.
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Figure 1
Example of HIV Risk Messaging from a Malawian Life Skills Textbook

Notes: Excerpted from the Form 4 Life Skills textbook used in Malawian secondary schools (highlighting added). The highlighted section suggests that the risk of contracting HIV from a single sex act is 100%. Author’s conversations with Malawi Ministry of Education officials confirm that the Life Skills course taught from Form 1 to Form 4 (the equivalent of US high school) is the only course that covers HIV in the country’s school system; this was the only explicit or implicit reference to HIV transmission rates found through an exhaustive review of the Life Skills textbooks and official curriculum.
Figure 2
Illustration of Tipping Point in Marginal Cost of Sexual Activity

Panel A: $P[HIV \text{ Infection—Number of Sex Acts } (y)]$ for Low and High Values of Per-Act Risk ($x$)

Panel B: $MC[\text{Sex Act } (y)—(y)]$ for Low and High Values of Per-Act Risk ($x$)

Notes: Panel A illustrates the total probability of HIV infection, as a function of the number of sex acts chosen, $y$, for different levels of the per-act risk, $x$. The solid line is initially steeper because the chance of contracting HIV from each act is higher. Both lines asymptote to 1 as $y$ goes to infinity; continuity and monotonicity therefore ensure that there exists a range (and hence at least one point) where the blue line is steeper. This leads to a tipping point combination of $y$ and $x$: below the tipping point, the marginal cost of risky sex is higher when per-act risks are higher, while above the tipping point the marginal cost is lower when the per-act risk is higher.

Panel B directly illustrates the average marginal costs for different ranges of $y$ given the two levels of the per-act risk; the mean marginal cost is higher for the lower per-act risk in the second portion of the graph, which is what generates the fatalistic range of responses.
Figure 3
Example Question about Subject’s HIV Risk Beliefs

E1a. If 100 men, who do not have HIV, each sleep with a woman who is HIV positive tonight and do not use a condom, how many of them do you think will have HIV after the night?

Number:  

E1b. If answer to E1a is 50
Do you really think that 50 of the men would get HIV, or are you just not sure?

1. I really think it’s 50
0. I’m just not sure

What is your best guess?

Notes: Example of one of the six different HIV-related expectations questions included on the survey. Enumerators were trained to ask a followup question along the lines of E1b if respondents answered 50% to any question; the data used in this paper replaces the initial response of 50 with the best guess if one was volunteered. The actual survey was conducted in Chichewa, the local language in Southern Malawi; questions were translated by bilingual experts, tested extensively, and backtranslated to ensure accuracy.
Figure 4
Measured Risk Beliefs over Time, by Study Arm
(Per-act HIV transmission rate for unprotected sex w/infected partner)

Notes: Each point represents the mean value of the risk beliefs for a given day; baseline control beliefs are hollow circles, endline control beliefs are solid circles, baseline treatment beliefs are hollow triangles, and endline treatment beliefs are solid triangles. The lines are linear fits of beliefs on date for a given date range and study arm. The light vertical line indicates the date of the training sessions when the survey enumerators were trained to provide the information treatment about HIV transmission risks. As shown on the plot, control-group baseline surveys were all conducted prior to this training session, with the exception of a handful of cleanup surveys. The pattern of Baseline beliefs suggests that the enumerators’ knowledge about the information treatment affected the data they recorded in the surveys. This theory is supported by a comparison of the Baseline Treatment beliefs (hollow triangles) with the Endline Control beliefs (solid circles). This compares the groups when both the respondents and enumerators had identical information sets: it was after the enumerators were taught the HIV risk information, the baseline survey took place before treatment-group respondents were exposed to the information treatment, and the control-group respondents were never exposed to the information treatment. The post-training session cleanup surveys for the control group also lend support to this theory (the low outlier comes from a day with just a single cleanup survey). Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed.
**Figure 5**

Histograms of Baseline HIV Infection Risk Beliefs, Control Group

**Panel A:** Per-Act Infection Risk from Unprotected Sex with an Infected Partner

**Panel B:** Per-Act Infection Risk from Unprotected Sex with a Randomly-Selected Partner

*Notes:* The two histograms plot the distribution of beliefs about the chance of contracting HIV from unprotected sex with either an infected partner (Panel A) or a randomly-selected person the respondent finds attractive (Panel B). Panel A has a large mass point at 100%. Panel B breaks up that mass point by accounting for the risk people perceive from unprotected sex with a randomly-selected partner, rather than conditioning on the partner being infected. Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed.
Notes: The two CDFs plot the distribution of beliefs about the chance of contracting HIV from a single unprotected sex act with a randomly-selected partner, separately for the treatment group (shaded red bars) and the control group (black outlined bars). This variable is constructed as $x_i = t_i \times p_i$, where $t_i$ is the perceived per-act HIV transmission rate from unprotected vaginal sex (for people of one’s own gender) and $p_i$ is the perceived prevalence of HIV among attractive members of the opposite sex from the local area. Panel A presents the raw data, while Panel B presents the data adjusted to correct for the contamination due to enumerator knowledge suggested by Figure 4. I run the regression $t_i = b_0 + b_1 AfterTraining_i + b_2 DaysAfterTraining_i + b_3 AfterTraining_i \times DaysAfterTraining_i + e_i$ and construct $t_i^{adj} = t_i^{resid} + \hat{b_0}$, bounding $t_i$ to lie within [0,1]. This preserves the scale on which the beliefs are measured. $p_i^{adj}$ is constructed likewise, and $x_i^{adj}$ is constructed as $x_i^{adj} = t_i^{adj} \times p_i^{adj}$. A comparison of the two panels reveals that the adjustment mitigates the large excess of treatment-group respondents reporting beliefs in the lowest category, but does not perfectly harmonize the two distributions. Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed.
Figure 7
First-Stage Effect of Treatment \((T)\) on Endline Risk Beliefs \((x)\),
by Baseline Risk Belief

Notes: The graph illustrates the first-stage estimate of the effect of the information treatment on endline (post-treatment) risk beliefs, decomposed by individuals’ baseline (pre-treatment) beliefs about HIV infection risks. The estimated effects on risk beliefs are negative for all levels of baseline beliefs because the true risk lies below the priors of virtually all respondents; the first stage is always negative, consistent with the monotonicity assumption. I estimate the underlying semiparametric regressions using the Robinson (1988) double-residual estimator to control for baseline values of the outcome and sampling strata; bandwidths are chosen to minimize the mean-squared error of the fitted values via the generalized cross-validation statistic of Loader (2004). See Section 4 for details on the estimation technique. The graph is restricted to Baseline Risk Belief values between 0.05 and 0.95 to mitigate boundary bias. Confidence intervals constructed via village-clustered bootstrap, with the Baseline Risk Belief variable re-generated for each resample to correct the confidence intervals for generated regressors. For each bootstrap sample, I trim observations with estimated densities below the minimum observed in the original sample. Baseline Risk Belief is the composite belief variable from Figure 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. Baseline Risk Belief is adjusted for non-constant time trends as in Panel B of Figure 6; omitting the adjustment does not change the qualitative results. Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed.
Figure 8
Reduced-Form Effect of Treatment (T) on Log Sex Acts in Past Week (ln(y)),
by Baseline Risk Belief

Notes: The graph illustrates the reduced form estimate of the effect of the information treatment on sexual behavior, decomposed by individuals’ baseline (pre-treatment) beliefs about HIV infection risks. The treatment effect is positive for most respondents but negative for people with the highest initial beliefs, suggesting rationally fatalistic behavior. I estimate the underlying semiparametric regressions using the Robinson (1988) double-residual estimator to control for baseline values of the outcome and sampling strata; bandwidths are chosen to minimize the mean-squared error of the fitted values via the generalized cross-validation statistic of Loader (2004). See Section 4 for details on the estimation technique. The graph is restricted to Baseline Risk Belief values between 0.05 and 0.95 to mitigate boundary bias. Confidence intervals constructed via village-clustered bootstrap, with the Baseline Risk Belief variable re-generated for each resample to correct the confidence intervals for generated regressors. For each bootstrap sample, I trim observations with estimated densities below the minimum observed in the original sample. Log sex in past week constructed as y' = ln(y + \sqrt{1 + y^2}) to account for zeroes. Baseline Risk Belief is the composite belief variable from Figure 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. Baseline Risk Belief is adjusted for non-constant time trends as in Panel B of Figure 6; omitting the adjustment does not change the qualitative results. Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed.
Figure 9
IV Estimates of the Elasticity of Sex Acts in Past Week (y) w.r.t. Endline Risk Beliefs (x), by Baseline Risk Belief

Notes: The graph illustrates the 2SLS estimate of the elasticity of sexual behavior with respect to endline (post-treatment) risk beliefs, decomposed by individuals’ baseline (pre-treatment) beliefs about HIV infection risks. The estimated elasticity is negative for most people but positive for the highest baseline risk beliefs, consistent with rationally fatalistic behavior. I estimate the underlying semiparametric regressions using the Robinson (1988) double-residual estimator to control for baseline values of the outcome and sampling strata; bandwidths are chosen to minimize the mean-squared error of the fitted values via the generalized cross-validation statistic of Loader (2004). See Section 4 for details on the estimation technique. The graph is restricted to Baseline Risk Belief values between 0.05 and 0.95 to mitigate boundary bias. Confidence intervals constructed via village-clustered bootstrap, with the Baseline Risk Belief variable re-generated for each resample to correct the confidence intervals for generated regressors. For each bootstrap sample, I trim observations with estimated densities below the minimum observed in the original sample. Log sex in past week constructed as $y' = \ln(y + \sqrt{1 + y^2})$ to account for zeroes. Baseline Risk Belief is the composite belief variable from Figure 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. Baseline Risk Belief is adjusted for non-constant time trends as in Panel B of Figure 6; omitting the adjustment does not change the qualitative results. Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed.
Figure 10
Multinomial Logit Estimates of Effect of Treatment on Perceived Likelihood of Having HIV Now (Panel A) in the Future (Panel B),
by Baseline HIV Transmission Risk Belief

Panel A: Unadjusted

Panel B: Adjusted to Correct for Enumerator-Knowledge Contamination

Notes: The graphs display the opposite of the mean marginal effects on the “No Likelihood” option from a multinomial logit of the categorical HIV status belief variable on a treatment indicator as well as controls for sampling strata and indicators for each category of the baseline value of the outcome; in Panel B no baseline data exists and so baseline data for “What is the likelihood that you have HIV now” are used as a proxy. Most changes are between some higher likelihood and “No Likelihood”, thus the marginal effects for the latter summarize the effect of the information treatment, in terms of changes in believing there is any chance that one currently has HIV or will get it in the future. The treatment significantly increases the rate at which people reported any likelihood of having HIV now or getting it in the future for the highest category of risk beliefs, but had no effect for the rest of the population. This suggests that the mechanism of the risk-seeking responses observed in the sample is consistent with the model of rationally fatalistic responses laid out in Section 2. The results are not changed qualitatively if the “Don’t know” category is excluded. Baseline Risk Belief is the composite belief variable from Figure 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. This is adjusted for non-constant time trends as in Panel B of Figure 6. Sample includes 1292 respondents who completed both baseline and endline surveys.
**Figure 11**

Differences in HIV Risk Factors by Baseline HIV Transmission Risk Belief

**Panel A:** Age

**Panel B:** Years Sexually Active

**Panel C:** Lifetime Sex Partners

**Panel D:** Perceives Any Likelihood of Being HIV-Positive

Notes: The graphs display the differences in baseline HIV risk factors between each risk category and the lowest one. People with the highest risk beliefs have consistently higher values for each risk factor; for all four graphs, the highest category is significantly different from the lowest category at the 0.10 level, and for three of the four the difference is significant at the 0.05 level. Baseline Risk Belief is the composite belief variable from Figure 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. This is adjusted for non-constant time trends as in Panel B of Figure 6. Sample includes 1292 respondents who completed both baseline and endline surveys.
Table 1
Demographic Covariate Baseline Balance

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Overall</th>
<th>Control</th>
<th>Treatment</th>
<th>C-T</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
<td>(5)</td>
</tr>
<tr>
<td><strong>Demographics</strong></td>
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<td></td>
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<tr>
<td>Male</td>
<td>1292</td>
<td>0.43</td>
<td>0.42</td>
<td>0.44</td>
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<tr>
<td>Married</td>
<td>1290</td>
<td>0.82</td>
<td>0.83</td>
<td>0.80</td>
<td>0.03</td>
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<tr>
<td>Age</td>
<td>1292</td>
<td>29.36</td>
<td>29.13</td>
<td>29.59</td>
<td>-0.46</td>
</tr>
<tr>
<td>Grew up in village where currently residing</td>
<td>1289</td>
<td>0.62</td>
<td>0.65</td>
<td>0.60</td>
<td>0.05</td>
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<tr>
<td>Years of education</td>
<td>1292</td>
<td>5.81</td>
<td>5.76</td>
<td>5.86</td>
<td>-0.10</td>
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<tr>
<td>Number of people in household</td>
<td>1292</td>
<td>4.95</td>
<td>5.04</td>
<td>4.87</td>
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<tr>
<td>Total children still living</td>
<td>1292</td>
<td>2.99</td>
<td>2.94</td>
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<tr>
<td>Desired future children</td>
<td>1289</td>
<td>1.36</td>
<td>1.31</td>
<td>1.41</td>
<td>-0.09</td>
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<td># media sources† used at least monthly</td>
<td>1292</td>
<td>1.18</td>
<td>1.16</td>
<td>1.20</td>
<td>-0.04</td>
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<td># common assets owned by household</td>
<td>1291</td>
<td>4.40</td>
<td>4.54</td>
<td>4.26</td>
<td>0.28</td>
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<tr>
<td>Household cash income past 30 days (PPP USD)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Baseline (C and T observed at different times of year)</td>
<td>1292</td>
<td>250.29</td>
<td>282.46</td>
<td>218.23</td>
<td>64.23**†</td>
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<tr>
<td>Endline (C and T observed simultaneously)</td>
<td>1292</td>
<td>190.28</td>
<td>201.94</td>
<td>178.66</td>
<td>23.29</td>
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<td>Household expenditure past 30 days (PPP USD)</td>
<td>1292</td>
<td>292.70</td>
<td>292.39</td>
<td>293.01</td>
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<td><strong>Religion</strong></td>
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<tr>
<td>Muslim</td>
<td>1292</td>
<td>0.07</td>
<td>0.09</td>
<td>0.06</td>
<td>0.02</td>
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<td>Christian</td>
<td>1292</td>
<td>0.89</td>
<td>0.89</td>
<td>0.89</td>
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<tr>
<td>Other</td>
<td>1292</td>
<td>0.04</td>
<td>0.03</td>
<td>0.05</td>
<td>-0.02</td>
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<td><strong>Ethnic Group</strong></td>
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<td>Nyanja</td>
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<td>0.47</td>
<td>0.46</td>
<td>0.48</td>
<td>-0.02</td>
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<td>Lomwe</td>
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<td>0.39</td>
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<td>Yao</td>
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<td>0.11</td>
<td>0.07</td>
<td>0.04</td>
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<td>Chewa</td>
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<td>0.04</td>
<td>0.05</td>
<td>0.03</td>
<td>0.02</td>
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<tr>
<td>Other</td>
<td>1292</td>
<td>0.03</td>
<td>0.04</td>
<td>0.02</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Notes: The t-tests shown in this table demonstrate that the sample is balanced on all observable demographics. The exception is income receipt at baseline due to seasonality; see (†) below.
† Media sources are newspapers, radio, and television.
‡ Baseline income differs between treatment and control respondents due to seasonal patterns in income receipt. Endline income is not significantly different for the two groups; baseline expenditure is also almost equal as a result of consumption smoothing.
Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed. Cluster-adjusted significance tests: * p< 0.1; ** p< 0.05; *** p<0.01.
Table 2
Sexual Activity Baseline Balance

<table>
<thead>
<tr>
<th>Panel A - Single-Question Recall</th>
<th>N (1)</th>
<th>Overall (2)</th>
<th>Control (3)</th>
<th>Treatment (4)</th>
<th>C-T (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Years since sexual debut</td>
<td>1275</td>
<td>13.15</td>
<td>13.10</td>
<td>13.20</td>
<td>-0.10</td>
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<tr>
<td>Total lifetime sex partners</td>
<td>1288</td>
<td>3.34</td>
<td>3.12</td>
<td>3.56</td>
<td>-0.44**</td>
</tr>
<tr>
<td>Months since last sex act</td>
<td>1252</td>
<td>4.98</td>
<td>4.73</td>
<td>5.23</td>
<td>-0.50</td>
</tr>
<tr>
<td>Any sex in the past 30 days</td>
<td>1281</td>
<td>0.73</td>
<td>0.74</td>
<td>0.73</td>
<td>0.01</td>
</tr>
<tr>
<td>Sex partners during past 30 days</td>
<td>1290</td>
<td>0.81</td>
<td>0.82</td>
<td>0.80</td>
<td>0.02</td>
</tr>
<tr>
<td>Total sex acts during past 30 days</td>
<td>1281</td>
<td>7.37</td>
<td>7.48</td>
<td>7.27</td>
<td>0.21</td>
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<tr>
<td>Any unpro. sex acts in the past 30 days</td>
<td>1281</td>
<td>0.67</td>
<td>0.67</td>
<td>0.66</td>
<td>0.00</td>
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<tr>
<td>Total unpro. sex acts in the past 30 days</td>
<td>1281</td>
<td>6.66</td>
<td>6.75</td>
<td>6.57</td>
<td>0.18</td>
</tr>
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</table>

Panel B - Retrospective Sex Diary - Sex Acts in Past 7 Days

<table>
<thead>
<tr>
<th></th>
<th>N (1)</th>
<th>Overall (2)</th>
<th>Control (3)</th>
<th>Treatment (4)</th>
<th>C-T (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any sex acts</td>
<td>1292</td>
<td>0.52</td>
<td>0.54</td>
<td>0.51</td>
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<td>Total sex acts</td>
<td>1292</td>
<td>1.71</td>
<td>1.80</td>
<td>1.62</td>
<td>0.18</td>
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<td>Any unpro. sex acts</td>
<td>1292</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.01</td>
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<tr>
<td>Total unpro. sex acts</td>
<td>1292</td>
<td>1.52</td>
<td>1.57</td>
<td>1.47</td>
<td>0.10</td>
</tr>
<tr>
<td>Sex with more than one partner</td>
<td>1292</td>
<td>0.01</td>
<td>0.02</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Total sex acts with non-primary partners</td>
<td>1292</td>
<td>0.02</td>
<td>0.03</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Any unpro. sex acts with non-primary partners</td>
<td>1292</td>
<td>0.01</td>
<td>0.01</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Total unpro. sex with non-primary partners</td>
<td>1292</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Notes: The t-tests presented in Column 5 suggest that the treatment and control group are well-balanced on observed sexual behavior. Because there are small differences between the two groups, however, controlling for baseline values of the outcome will reduce in less-biased regression estimates of treatment effects. Panel A shows data collected by the standard single-question recall method. Panel B shows data collected by a retrospective sex “diary” that walks respondents through the previous 7 days and asks them questions about a range of activities, both sexual and non-sexual, and collects details for each sex act. Sample is 1292 people from 70 villages for whom both baseline and endline surveys were successfully completed. Cluster-adjusted significance tests: * p< 0.1; ** p< 0.05; *** p<0.01.
### Table 3
Regression Estimates of Effect of HIV Transmission Rate Information on HIV Risk Beliefs

<table>
<thead>
<tr>
<th>Perceived HIV Transmission Rate, if Partner Infected</th>
<th>Perceived HIV Prevalence</th>
<th>Composite Beliefs: P(Contract HIV from Unprotected Sex w/Random Attractive Person)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-0.408***</td>
<td>-0.048***</td>
<td>-0.381***</td>
</tr>
<tr>
<td>(0.019)</td>
<td>(0.006)</td>
<td>(0.016)</td>
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<tr>
<td>Observations</td>
<td>1,284</td>
<td>1,284</td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.273</td>
<td>0.036</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Perceived HIV Transmission Rate, if Partner Infected</th>
<th>Perceived HIV Prevalence</th>
<th>Composite Beliefs: P(Contract HIV from Unprotected Sex w/Random Attractive Person)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-0.384***</td>
<td>-0.045***</td>
<td>-0.371***</td>
</tr>
<tr>
<td>(0.019)</td>
<td>(0.006)</td>
<td>(0.016)</td>
</tr>
<tr>
<td>Observations</td>
<td>1,281</td>
<td>1,283</td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.315</td>
<td>0.066</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Perceived HIV Transmission Rate, if Partner Infected</th>
<th>Perceived HIV Prevalence</th>
<th>Composite Beliefs: P(Contract HIV from Unprotected Sex w/Random Attractive Person)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-0.316***</td>
<td>-0.022*</td>
<td>-0.336***</td>
</tr>
<tr>
<td>(0.023)</td>
<td>(0.011)</td>
<td>(0.018)</td>
</tr>
<tr>
<td>Observations</td>
<td>1,281</td>
<td>1,283</td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.314</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Control Mean(De. Var) | 0.742 | 0.082 | 0.905 | 0.176 | 0.485 | 0.463 | 0.351 | 0.424 |
Control SD(De. Var) | 0.318 | 0.162 | 0.198 | 0.264 | 0.290 | 0.265 | 0.268 | 0.263 |

Notes: This table shows the information treatment has a strong negative effect on HIV risk beliefs that is robust different regression specifications. The treatment group received this information while the control group did not. Respondents update all their HIV-related beliefs, not just the one covered by the information treatment (the annual risk of infection from unprotected sex with an infected partner). This suggests that people learned and processed the information, and updated their other beliefs based on their new knowledge. All regressions include controls for sampling strata (distance category X gender). Panel A uses a simple regression of the endline value of the belief variable; Panel B adds controls for raw baseline values of the belief variable (not adjusted for enumerator contamination); Panel C uses the change in the belief variable from baseline to endline as the outcome.

† The question asked respondents to imagine couples having typical sexual behavior over the course of one year.
‡ Prevalence belief variables are questions specifically about members of the opposite sex.
Sample includes 1292 respondents who completed both baseline and endline surveys. Heteroskedasticity-robust standard errors, clustered by village, in parentheses. * p< 0.1; ** p< 0.05; *** p< 0.01
Table 4  
Regression Estimates of the Effect of Information about HIV Transmission Risks on Sexual Behavior

<table>
<thead>
<tr>
<th></th>
<th>Any Sex in Past Week</th>
<th>Log Sex Acts in Past Week</th>
<th>Log Unprotected Sex Acts in Past Week</th>
<th>Log Sex Partners in Past 30 Days</th>
<th>Log Condoms Acquired in Past 30 Days</th>
<th>Log Condoms Purchased</th>
<th>Log Overall Sexual Activity Index†</th>
<th>Log Diary Sexual Activity Index†</th>
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</thead>
<tbody>
<tr>
<td>Treatment Group</td>
<td>0.050**</td>
<td>0.101**</td>
<td>0.071</td>
<td>0.012</td>
<td>0.080</td>
<td>0.054</td>
<td>0.063*</td>
<td>0.057**</td>
</tr>
<tr>
<td></td>
<td>(0.024)</td>
<td>(0.047)</td>
<td>(0.045)</td>
<td>(0.019)</td>
<td>(0.075)</td>
<td>(0.105)</td>
<td>(0.032)</td>
<td>(0.024)</td>
</tr>
<tr>
<td>Observations</td>
<td>1.292</td>
<td>1.292</td>
<td>1.292</td>
<td>1.290</td>
<td>1.283</td>
<td>1.286</td>
<td>1.261</td>
<td>1.292</td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.238</td>
<td>0.277</td>
<td>0.260</td>
<td>0.288</td>
<td>0.140</td>
<td>0.047</td>
<td>0.378</td>
<td>0.225</td>
</tr>
<tr>
<td>Ctrl Mean(Dep. Var)</td>
<td>0.490</td>
<td>1.67</td>
<td>1.48</td>
<td>0.77</td>
<td>2.52</td>
<td>5.08</td>
<td>-0.03</td>
<td>-0.02</td>
</tr>
<tr>
<td>Ctrl SD(Dep. Var)</td>
<td>0.500</td>
<td>2.39</td>
<td>2.29</td>
<td>0.58</td>
<td>9.65</td>
<td>6.59</td>
<td>0.99</td>
<td>1.03</td>
</tr>
</tbody>
</table>

Notes: Results illustrate that the information treatment had a small but statistically-significant effect on sexual behavior, increasing risky activities by 5 to 10 percentage points for most outcomes. I can reject effects above 20 percentage points in magnitude.

† The Sexual Activity Index variables are weighted averages of normalized values of all available outcome measures (Column 7) or just the outcomes measured on the Sex Diary, which are measured with less noise (Column 8). The weights used are factor loadings for the first principal component of the outcomes for the control group. Alternative indices using equal weights yield comparable, but slightly smaller, magnitudes.

Logged variables are constructed as \( y' = \ln(y + \sqrt{1 + y^2}) \) to account for zeroes. All regressions include controls for sampling strata (distance category X gender). All regressions also control for baseline values of the outcome variable; the exception is Log Condoms Purchased (Column 6), where baseline Log Condoms Acquired in Past 30 Days was used as a proxy because condoms were not sold at baseline. Sample includes 1292 respondents who completed both baseline and endline surveys. Heteroskedasticity-robust standard errors, clustered by village, in parentheses. * p< 0.1; ** p< 0.05; *** p<0.01.
Table 5
OLS and 2SLS Estimates of the Partial Effect of Endline Risk Beliefs on Sexual Activity

<table>
<thead>
<tr>
<th>Any Sex in Past Week</th>
<th>Log Sex Acts in Past Week</th>
<th>Log Unprotected Sex Acts in Past Week</th>
<th>Log Sex Partners in Past 30 Days</th>
<th>Log Condoms acquired in past 30 days</th>
<th>Log Condoms Purchased</th>
<th>Log Overall Sexual Activity Index</th>
<th>Log Diary Sexual Activity Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panel A: OLS Estimates (Control Group Only)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endline Risk Belief</td>
<td>0.155*** (0.054)</td>
<td>0.175* (0.102)</td>
<td>0.106 (0.103)</td>
<td>0.196*** (0.058)</td>
<td>0.118 (0.172)</td>
<td>-0.337 (0.224)</td>
<td>0.318*** (0.100)</td>
</tr>
<tr>
<td>Observations</td>
<td>627</td>
<td>627</td>
<td>627</td>
<td>626</td>
<td>626</td>
<td>617</td>
<td>617</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.210</td>
<td>0.277</td>
<td>0.240</td>
<td>0.258</td>
<td>0.165</td>
<td>0.049</td>
<td>0.340</td>
</tr>
<tr>
<td>Panel B: 2SLS Estimates</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endline Risk Belief</td>
<td>-0.260** (0.121)</td>
<td>-0.562** (0.241)</td>
<td>-0.412* (0.232)</td>
<td>-0.043 (0.102)</td>
<td>-0.375 (0.402)</td>
<td>-0.256 (0.535)</td>
<td>-0.327** (0.159)</td>
</tr>
<tr>
<td>Observations</td>
<td>1,252</td>
<td>1,252</td>
<td>1,252</td>
<td>1,250</td>
<td>1,246</td>
<td>1,222</td>
<td>1,252</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.208</td>
<td>0.256</td>
<td>0.253</td>
<td>0.277</td>
<td>0.129</td>
<td>0.046</td>
<td>0.361</td>
</tr>
<tr>
<td>1st-Stage F-Statistic</td>
<td>222.0</td>
<td>220.7</td>
<td>221.3</td>
<td>222.7</td>
<td>221.3</td>
<td>218.1</td>
<td>226.5</td>
</tr>
</tbody>
</table>

Notes: 2SLS estimates use the randomized treatment group assignment as an instrumental variable for endline beliefs. The results indicate that the elasticity of sexual activity with respect to HIV risk beliefs is between -0.3 and -0.6. OLS estimates use the endline data for the control group only, to estimate the relationship that would be observed in the absence of any exogenous variation in risk beliefs.

† The Sexual Activity Index variables are weighted averages of normalized values of all available outcome measures (Column 7) or just the outcomes measured on the Sex Diary, which are measured with less noise (Column 8). The weights used are factor loadings for the first principal component of the outcomes for the control group. Alternative indices using equal weights yield comparable, but slightly smaller, magnitudes.

Logged variables are constructed as $y' = \ln(y + \sqrt{1+y^2})$ to account for zeroes. Endline Risk Belief is the composite belief variable from Column 7 of Table 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. All regressions include controls for sampling strata (distance category X gender) and baseline values of risk beliefs. All regressions also control for baseline values of the outcome variable; the exception is Log Condoms Purchased (Column 6), where baseline Log Condoms Acquired in Past 30 Days was used as a proxy because condoms were not sold at baseline. Sample includes 1292 respondents who completed both baseline and endline surveys. Heteroskedasticity-robust standard errors, clustered by village, in parentheses. * p< 0.1; ** p< 0.05; *** p<0.01.
Table 6
Non-Monotonic Responses to Information Treatment Effects by Baseline Risk Beliefs

<table>
<thead>
<tr>
<th>Treatment (T)</th>
<th>0.101**</th>
<th>0.320***</th>
<th>0.123*</th>
<th>0.070</th>
<th>0.136**</th>
<th>0.095</th>
<th>0.412</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(0.047)</td>
<td>(0.083)</td>
<td>(0.072)</td>
<td>(0.057)</td>
<td>(0.060)</td>
<td>(0.062)</td>
<td>(0.309)</td>
</tr>
<tr>
<td>T*(Baseline Risk Belief [0-1])†</td>
<td>-0.499***</td>
<td>-0.477***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.162)</td>
<td>(0.168)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T*(Male)</td>
<td>-0.049</td>
<td>-0.039</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.131)</td>
<td>(0.155)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T*(Baseline Log Sex Acts in Past Wk.)</td>
<td>0.035</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.051)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T*(Ever Exposed to HIV)</td>
<td>-0.151</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.113)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T*(Any Chance I am HIV-positive)</td>
<td>0.009</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.115)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T Interacted with Other Baseline Covariates‡</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observations</td>
<td>1.292</td>
<td>1.275</td>
<td>1.292</td>
<td>1.292</td>
<td>1.275</td>
<td>1.277</td>
<td>1.245</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.277</td>
<td>0.284</td>
<td>0.277</td>
<td>0.277</td>
<td>0.277</td>
<td>0.276</td>
<td>0.345</td>
</tr>
</tbody>
</table>

Notes: Results illustrate that there is substantial heterogeneity in responses to the information treatment by baseline HIV risk beliefs, but not by any other baseline covariate. This heterogeneity is robust to including interactions between the treatment indicator and a wide range of other baseline covariates.

†Baseline Risk Belief is the composite belief variable from Column 7 of Table 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. This is adjusted for non-constant time trends as in Panel C of Table 5; omitting the adjustment does not change the qualitative results.

‡Other baseline covariates include immediate and delayed word recall [each 0-10], numeracy score [0-3], score on Raven's progressive matrices [0-3], lifetime sex partners, whether respondent had any sex in the past week, and indicators for marital status, age category, ethnic group, education level, frequency of listening to the radio, frequency of watching television, frequency of reading the newspaper.

All regressions include controls for baseline values of the outcome, and sampling strata (distance category X gender). In each specification, the factor being interacted with the treatment dummy also enters into the regression in levels. Logged variables are constructed as \( y' = \ln(y + \sqrt{1 + y^2}) \) to account for zeroes. Sample includes 1292 respondents who completed both baseline and endline surveys.

Heteroskedasticity-robust standard errors, clustered by village, in parentheses. * p < 0.1; ** p < 0.05; *** p < 0.01. Standard errors in Columns 2 and 7 are cluster-bootstrapped to correct for generated regressors.