

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

Jason T. Kerwin^{*}
University of Michigan
October 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 show that fatalism is a rational response to sufficiently high health risks if people do not have perfect control over all possible exposures, and if the health risk 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 using a randomized field experiment that provided information on HIV transmission risks to people in Malawi. Average risk responses are self-protective and statistically significant, but small in magnitude: the mean risk elasticity of sexual behavior is roughly -0.6. Consistent with the model, this elasticity varies sharply by baseline risk beliefs. I develop a method of decomposing 2SLS estimates of the risk elasticity of sexual behavior by people's baseline risk beliefs, and find that the risk elasticity varies 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, which is consistent with fatalism.

^{*}University of Michigan Department of Economics and Population Studies Center. Email jtkerwin@umich.edu. I am grateful to Rebecca Thornton, Jeff Smith, John DiNardo, David Lam, Matias Cattaneo, Bob Willis, Mel Stephens, John Bound, Miles Kimball, Susan Watkins, Ophira Vishkin, Evan Herrnstadt, Joe Golden, Audrey Dorélien, Laura Derksen, Eric Chyn, Olga Malkova, Justin Ladner, and seminar participants at the University of Michigan, MEA, and PacDev for their valuable feedback. 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 remaining mistakes 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 declines in the risks associated with certain behaviors are often offset by rational increases in risk-taking. Empirical research on risk compensation typically assumes that people are uniformly risk-avoiding – that when the per-act risk of an activity goes up, people 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 per-act risks rise.

Strictly risk-avoiding behavior is always rational if the expected cost of the 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 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-

¹ This 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.

² This 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 2012) 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.

³ This 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.

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 a rural area of Southern Malawi, an area with a severe HIV epidemic where qualitative evidence suggests that some people are responding fatalistically to the virus.⁴ 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, and 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.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 estimate is larger in magnitude than estimates of the response of sexual behavior to the prevalence of HIV in sub-Saharan Africa, and comparable to estimates for the United States. However, because people do not accurately know the 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.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, and rejects a monotonically negative response. 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

⁴ E.g. Kaler (2003), Kaler and Watkins (2010)

⁵ People in Malawi greatly overestimate how easily HIV is transmitted: the actual rate is just 0.1%.

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.

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. This fatalistic group also has a higher-than-average baseline number of lifetime sex partners, and elevated measures for other HIV risk factors as well, suggesting that they may be more important in driving the overall prevalence of HIV. This means that the effect of the status quo policy – in 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 understanding 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 world⁷ as well as in developing countries.⁸ I demonstrate that these subjective beliefs 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 relates to research on rational models of HIV testing and sexual behavior. 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

⁷ E.g. Lillard and Willis (2001), Manski (2004)

⁸ Attanasio (2009)

logic applies to those who learn they are HIV-negative. Experimental research on HIV testing and sexual behavior has found conflicting results: Thornton (2008) finds zero effects for HIV-negatives and small reductions in risk-taking for HIV-positives in Malawi, while Gong (2013), studying Kenya, finds that people who are surprised by an HIV-positive result increase their level of risky sexual behavior as a result. Both results are reconcilable with a model in which HIV-positive people are altruistic, but some are fatalistic about transmitting the virus to their sex partners.

Fourth, it helps reconcile the substantial responses to HIV risks found in America with very small ones in Africa.⁹ 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.¹⁰ 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,¹¹ even though overall responses are small in magnitude.

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 discuss the mechanisms behind my results and address some potential limitations of this paper. Section 6 concludes. All appendix material can be found in the Online Appendix to the paper.¹²

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

⁹ See e.g. Ahituv et al. (1996) for estimates of HIV risk compensation in the United States and Oster (2012) for Africa.

¹⁰ White and Stephenson (2014) find that 90% of gay men in the US believe the prevalence of HIV among their sex partners to be 10% or less. While no data for other African countries is available, multiple different studies across Malawi have found the perceived prevalence of HIV to be 40% or higher on average (Delavande and Kohler 2009; Kerwin et al. 2011); the same is true in the data for this study.

¹¹ Godlonton et al. (2012) 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.

¹² http://www-personal.umich.edu/~jtkerwin/Papers/JMP/Kerwin_JMPAppendix_Latest.pdf

to risks, HIV or otherwise, relies on an 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 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 depending on an agent’s risk beliefs and risk-taking time profile. In Section 2.1, I lay out the basic form of the model. I focus on the function that aggregates per-act risks into a subjective belief about HIV infection, and in particular on the properties it must satisfy in order to produce sensible probabilities. Using those properties, I then derive the key comparative static, which is the response of sexual risk-taking to the perceived per-act risk of HIV infection (Section 2.2). I show that this response is initially self-protective (consistent with the previous empirical literature) but becomes fatalistic for sufficiently-high per-act risks.

2.1 Model Basics

In this model, I assume that people weigh the benefits of choosing a level of risky sex, y , against its costs. These costs include both a fixed per-act price (or time cost, or emotional cost) q , and a stochastic component due to the risk of HIV infection. An agent’s *perceived* risk per sex act or “riskiness” 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 risky acts that are unavoidable, m_1 . The latter captures accidental exposures through things like condom breakage as well as situations where an agent may lack the bargaining power to turn down some future sex acts.

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 health conditions, and depends on perceived rather than actual irreversibility. 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 y 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 focus on a one-shot, static model, collapsing the future into the cost of HIV infection c . The results in this section can be generalized to a multi-period setting – see Appendix ID for details. The single-period optimization problem is:

$$\max_{y \geq 0} \{U(y; x, m_0, m_1, q, c)\} = \max_{y \geq 0} \{B(y) - qy - P(x, y + m_0 + m_1)c\} \quad (1)$$

By the assumption that y is continuous, the maximand $U(n; m_0, m_1, p, c, x)$ is the 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.¹³ The subjective probability also approaches 1 as riskiness rises toward 1 and total risk-taking goes to infinity.¹⁴

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 ID.

For most possible functional forms of $B(\cdot)$ and $P(\cdot, \cdot)$ this optimization problem will have no 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 $y \rightarrow \infty$. A sufficient condition for interior optima is that $q > 0$, so there is some fixed price or time cost to risky sex.¹⁵

2.2 Comparative Statics

Given the existence of an interior solution, we are interested in a specific

¹³ 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 $P_2(0, y + m_0 + m_1) = 0$.

¹⁴ $P \rightarrow 1$ as $y + m \rightarrow \infty$ as long as $x > 0$, and $P = 1$ if $x = 1$ and $y + m_0 + m_1 \neq 0$.

¹⁵ See Appendix IA for a proof.

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:

$$\begin{aligned} B'(y^*) - q - P_2(x, y^* + m_0 + m_1)c &= 0 \\ B''(y^*) - P_{22}(x, y^* + m_0 + m_1)c &\leq 0 \end{aligned}$$

The first-order condition is equivalent to there being a function $G(y^*, x, m, q, c) = B'(y^*) - q - P_2(x, y^* + m) * c = 0$. Therefore the implicit function theorem allows us to compute the comparative static for changes in y^* in response to changes in x :

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

The denominator is just the left-hand side of the second-order condition, and is thus negative.¹⁶ Since $c > 0$, $\text{sign}(\partial y^*/\partial x) = -\text{sign}(P_{21}(x, y^* + m_0 + m_1))$. As noted in Section 1, it is typical 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 this case $P_{21} = 1$ and $P_{21}(x, y^* + m_0 + m_1) > 0$ 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 riskiness of each act rises.

However, the linear approximation specified for the functional form of $P(x, y + m_0 + m_1)$ does not, in general, 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. This is the case when a condition is readily curable: future or past risks will not aggregate into a combined probability of being infected, because the agent will just cure their condition (setting $P(x, 0 + m_0 + m_1) = 0$). 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. One way of imposing that probabilities are sensible 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

¹⁶ 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.

$\pi_{12} > 0$ if $y + m_0 + m_1 < \frac{1}{-\ln(1-x)}$ and $\pi_{12} < 0$ if $y + m_0 + m_1 > \frac{1}{-\ln(1-x)}$. 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 $y + m$. 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 $P(r, y + m)$ that satisfies the basic conditions laid out in Section 2.1. This fact is proven formally in Appendix IB, 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.¹⁷ Formally, this can be written as follows:

Proposition 1

$\exists \tilde{x} = \tilde{x}(y+m_0+m_1)$ s.t. $P_{12}(x, y+m_0+m_1) > 0$ if $x < \tilde{x}$ and $P_{12}(x, y+m_0+m_1) < 0$ 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 I 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 $y + m = 0$, and guarantees that the tipping point value \tilde{x} that changes the sign of P_{12} to negative will be somewhere below 1. Proposition 1 then

¹⁷ The results here technically rely on $P(x, y + m)$ being continuous, but as discussed in Appendix IC it is possible to reach similar conclusions even if people use heuristic methods for aggregating risks into total probabilities that are not continuous.

implies that $\frac{\partial y^*}{\partial x}$ will itself have a tipping point:

Proposition 2: Comparative static of y with respect to 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. It is a purely rational alternative to the psychologically-driven fatalism derived by Caplin (2003). In his model, people do not compensate away from extremely high risks because not responding lets them ignore the problem and not experience the stress and fear associated with it. In mine, they 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.¹⁸ 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 messaging targeted at HIV-positive people emphasizes the risk of “reinfection” with a different strain of HIV.¹⁹ Actual cases of reinfection are rare enough that the medical importance of this possibility is unclear (Smith et al. 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.²⁰

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

¹⁸ See e.g. the “Quitting Timeline” from the British National Health Service (2013).

¹⁹ For example the webpage HIV Reinfection - Positive Prevention - Reinfection (2007) at <http://aids.about.com/cs/safesex/a/reinfection.htm>

²⁰ There is also suggestive empirical evidence of fatalism in the African HIV epidemic. Wilson et al. (2012) find that men in Kenya reduce their levels of sexual activity after being circumcised. The authors suggest that this is consistent with a decline in fatalism, but without direct evidence on the men's beliefs about the transmission rate of the virus, they cannot test this theory.

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 risky behavior. In my empirical analysis in Section 4, I will 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. 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 (RCT) 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.²¹ Participants from half of the villages, chosen at random, were also read an information script that presented the actual annual risk of HIV transmission in serodiscordant

²¹ Knowledge 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). The latter figure may be an underestimate: in my survey data, virtually all respondents stated that condoms provided at least some risk benefit.

couples (relationships with one HIV-positive and one HIV-negative partner) 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 (NSO) GIS files for the 2008 Census. I began by removing all duplicate village entries from the dataset.²² 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.²³ 24 of the sampled villages (34%) were within 2 km of a trading center²⁴; another 24 (34%) were within 2 and 5 km from a trading center; 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; it is common for people in this area of Malawi to travel during the agricultural off-season to look for casual wage labor. 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

²² The 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.

²³ Trading centers were identified based on the 2008 Malawi Population and Housing Census, which codes peri-urban areas outside the main cities with enumeration area numbers from 800 to 899. I included trading centers both inside the TA as well as in other nearby parts of the Southern Region. 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, several more trading centers in the local area were included, that do not have enumeration area codes between 800 and 899 but that are nonetheless major centers for trade and nightlife.

²⁴ In 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 to drink and search for sex partners.

sexually active adults, the survey contained an early screening question that eliminated people who had never had sex from the sample. This included 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 1292.²⁵ 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; these results are robust to running the regressions as logits instead of LPMs.²⁶ There is also no evidence of differential attrition by baseline covariates, which I examine by interacting the treatment indicator with different baseline variables.²⁷

Baseline summary statistics for the overall sample, as well as a comparison of the treatment and control groups, are presented in Table 1.²⁸ 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 largely attributed to seasonality in income combined with the differential timing of the baseline surveys (for reasons discussed in Section 3.2 below, the control group baseline surveys were done first and the 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 than 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

²⁵ See Appendix Table III-1 for detailed figures on the number of people in each study arm and sampling stratum.

²⁶ See Appendix Table III-2.

²⁷ See Appendix Table III-3.

²⁸ 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 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 II.

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.²⁹ 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.³⁰ 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.³¹ 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. It is 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 serodiscordant 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-

²⁹ The actual figure for the Wawer et al. study is 235 couples, 188 of which never used condoms when they had sex (results are not broken out by condom use, but condom use was very inconsistent and had no impact on the estimated transmission rate). The time period was actually 10 months, with some couples being observed for multiple time windows. This was reduced to 100 couples over the course of 1 year for the sake of clarity and simplicity, and to match the 10% per year figure cited by Malawi's National AIDS Commission.

³⁰ This is the annual transmission rate cited by the Malawi National AIDS Commission. The exact annual rate implied by the Wawer results is 12%. The Hollingsworth et al. (2008) reanalysis of the Wawer data finds an annual transmission rate of 10.6% from asymptomatic partners (HIV-positive sex partners who have not just recently contracted the virus and do not yet have AIDS), which are the majority of cases, but does not provide an overall average.

³¹ The questions respondents asked were recorded on the baseline survey. All my results are robust to excluding respondents who asked any follow-up questions.

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 was based on Godlonton et al. (2012). 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 retrospective sexual “diary” that walks respondents back through the previous seven days to collect data about a range of activities, focusing specifically on sex. It then asks detailed questions about each reported sex act.

The baseline and endline surveys contained two broad categories of self-reported sexual behavior.³² The first were single-question recall measures, for example: “In the past 30 days, how many total times did you have sex, including serious and non-serious partners?” The second comprised a detailed retrospective sexual diary, which 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 gave to or received from their partner,³³ 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.

This 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 et al. 2001). Luke et al. (2009) 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

³² Throughout this paper I use “sex” to refer to vaginal intercourse. Other forms of sexual activity are quite uncommon in Malawi are potentially sensitive topics (cf. Kerwin, Foley, and Thornton 2014), so they were not included in the survey.

³³ The 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.

substantially more heaping at multiples of 5 in the single-question recall variable³⁴. Given the lower quality of the single-question recall variables, and because I used the sex diary variables in an earlier working paper that I wrote prior to the experiment (Kerwin 2012WP), 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 part of the motivation for controlling 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 Godlonton et al. 2012, Kling et al. 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 in Section 3.2 above provides more accurately-measured outcomes than the single-question recall variables that comprise my other available outcome variables. An alternative index includes both the sex diary outcomes as well as all other outcomes that can be constructed from the survey.

Each index is constructed separately for the baseline and endline waves by normalizing all component variables (subtracting the sample mean and the dividing by

³⁴ See Appendix IV for histograms and a discussion of the implications of heaping for regression estimates.

the sample 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.³⁵

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.³⁶ 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 et al. 2011, Delavande 2014). Specifically in Malawi, Delavande and Kohler (2009) have developed a method of eliciting subjective expectations that they have shown performs very well. Their approach, implemented starting with the 2006 wave of the Malawi Longitudinal Study of Families and Health (MLSFH) relies on training respondents to report subjective beliefs by choosing how many of ten beans to place on a plate indicating a belief that an event will happen (with 0 beans meaning a 0% chance and 10 beans indicating a 100% chance), using common events in Malawi as reference points.

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

³⁵ I also explored unweighted averages; these produce similar results with slightly smaller magnitudes.

³⁶ E.g. Ahituv et al. (1996) in the US and Oster (2012) and Juhn et al. (2009) in Africa

having sex with an HIV-positive man.³⁷

I use these concrete expectation 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 areas³⁸ as well as in areas of rural southern Malawi near my study site.³⁹ 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.⁴⁰ 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 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.⁴¹ 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,⁴² and 85.9% per act using Delavande and Kohler’s method.

3.5 Focal probabilities

One potential concern with eliciting subjective expectations is the tendency for probabilities to heap at the “focal” probability of 50%. The typical interpretation, cited

³⁷ Six HIV risk belief variables that 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.

³⁸ Chinkhumba et al. (2012)

³⁹ Godlonton et al. (2012), Kerwin et al. (2011)

⁴⁰ Author’s calculations based on Chinkhumba et al. (2012)

⁴¹ The annual question for DK 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%.

⁴² The measured probability differs for the treatment group; see Section 4.3.2 for details.

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 et al. 2010). 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. The measure of risk beliefs I rely on uses the results of the main question, but replaces 50% with the expectation measured in the followup question for people who say they are just not sure, and change their mind when prompted for their best guess.

3.6 Enumerator-knowledge contamination of measured beliefs

As described in Section 3.1 above, 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 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 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 III-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 the enumerator effects, no matter what specification is used.

There are two potential explanations for this pattern. One is that different knowledge may have lead 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 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 led 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 yellow 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 yellow 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 yet been told the HIV risk information). This is reflected in the recorded values, which look the same in the two groups.⁴³

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:

$$x_{i0} = \rho_0 + \rho_1 Date_i + \rho_2 After_i + \rho_3 After_i * Date_i + v_i$$

$Date_i$ 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 $x_{i0}^{Adj} = x_{i0}^{resid} - \hat{\rho}_0$, 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 III-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

⁴³ Panel B of Appendix Table III-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.

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.

3.7 Composite belief measures

I also construct composite measures of the perceived risk of HIV infection from unprotected sex with any partner, rather than one that is specifically HIV-positive. This involves multiplying the perceived prevalence of HIV by the perceived risk of contracting the virus conditional on an HIV-positive partner. I focus on the product of the perceived per-act risk of HIV transmission from unprotected sex with an infected part and the perceived prevalence of HIV among attractive people of the opposite gender. I do this for three reasons.

First, I use perceived HIV prevalence among attractive people of the opposite sex to mitigate concerns about people's *self*-beliefs about risks differing 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 (e.g. Wiswall and Zafar 2014). For risks, this commonly takes the form of unrealistically optimistic beliefs (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 total 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, using the product of per-act transmission risks and HIV prevalence gives a natural measure of how risky a respondent believes unprotected sex to be. While this will not apply to any specific sex partner or prospective sex partner, it does capture the general level of HIV infection risk that respondents believe they face when having unprotected sex. If people think that the transmission rate of HIV is high, but that almost none of their prospective sex partners is infected, then the effective perceived risk they face is in fact quite low.

Third, relying on variation in the other beliefs allows us 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 than 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 would hide 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. 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 above in Section 3.7. In each panel the blue line shows the control group's beliefs while the red 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 had 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 has decreased even further, by 41 percentage points.⁴⁴ Note that the respondents do not update their beliefs perfectly: the actual annual transmission rate is about 10%; just 2% of the sample reports beliefs low enough to be consistent with the true values (given that the questions have a minimum granularity of 1%). Panels B and C present alternative estimates of the treatment effects, 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 condom-protected sex and about 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 or on 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 (1991) 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 (a difference-in-differences or value-added regression). 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 my outcome variables, this is the preferred estimator for my

⁴⁴ 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.

sample. The specifications used in this paper will also control for the stratification cells (combinations of distance categories and gender) used to draw the original sample, which can be shown to improve statistical efficiency (Bruhn and McKenzie 2009). My regressions have the following form:

$$y_i^e = \alpha + \beta T_i + \gamma y_i^b + \eta Z_i + e_i$$

where y_i^e is the endline value of the outcome variable, T_i is an indicator of whether the respondent was in the treatment group, y_i^b is the baseline value of the outcome variable, Z_{i0} 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.⁴⁵ 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 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.⁴⁶ In the regression

$$y_i^e = \alpha + \delta x_i^e + \gamma y_i^b + \eta Z_i + e_i,$$

$\hat{\delta}$ is an estimate of $\partial y / \partial x$, the partial effect of risk beliefs on risky sex. The results of these regressions are shown in Panel A of Table 5, and discussed below. However, for these estimates to be consistent, x_i^e 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

⁴⁵ Because many outcomes contain zeroes, I use the inverse hyperbolic sine transformation of Burbidge et al. (1988) rather than logging the variable directly, constructing $\log_{ihs}(y) = \ln(y + \text{sqrt}(y^2 + 1))$.

⁴⁶ 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 as well.

based in part on sexual experience, and sexual experience is highly autocorrelated. Another, noted by Oster (2012), is that the subjective risk will probably have some association with the actual prevalence of HIV – but that 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_i^e . T_i is plausibly excludable. 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 in the first-stage regressions is roughly 220 for all specifications.⁴⁷ This allows me to estimate two-stage regressions as follows:

$$\begin{aligned} x_i^e &= \alpha + \beta T_i + \gamma y_i^b + \rho x_i^b + \eta Z_i + e_i \\ y_i^e &= \alpha + \delta \hat{x}_i^e + \gamma y_i^b + \rho x_i^b + \eta Z_i + e_i \end{aligned}$$

x_i^b is included as a control in the first stage in order to improve efficiency and reduce bias, for the same reason discussed in Section 4.1 above.

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, and 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).

⁴⁷ 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.

This result is closer to the Ahituv et al. (1996) estimates for the US, which find elasticities of about -0.2 for binary outcomes. Using a continuous measure, and focusing on gay men in San Francisco, Auld (2006) estimates an elasticity of -0.5. However, my results are not strictly speaking 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.

Both the population-average ITT effects and marginal effects fit a model of self-protection, 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 Heterogeneous impacts 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:

$$y_i^e = \alpha + \beta T + \delta_1 w_i^1 + \sum_{j=2}^J [\beta^{T w^j} T_i w_i^j + \delta_j w_i^j] + \gamma y_i^b + \eta Z_i + e_i$$

Here w_i^1, \dots, w_i^J are a set of J baseline covariates. My primary focus is on heterogeneity by baseline risk beliefs (x_i^b). 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). 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.

In Columns 3 through 6 I look for heterogeneous responses by gender,⁴⁸ baseline sexual activity, perceived previous exposure to HIV,⁴⁹ and whether the respondent believes he or she may currently be HIV-positive.⁵⁰ 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:

$$\begin{aligned} y_i^e &= \beta^T + f^T(w_i) + \gamma^T y_i^b + \eta^T Z_i + \varepsilon_i \text{ if Treatment} = 1 \\ y_i^e &= \beta^C + f^C(w_i) + \gamma^C y_i^b + \eta^C Z_i + \nu_i \text{ if Treatment} = 0 \end{aligned}$$

These regressions give me estimates of $\mathbb{E}[y|T=1]$ and $\mathbb{E}[y|T=0]$ for each value of w_i . Thus taking the difference gives me estimates of the w -specific treatment effect $\hat{\tau}_y(w_i) = \hat{f}^T(w_i) - \hat{f}^C(w_i)$.⁵¹

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

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

The conditional expectations of y_i^e given w_i and of the controls given w_i are estimated by separate non-parametric regressions for each variable. These estimates are plugged in to the equation above, which is estimated by OLS. Finally, the parametric component of y_{i1} is removed using the estimates of γ^C and η^C , allowing the function $f^C(w_i)$ to be estimated non-parametrically. I choose data-driven bandwidths to minimize the mean-

⁴⁸ 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 matrilineal villages, which grant women more power to divorce their husbands and hence may increase bargaining power within relationships as well (Schatz, 2005).

⁴⁹ 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.

⁵⁰ 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.

⁵¹ A purely non-parametric version of this estimator is used in the Benneer et al. (2011) study of behavioral responses to information about arsenic in drinking water.

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 fix 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 repetition, I repeat the procedure of constructing the adjusted belief variable to correct my estimated confidence intervals for generate regressors. In each bootstrap sample, I trim observatins 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 in Figure 7 show that the change in risk beliefs is largest for people with the highest beliefs, and drops fairly steadily as baseline beliefs fall.⁵² 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 semi-parametric 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 results shown in Figure 8 are consistent with those from the linear approximation in Table 6: the treatment effect is initially positive, and then becomes

⁵² Near the low end of the scale the estimated dx/dT is larger in magnitude than the baseline beliefs, x^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 belief spectrum. If some of the respondents in the high tail at baseline were actually lower on the belief spectrum, my results will understand the extent of fatalism among the people whose initial beliefs were actually high.

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 ≥ 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 ways of specifying the regressions, 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 Heterogeneous marginal effects of risk beliefs on behavior

My theoretical model 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 some baseline covariate $w_i = w^k$. It is possible to construct an estimator of the group k -specific marginal effect $\hat{\delta}_{IV}^k = \hat{\delta}_{IV}^k(w^k)$, which will in general be a function of w^k . Since T 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:

$$\begin{aligned} x_i^e &= \alpha^x + \beta^x T_i + \gamma^x y_i^b + \delta^x Z_i + e_i \text{ for } w_i = w^k \\ y_i^e &= \alpha^y + \beta^y T_i + \gamma^y y_i^b + \delta^y Z_i + v_i \text{ for } w_i = w^k \end{aligned}$$

where w_i is the baseline belief variable and w_k represents each of its values I then construct

$$\hat{\delta}_{ILS,j}^k(w^k) = \frac{\hat{\beta}^y(w^k)}{\hat{\beta}^x(w^k)} \xrightarrow{p} \frac{\frac{dy}{dT}(w^k)}{\frac{dx}{dT}(w^k)} = \frac{dy}{dx}(w^k),$$

where convergence in probability comes from Slutsky’s theorem.⁵³ I will estimate the w_k -specific treatment effects $\hat{\beta}^y(w^k)$ and $\hat{\beta}^x(w^k)$ using $\tau^y(w^k)$ and $\tau^x(w^k)$ 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 better than the 0.01 level: using the standard Bonferroni adjustment gives a p-value below 0.02. The bracketed approach thus suggests that the top octile, or 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. Section 5.1 explores the mechanisms behind the fatalistic responses found in this population. In Section 5.2, I examine whether baseline risk beliefs are capturing other observed variation in the baseline data, such as education or sexual activity. Section 5.3 describes the socio-sexual context Section 5.4 addresses several potential limitations of this study, in particular problems of multiple comparisons. In Section 5.5 I discuss

⁵³ 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_i and the strength of the first stage for w_i . See Appendix VII for a derivation.

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 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. The results in Section 4 cannot distinguish between these two mechanisms.

One way of evaluating which mechanism is at work is to examine whether the effect of the information treatment varied based on people’s beliefs about their current HIV status. 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.⁵⁴ This result does not differ for people who are in the highest category of risk beliefs (not shown).

Alternatively, the information treatment may shift people’s beliefs about their current HIV status, or about whether they will contract HIV in the future. Using 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.⁵⁵ These consider the different likelihood values, as well as “Don’t Know”, as discrete choices. I estimate these regressions separately for the respondents in the highest quantile of risk beliefs, and for those in the lower eight quantiles. Figure 10 reports the mean marginal effects from these regressions.

I find evidence for both mechanisms: the information increases the probability that people with high initial risk beliefs will say there is “No likelihood” they currently have HIV by 18 percentage points compared to a control-group mean of 64%. The effect is even stronger for saying there is “No likelihood” of contracting HIV in the future, decreasing it by 19 percentage points – a decline of nearly one half relative to the control group mean of 43%. 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

⁵⁴ See Section 4.5 for a description of how this variable is defined.

⁵⁵ No data for perceived likelihood of contracting HIV in the future was collected at baseline, so the baseline data for the respondent’s perceived likelihood of having HIV currently was used as a proxy.

of getting HIV in the future are also robust to conditioning on respondents saying there is no likelihood that they currently have HIV.

5.2 Other Possible Sources of Heterogeneity

One potential concern with the results shown in Figures 7 to 9 is that respondents' baseline beliefs are not assigned at random, and therefore may be correlated with their other attributes. For example, people form their risk beliefs partly through experience with sexual partners, and 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, and so 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, rather than from beliefs themselves.

To examine this issue, 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.⁵⁶ This regression (omitted for space) has an R-squared of 0.067: a substantial portion of people's risk beliefs are not attributable to their baseline characteristics. I also repeat the analysis of 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 Socio-Sexual Context

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.

⁵⁶ The exact variables used 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.

Both types of change are reasonable to expect in this setting, because Southern Malawi has high rates of perceived and actual infidelity. 18% of married women think their spouse is unfaithful, and 10% of married men (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 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). At 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 a 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.

5.4 Potential Limitations

As discussed in Section 3.2, this paper 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 were studying a specific treatment (a cash incentive to stay in school for teenage girls) that may have lead to differential self-report bias, since people Malawi commonly perceive sexual activity by school-aged girls to lead automatically to dropout (Grant 2012). Studying a treatment that is unlikely to lead to differential self-report bias (the effect of economic shocks on sexual activity), Gong et al. (2014) find that STI incidence measures and self-reports yield similar estimates of treatment effects. Furthermore, my approach has the advantage of capturing changes in behavior among high-risk individuals, which cannot be done when using STIs as

outcome measures unless treatable STIs are used and individuals are treated for existing STIs at baseline.

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.

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 WP). I also use the same primary outcome variable (total sex acts in the past week as measured on the 7-day retrospective sex diary) as well as the same risk belief variable (the risk of HIV infection from a single unprotected sex act with a randomly-selected attractive person from the local community) as I employed in the preliminary empirical analysis in that paper.

5.5 Epidemiological Implications

The randomized treatment provided by my experiment – information about the true risk of HIV transmission – slightly increased 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 et al. 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.

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 marginal effects as summaries of population-level health behaviors. 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 marginal effect of HIV risk beliefs on sexual behavior 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; 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 Oster's 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 beliefs about risk 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 per-act risks 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 by people who believe that they are doomed to contract HIV in the future (for example because of condom breaks) rather than those

who think they already have the virus. This may be because of the high availability of HIV testing in the local area.

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 the extent to which 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 overestimates may be crucial for understanding the continued spread of the African HIV epidemic.

References

- Ahituv, A., Hotz, V. J., & Philipson, T. (1996). The responsiveness of the demand for condoms to the local prevalence of AIDS. *The Journal of Human Resources*, 31(4), 869–897.
- Attanasio, O. (2009). Expectations and perceptions in developing countries: Their measurement and their use. *American Economic Review*, 99(2), 87–92.
- Baird, S. J., Garfein, R. S., McIntosh, C. T., & Özler, B. (2012). Effect of a cash transfer programme for schooling on prevalence of HIV and herpes simplex type 2 in Malawi: a cluster randomized trial. *The Lancet*, 1–10. doi:10.1016/S0140-6736(11)61709-1
- Belli, R. F., Shay, W. L., & Stafford, F. P. (2001). Event history calendars and question list surveys: A direct comparison of interviewing methods. *Public Opinion Quarterly*, 65(1), 45–74. doi:10.1086/320037
- Becker, G. S., & Murphy, K. M. (1988). A theory of rational addiction. *The Journal of Political Economy*, 675–700.
- Benneer, L. S., Tarozzi, A., Pfaff, A., Soumya, H. B., Ahmed, K. M., & van Geen, A. (2011). *Bright lines, risk beliefs, and risk avoidance: Evidence from a randomized intervention in Bangladesh* (SSRN Scholarly Paper No. ID 1695115). Rochester, NY: Social Science Research Network.
- Black, D. A., & Smith, J. A. (2006). Estimating the returns to college quality with multiple proxies for quality. *Journal of Labor Economics*, 24(3), 701–728.
- Burbidge, J. B., Magee, L., & Robb, A. L. (1988). Alternative Transformations to Handle Extreme Values of the Dependent Variable. *Journal of the American Statistical Association*, 83(401), 123–127. doi:10.1080/01621459.1988.10478575
- Bruhn, M., & McKenzie, D. (2009). In pursuit of balance: Randomization in practice in development field experiments. *American Economic Journal: Applied Economics*, 1(4), 200–232.
- Caplin, A. (2003). Fear as a policy instrument. *Time and Decision: Economic and Psychological Perspectives on Intertemporal Choice*, 441–458.
- Card, D., & Giuliano, L. (2013). *Does gifted education work? For whom?* (Working Paper). University of California, Berkeley.
- Chinkhumba, J., Godlonton, S., & Thornton, R. L. (In Press). Demand for medical male circumcision. *American Economic Journal: Applied Economics*.
- Conroy, A. A. (2014). Marital Infidelity and Intimate Partner Violence in Rural Malawi: A Dyadic Investigation. *Archives of Sexual Behavior*, 43(7), 1303–1314. doi:10.1007/s10508-014-0306-2
- De Walque, D., Dow, W. H., Nathan, R., Abdul, R., Abilahi, F., Gong, E., Isdahl, Z., Jamison, J., Jullu, B., & Krishnan, S. (2012). Incentivising safe sex: a randomised trial

- of conditional cash transfers for HIV and sexually transmitted infection prevention in rural Tanzania. *BMJ Open*, 2(1).
- De Walque, D., Dow, W. H., & Gong, E. (2014). *Coping with risk : the effects of shocks on reproductive health and transactional sex in rural Tanzania* (Policy Research Working Paper Series No. 6751). The World Bank.
- Deaton, A. S. (2009, January). *Instruments of development: Randomization in the tropics, and the search for the elusive keys to economic development*. Lecture presented at the British Academy.
- Delavande, A. (2014). Probabilistic Expectations in Developing Countries. *Annual Review of Economics*.
- Delavande, A., & Kohler, H. P. (2009). Subjective expectations in the context of HIV/AIDS in Malawi. *Demographic Research*, 20(31), 817–875.
- Delavande, A., Giné, X., & McKenzie, D. (2011). Measuring subjective expectations in developing countries: A critical review and new evidence. *Journal of Development Economics*, 94(2), 151–163. doi:10.1016/j.jdeveco.2010.01.008
- Donner, A., & Klar, N. (2000). *Design and analysis of cluster randomization trials in health research*. London Arnold Publishers.
- Dupas, P. (2011). Do teenagers respond to HIV risk information? Evidence from a field experiment in Kenya. *American Economic Journal: Applied Economics*, 3(1), 1–34.
- Fan, J., & Gijbels, I. (1996). *Local polynomial modelling and its applications*. London: Chapman & Hall.
- Fishbein, M., & Pequegnat, W. (2000). Evaluating AIDS prevention interventions using behavioral and biological outcome measures. *Sexually Transmitted Diseases*, 27(2), 101–110.
- Frison, L., & Pocock, S. J. (1992). Repeated measures in clinical trials: analysis using mean summary statistics and its implications for design. *Statistics in Medicine*, 11(13), 1685–1704.
- Galvin, S. R., & Cohen, M. S. (2004). The role of sexually transmitted diseases in HIV transmission. *Nature Reviews Microbiology*, 2(1), 33–42. doi:10.1038/nrmicro794
- Godlonton, S., Munthali, A., & Thornton, R. (2012). *Behavioral response to information? Circumcision, information, and HIV prevention* (Working Paper). Ann Arbor, MI: University of Michigan.
- Gong, E. (2010). *HIV testing & risky sexual behavior* (Working Paper).
- Grant, M. J. (2012). Girls' schooling and the perceived threat of adolescent sexual activity in rural Malawi. *Culture, Health & Sexuality*, 14(1), 73–86.
- Gray, R., Sempijja, V., Shelton, J., Serwadda, D., Nalugoda, F., Kagaayi, J., ... Wawer, M. J. (2011). The contribution of HIV-discordant relationships to new HIV infections in Rakai, Uganda: *AIDS*, 25(6), 863–865. doi:10.1097/QAD.0b013e3283448790

- Heckman, J. J. (1996). Randomization as an Instrumental Variable. *The Review of Economics and Statistics*, 336–341.
- HIV Reinfection - Positive Prevention - Reinfection. (2007, July 17). *About.com AIDS / HIV*. Retrieved March 25, 2014, from <http://aids.about.com/cs/safesex/a/reinfection.htm>
- Hollingsworth, T. D., Anderson, R. M., & Fraser, C. (2008). HIV-1 transmission, by stage of infection. *Journal of Infectious Diseases*, 198(5), 687–693. doi:10.1086/590501
- Holmes, K. K., Levine, R., & Weaver, M. (2004). Effectiveness of condoms in preventing sexually transmitted infections. *Bulletin of the World Health Organization*, 82(6), 454–461. doi:10.1590/S0042-96862004000600012
- Hudomiet, P., Kezdi, G., & Willis, R. J. (n.d.). Stock market crash and expectations of American households. *Journal of Applied Econometrics*.
- Juhn, C., Kalemli-Ozcan, S., & Turan, B. (2009). *HIV and fertility in Africa: First evidence from population based surveys* (Working Paper No. Discussion Paper No. 4473). Institute for the Study of Labor.
- Kaler, A. (2003). “My girlfriends could fill a yanu-yanu bus”: Rural Malawian men’s claims about their own serostatus. *Demographic Research, Special Collection*(1).
- Kalichman, S. C., Kelly, J. A., Morgan, M., & Rompa, D. (1997). Fatalism, current life satisfaction, and risk for HIV infection among gay and bisexual men. *Journal of Consulting and Clinical Psychology*, 65(4), 542–546. doi:10.1037/0022-006X.65.4.542
- Kerwin, J. T. (2012). “*Rational fatalism*”: *Non-monotonic choices in response to risks* (Working Paper). Ann Arbor: University of Michigan.
- Kerwin, J. T., Thornton, R. L., & Foley, S. M. (2014). Prevalence of and Factors Associated with Oral Sex among Rural and Urban Malawian Men. *International Journal of Sexual Health*, 26(1), 66–77. doi:10.1080/19317611.2013.830671
- Kerwin, J. T., Thornton, R. L., Foley, S. M., Chinkhumba, J., & Chibwana, A. (2011). *Situational analysis of sexual behaviors and alternative safer sex strategies in-depth interview dataset*. University of Michigan and University of Malawi College of Medicine.
- Kling, J. R., Liebman, J. B., & Katz, L. F. (2007). Experimental analysis of neighborhood effects. *Econometrica*, 75(1), 83–119. doi:10.1111/j.1468-0262.2007.00733.x
- Kremer, M. (1996). Integrating behavioral choice into epidemiological models of AIDS. *The Quarterly Journal of Economics*, 111(2), 549–573. doi:10.2307/2946687
- Lillard, L., & Willis, R. (2001). *Cognition and wealth: The importance of probabilistic thinking* (Working Paper No. WP 2001-007) (pp. 48109–1220). University of Michigan Retirement Research Center.
- Loader, C. (2004). Smoothing: local regression techniques. In *Handbook of Computational Statistics* (pp. 571–596). Springer.

- Luke, N., Clark, S., Zulu, E., Westlands, N., & Eliya, K. (2009). The Relationship History Calendar: Improving the scope and quality of data on youth sexual behavior. *Brown University Typescript*.
- Malawi National AIDS Commission. (2003). *National HIV/AIDS policy: A call for renewed action*. Lilongwe: Malawi National AIDS Commission (NAC).
- Malawi National Statistical Office, & Opinion Research Company's Macro International (ORC-MACRO). (2010). Malawi demographic and health Survey 2010. Calverton, MD: MEASURE DHS.
- Manski, C. F. (2004). Measuring Expectations. *Econometrica*, 72(5), 1329–1376. doi:10.1111/j.1468-0262.2004.00537.x
- McKenzie, D. (2012). Beyond baseline and follow-up: The case for more T in experiments. *Journal of Development Economics*, 99(2), 210–221.
- National Health Service. (2013). Quitting timeline | Why Quit | NHS SmokeFree. Retrieved July 19, 2013, from <http://smokefree.nhs.uk/why-quit/timeline/>
- O'Donoghue, T., & Rabin, M. (2001). Risky Behavior among Youths: Some Issues from Behavioral Economics. *NBER Chapters*, 29–68.
- Office of the Secretary. (1979). *The Belmont Report*. Bethesda, MD: National Commission for the Protection of Human Subjects of Biomedical Behavioral Research.
- Oster, E. (2012). HIV and sexual behavior change: Why not Africa? *Journal of Health Economics*, 31(1), 35–49. doi:10.1016/j.jhealeco.2011.12.006
- Özler, B. (2013). *Economists have experiments figured out. What's next? (Hint: It's Measurement)*. Impact Evaluations. Retrieved August 5, 2013, from <http://blogs.worldbank.org/impactevaluations/economists-have-experiments-figured-out-what-s-next-hint-it-s-measurement>
- Padian, N. S., McCoy, S. I., Balkus, J. E., & Wasserheit, J. N. (2010). Weighing the gold in the gold standard: challenges in HIV prevention research. *AIDS*, 24(5), 621–635. doi:10.1097/QAD.0b013e328337798a
- Philipson, T. J., & Posner, R. A. (1993). *Private choices and public health: The AIDS epidemic in an economic perspective*. Harvard University Press Cambridge, MA.
- Schatz, E. (2005). 'Take your mat and go!': Rural Malawian women's strategies in the HIV/AIDS era. *Culture, Health & Sexuality*, 7(5), 479–492.
- Smith, D. M., Richman, D. D., & Little, S. J. (2005). HIV Superinfection. *Journal of Infectious Diseases*, 192(3), 438–444. doi:10.1086/431682
- Sterck, O. (2012). Should prevention campaigns disclose the transmission rate of HIV/AIDS? Theory and application to Burundi. *SSRN eLibrary*.
- Stock, J. H., & Yogo, M. (2005). *Testing for weak instruments in linear IV regression*. In Identification and Inference for Econometric Models: A Festschrift in Honor of Thomas Rothenberg (pp. 80–108). Cambridge University Press.

- Thornton, R. L. (2008). The Demand for, and Impact of, Learning HIV Status. *American Economic Review*, 98(5), 1829.
- Viscusi, W. K. (1990). Do smokers underestimate risks? *Journal of Political Economy*, 98(6), 1253–1269.
- Wawer, M. J., Gray, R. H., Sewankambo, N. K., Serwadda, D., Li, X., Laeyendecker, O., ... Quinn, T. C. (2005). Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. *The Journal of Infectious Diseases*, 191, 1403–1409.
- Weinstein, N. D., & Klein, W. M. (1996). Unrealistic optimism: Present and future. *Journal of Social and Clinical Psychology*, 15(1), 1–8. doi:10.1521/jscp.1996.15.1.1
- White, D., & Stephenson, R. (2014). *Correlates of Perceived HIV Prevalence and Associations with HIV Testing Behavior among MSM in the United States* (Working Paper). Boston, MA: Population Association of America Annual Meeting 2014.
- Wilson, N., Xiong, W., & Mattson, C. (2012). *Is sex like driving? Risk compensation Associated with Male Circumcision in Kisumu, Kenya* (Working Paper).
- Wiswall, M., & Zafar, B. (in press). Determinants of college major choice: Identification using an information experiment. *Review of Economic Studies*.

Figure 1

Example of HIV Risk Messaging from a Malawian Life Skills Textbook

Case study

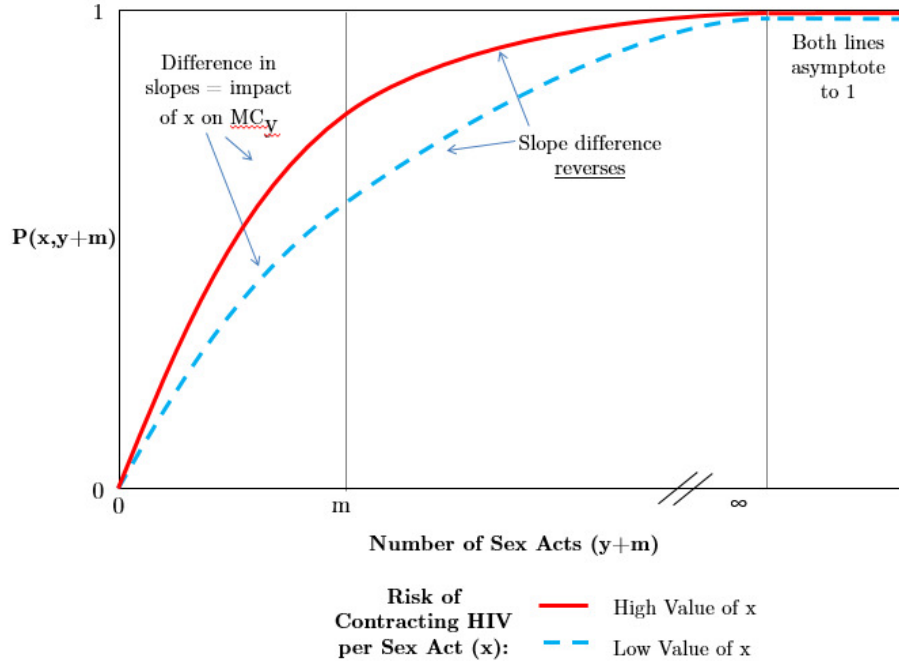
Read the case study below and answer the questions that follow.

Nabetha suffered from oral thrush only a month after her first sexual encounter. The infection persisted over a long period of time. The doctor recommended a HIV test. She tested positive. The results devastated her. She decided she would not suffer alone but infect as many people as possible. She worked very hard towards this end. She died three years after the diagnosis with HIV.

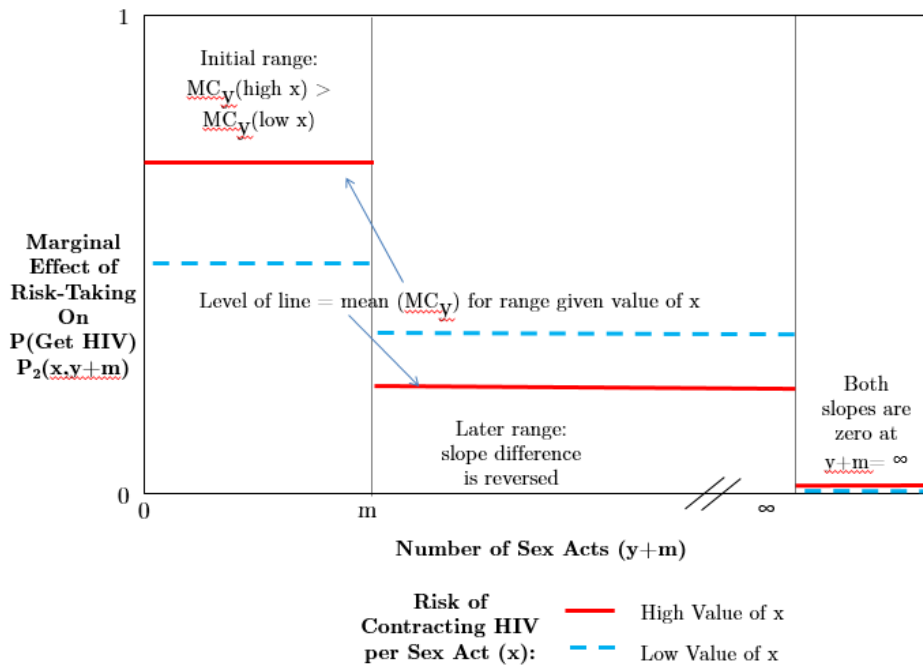
Notes: Excerpted from the Form 4 Life Skills textbook used in Malawian secondary schools (highlighting added). The highlighted section suggests that the risk of contracting HIV from a single sex act is 100%. Author's conversations with Malawi Ministry of Education officials confirm that the Life Skills 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 text books and official curriculum.

Figure 2

Illustration of Tipping Point in Marginal Cost of Sexual Activity
 $P[\text{HIV Infection}|\text{Number of Sex Acts } (y)]$ for Low and High Values of Per-Act Risk (x)



Panel A: $P[\text{HIV Infection}|\text{Number of Sex Acts } (y)]$ 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 which rational responses to risks are fatalistic (risk-seeking) rather than self-protective (risk-avoiding).

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

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?

#	#	#
---	---	---

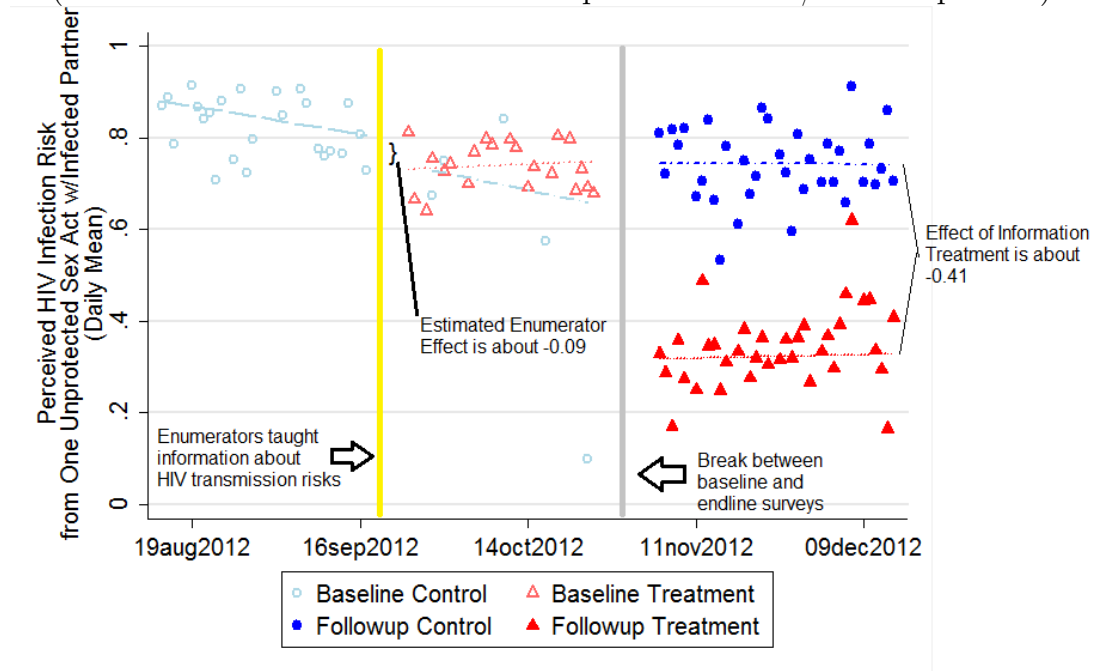
Example Question about Subject Risk Beliefs

Notes: Example of one of the six different HIV-related expectations questions used in the analysis. 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

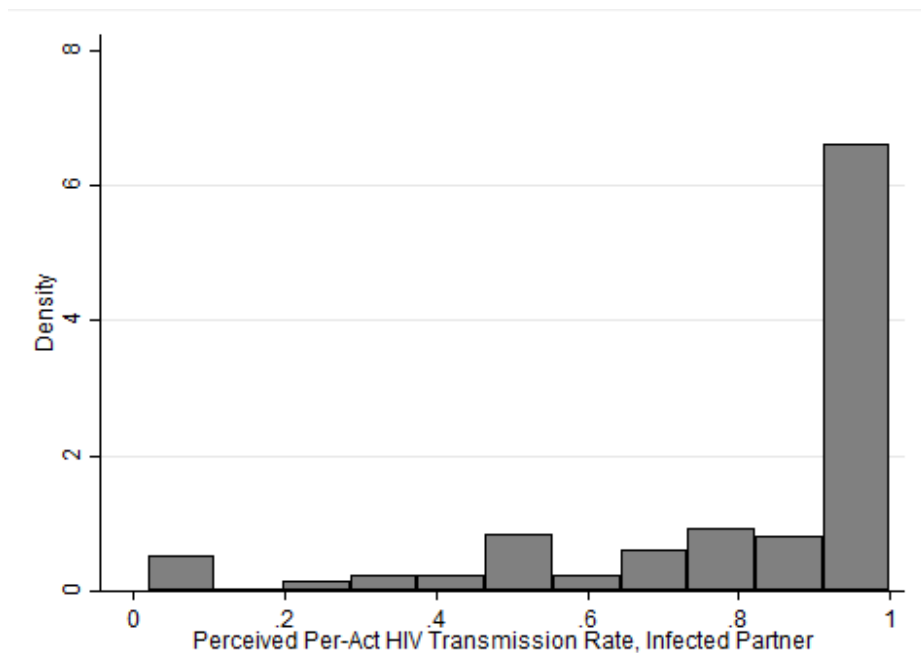
(Per-act HIV transmission rate for unprotected sex w/infected partner)



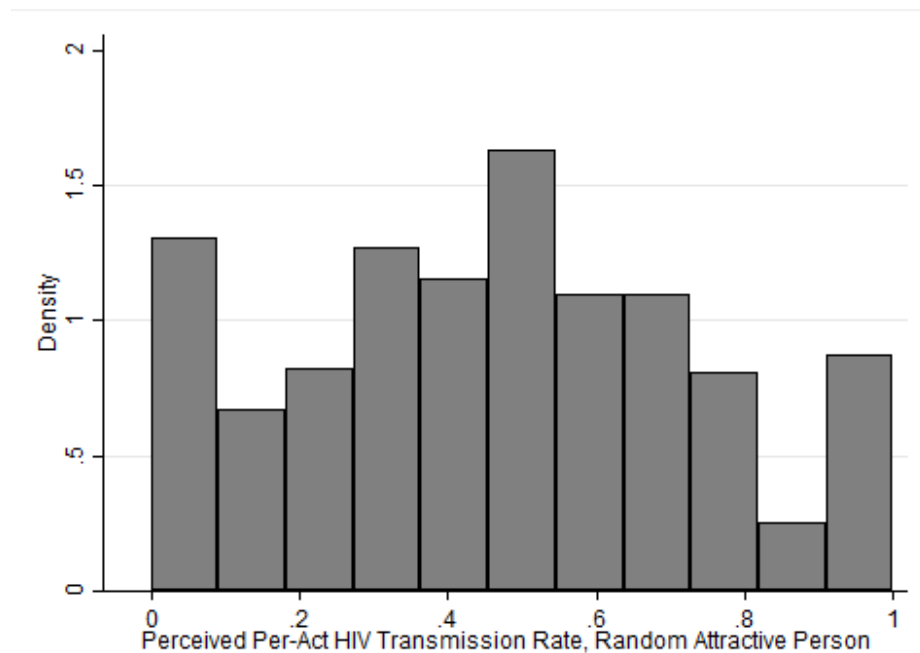
Notes: Each point represents the mean value of the risk beliefs for a given day; baseline control beliefs are hollow circles, endline control beliefs are solid circles, baseline treatment beliefs are hollow triangles, and endline treatment beliefs are solid triangles. The lines are linear fits of beliefs on date for a given date range and study arm. The yellow 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 (light red) with the Endline Control beliefs (dark blue). 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

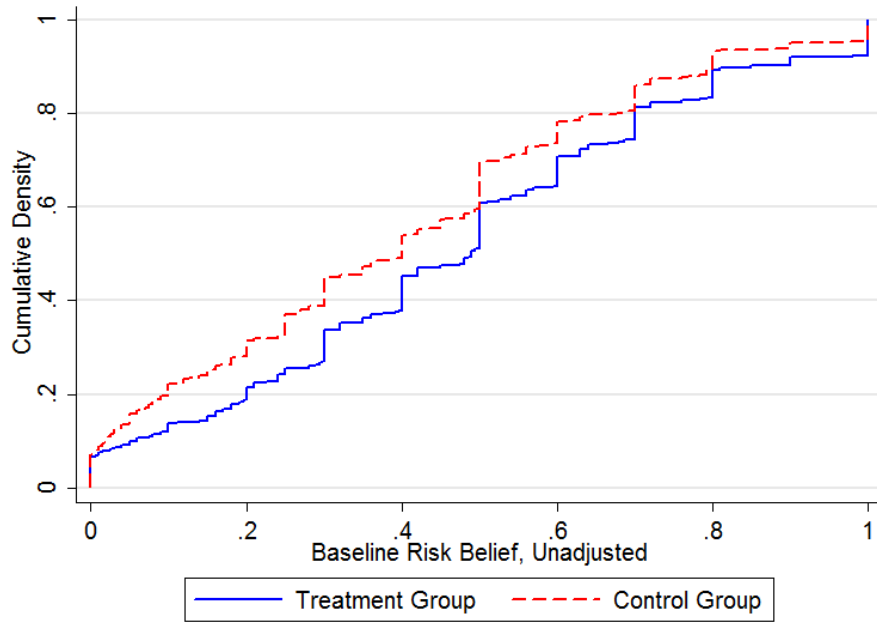


Panel B – Per-Act Infection Risk from Unprotected Sex with a Randomly-Selected Partner

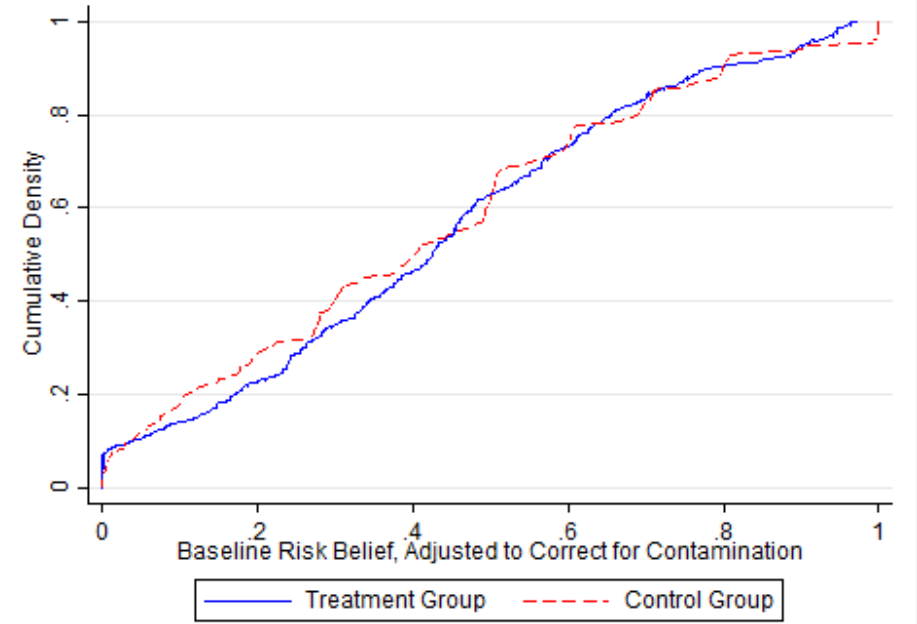
Notes: The two histograms plot the distribution of beliefs about the chance of contracting HIV from unprotected sex with either an infected partner (Panel A) or a randomly-selected person the respondent finds attractive (Panel B). Panel A has a large mass point at 100%. Panel B breaks up that mass point 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.

Figure 6

CDFs of Baseline Beliefs about Per-Act HIV Infection Risk from a Random Attractive Sex Partner, by Study Arm



Panel A – Unadjusted

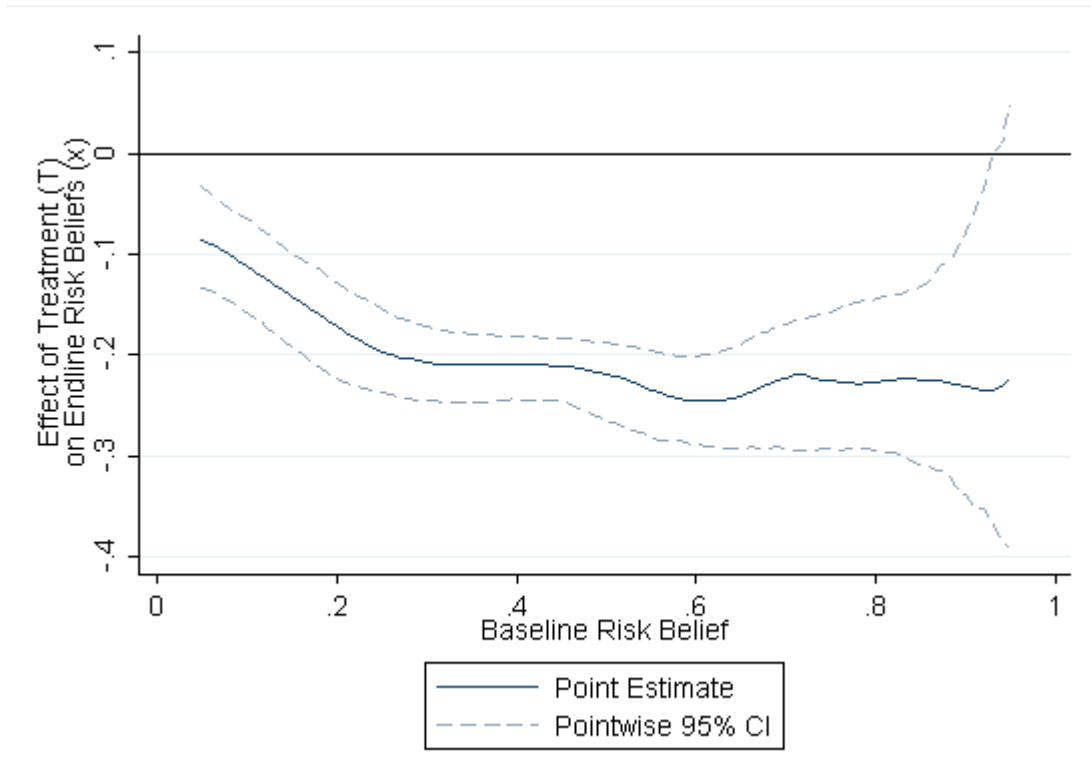


Panel B – Adjusted to Correct for Enumerator-Knowledge Contamination

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 $x_i = t_i * p_i$, 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

$t_i = \beta_0 + \beta_1 \text{AfterTraining}_i + \beta_2 \text{DaysAfterTraining}_i + \beta_3 \text{AfterTraining}_i * \text{DaysAfterTraining}_i + \varepsilon_i$ and construct $t_i^{adj} = t_i^{resid} + \hat{\beta}_0$. p_i^{adj} is constructed likewise, and $x_i^{adj} = t_i^{adj} * p_i^{adj}$, and bound x_i^{adj} to lie within $[0, 1]$. This preserves the scale on which the beliefs are measured. 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[‡]
 Semi-Parametric Estimates



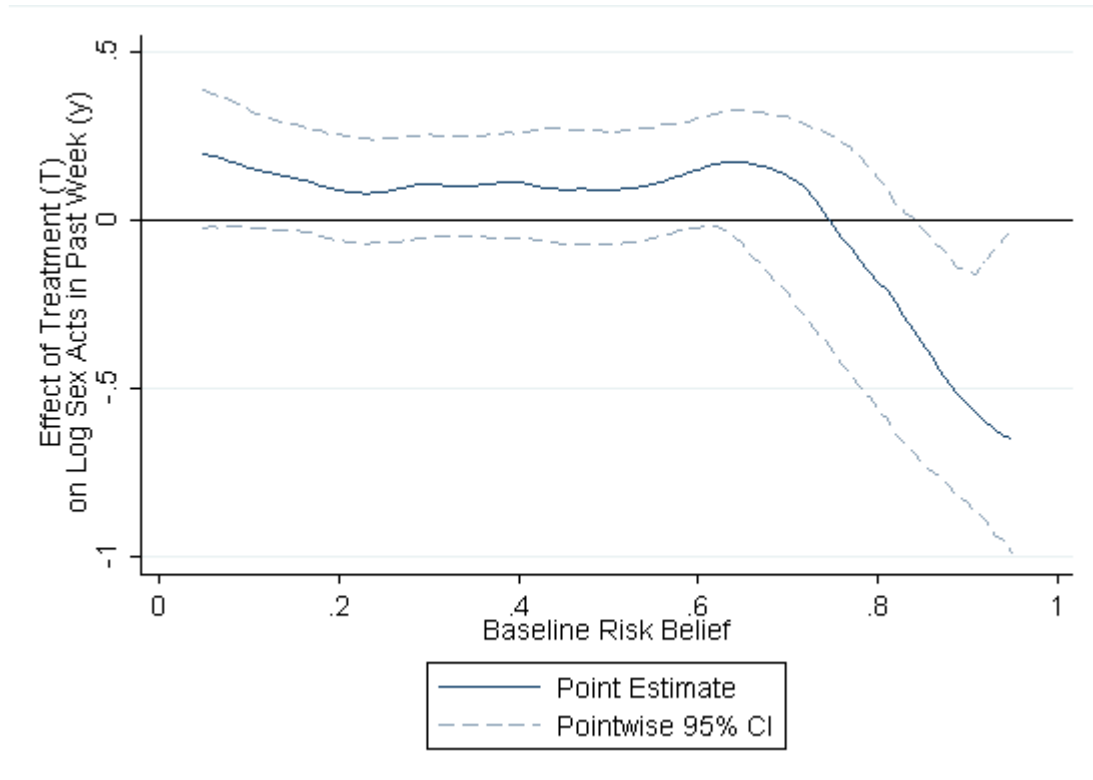
Notes: The graph illustrates the first-stage estimate of the effect of the information treatment on endline (post-treatment) risk beliefs, decomposed by individuals' baseline (pre-treatment) beliefs about HIV infection risks. The estimated effects on risk beliefs are negative for all levels of baseline 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[‡]
 Semi-Parametric Estimates



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.

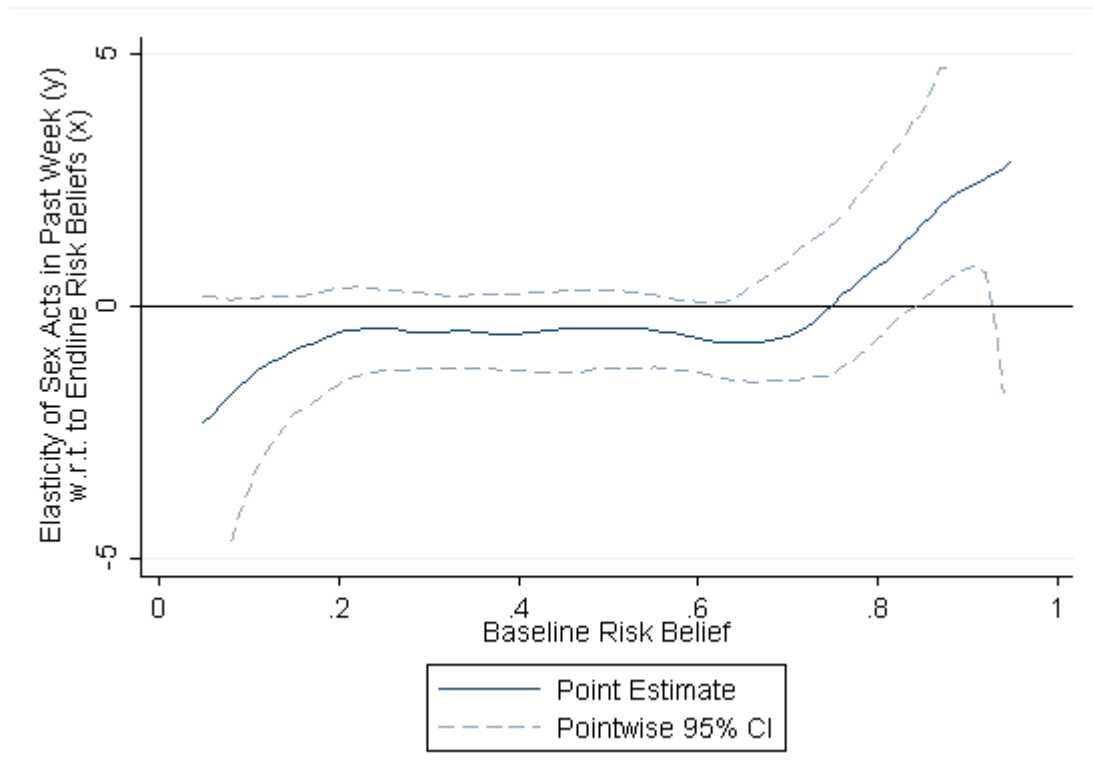
[†] Log sex in past week constructed as $y' = \ln(y + \sqrt{1 + y^2})$ to account for zeroes.

[‡] 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[‡]
Semi-Parametric Estimates



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.

The outcome used is log sex in past week (constructed as $y' = \ln(y + \sqrt{1 + y^2})$ to account for zeroes) which allows the estimates to be interpreted as elasticities.

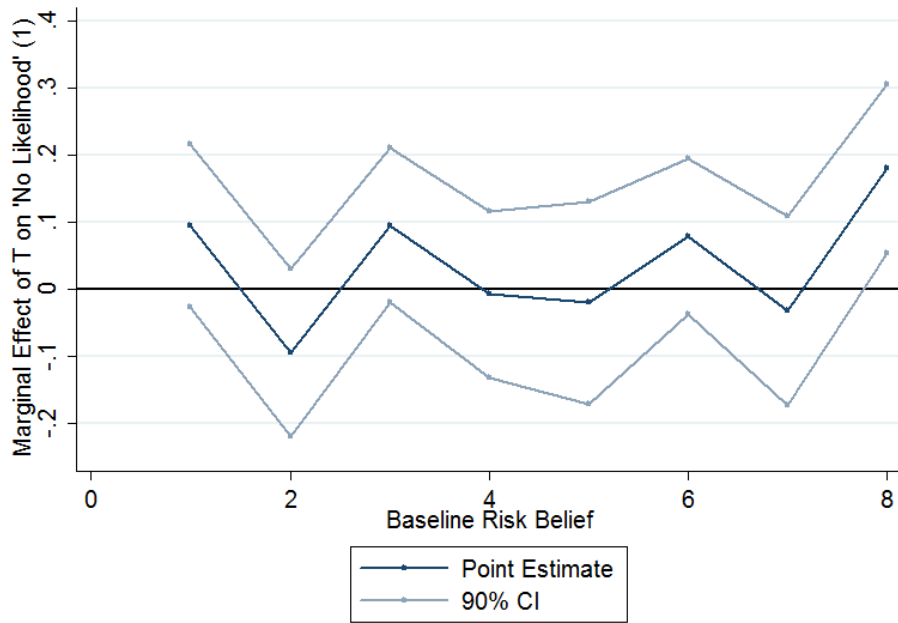
[‡] Baseline Risk Belief is the composite belief variable from Figure 6: the perceived chance of contracting HIV from a single unprotected sex act with a randomly-chosen attractive person of the opposite sex from the local area. Baseline Risk Belief is adjusted for non-constant time trends as in Panel 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.

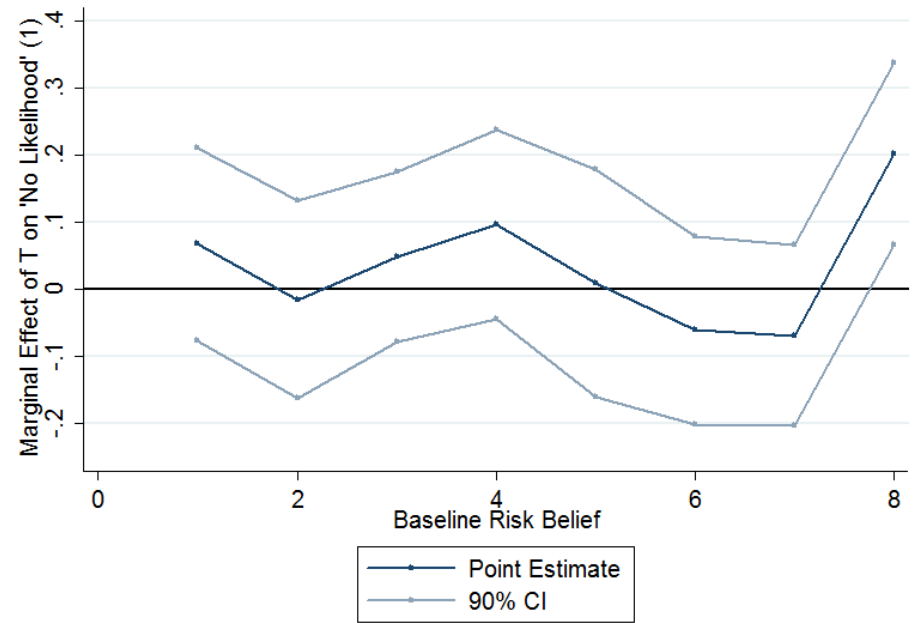
Figure 10

Multinomial Logit Estimates of Effect of Treatment on Perceived Likelihood of Having HIV Now (Panel A) in the Future (Panel B), by Level of Baseline HIV Transmission Risk Belief[‡]

1 = “No Likelihood”, 2 = “Low Likelihood”, 3 = “Medium Likelihood”, 4 = “High Likelihood”, 98 = “Don’t Know”



Panel A – Effect on Reporting “No Likelihood” of Currently Having HIV



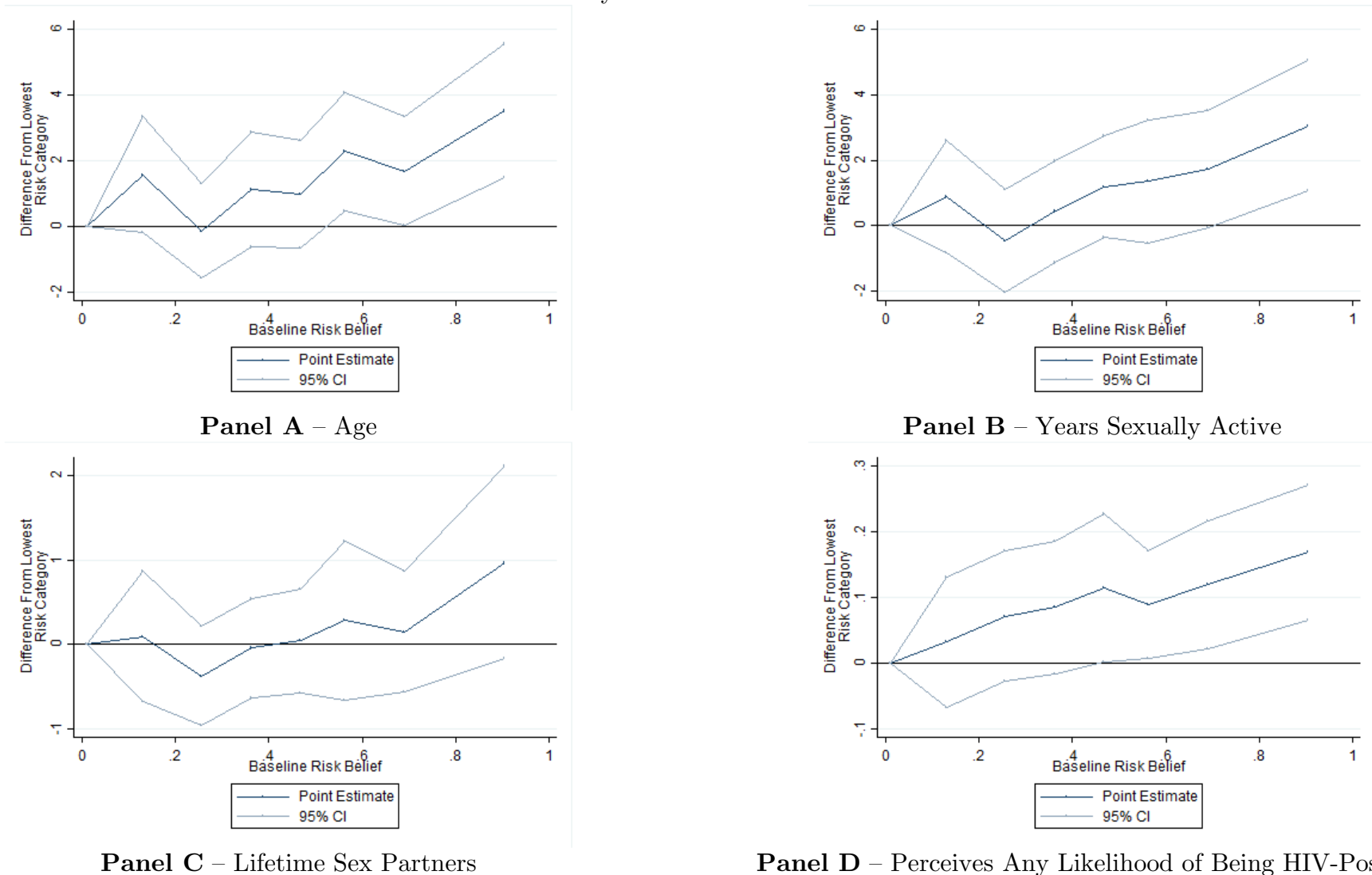
Panel B – Effect on Reporting “No Likelihood” of Getting HIV in the Future

Notes: The graphs display 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. The treatment significantly increased the rate at which people reported no 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. Results are not changed qualitatively by the exclusion of the “Don’t know” category.

[‡]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. Heteroskedasticity-robust 95% confidence intervals, clustered by village, in blue.

Figure 11
Differences in HIV Risk Factors by Level of Baseline HIV Transmission Risk Belief†



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. Sample includes 1292 respondents who completed both baseline and endline surveys. Heteroskedasticity-robust 95% confidence intervals, clustered by village, in blue.

†Baseline Risk Belief is the composite belief variable from Figure 6.

Table 1
Demographic Covariate Baseline Balance

	N	Overall	Control	Treatment	C-T
	(1)	(2)	(3)	(4)	(5)
<u>Demographics</u>					
Male	1292	0.43	0.42	0.44	-0.01
Married	1290	0.82	0.83	0.80	0.03
Age	1292	29.36	29.13	29.59	-0.46
Grew up in village where currently residing	1289	0.62	0.65	0.60	0.05
Years of education	1292	5.81	5.76	5.86	-0.10
Number of people in household	1292	4.95	5.04	4.87	0.17
Total children still living	1292	2.99	2.94	3.05	-0.11
Desired future children	1289	1.36	1.31	1.41	-0.09
# media sources [†] used at least monthly	1292	1.18	1.16	1.20	-0.04
# common assets owned by household	1291	4.40	4.54	4.26	0.28
Household cash income past 30 days (PPP USD)					
Baseline (C and T observed at different times of year)	1292	250.29	282.46	218.23	64.23**‡
Endline (C and T observed simultaneously)	1292	190.28	201.94	178.66	23.29
Household expenditure past 30 days (PPP USD)	1292	292.70	292.39	293.01	-0.62
<u>Religion</u>					
Muslim	1292	0.07	0.09	0.06	0.02
Christian	1292	0.89	0.89	0.89	-0.01
Other	1292	0.04	0.03	0.05	-0.02
<u>Ethnic Group</u>					
Nyanja	1292	0.47	0.46	0.48	-0.02
Lomwe	1292	0.37	0.34	0.39	-0.05
Yao	1292	0.09	0.11	0.07	0.04
Chewa	1292	0.04	0.05	0.03	0.02
Other	1292	0.03	0.04	0.02	0.02

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

	N	Overall	Control	Treatment	C-T
	(1)	(2)	(3)	(4)	(5)
Panel A - Single-Question Recall					
Years since sexual debut	1275	13.15	13.10	13.20	-0.10
Total lifetime sex partners	1288	3.34	3.12	3.56	-0.44**
Months since last sex act	1252	4.98	4.73	5.23	-0.50
Any sex in the past 30 days	1281	0.73	0.74	0.73	0.01
Sex partners during past 30 days	1290	0.81	0.82	0.80	0.02
Total sex acts during past 30 days	1281	7.37	7.48	7.27	0.21
Any unpro. sex acts in the past 30 days	1281	0.67	0.67	0.66	0.00
Total unpro. sex acts in the past 30 days	1281	6.66	6.75	6.57	0.18
Panel B - Retrospective Sex Diary - Sex Acts in Past 7 Days					
Any sex acts	1292	0.52	0.54	0.51	0.03
Total sex acts	1292	1.71	1.80	1.62	0.18
Any unpro. sex acts	1292	0.47	0.47	0.47	0.01
Total unpro. sex acts	1292	1.52	1.57	1.47	0.10
Sex with more than one partner	1292	0.01	0.02	0.01	0.01
Total sex acts with non-primary partners	1292	0.02	0.03	0.01	0.02
Any unpro. sex acts with non-primary partners	1292	0.01	0.01	0.00	0.00
Total unpro. sex with non-primary partners	1292	0.01	0.01	0.01	0.00

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

	Perceived HIV Transmission Rate, if Partner Infected				Perceived HIV Prevalence		Composite Beliefs: P(Contract HIV from Unpro. Sex w/Random Attractive Person [‡])	
	One Act		One Year [†]		All Local People [‡]	Attractive Local People [‡]	One Act	One Year [†]
	Unprotected	W/Condom	Unprotected	W/Condom			(7)	(8)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A – Differences at Endline, without Controlling for Baseline Beliefs								
Treatment Group	-0.408*** (0.019)	-0.048*** (0.006)	-0.381*** (0.016)	-0.089*** (0.013)	-0.163*** (0.018)	-0.055*** (0.018)	-0.197*** (0.015)	-0.193*** (0.016)
Observations	1,284	1,284	1,284	1,284	1,269	1,269	1,268	1,269
Adjusted R-squared	0.273	0.036	0.300	0.061	0.074	0.012	0.144	0.134
Panel B – Differences at Endline, Controlling for Baseline Beliefs								
Treatment Group	-0.384*** (0.019)	-0.045*** (0.006)	-0.371*** (0.016)	-0.071*** (0.012)	-0.162*** (0.016)	-0.047*** (0.015)	-0.182*** (0.014)	-0.185*** (0.015)
Observations	1,281	1,283	1,276	1,276	1,257	1,254	1,252	1,251
Adjusted R-squared	0.315	0.066	0.328	0.142	0.157	0.081	0.200	0.182
Panel C – Difference-in-Differences								
Treatment Group	-0.316*** (0.023)	-0.022* (0.011)	-0.336*** (0.018)	-0.010 (0.016)	-0.154*** (0.018)	-0.028 (0.019)	-0.127*** (0.020)	-0.148*** (0.020)
Observations	1,281	1,283	1,276	1,276	1,257	1,254	1,252	1,251
Adjusted R-squared	0.149	0.002	0.225	0.008	0.046	0.006	0.049	0.066
Mean(Dep. Var)	0.742	0.0818	0.905	0.176	0.485	0.463	0.351	0.424
SD(Dep. 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

	Any Sex in Past Week (1)	Log [‡] Sex Acts in Past Week (2)	Log [‡] Unprotected Sex Acts in Past Week (3)	Log [‡] Sex Partners in Past 30 Days (4)	Log [‡] Condoms acquired in past 30 days (5)	Log [‡] Condoms Purchased (6)	Log [‡] Overall Sexual Activity Index [†] (7)	Log [‡] Diary Sexual Activity Index [†] (8)
Panel A: OLS Estimates (Control Group Only)								
Endline Risk Belief [‡]	0.155*** (0.054)	0.175* (0.102)	0.106 (0.103)	0.196*** (0.058)	0.118 (0.172)	-0.337 (0.224)	0.318*** (0.100)	0.180** (0.078)
Observations	627	627	627	626	626	626	617	627
R-squared	0.210	0.277	0.240	0.258	0.165	0.049	0.340	0.219
Panel B: 2SLS Estimates								
Endline Risk Belief [§]	-0.260** (0.121)	-0.562** (0.241)	-0.412* (0.232)	-0.043 (0.102)	-0.375 (0.402)	-0.256 (0.535)	-0.327** (0.159)	-0.317** (0.122)
Observations	1,252	1,252	1,252	1,250	1,243	1,246	1,222	1,252
R-squared	0.208	0.256	0.253	0.277	0.129	0.046	0.361	0.196
1 st -Stage F-Statistic	222.0	220.7	221.3	222.7	221.3	218.1	226.5	221.6

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. 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. * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table 5
 OLS and 2SLS Estimates of the Partial Effect of Endline Risk Beliefs on Sexual Activity

	Any Sex in Past Week (1)	Log [‡] Sex Acts in Past Week (2)	Log [‡] Unprotected Sex Acts in Past Week (3)	Log [‡] Sex Partners in Past 30 Days (4)	Log [‡] Condoms acquired in past 30 days (5)	Log [‡] Condoms Purchased (6)	Log [‡] Overall Sexual Activity Index [†] (7)	Log [‡] Diary Sexual Activity Index [†] (8)
Panel A: OLS Estimates (Control Group Only)								
Endline Risk Belief [‡]	0.155*** (0.054)	0.175* (0.102)	0.106 (0.103)	0.196*** (0.058)	0.118 (0.172)	-0.337 (0.224)	0.318*** (0.100)	0.180** (0.078)
Observations	627	627	627	626	626	626	617	627
R-squared	0.210	0.277	0.240	0.258	0.165	0.049	0.340	0.219
Panel B: 2SLS Estimates								
Endline Risk Belief [§]	-0.260** (0.121)	-0.562** (0.241)	-0.412* (0.232)	-0.043 (0.102)	-0.375 (0.402)	-0.256 (0.535)	-0.327** (0.159)	-0.317** (0.122)
Observations	1,252	1,252	1,252	1,250	1,243	1,246	1,222	1,252
R-squared	0.208	0.256	0.253	0.277	0.129	0.046	0.361	0.196
1 st -Stage F-Statistic	222.0	220.7	221.3	222.7	221.3	218.1	226.5	221.6

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. 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. * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table 6
Non-Monotonic Responses to Information Treatment Effects by Baseline Risk Beliefs

	Outcome: Log [§] Sex Acts in Past Week						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Treatment (T)	0.101** (0.047)	0.320*** (0.083)	0.123* (0.072)	0.070 (0.057)	0.136** (0.060)	0.095 (0.062)	0.412 (0.309)
T*(Baseline Risk Belief [0-1]) [†]		-0.499*** (0.162)					-.477*** (0.168)
T*(Male)			-0.049 (0.131)				-0.039 (0.155)
T*(Baseline Log Sex Acts in Past Wk.)				0.035 (0.051)			-0.075 (0.112)
T*(Ever Exposed to HIV)					-0.151 (0.113)		-0.105 (0.119)
T*(Any Chance I am HIV-positive)						0.009 (0.115)	0.075 (0.122)
T Interacted with Other Baseline Covariates [‡]	No	No	No	No	No	No	Yes
Observations	1,292	1,275	1,292	1,292	1,275	1,277	1,245
R-squared	0.277	0.284	0.277	0.277	0.277	0.276	0.345

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.

[§] Logged variables are constructed as $y' = \ln(y + \sqrt{1 + y^2})$ to account for zeroes.