Scared Straight or Scared to Death?
The Effect of Risk Beliefs on Risky Behaviors

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Abstract

This paper develops and tests a model of risk compensation that allows for "fatalism": higher risks lead to *more* risk-taking, rather than less. Fatalism can be rational if the risk of each act exceeds a threshold value. I test this prediction by randomizing the provision of information about HIV risks in Malawi, and use a novel method to decompose the risk elasticity of sexual risk-taking by people's initial risk beliefs. Matching the model's predictions, this elasticity varies from -2.3 for the lowest to 2.9 for the highest beliefs. Fatalistic people, who have a positive elasticity, comprise 14% of the population.
JEL: I12, I15, J10, O12

Risk compensation is central to our understanding of how people make decisions about potentially dangerous activities. Beginning with Peltzman's 1975 study of automobile regulation, economists have realized that a decline in the risk associated with a particular behavior is often offset by a rational increase in risk-taking. In line with this, empirical research on risk

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compensation typically assumes that people are uniformly risk-avoiding, or "self-protective": when the per-act risk of an activity goes up, people are presumed to become more cautious.

This paper develops and tests an alternative model where rational responses to health risks are sometimes risk-seeking, or "fatalistic" – where the optimal choice may be to *increase* one's risk-taking when the per-act risk rises. Consider the example of HIV. An increase in the risk of each sex act affects not only the marginal acts the agent is deciding over, but also a stock of previously-chosen acts over which the agent no longer has any control. If the perceived risk of contracting HIV from each act rises, this raises the marginal cost of additional sex acts. However, a rise in the perceived risk also increases the probability that the agent *already* has HIV, which *decreases* the marginal cost of more risky sex. When this second effect dominates, increases in perceived risks will lead to more risk-taking rather than less. In other words, rational agents will become fatalistic. 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 alone will not prevent people from becoming fatalistic.

The possibility of rational fatalism is ignored by much of the empirical literature on risk compensation, which assumes that the probability (and thus the expected cost) of having HIV can be approximated by a linear function (see e.g. Viscusi 1990, Philipson and Posner 1993, Ahituv, Hotz and Philipson 1996 and Oster 2012). Previous theoretical work has shown that if agents can compute the true probability of being infected with HIV, rational responses to risks are fatalistic rather than self-protective for certain individuals (Kremer 1996; O'Donoghue and Rabin 2001; Sterck 2014). These results are all driven by the same principle, which is that the per-act risk of HIV affects the expected damage not just of the marginal sex act but of all previous sex acts. The theoretical framework I develop in this paper shows that rational fatalism is in fact a very general property of risk compensation. If agents optimize using any plausible expected cost function – any function where the total chance of having HIV is capped at 100% – sufficiently high risk beliefs encourage risk-seeking behavior.

I test this model of rational fatalism by conducting a field experiment in southern Malawi, an area with high rates of HIV infection. Malawi is an ideal setting to study rational fa-

talism because qualitative evidence suggests that some people are responding fatalistically to the virus (Kaler 2003, Kaler and Watkins 2010), and because HIV prevention education emphasizes that the risk of contracting HIV is extremely high (Kadyoma et al. 2012). The experiment randomly assigned 1,292 people to either be a control group, or to receive information about the true risk of HIV infection, which is much lower than people's *ex ante* beliefs.

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.[1] Using the experimental treatment as an instrumental variable, I estimate that the risk belief elasticity of sexual activity is small but statistically significant at about -0.6. This elasticity implies that a 10% increase in the perceived chance of contracting HIV from unprotected sex would cause a 6% decline in the amount of risky sex people choose to have.

However, this average elasticity is misleading. The model predicts that responses to risks will vary sharply by people's initial risk beliefs: people with sufficiently high risk beliefs will become fatalistic, and have a positive rather than a negative elasticity. The mean elasticity will therefore mask important variation across the population. I test this implication of the model by examining the pattern of heterogeneity in the risk elasticity by people's baseline (pre-treatment) beliefs. To do this I develop a method for decomposing instrumental-variables estimates by exogenous covariates and show that my method gives consistent estimates of the underlying conditional parameter.

This novel decomposition technique 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. People with positive elasticities, who respond fatalistically to HIV risks, make up 13.8% of the population. The fatalistic group has higher-than-average risk factors for HIV, such as years of sexual experience and perceived HIV-positive status.

These fatalistic responses, concentrated among people who are at above-average risk for contracting HIV, suggest that policies which encourage people to greatly overestimate HIV

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

transmission risks have ambiguous ethical and epidemiological implications. Such "scared straight" approaches are common in HIV prevention programs in Malawi and around the world. The presumption is that people will respond to exaggerated claims about HIV transmission risks in a uniformly self-protective way. Instead, over an eighth of the population responds fatalistically, taking more risks instead of fewer – and that group is at greater risk of contracting HIV than the rest of the population.[2] While it is not clear whether the optimal policy would be to tell everyone about the true risk of HIV transmission, one implication of my findings results is that HIV prevention campaigns should state that the transmission rate is less than 100%.[3] This would reduce the extent to which HIV prevention efforts unintentionally promote fatalistic behavior.

My results suggest that the fatalistic responses I observe in my sample are a general phenomenon, rather than being specific to HIV in southern Malawi, for two reasons. First, I am able to show that the heterogeneity in responses I observe is driven solely by variation in people's risk beliefs, and not by correlations between risk beliefs and other characteristics. Second, I document changes in the perceived chance of currently having HIV (or of getting it in the future) that are consistent with the core mechanism of my model.

These findings suggest that rational fatalism will affect choices about any condition that, like HIV, has three basic features which drive my model's results. First, people must perceive the condition to be binary. You either have HIV or not, and you can't get it more than once.[4] Second, the condition must be irreversible, so that all risks you take aggregate into a single probability. Third, the condition must be imperfectly observable - either because getting tested is difficult or because of unavoidable future exposures. This prevents the probability from resetting to zero, which would keep the expected cost function from approaching 100%.

Fatalism is therefore sometimes rational for many conditions, ranging from other incur-

[2]The elevated risk factors for the fatalistic group could also mean that scared-straight policies greatly exacerbate the overall HIV epidemic. Some epidemiological models of HIV attribute the sustained epidemic to a "core" of high-activity individuals, who could be disproportionately fatalistic. Alternative models focus on prevalence of concurrent partnerships across the entire population. The importance of fatalistic people in driving the HIV epidemic depends on which model is correct.

[3]The only mention of HIV transmission in the textbooks used to teach HIV prevention suggests that a single HIV exposure leads to infection for sure (Kadyoma et al. 2012, p.61).

[4]This may not be strictly true (see Section 1.2 for a discussion of HIV "reinfection") but what matters for rational fatalism is not the medical reality but that people *perceive* HIV infection to be binary.

able STIs like HSV-2 to exposure to carcinogens. Fatalism may also arise in the context of short-run decisions about curable diseases, such as the choice of whether to use bednets to prevent malaria. Malaria can be tested for and cured, but doing so takes time; in the short run, it is possible to get bitten enough times that you are convinced getting sick is inevitable. A mosquito-infested environment may also trigger fatalism by making it seem impossible to avoid all future bites.

My results therefore militate against many programs that rely on scared straight-style messaging to encourage safer behavior. Emphasizing that an activity's risks are extremely high – especially when they actually are not – can backfire, causing fatalism and increased risk-taking. This backfiring is likely to occur for binary, irreversible conditions for which one's status is imperfectly observed.

This paper contributes to three 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 effect of perceived risks on risk-taking behavior. Moreover, it shows risk compensation often cannot be meaningfully summarized by a mean elasticity, because people with very high initial risk beliefs may respond positively (fatalistically) to risks. Future empirical work on risk compensation should take this possible non-monotonicity into account.

Second, it contributes to a growing empirical literature that studies how people's subjective expectations affect their behavior. Expectations have long played an important role in economic models, but recent research has shown that it is possible to collect meaningful information on people's subjective expectations both in the developed world (e.g. Manski 2004) as well as in developing countries (e.g. Attanasio 2009; Delavande, Giné and McKenzie 2011; Delavande 2014). These subjective beliefs have consequences: Delavande and Kohler (2016) use data from Malawi show that subjective expectations about HIV risks drive individuals' choices about their number of sexual partners. I take this literature to its logical conclusion, providing the first experimental evidence that subjective expectations about risks have a measurable, causal effect on people's behavior. My results lend additional credence to the broader idea that we should be asking people about their subjective beliefs rather than assuming they know the true probabilities of events.

Third, it helps reconcile the substantial responses to HIV risks found in America (Ahi-

tuv, Hotz and Philipson 1996) with very small ones in Africa (Oster 2012). Self-protective responses by the majority of people may be offset by opposite-signed, fatalistic responses by a subset of the population, yielding an average response that is self-protective but low in magnitude. This is particularly plausible because gay men in the US perceive the prevalence of HIV to be much lower than Africans do (White and Stephenson 2014). The same reasoning may also help explain why recent field experiments in Africa have found large responses to information about HIV risks for specific population groups, despite the small overall risk responses (Chinkhumba, Godlonton and Thornton 2014, Derksen, Muula and van Oosterhout 2014, Dupas 2011, Godlonton, Munthali and Thornton 2015).

The remainder of this paper is organized as follows: I begin in Section 1 by laying out a general model of risk compensation, showing the conditions under which rational fatalism will occur. Section 2 describes the randomized field experiment that I conducted to test the implications of this model, as well as the data on risk beliefs and sexual risk-taking that I rely on. Section 3 lays out my empirical strategy and results, and also discusses the mechanisms behind my findings. In Section 4 I address some of the potential limitations of this paper, and in Section 5 I discuss the implications of my findings for the design of HIV prevention policies. Section 7 concludes. All appendix material can be found in the Online Appendix to the paper.

1 A Model of Risk Compensation

This section develops a general model of risk compensation, using the context of the HIV epidemic as a specific motivating example. Agents choose a level of risky sex, y by comparing the benefits $B(y)$ against costs that include both a fixed component, qy, and a stochastic component, Pc. The stochastic component is the product of the probability of having HIV, P, and the utility cost of being infected, c. A certain number of risky acts are unavoidable: agents have m_0 sex acts that have occurred since their most-recent HIV test, and m_1 future sex acts that are unnavoidable,[5] so total risk-taking is $n = y + m_0 + m_1$.

[5] m_1 captures accidental exposures through things like condom breakage, situations where an agent may lack the bargaining power to turn down some future sex acts, imperfect self control, and so forth.

To focus the exposition on the mechanism that drives fatalistic risk responses, rather than on mathematical derivations, I model the agent's choice as a one-shot, static decision. This collapses the future into the expected cost of HIV infection $P(x, y + m_0 + m_1)c$. The results in this section can be generalized to a multi-period setting – see Appendix A.4 for details. Thus the optimization problem is:

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

My model differs from the majority of the risk compensation literature in a simple but crucial way: I restrict P to reasonable values. Most empirical work on risk compensation uses the simplifying assumption that P is a linear function of the perceived risk per sex act, x, and the total number of risky acts, n. This assumption can generate probabilities that are not sensible: the total chance of having HIV, in my example, can exceed 100%.

I impose the most general possible restriction that avoids this problem, by requiring that $P = P(x, n)$ have a concave shape that asymptotes to a probability of 1. This corresponds to the intuitive notion that agents cannnot contract HIV more than once. This assumption drives my core result, which is that the comparative static of y with respect to x – the derivative of risk-taking with respect to per-act risks – is not always negative, or self-protective. The sign of the comparative static becomes positive, or fatalistic, if an agent's risk beliefs and stock of unavoidable risks are sufficiently high. This happens because the marginal cost of risk-taking will approach zero as the total chance of HIV infection gets close to 100%. Previous research has shown that this result holds when agents optimize with respect to the true total probability of HIV infection (Kremer 1996; O'Donoghue and Rabin 2001; Sterck 2014), which in this case would mean using the binomial distribution. I show that the change of the comparative static from self-protective to fatalistic will occur for *any* reasonable function that describes the total probability of being infected with HIV.

Throughout the model I treat HIV infection as irreversible, so that all risky acts aggregate into a single probability P. This is true of HIV if testing is unavailable, so that it is not possible to find out you are uninfected and reset the probability to zero. It is also true if we focus only on inevitable future exposures to the virus. It will only hold for certain other risks,

and depends on perceived rather than actual irreversibility of the condition. For example, if people perceive lung cancer to be a binary and irreversible condition, the model results will go through, but if a condition is widely known to be curable, such as Chlamydia, then they will not.

1.1 Comparative statics

For most possible functional forms of $B(\cdot)$ and $P(\cdot,\cdot)$ this optimization problem has 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 $y \to \infty$.[6] A sufficient condition for the existence of an interior optimum is that $q > 0$, so there is some fixed price or time cost to risky sex, i.e. $q \neq 0$.[7]

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

$$B'(y^*) - q - P_2(x, y^* + m_0 + m_1)c = 0 \tag{2}$$

$$B''(y^*) - P_{22}(x, y^* + m_0 + m_1)c \leq 0 \tag{3}$$

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

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

The denominator is just the left-hand side of the second-order condition, and is thus weakly

[6] The results in this section technically rely on the continuous differentiability of $P(x, y + m_0 + m_1)$. In Appendix A.3 I show that similar conclusions hold even for non-continuous risk-aggregation heuristics.

[7] q could be either a time cost or a monetary price paid for transactional sex. See Appendix A.1 for a proof.

negative.[8] Since $c > 0$, $\text{sign}(\partial y^*/\partial x) = -\text{sign}(P_{21}(x, y^* + m_0 + m_1))$. If we approximate P by a linear function, $P(x, y + m_0 + m_1) \approx x(y + m_0 + m_1)$, as is typical in the literature[9], then $P_{21} = 1 > 0$ always, so $\partial y^*/\partial x < 0$. This implies that behavior is uniformly self-protective: people always choose fewer risky acts as the per-act risk of each act rises.

1.2 Rational fatalism

Instead of imposing a linear functional form, I allow $P(x, y+m_0+m_1)$ to take any reasonable functional form that agents might use to add up risks. Critically, 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.[10] The subjective probability also approaches 1 as riskiness rises toward 1 or as total risk-taking goes to infinity.[11]

Any such function necessarily has a tipping point in its cross-partial derivative, P_{21}. The sign of the cross-partial derivative is initially positive, and becomes negative if x and $y + m_0 + m_1$ exceed a critical value. I prove this fact formally in Appendix A.2, and the mechanism behind the result is shown graphically in Figure 1.

[Figure 1 about here]

Figure 1 plots the total probability of contracting HIV (Panel A) and its first derivative with respect to x (Panel B). For this illustration I use the actual probability that comes from the binomial distribution, $P(x, y + m_0 + m_1) = 1 - (1 - x)^{y+m_0+m_1}$. Panel A plots

[8] I assume strict negativity, since otherwise $\partial y^*/\partial x$ is undefined. However, all the results in this section hold as the second-order condition approaches 0 from below.

[9] This is done explicitly in Oster (2012) and implicitly by Viscusi (1990), Philipson and Posner (1993), and Ahituv, Hotz and Philipson (1996) , for example.

[10] 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 $P_2(0, y + m_0 + m_1) = 0$.

[11] $P \to 1$ as $y + m \to \infty$ as long as $x > 0$, and $P = 1$ if $x = 1$ and $y + m_0 + m_1 \neq 0$.

the number of risky acts chosen on the horizontal axis and the total subjective probability of contracting HIV on the vertical axis. Panel B plots the marginal cost, which is the first derivative of the subjective probability with respect to the level of risk-taking. 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 solid 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 solid red line is *lower* than that of the dashed blue line. For this illustration I chose parameter values that set that tipping point to be at a value of 13, which I assume to be $m_0 + m_1$. In this example, then, the marginal cost of the sex acts the agent has control over (y) is higher for the agent with the *lower* perceived risk.

This result is summarized in the following proposition:

Proposition 1 (Tipping point in P_{21})

$$\exists \tilde{x} = \tilde{x}(y + m_0 + m_1) \ s.t. \ :$$

$$P_{21}(x, y + m_0 + m_1) \begin{cases} > 0, x < \tilde{x} \\ = 0, x = \tilde{x} \\ < 0, x > \tilde{x} \end{cases}$$

The cross-partial derivative of P with respect to x and $y + m_0 + m_1$ is initially positive and becomes negative when the per-act risk x becomes high enough.

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

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

$$\exists \tilde{x} = \tilde{x}(y + m_0 + m_1) \ s.t. \ :$$

$$\frac{\partial y^*}{\partial x} \begin{cases} < 0, x < \tilde{x} \\ = 0, x = \tilde{x} \\ > 0, x > \tilde{x} \end{cases}$$

Below the threshold value of the per-act HIV infection risk $\tilde{x}$, rational agents will behave self-protectively (reducing their risk-taking in response to increased risks); above $\tilde{x}$ they will behave fatalistically (increasing their risk-taking in response to increased risks).

This result is somewhat counterintuitive, but it captures a fairly simple logical conclusion: if the risks are sufficiently high and I can't totally avoid exposure, there is no value to limiting how much sex I have; I am doomed no matter what.[12]

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.

This sort of rationally fatalistic response is a potential issue for a wide range of decisions. Anti-smoking campaigns, to take one example, often feature "Benefit Timelines" that emphasize the health benefits that accrue to ex-smokers 20 minutes after quitting, 24 hours, 3 months, and so forth (e.g. National Health Service 2013). These timelines can be under-

[12]This is a purely rational alternative to the psychologically-driven fatalism derived by Caplin (2003). It is also similar in spirit to the Becker and Murphy (1988) rational addiction model in that choices are linked across periods by the effect of past behavior on the marginal utility of current choices. This analogy is made even more clear by the multi-period formulation of the model in Appendix A.4.

stood as a way to combat the possibility that smokers will think they are doomed to eventual cancer, no matter what they now decide. Similar to the benefit timelines in logic, HIV prevention messaging targeted at HIV-positive people emphasizes the risk of "reinfection" with a different strain of HIV (e.g. Cichocki 2014). Actual cases of reinfection are rare enough that the medical importance of this possibility is unclear (Smith, Richman and Little 2005), but one goal of this kind of messaging is to avoid a rise in risky sex by selfishly rational people who believe they have nothing to lose. Indeed, there is suggestive evidence that fatalistic reasoning about HIV infection is important in sub-Saharan Africa's HIV epidemic (Barnett and Blaikie 1992; Kaler 2003; Kaler and Watkins 2010; Wilson, Xiong and Mattson 2014).[13]

One consequence of Proposition 2 is that the linear relationship between x and y^* typically estimated in empirical analyses of risk responses is often misspecified, since y^* is in general a non-monotonic function of x. Estimated average partial effects of x on y^* will thuse usually include both positive and negative ranges of $\partial y^*/\partial x$, which will tend to push the average toward zero. Proposition 2 also yields a direct empirical test of the model: risk elasticities should become fatalistic (positive-signed) for sufficiently-high risk beliefs.

2 Data and Experimental Design

I test the model laid out in Section 1 using a field experiment I conducted in the Zomba District of Malawi's Southern Region. The experiment took place in Traditional Authority (TA) Mwambo from August to December 2012, using a final sample (after attrition) of 1,292 individuals. Each participant was interviewed twice: once for a baseline survey, and again for an endline survey conducted 1-3 months later.

I constructed the survey sample by randomly selecting 70 villages from TA Mwambo and then randomly selecting roughly 30 adults aged 18-49 from each village.[14] The village sample was stratified by distance from the nearest trading center; within each village, the sample of adults was stratified by gender. The baseline sample comprised 1,503 sexually-active adults. After a minimum delay of 30 days, the enumerator team attempted to recontact all 1,503

[13] It is also 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 (see Appendix A.5).

[14] See Appendix B.1 for details of the sample selection process.

sexually-active respondents from the baseline survey, successfully finding 1,292.[15]

Baseline demographic statistics for the overall sample, as well as a comparison of the treatment and control groups, can be found in Appendix Table B.2. The summary statistics are consistent with the randomization having successfully generated balanced treatment and control groups. 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.[16] There is also no evidence of differential attrition by baseline covariates, which I examine by interacting the treatment indicator with different baseline variables.[17]

2.1 Information treatment

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.[18] Participants from half of the villages, chosen at random, were assigned to the treatment group. They were read an information script that told them that the actual risk of HIV transmission in serodiscordant[19] couples that have unprotected sex is 10% per year (Wawer et al. 2005). I used the annual risk for the information treatment because it is simpler to explain than the per-act risk, which is very small, and also because it is the figure available on the Malawi National AIDS Commission's website (Malawi National AIDS Commission 2009).[20] The information treatment was administered by the survey enumerators in a one-on-one setting, and the information was presented both orally and visually.[21]

To minimize the risk of contaminating the control villages, all the baseline treatment surveys were done after the baseline control surveys were completed, following Godlonton,

[15] See Appendix Table B.1 for detailed figures on the number of people in each study arm and sampling stratum.

[16] See Appendix Table B.4.

[17] See Appendix Table B.5.

[18] 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).

[19] Relationships with one HIV-positive and one HIV-negative partner.

[20] For a discussion of the ethical dimensions of teaching people the true risk of HIV transmission, see Appendix C.

[21] See Appendix D for a detailed description of how the information was presented.

Munthali and Thornton (2015). The survey enumerators were only taught to administer the information intervention after all the control surveys were completed.

2.2 Measures of sexual behavior

My primary outcome measure is self-reported sexual behavior as recorded using a detailed retrospective sexual diary. The diary walks respondents through the previous seven days beginning with yesterday. On each day, respondents were asked what time they woke up, how much alcohol they had, whether they were menstruating (or for men, whether their sex partner was menstruating), how many times they had sex, and the time they went to sleep. Then, for each reported sex act, they were asked detailed questions such as the time of day, the length of the act, condom use, and whether the sex act was with their primary sex partner or a different partner. The surveys also contained single-question recall measures of sexual behavior, for example: "In the past 30 days, how many total times did you have sex, including serious and non-serious partners?"[22] As an additional measure of sexual risk-taking, enumerators sold respondents condoms at a subsidized price immediately after the endline survey. This the price (three condoms for MK5, or about ten cents) was a sizeable subsidy relative to the retail price of condoms at local stores, but the vast majority of respondents who had acquired condoms in the period leading up to the endline survey got them for free.[23] The condom sales measure was only collected at the endline survey.

The improved accuracy of the sex diary over other methods is reflected in the data captured by the surveys. The two variables record fairly similar average levels of sexual activity, but their distributions of the two variables are very different; there is substantially more heaping at multiples of 5 in the single-question recall variable.[24] Given the lower quality of the single-question recall variables, and since I focused on the sex diary variables in an

[22]The diary-based approach to measuring sexual behavior has been validated through previous work on sexual behavior in southern Malawi (Kerwin et al. 2011), and builds on research that shows that calendar-based methods reduce recall bias compared with single-question recall methods (Belli, Shay and Stafford 2001, Luke, Clark and Zulu 2011). Throughout this paper, I use the word "sex" to refer to heterosexual vaginal intercourse. Other forms of sexual activity are extremely uncommon in Malawi and are potentially sensitive topics (cf. Kerwin, Thornton and Foley 2014), so they were not included in the survey.

[23]Liquidity constraints were relieved by giving respondents six coins worth MK5 and allowing a maximum six condoms to be purchased.

[24]See Appendix E for histograms and a discussion of the implications of heaping for regression estimates.

earlier working paper I wrote prior to the experiment (Kerwin 2012), my preferred outcome measures come from the sex diary.

To address the issue of multiple comparisons and to improve the precision of estimates I construct combined outcome indices (Kling, Liebman and Katz 2007). Since some outcomes are measured with greater error or lack baseline data (e.g. condom sales were only done at endline), I construct two different sexual risk indices. The first uses only outcomes from the retrospective sexual diary, which I argue provides more accurately-measured outcomes than the single-question recall variables. An alternative index includes the sex diary outcomes as well as all other sexual risk-taking outcomes, including the condom sales.

Each index is constructed separately for the baseline and endline waves by normalizing all component variables (subtracting the control-group mean and then dividing by the control-group standard deviation). The normalization is reversed in sign for condom use, condom acquisition, and condoms purchased, for which positive numbers imply less risk-taking. These normalized values are then averaged for each respondent, weighted by the factor loadings for the first principal component of the matrix of the data for the control group. This follows Black and Smith (2006) in assuming that there is a single underlying sexual activity factor, and that the different outcomes measured in the data are noisy signals of that factor; the procedure selects the linear combination of the data that gives the best estimate of the underlying sexual activity factor.[25]

Table 1 presents baseline 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.[26] All the differences are fairly small in magnitude, but none of the variables has exactly equal means across the treatment and control groups at baseline. This is one reason my analyses control for respondents' baseline values of self-reported sex.

[Table 1 about here]

[25] I also explored unweighted averages; these produce similar results with slightly smaller magnitudes.

[26] This table presents only the main outcomes used in the paper. For balance statistics for the full set of sexual activity outcome measures, see Appendix Table B.3.

2.3 Measures of risk beliefs

The central prediction of the model I outline in Section 1 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.[27] 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. All the questions were gender-specific: for instance, when men were asked about HIV transmission they were asked about 100 men having sex with an HIV-positive woman, and likewise women were asked about 100 women having sex with an HIV-positive man.[28] The concrete style of expectation question I use on my survey has been validated through extensive use in previous research across a variety of contexts in Malawi, including in urban areas[29] as well as in areas of rural southern Malawi near my study site.[30,31] In the cross-section, subjective perceptions of HIV transmission risks are strongly correlated with sexual activity (see Panel B of Table 4), suggesting that they are relevent for understanding choices about sexual behavior.

One potential concern with eliciting subjective expectations is the tendency for probabilities to heap at the "focal" probability of 50%. People commonly use 50% (or in my case, report half of the total denominator), when they are simply unsure about the answer (Delavande and Kohler). To address this issue, I follow the Health and Retirement Study by prompting respondents who report beliefs of 50% with a followup question about whether they really believed the chance was 50%, or if they were just not sure (Hudomiet, Kézdi and

[27] An emerging literature has shown that it is feasible to collect meaningful data on subjective beliefs about probabilities using surveys in the developing world (e.g. Attanasio 2009, Delavande, Giné and McKenzie 2011, Delavande 2014). Delavande and Kohler (2009) have developed a method of eliciting subjective expectations using visual aids that they show performs very well in Malawi.

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

[29] Chinkhumba, Godlonton and Thornton (2014)

[30] Godlonton, Munthali and Thornton (2015), Kerwin et al. (2011)

[31] These questions perform comparably to Delavande and Kohler's approach; see Appendix F for details.

Willis 2011). Respondents who said they were just not sure were then prompted for their best guess. In my measure of risk beliefs I use the response to the followup question for people who change their answer.

As noted in Section 2.1, the enumerators were only trained to provide the information intervention after the baseline interviews for the control group were finished. This was done to minimize any chance of the information intervention contaminating the control group. However, it also meant that this was the first time the enumerators themselves were taught the true risk of HIV transmission. As a result, enumerators brought different beliefs with them into the baseline treatment and control surveys. This had a relatively small but statistically-significant effect on the measured beliefs of treatment-group respondents at baseline. As discussed in detail in Kerwin (2016), treatment group respondents have lower measured baseline values for all risk variables because the enumerators' knowledge affected the recorded values. Consistent with enumerator knowledge affecting measured beliefs, the control group's beliefs are also lower at endline, after the enumerators have been taught the risk information. To correct for this issue, I adjust reported beliefs based on time trends with a trend break. My results are not sensitive to this correction.

2.4 Composite belief measures

My analysis focuses on a composite measure of the perceived risk of contracting HIV from unprotected sex with a randomly-chosen potential sex partner. This is the product of two variables: 1) the perceived per-act risk of HIV transmission from unprotected sex with an infected partner; and 2) the perceived prevalence of HIV among attractive people of the opposite gender. I use this composite variable for three reasons. First, it is the same risk belief variable I used in the working paper that laid out the key theoretical results that motivated this project (Kerwin 2012). Second, using the perceived HIV prevalence among attractive people of the opposite sex mitigates the concern that people's self-beliefs about risks may differ from their beliefs about the risks faced by the rest of the population. Recent research on subjective expectations has highlighted that people's self-beliefs can be very different from what they believe about people in general and that people are more responsive to self-beliefs (e.g. Wiswall and Zafar 2014). While I cannot totally eliminate the potential

for differences between self-beliefs and general beliefs, focusing on the risk from unprotected sex with a random attractive member of the opposite sex (rather than all local people of the opposite sex) is likely to be a superior measure of the level of risk people feel they actually face. Third, relying on variation in both the prevalence and transmission beliefs allows me to avoid one of the shortcomings of using perceived per-act HIV risks, which is that they are extremely concentrated in the right tail (Figure 2, Panel A).[32]

[Figure 2 about here]

The resulting product also has a natural interpretation: it is how risky people perceive any given sex act to be if they do not know the HIV status of their partner, given their perceptions about the prevalence of HIV among potential sex partners and the transmission rate of the virus. Panel B of Figure 2 shows the distribution of this combined variable, which has a much smaller mass point at 100%.

3 Empirical Results

The information treatment has large effects on respondents' risk beliefs. Table 2 shows the endline treatment-control differences for all the measures of people's beliefs about HIV transmission and prevalence. People begin with extremely high risk beliefs: the control group believes that a unprotected single sex act with a randomly-chosen sex partner has a 4 in 10 chance of giving them HIV.[33]

[Table 2 about here]

[32] At baseline, over four in ten respondents believe that the per-act risk of HIV transmission from unprotected sex is 100%. This mass point at the top of the belief distribution hides the fact that people in the highest category of 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.

[33] These exaggerated risk perceptions are consistent with what students are taught in schools in Malawi. The textbooks for the course that covers HIV prevention in secondary school (Life Skills) reference the transmission rate only once. Page 61 of Kadyoma et al. (2012) describes a young woman who contracted HIV the first time she had sex, implying a transmission rate of 100%. Contracting HIV after a single exposure is possible, but uncommon; the book provides no information to put this example into context.

The treatment reduces the perceived annual risk of HIV infection from unprotected sex by 38 percentage points. The treatment group's beliefs about the per-act risk decrease even further, by 41 percentage points.[34] Note that the respondents do not update their beliefs perfectly: the actual annual transmission rate is about 10%; just 2% of the treatment group reports beliefs that low.[35]

The updating of beliefs is illustrated graphically in Figure 3. Beliefs evolve between survey waves in both the control group (the hollow dots) and the treatment group (solid dots). The changes in control-group beliefs are not totally random - beliefs tend to move toward the middle of the distribution in the endline survey, relative to the baseline survey. While this could reflect the evolution of risk perceptions in my sample, it is also likely driven by ceiling and floor effects, mean-reverting measurement error, and the enumerator knowledge contamination issue mentioned above in Section 2.3. The effect of the treatment on risk beliefs is evident from the large downward shift in the treatment group belief distribution relative to the control group distribution. My identification relies on this experimental change in risk beliefs, rather than the endogenous changes in risk beliefs observed in the control group.

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

These reductions in risk beliefs led to an increase in sexual activity. Table 3 presents

[34]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.

[35]In light of potential contamination of respondents' baseline risk perceptions, I also explore alternative specifications for this analysis. These confirm that these results are robust to controlling for baseline values of the outcome variable and running a difference-in-differences respectively (not shown).

reduced-form regressions of endline sexual activity y_i^e on the treatment indicator T_i, using the following specification:

$$y_i^e = \alpha + \beta T_i + \gamma y_i^b + Z_i'\eta + e_i \tag{5}$$

All my regressions control for baseline values of the outcome variable y_i^b.[36] Frison and Pocock (1992) show that this generates estimated treatment effects with a lower variance than either a) relying the endline values of the outcome alone or b) using changes in the outcome (i.e. a difference-in-differences). When there are baseline differences in outcomes across study arms, this approach also generates estimates with a lower bias than either alternative. (See Appendix G for a mathematical derivation). Controlling for the baseline value of the outcome will reduce the bias anytime the outcome variable is not exactly equal across study arms – even if the difference is not statistically significant. Since there are small but non-zero differences in the means of outcome variables across study arms, this is the preferred estimator for my sample. My regressions also control for Z_i, a vector of categorical dummy variables for the sampling strata (combinations of distance categories and gender), which improves statistical efficiency (Bruhn and McKenzie 2009). e_i is mean-zero error term. Continuous outcomes are presented in logs so the coefficient estimates can be interpreted as percentage-point changes.[37]

[Table 3 about here]

The impact of the treatment on sexual activitty 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

[36] For the condom sales there is no baseline data; instead I use baseline condom acquisitions (the same control as in column 5) as a proxy.

[37] Because many outcomes contain zeroes, I use the inverse hyperbolic sine transformation of Burbidge, Magee and Robb (1988) rather than logging the variable directly, constructing $log_{ihs}(y) = ln(y + \sqrt{y^2 + 1})$. Interpreting the semi-log coefficients as percentage point changes technically requires the adjustment recommended by Kennedy 1981: $\hat{\beta}_{pp} = e^{\hat{\beta} - \frac{1}{2}SE(\hat{\beta})^2} - 1$. However, this adjustment makes a trivial difference for all my estimates because the estimated standard errors are fairly small.

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.

3.1 The mean risk belief elasticity of sexual behavior

The effect of this specific information treatment on sexual behavior is less generalizable than the elasticity of sexual risk-taking with respect to HIV risk beliefs, which can be used to design other policy interventions involving responses to HIV infection risks. Consider the OLS regression

$$y_i^e = \alpha + \delta x_i^e + \gamma y_i^b + Z_i'\eta + e_i \tag{6}$$

$\hat{\delta}$ is an estimate of $\partial y^*/\partial x$, the partial effect of risk beliefs on risky sex. However, for this estimate to be consistent, x_i^e must be independent of the error term. This is unlikely to be true, because beliefs and behavior tend ot be codetermined.

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 from the second-stage regression. Because the treatment was randomized, membership in the treatment group should have no association with sexual behavior other than through the information treatment. Furthermore, the information treatment is very unlikely to affect sexual behavior through any channel other than individuals' risk beliefs: it does not contain any guidance or information about sex. The instrument also easily satisfies the relevance condition. The F-statistic on T_i in the first-stage regressions is roughly

220 for all specifications.[38] This allows me to estimate two-stage regressions as follows:

$$x_i^e = \alpha^x + \beta T_i + \gamma^x y_i^b + \rho^x x_i^b + Z_i'\eta^x + e_i \tag{7}$$

$$y_i^e = \alpha^y + \delta \hat{x}_i^e + \gamma^y y_i^b + \rho^y x_i^b + Z_i'\eta^y + v_i \tag{8}$$

x_i^b is included as a control in the first stage in order to improve efficiency and reduce bias, for the same reason that I control for y_i^b.

The 2SLS and OLS estimates are shown in Panels A and B of Table 4 respectively. The OLS regressions are estimated on the control group only. The OLS results have a uniform positive bias relative to 2SLS, confirming that OLS is not consistent in this context.[39] 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.

[Table 4 about here]

The elasticity of sex acts in the past week with respect to HIV risk beliefs is approximately -0.6. The other elasticities are smaller in magnitude: they are mostly around -0.3, which is the estimate yielded by the sexual activity index method. These results are much larger than Oster (2012), which estimates prevalence elasticities of about -0.01 to -0.02 for binary outcomes (compared with -0.3 for my binary outcome in column 1). My estimates are closer to the Ahituv, Hotz and Philipson (1996) estimates for the US: they find elasticities of about -0.2 for binary outcomes. My estimates for continuous outcomes are also close to those found in US studies: focusing on gay men in San Francisco, Auld (2006) estimates a prevalence

[38] It is not possible to conduct the typical formal test for weak instruments from Stock and Yogo (2005) unless the number of excluded instruments is at least two more than the number of endogenous regressors. 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.

[39] This matches the findings of 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.

elasticity of sexual acticity of -0.5. However, my results are not directly comparable with this earlier work, which uses the true prevalence as the regressor of interest. People do not accurately know the true prevalence, so changes in the true prevalence are unlikely to show up 1-for-1 as changes in perceived prevalence. This means that the implied prevalence elasticities from my results are likely to be smaller than those for the US, and closer to the Oster (2012) findings.

The population-average reduced form effects and elasticities both fit a model of self-protective risk-compensation, which is consistent with the existing literature. However, the specifications in Tables 3 and 4 impose common effects across all respondents, and hence across all levels of risk beliefs. This is at odds with the model laid out in Section 1, which holds that the elasticity will vary in magnitude as well as sign across the population.

3.2 Heterogeneity in the reduced-form effect of the risk information treatment

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

$$y_i^e = \alpha + \beta T + \sum_{j=1}^{J} \left[\beta^{Tw^j} T_i w_i^j + \delta_j w_i^j\right] + \gamma y_i^b + Z_i'\eta + e_i \tag{9}$$

Here $w_i^1, ..., w_i^J$ 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 5. Responses to the information treatment are

strongly heterogeneous by baseline risk beliefs (Column 2). Using this linear specification, people with baseline risk beliefs of 0% respond to the information treatment by increasing their sex acts per week by 32%. For people with baseline beliefs of 100%, the response is lower by 50%, meaning that weekly sexual activity *declines* by 18%. I can reject that responses for people with high risk beliefs are the same as for those with low beliefs at the 1% level; the negative response for people with the highest risk beliefs is statistically significant at the 10% level (p=0.052). The positive treatment effect for people who have baseline beliefs of 0% suggests that a linear specification for the treatment effect heterogeneity is misspecified, since their risk beliefs should increase rather than decrease in response to the treatment. This lends further support to the flexible analyses I conduct below.

[Table 5 about here]

The specification in Table 5 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. I relax tis restriction by estimating semiparametric regressions of dy/dT by baseline risk beliefs for the treatment and control groups:

$$y_i^e = \beta^T + f^T(w_i) + \gamma^T y_i^b + Z_i'\eta^T + \varepsilon_i \text{ if Treatment} = 1 \tag{10}$$

$$y_i^e = \beta^C + f^C(w_i) + \gamma^C y_i^b + Z_i'\eta^C + \nu_i \text{ if Treatment} = 0 \tag{11}$$

These regressions give me estimates of $\mathbb{E}[y|T=1]$ and $\mathbb{E}[y|T=0]$ for each value of w_i.[40] Thus taking the difference gives me estimates of the w_i-specific treatment effect $\hat{\tau}_y(w_i) = \hat{f}^T(w_i) - \hat{f}^C(w_i)$.[41]

I implement the semiparametric regressions using the Robinson (1988) double residual estimator for partially linear regressions.[42] I choose data-driven bandwidths to minimize

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

[41] A purely nonparametric version of this estimator is used in the Bennear et al. (2013) study of behavioral responses to information about arsenic in drinking water.

[42] The underlying semiparametric regressions do not have ias problems because they are fit using local linear regressions (Fan and Gijbels 1996). 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).

the mean-squared prediction error using the generalized cross-validation (GCV) statistic of Loader (2004).

I construct confidence intervals for these estimates via a clustered bootstrap with 1000 repetitions; for each bootstrap repetition, I repeat the procedure of adjusting belief variable to correct my estimated confidence intervals for the fact that it is a generated regressor. In each bootstrap sample, I trim observations with estimated densities lower than the minimum observed in the original dataset. The original sample has no estimated densities that are near zero, so my point estimates do not have trimming issues. Replicating the results while trimming at zero instead does not appreciably change the estimates, suggesting that very few observations have extremely small estimated densities.

I apply this approach to heterogeneity in my reduced-form regressions of treatment effects on sexual activity, estimating a function $\tau_y(x_i^b)$. Panel A of Figure 4 graphs the results. The semiparametric reduced-form estimates are consistent with those from the linear approximation in Table 5: the treatment effect is initially positive, and then becomes negative for people with extremely high baseline risk beliefs. For people with the highest baseline beliefs, I can reject the null that the treatment effect is ≥ 0 at the 1% level.

[Figure 4 about here]

3.3 Heterogeneity in the risk belief elasticity of sexual behavior

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

To do this, I develop an estimation strategy that can decompose instrumental-variables estimates by any baseline covariate w_i. I begin by defining subgroup k of the sample as those individuals with $w_i = w^k$. Since T_i and w_i are independent, the treatment remains a valid instrument for this subsample. Selection on right-hand side variables likewise does not affect the consistency of an estimator, so any valid instrumental variables estimator for

the whole sample will be valid for this subsample (Heckman 1996). While I could rely on 2SLS estimation, in general I will want to estimate the relationships semiparametrically, so I instead use the indirect least squares (ILS) estimator. I estimate the following separate regressions:

$$y_i^e = \alpha^y + \beta^y T_i + \gamma^y y_i^b + Z_i'\delta^y + v_i \text{ for } w_i = w^k \tag{12}$$

$$x_i^e = \alpha^x + \beta^x T_i + \gamma^x y_i^b + Z_i'\delta^x + e_i \text{ for } w_i = w^k \tag{13}$$

with w_i being the baseline belief variable and w_k represents each of its values. Equation 12 yields the reduced-form estimates described above. Estimating equation 13 yields semiparametric decompositions of the first-stage effect of the information treatment on endline risk beliefs by individuals' baseline risk beliefs. The effect of the information treatment on endline risk beliefs is uniformly negative; see Appendix H for the results of this decomposition. I then construct

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

where convergence in probability comes from Slutsky's theorem. The overall LATE can be recovered from these w_i-specific LATEs by taking a weighted average of them, where the weights are the product of the share of the data that has a given value of w^k and the strength of the first stage for w^k.[43]

I estimate the w_k-specific treatment effects $\hat{\beta}^x(w^k)$ and $\hat{\beta}^y(w^k)$ using $\tau^x(w^k)$ and $\tau^y(w^k)$ as described above. While it is possible to construct analytic standard errors for ILS, I rely instead on cluster-bootstrapped confidence intervals since my preferred underlying estimator is already semiparametric and has standard errors without a known analytical form.

The results of this procedure, using the log of sex acts in the past week as the outcome variable, are shown in Panel B of Figure 4. These elasticities are consistent with the theoretical framework from Section 1, 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. The risk elasticity of risky sex is greater than zero for 13.8% of people, and it varies from -2.3 for the lowest risk beliefs to 2.9 for the highest ones. This

[43] See Appendix I for a derivation.

evidence suggests that the underlying relationship between y and x is U-shaped relationship. However, I am unable to recover the underlying function: I can estimate heterogeneity in the endline risk belief elasticity of risky sex only by *baseline* risk beliefs, not by endline beliefs.

3.4 Mechanisms for fatalistic responses

The theoretical framework in Section 1 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. If the first mechanism alone is driving the fatalism measured in my 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 5 shows that there is no statistically-significant difference in the treatment effect by people's baseline beliefs about their HIV status.[44] Another implication of the first mechanism is that HIV testing should reduce fatalism. I find evidence for this in my sample. Column 8 of Table 5 shows that fatalistic responses are very strong among people who report never having been tested for HIV; people who have had a test are less fatalistic.

Another implication of the model is that the information treatment should shift people's beliefs about their current HIV status or about whether they will contract HIV in the future. To examine this, I use endline data about respondent's perceived likelihoods of current or future HIV infection. I run multinomial logits of the endline perceived likelihood variables on a treatment indicator, controlling for sampling strata and categorical indicators for the values of the baseline perceived likelihood variable.[45] These consider the different likelihood values, as well as "Don't Know," as discrete choices. I estimate these regressions separately for each quantile of risk beliefs. Figure 5 reports the mean marginal effects on people reporting there is "No Likelihood" that they have HIV from these regressions, multiplied by negative 1. These can be interpreted as the effect of the information treatment on people believing

[44] This result does not differ by baseline risk beliefs (not shown).

[45] 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.

there is any chance that they have HIV now (Panel A) or will get it in the future (Panel B).

[Figure 5 about here]

I find evidence for both potential mechanisms for fatalism. The information treatment decreases the probability that people with high initial risk beliefs think there is any chance they currently have HIV by 18 percentage points compared to a control-group mean of 38%. The effect on perceiving there is any chance that you will contract HIV in the future is even stronger: it decreases by 19 percentage points.[46] This suggests that the results presented in Figure 4 can indeed be explained by reductions in fatalism among the highest-risk group.

These results help may explain the small measured responses of sexual behavior to HIV testing. Thornton (2008) finds zero average effects for HIV-negatives and very small average reductions in risk-taking for HIV-positives in Malawi. One possible explanation for these small responses is that people's high perceived risk of contracting HIV means that testing has a limited effect on their perceived lifetime risk of becoming HIV positive: even if a person tests negative today, she may continue to think that contracting HIV is highly likely in the future. Likewise, a current positive test may not be a substantial surprise. Consistent with this argument, Gong (2015), studying people in urban Kenya, finds that responses to HIV testing vary by people's priors about their HIV status. People who are surprised by a test result respond in a selfishly rational manner, with large increases in risk-taking when people are surprised by positive test results and large declines in risk-taking in response to surprise negative test results. My results imply that HIV testing alone may not 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.

[46] The results on the perceived chance of getting HIV in the future are also robust to conditioning on respondents saying there is no likelihood that they currently have HIV.

4 Potential Limitations

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

The results shown in Figure 4 show that the HIV risk elasticity of sexual activity varies by respondents' baseline risk beliefs. However, these beliefs are not assigned at random, and therefore may be correlated with the respondents' other characteristics. It is therefore possible that some of the heterogeneity in risk responses is coming from other factors correlated with risk beliefs, rather than from the beliefs themselves.

To explore this possibility, I repeat the analysis from Column 2 of Table 5, interacting the treatment with various other baseline variables instead of the risk belief variable. In Columns 3 through 7 I look for heterogeneous responses by gender, baseline sexual activity, perceived previous exposure to HIV, whether the respondent believes he or she may currently be HIV-positive, and whether the respondent has ever had an HIV test. There is also no statistically-significant heterogeneity by any of these factors. Column 9 of the table replicates Column 2, but all also interacts the treatment indicator with an extensive list of baseline variables.[47] The results 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.

4.2 Social desirability bias

A separate potential limitation of this paper is social desirability bias: I rely on self-reported sexual behavior to measure of sexual risk-taking, rather than objective measures of STI status, which may not yield accurate estimates of treatment effects due to social desirability bias (Baird et al. 2012). Social desirability bias is unlikely drive my results for two reasons.

[47] These variables include all those used in Columns 3 through 7 of the table as well as 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.

First, my information treatment should not have induced differential biases in self-reports across study arms: it provided no direct modeling of "good" behavior nor encouragement to behave in a specific way.[48] Second, any differential pattern of social desirability bias would also have to be heterogeneous by baseline risk beliefs in the same way predicted by my model.

For the purposes of this paper, self-reported sex also has an important advantage compared with using STIs to measure sexual risk-taking. The STI most commonly used to measure sexual activity is HSV-2, which is untreatable. Since I could only measure risk-taking by the initiallity-unifected, this would effectively screen out some of the high-risk individuals who are crucial for my analysis.

4.3 Sensitivity analyses

A final potential limitation is that my analyses of heterogeneous treatment effects are potentially subject to the Deaton (2009) critique that subgroup analyses can constitute ex post "fishing expeditions." However, that concern is mitigated due to the fact that my main theoretical results were laid out in earlier work done prior to the experiment (Kerwin 2012). I also use identical definitions for my primary outcome variable and my risk belief variable in this paper and in the earlier working paper, limiting the number of researcher degrees of freedom involved in my analysis. I provide two additional checks that reinforce the idea that my results are not driven by spurious relationships.

First, my results are robust to a number of potential variations in my approach. In particular, the same patterns is present if halve all the bandwidths used in the semiparametric estimation process (see Appendix Figures J.1 to J.3). The patterns are also robust to replacing the Robinson estimator with an alternative approach that divides the baseline risk variable into quantiles and interacts indicators for those quantiles with the treatment dummy. Appendix Figure J.4 shows that the reduced-form estimates have the same pattern under the bracketed approach, with negative treatment effects for the highest risk beliefs; Appendix Figure J.5 likewise shows that people with the highest risk beliefs have positive elasticities. This approach is substantially less computationally intensive than the Robinson

[48] de Walque, Dow and Gong (2014), studying a treatment that is not expected to cause differential social desirability bias, find that self-reports have a limited bias relative to STI measures.

estimator, allowing me to show that pattern of reduced-form estimates is robust to a wide range of alternative specifications. In Appendix J, I test a number of alternative methods of handling the baseline risk beliefs, and a number of different outcome measures and estimation strategies, showing that my main result is unaffected by these changes.[49]

Second, the statistically-significant negative effect of the information on risk-taking for fatalistic individuals is robust to correcting for multiple comparisons. The graphs in Figure 4 use pointwise confidence intervals, rather than simultaenous ones. This leaves open the possibility that the statistically-significant positive elasticity for people with high risk beliefs results from multiple comparisons. To address this possibiity, I rely again on the bracketed approach, and apply the conservative Bonferroni correction to the p-values, multiplying them by eight (which is the number of brackets). The adjusted p-value for the highest bracket is below 0.02.

5 Implications for HIV Prevention Policy

The information about the true risk of HIV transmission slightly increases sexual activity for most people, but sharply decreases it for people with the highest risk beliefs. The effect of the information treatment on overall HIV transmissions is therefore ambiguous, because some research suggests that HIV transmission depends strongly on high-activity groups (e.g. Koopman, Simon and Riolo 2005). If this theory is correct, high-activity individuals who are responsible for keeping the epidemic alive and spreading it to the rest of the population. Competing theories instead attribute the sustained HIV epidemic to concurrent sexual partnerships (e.g. Epstein and Morris 2011). Determining the overall effect of the information treatment on the HIV epidemic would require detailed knowledge not only which of these two basic epidemiological models best fits HIV in southern Malawi, but also the parameter values that fit the model to the data. Such an exercise is beyond the scope of this paper. However, it is informative to look at how risk factors for HIV

[49] In particular, my core finding - that the behavioral response to the information treatment is reversed for people with the highest initial risk beliefs - is robust to a) redefining the risk belief variable to be an index of all of the risk belief variables in the survey (Appendix Figure J.10), b) dropping the controls from the regression (Appendix Figure J.11), and c) redefining the outcome variable to be an index of all the possible outcome variables (Appendix Figure J.19).

transmission vary with the baseline beliefs that determine who responds fatalistically to the information treatment.

Figure 6 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 - depending on the underlying epidemiological model at work. A targeted information campaign, that restricted access to the information only to fatalistic people, could be even more beneficial; however, it may be difficult to prevent the information from spreading to other groups.

[Figure 6 about here]

An alternative to an information campaign that tells people about the true risk of HIV infection would be one that simply informs people that the transmission rate is not 100%. This would reduce most people's risk perceptions, but would have the largest effect for people with the most-exaggerated beliefs - who are the same individuals that behave fatalistically on average. A potential example would be to inform people that serodiscordance is fairly common. Since most people believe that it is impossible, this would lower the perceived transmission rate of the virus.

6 External Validity

The point estimates in Section 3 are representative of sexually-active adults in the region where the experiment took place. These are primarily married individuals: marriage rates are very high in southern Malawi (over 80% of my respondents are married).[50] In Section

[50]Changes in sexual activity by married people in response to HIV risks are plausibe in this setting because southern Malawi has high rates of perceived and actual infidelity; see Appendix K for details. The estimated rates of fatalism in my sample could therefore plausibly be lower bounds, since my sample does

4.1 I showed that responses to the information treatment, and hence the risk belief elasticity of risk-taking, vary only by individuals' baseline beliefs, and not by other fixed covariates. Taken literally, this suggests that the estimated elasticities should be valid for other areas that have similar demographics to my sample - which would include most of southern Malawi. Extrapolating to other high-HIV-prevalence settings in Africa may also be plausible: my results are for a sample of primarily married people, and married people play an important for the HIV epidemic across Africa.[51]

Beyond the elasticity values I estimate in this paper, the underlying phenomenon that I document - a tipping point value of the perceived risk, above which risk compensation changes from self-protective to fatalistic - is likely to generalize to many settings. The mechanism tests in Section 3.4 show that fatalistic people become less likely to think they are HIV-positive as a result of the information treatment. This is consistent with the heterogeneity in the risk elasticities being driven by the effect of changes in the per-act risk on the stock of accumulated acts as well as the flow of new acts. Rational fatalism is therefore likely to arise for other conditions that share this underlying mechanism.

7 Conclusion

Empirical research on behavioral responses to health risks has traditionally assumed that responses to risks are uniformly self-protective, and focused on mean elasticities as summaries of risk compensation across a population. I use a randomized field experiment in rural southern Malawi to explore the validity of this assumption in the context of 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.

While I find that the mean elasticity is about -0.6, this average masks significant variation across the popular. I develop a method to allow for heterogeneity in instrumental variables

not target high-risk individuals. A related issue is that both my model and my elasticity estimates assume that people can independently choose how much sex they have; see Appendix L for a discussion of these general equilibrium issues.

[51] Up to 70% of new HIV infections occur within married couples (Gray et al. 2011).

estimates of parameter values and use it to break down the elasticity by people's initial risk beliefs. The effect of risk beliefs on risky sex is negative (consistent with self-protective responses) for people who initially hold low risk beliefs, and becomes positive (consistent with fatalism) as initial risk beliefs become sufficiently high.

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

My results imply that the use of mean elasticities as a way to summarize the response of health behaviors to health risks may be misleading. In the case of HIV in particular, some epidemiologists argue that aggregate HIV transmission is dominated by high-sexual activity individuals. If this model (rather than competing explanations such as concurrent sexual partnerships) is accurate, 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. When 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; they look worse in terms of other HIV risk factors as well. While it is uncertain how spreading information about the transmission rate of HIV would affect the number of new HIV cases, one definitive implication of my results is that HIV prevention campaigns should make it clear that the transmission rate is less than 100%.

I am able to show that the heterogeneity in risk compensation I document comes from

risk beliefs themselves and not other correlated factors such as demographics. Moreover, fatalistic people reduce their perceived chance of having HIV in response to the information treatment, which is consistent with the mechanism underlying the model. Hence my core result - that the rational response to an increase in risk is sometimes fatalistic - is likely to hold for risk compensation in response to conditions that are binary, irreversible, and imperfectly observed. These include other incurable STIs like HSV-2, exposure to cancer-causing chemicals, and possibly even short-run responses to malaria.

The extent to which mean elasticities are a useful summary statistic for risk compensation for these conditions will depend on how many people hold extreme risk beliefs, 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 infection into rational epidemic models of HIV and other infectious diseases, rather than just assuming agents understand the true prevalence and transmission rate of the virus. Such models should also allow for responses to perceived risks to be heterogeneous by the level of the perceived risk, rather than imposing that they are the same across the whole population.

The formation of people's risk beliefs is another important area for study. While anecdotal evidence suggests that people learn about HIV in school, the exact process by which many people arrive at gross overestimates of the prevalence and transmission rate of the virus is still unknown. Given that overestimating HIV risks seems to scare people to death, rather than scaring them straight, getting at the source of these overestimates may be crucial for understanding the continued spread of HIV in Africa.

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Table 1
Sexual Activity Baseline Balance

	N	Overall	Control	Treatment	C-T
	(1)	(2)	(3)	(4)	(5)
Any Sex in Past Week	1292	0.52	0.54	0.51	-0.03
Sex Acts in Past Week	1292	1.71	1.80	1.62	-0.18
Unprotected Sex Acts in Past Week	1292	1.52	1.57	1.47	-0.10
Sex Partners in Past 30 Days	1290	0.81	0.82	0.80	-0.02
Condoms Acquired in Past 30 Days	1288	4.13	4.74	3.53	-1.21
Overall Sexual Activity Index†	1277	0.00	0.03	-0.02	-0.04
Diary Sexual Activity Index†	1292	-0.04	-0.01	-0.06	-0.05

Notes: Balance statistics for the main outcome variables used in the paper. For balance statistics for the other sexual activity variables that are used to construct the index variables, see Appendix Table B.3
† The Sexual Activity Index variables are weighted averages of normalized values of all available outcome measures (Overall Index) or just the outcomes measured on the Sex Diary, which are measured with less noise (Diary Index). The weights used are factor loadings for the first principal component of the outcomes for the control group. Sample includes 1,292 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

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				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment Group	-0.384***	-0.045***	-0.371***	-0.071***	-0.162***	-0.047***	-0.182***	-0.185***
	(0.019)	(0.006)	(0.016)	(0.012)	(0.016)	(0.015)	(0.014)	(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
Control Mean(Dep. Var)	0.742	0.082	0.905	0.176	0.485	0.463	0.351	0.424
Control SD(Dep. Var)	0.318	0.162	0.198	0.264	0.290	0.265	0.268	0.263

Notes: 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 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed. Heteroskedasticity-robust standard errors, clustered by village, in parentheses: * p<0.1; ** p<0.05; *** p<0.01.

Table 3

Regression Estimates of the Effect of Information about HIV Transmission Risks on Sexual Behavior

	Any Sex in Past Week	Log Sex Acts in Past Week	Log Unprotected Sex Acts in Past Week	Log Sex Partners in Past 30 Days	Log Condoms Acquired in Past 30 Days	Log Condoms Purchased	Log Overall Sexual Activity Index†	Log Diary Sexual Activity Index†
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment Group	0.050**	0.101**	0.071	0.012	0.080	0.054	0.063*	0.057**
	(0.024)	(0.047)	(0.045)	(0.019)	(0.075)	(0.105)	(0.032)	(0.024)
Observations	1,292	1,292	1,292	1,290	1,283	1,286	1,261	1,292
Adjusted R-squared	0.238	0.277	0.260	0.288	0.140	0.047	0.378	0.225
Ctrl Mean(Dep. Var)	0.490	1.67	1.48	0.77	2.52	5.08	-0.03	-0.02
Ctrl SD(Dep. Var)	0.500	2.39	2.29	0.58	9.65	6.59	0.99	1.03

Notes: 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. All regressions include controls for sampling strata (distance category X gender).

† 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.

Sample includes 1,292 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

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

	Any Sex in Past Week	Log Sex Acts in Past Week	Log Unprotected Sex Acts in Past Week	Log Sex Partners in Past 30 Days	Log Condoms Acquired in Past 30 Days	Log Condoms Purchased	Log Overall Sexual Activity Index†	Log Diary Sexual Activity Index†
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: 2SLS Estimates								
Endline Risk Belief	-0.260**	-0.562**	-0.412*	-0.043	-0.375	-0.256	-0.327**	-0.317**
	(0.121)	(0.241)	(0.232)	(0.102)	(0.402)	(0.535)	(0.159)	(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
Panel B: OLS Estimates (Control Group Only)								
Endline Risk Belief	0.155***	0.175*	0.106	0.196***	0.118	-0.337	0.318***	0.180**
	(0.054)	(0.102)	(0.103)	(0.058)	(0.172)	(0.224)	(0.100)	(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

Notes: 2SLS estimates use the randomized treatment group assignment as an instrumental variable for endline beliefs. 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. 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. Endline Risk Belief is 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.

† 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.

Sample includes 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed. Heteroskedasticity-robust standard errors, clustered by village, in parentheses: * p<0.1; ** p<0.05; *** p<0.01.

Table 5

Heterogeneity in Effects of Information Treatment by Baseline Risk Beliefs and Other Baseline Covariates

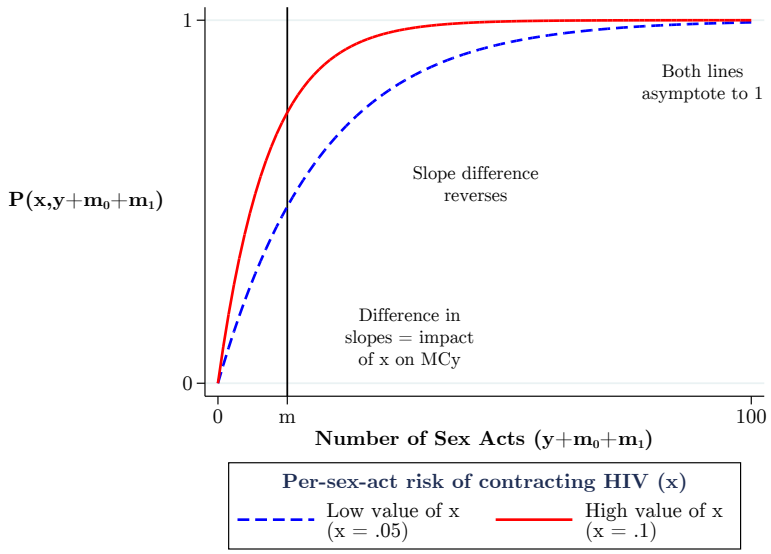
	Outcome: Log Sex Acts in Past Week								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Treatment (T)	0.101**	0.325***	0.123*	0.070	0.136**	0.101	0.040	0.619***	0.340
	(0.047)	(0.083)	(0.072)	(0.057)	(0.060)	(0.062)	(0.131)	(0.210)	(0.341)
T*(Baseline Risk Belief [0-1])†		-0.514***						-1.248***	-0.507***
		(0.165)						(0.445)	(0.182)
T*(Male)			-0.049						0.011
			(0.131)						(0.145)
T*(Baseline Log Sex Acts in Past Wk.)				0.035					-0.117
				(0.051)					(0.108)
T*(Ever Exposed to HIV)					-0.151				-0.060
					(0.113)				(0.123)
T*(Any Chance I am HIV-positive)						-0.007			0.010
						(0.117)			(0.129)
T*(Ever Had HIV Test)							0.063	-0.344	-0.033
							(0.147)	(0.228)	(0.155)
T*(Ever Had HIV Test)*(Baseline Risk Belief)								0.857*	
								(0.489)	
T Interacted with Other Baseline Covariates‡	No	No	No	No	No	No	No	No	Yes
Observations	1,292	1,275	1,292	1,292	1,275	1,277	1,225	1,210	1,184
R-squared	0.277	0.284	0.277	0.277	0.277	0.276	0.281	0.291	0.307

Notes: 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. Baseline Risk Belief is 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, and is adjusted for non-constant time trends.

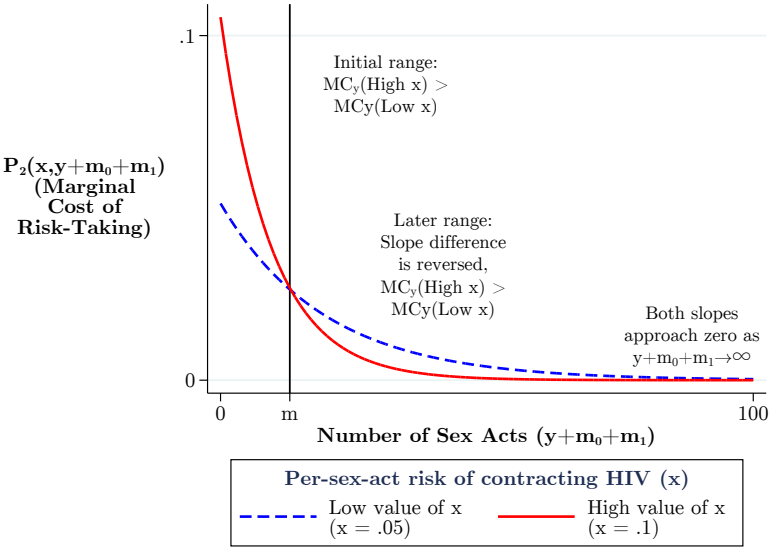
‡Other baseline covariates include immediate and delayed word recall [each 0-10], numeracy score [0-3], score on Raven's progressive matrices [0-3], lifetime sex partners, whether respondent had any sex in the past week, and indicators for marital status, age category, ethnic group, education level,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

Sample includes 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed. 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.

Figure 1
Illustration of Tipping Point in Marginal Cost of Sexual Activity



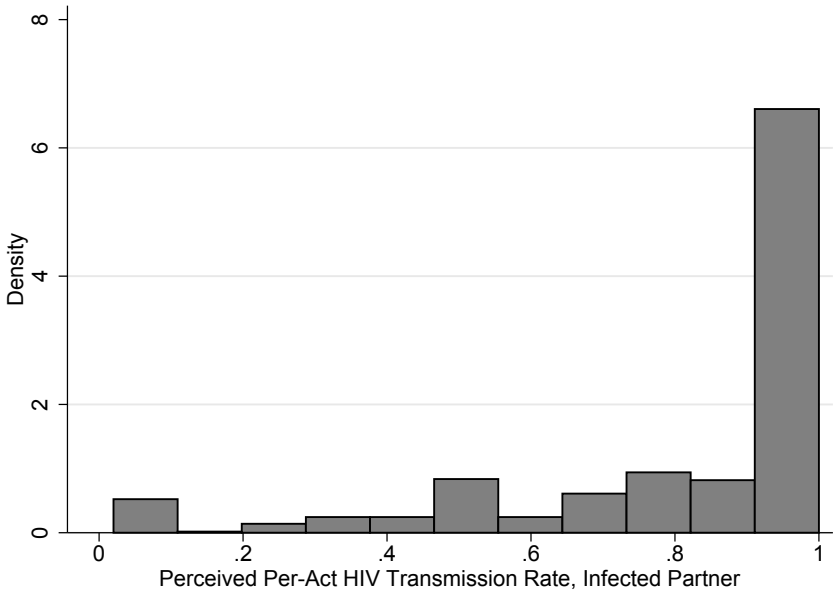
Panel A: P[HIV Infection|Number of Sex Acts] for Low and High Values of Per-Act Risk



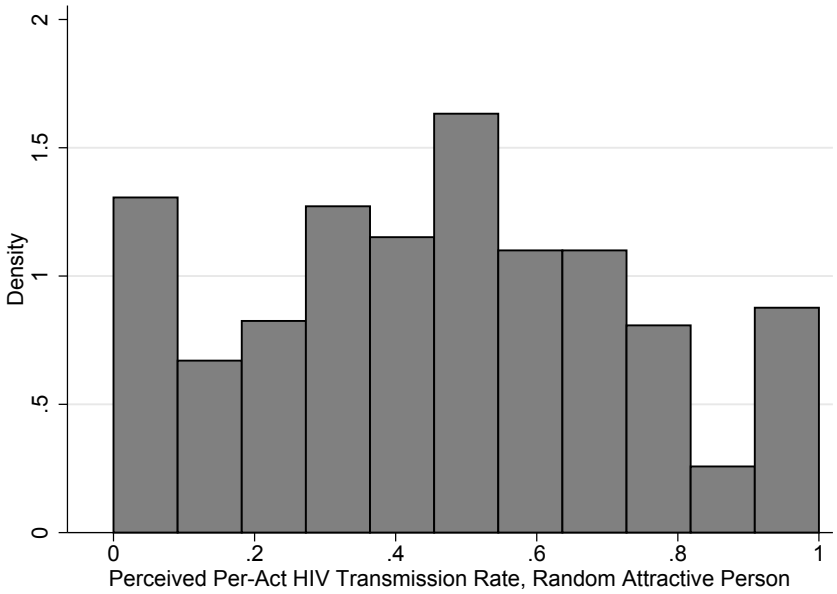
Panel B: MC(Sex Act |Number of Sex Acts] for Low and High Values of Per-Act Risk

Notes: Panel A illustrates the total probability of HIV infection, as a function of the number of sex acts chosen, $y + m_0 + m_1$, for different levels of the per-act risk, x. The illustration uses the true function $\pi(x, y + m_0 + m_1) = 1 - (1 - x)^{y+m_0+m_1}$ with $m_0 + m_1$ set to a value of 13, but the same conclusions hold for any reasonable risk aggregation function $P(x, y + m_0 + m_1)$. The dashed blue line shows a low value of the per-act risk ($x = 0.05$) and the solid red line shows a a high value ($x = 0.10$).
Panel B directly illustrates the marginal costs for different ranges of y given the two levels of the per-act risk; the marginal cost is larger for the *lower* per-act risk in the second portion of the graph, which is what generates the fatalistic range of responses.

Figure 2
Histograms of Baseline HIV Infection Risk Beliefs, Control Group



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

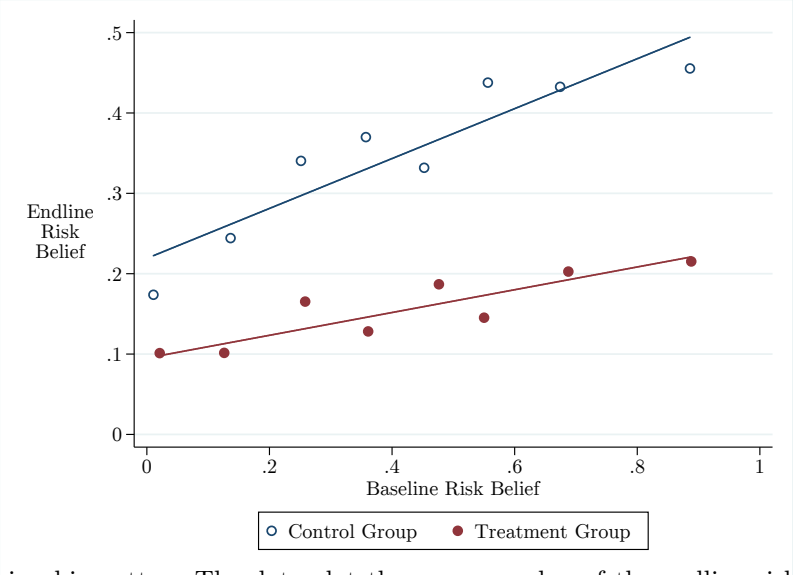


Panel B: Per-Act Infection Risk from Unprotected Sex with a Randomly-Selected Partner

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 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed.

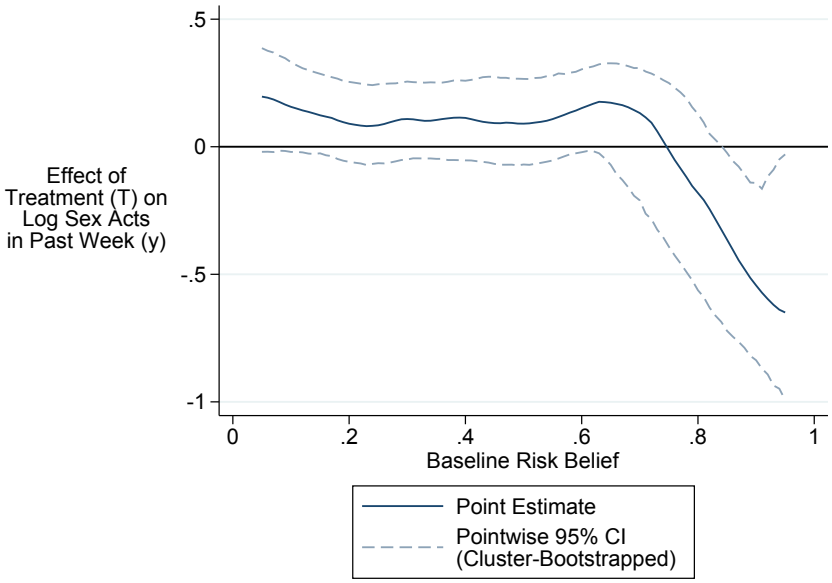
Figure 3

Binned Scatterplot of Endline Risk Beliefs by Baseline Risk Beliefs

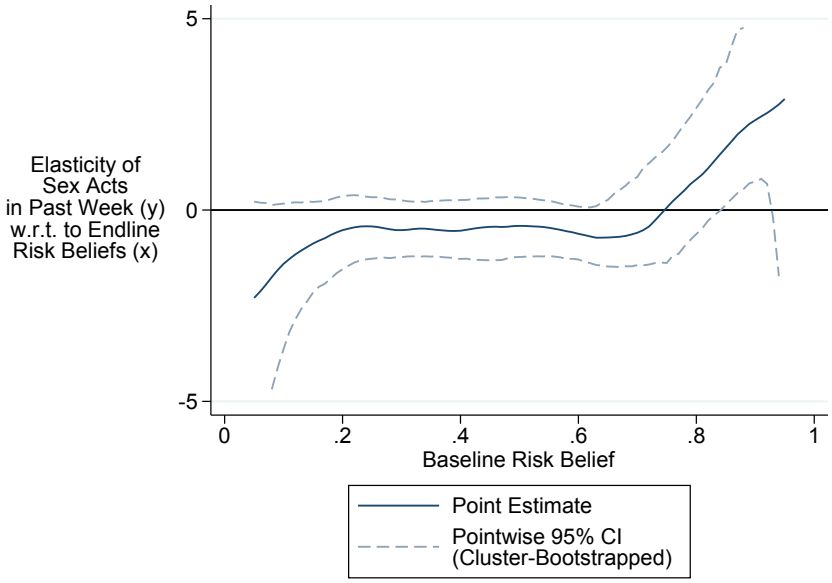


Notes: Created using binscatter. The dots plot the average value of the endline risk belief for each bin of the baseline risk belief, for both the control group (hollow dots) and the treatment group (solid dots). The lines show the linear best fit of the endline risk belief as a function of baseline risk beliefs for each study arm. Risk belief variable is the chance of contracting HIV from a single unprocted sex act with a randomly-selected sex partner. Sample is 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed.

Figure 4
Decompositions of Treatment effect and Risk Elasticity by Baseline risk Beliefs



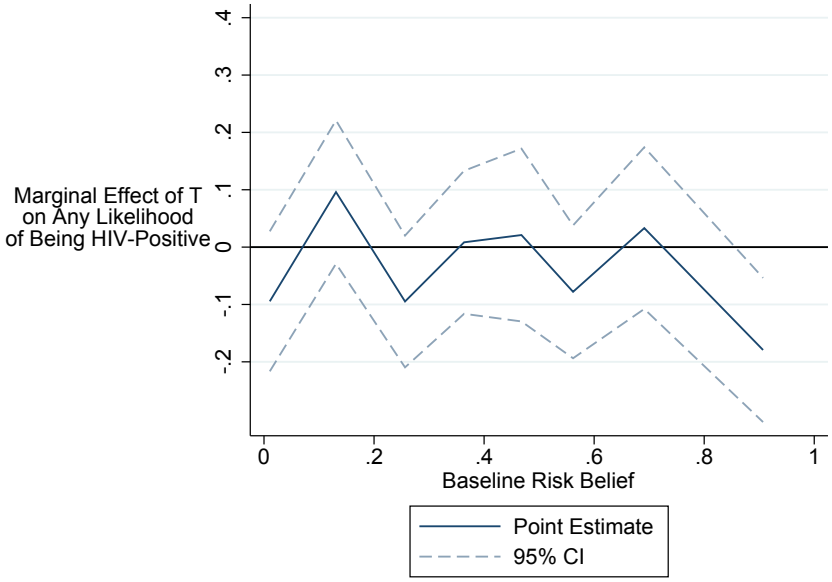
Panel A: Reduced-Form Effect of Information Treatment (T) on Log Sex Acts in Past Week ($\ln(y)$), by Baseline Risk Belief



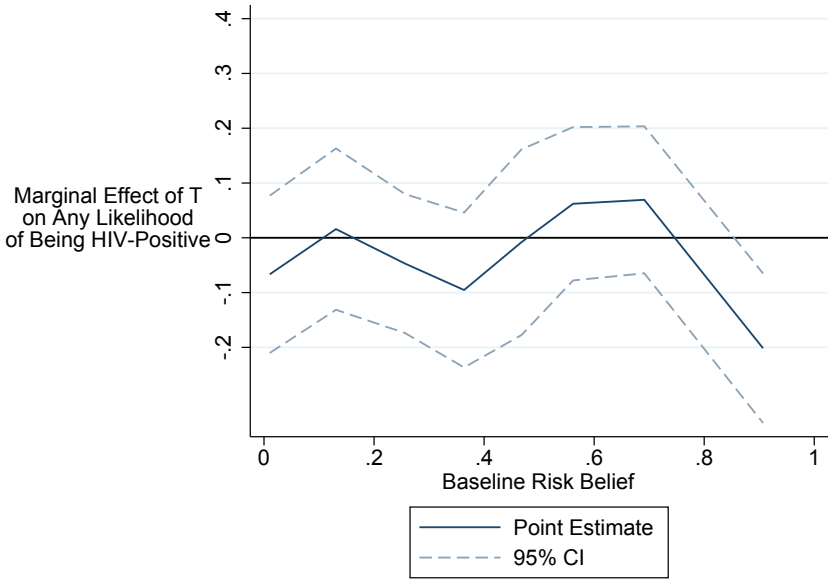
Panel B: IV Estimates of the Elasticity of Sex Acts in Past Week (y) w.r.t. Endline Risk Beliefs (x), by Baseline Risk Belief

Notes: The underlying semiparametric regressions use 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). Log sex in past week constructed as $y' = \ln(y + \sqrt{1+y^2})$ to account for zeroes. Baseline Risk Belief is 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, and is adjusted for non-constant time trends.
Sample is 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed.

Figure 5
Multinomial Logit Estimates of Effect of Treatment on
Perceived Likelihood of Having HIV, by Baseline HIV Transmission Risk Belief



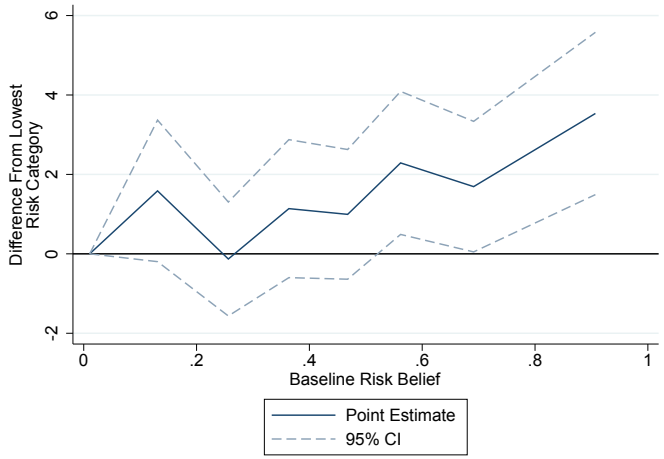
Panel A: Perceived Likelihood of Having HIV Now



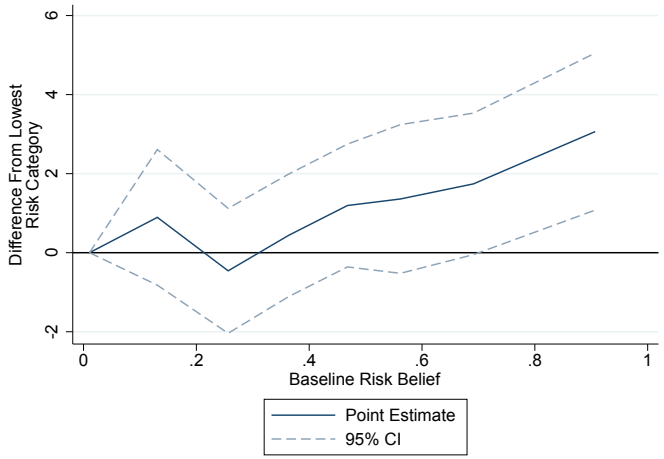
Panel B: Perceived Likelihood of Getting HIV in the Future

Notes: The graphs display the mean marginal effects (times negative one) on the "No Likelihood" option from a multinomial logit of the categorical HIV status belief variable on a treatment indicator, controlling 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 Panel A is used as a proxy. Baseline Risk Belief is 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, and is adjusted for non-constant time trends.
Sample includes 1,292 respondents from 70 villages who completed both baseline and endline surveys.

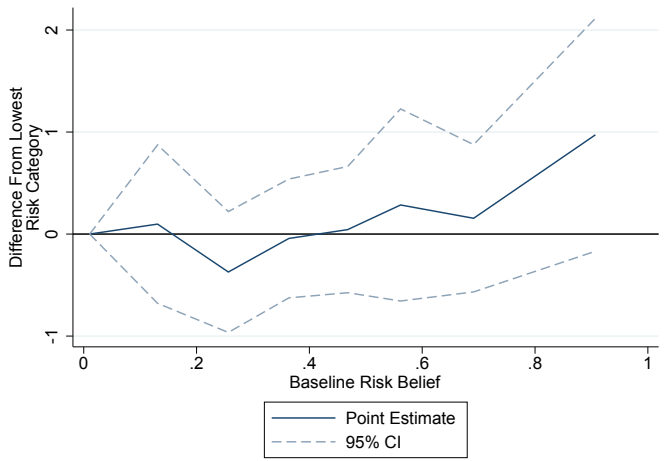
Figure 6
Differences in HIV Risk Factors by Baseline HIV Transmission Risk Belief



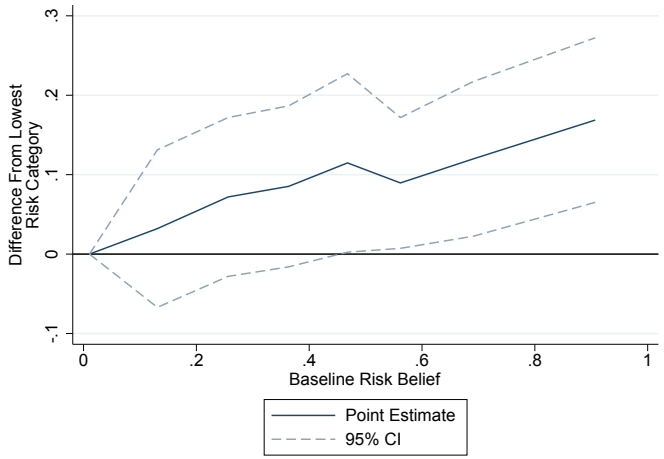
Panel A: Age



Panel B: Years Sexually Active



Panel C: Lifetime Sex Partners



Panel D: Perceives Any Likelihood of Being HIV-Positive

Notes: The graphs display the differences in baseline HIV risk factors between each risk category and the lowest one. Baseline Risk Belief is 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, and is adjusted for non-constant time trends.
Sample includes 1,292 people from 70 villages for whom both baseline and endline surveys were successfully completed.