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CAN HAVING FEWER PARTNERS INCREASE PREVALENCE OF AIDS?

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ABSTRACT

Under asymmetric information about sexual history, sexual activity creates externalities. Abstinence by those with few partners perversely increases the average probability of HIV infection in the pool of available partners. Since this increases prevalence among the high activity people who disproportionately influence the disease's future spread, it may increase long-run prevalence. Preliminary calculations using standard epidemiological models and survey data on sexual activity suggest that most people have few enough partners that further reductions would increase steady-state prevalence. To the extent the results prove robust, they suggest that public health messages will be more likely to reduce steady-state prevalence and create positive externalities if they stress condom use rather than abstinence.

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Introduction

Since 1981, three hundred and fifty thousand Americans have been diagnosed with AIDS. Fifteen million people are estimated to be infected with HIV worldwide. The disease raises a host of policy questions: Should public health messages stress condom use or reductions in the number of partners? Should they be targeted to high-risk people or broadcast more generally? Should the government promote changes in behavior at all? This paper combines techniques from economics and epidemiology to address these questions. In particular, it identifies conditions under which changes in individual sexual behavior create externalities by increasing or decreasing others' risk of infection. Assuming people lack information about the sexual history of potential partners, condom use is likely to create positive externalities. Reductions in the rate of partner change by those with few partners, however, may perversely increase others' chance of infection, and raise steady-state prevalence in the population as a whole.

These externalities from sexual behavior are due to asymmetric information about sexual history. In a hypothetical case in which sexual history were publicly observable, those who did not use condoms in the past, had many previous partners, or had especially risky previous partners would be considered less desirable partners, and in equilibrium they would have high risk partners. This would create an extra incentive for people who expected future partners to use condoms and seek low risk partners.

However, if people lack information about others' sexual history, private and social costs of risky behavior will differ because those who behave safely will not be rewarded with

safer partners in the future. Condom use will create positive externalities by improving the future pool of partners. Perversely, however, reductions in the frequency of partner change by those with less than a weighted average number of partners per period will create negative externalities by worsening the pool of available partners. This is because reductions in the frequency of partner change by low activity people increase the chance that others will match with high activity people. High activity people in particular will be more likely to match with each other and become infected. Since increases in prevalence among high activity people have a disproportionate impact on the future spread of the disease, reductions in the frequency of partner change by low activity people may increase long run prevalence in the population as a whole.

Calculations based on standard epidemiological models and surveys of sexual behavior suggest that this possibility is more than a theoretical curiosity. Under the standard susceptible-infected (SI) model, the rate of growth of a new epidemic, denoted R_0, is proportional to $\mu + \sigma^2/\mu$, where μ and σ^2 denote the mean and variance of the number of partners per period [Anderson and May, 1991]. It is straightforward to show that reductions in the number of partners by those with fewer than $(\mu + \sigma^2/\mu)/2$ partners per period will increase R_0. Survey data suggest that the distribution of numbers of partners has high variance and is extremely skewed, so that most of the population is below this cutoff. For example, in a comprehensive survey of English heterosexuals [Johnson et al, 1993], more than 90% of the population has fewer than the cutoff number of partners. Simulations suggest that if everybody who had one partner every five years reduced their frequency of partner change by

five percent, steady-state prevalence would increase by seven percent. If people have some information about others' rate of partner change, as in Kaplan, Cramton, and Paltiel [1989], then high activity people will be disproportionately likely to match with each other, as in the preferred mixing model of Jacquez et al. [1988]. Even in this case, however, most people in low prevalence populations would create negative externalities and increase steady-state prevalence by reducing their rate of partner change. In order to internalize this externality, people must be able to observe not only potential partners' frequency of partner change, but also the sexual history of potential partners' previous partners.

Several caveats should be noted. The proportion of the population that would create negative externalities by having fewer partners falls with prevalence, so these perverse effects are unlikely to arise among IV-drug users or people in very high risk areas of Africa. Moreover, reductions in the number of partners will temporarily reduce prevalence, even if they increase prevalence in the long-run. In general, since the SI model and the preferred mixing model abstract from important features of the epidemic, the results should be considered provisional. However, to the extent that they prove robust under more complex models, the results suggest that public health messages are more likely to reduce the steady state prevalence and create positive externalities if they stress condom use rather than abstinence.

In this paper, I examine both how changes in sexual behavior affect total prevalence

and how they create externalities by affecting others' prevalence.[2] Standard economic welfare analysis suggests people should be able to trade off their own risk of disease against other objectives, but that to the extent that their actions affect others, society may wish to intervene. Thus, for example, many economists would oppose government action to discourage mountain climbing, because mountain climbers voluntarily assume the risk, but support cigarette taxes because second-hand smoke imposes dangers on others. Public health officials, on the other hand, may care about total prevalence as well as externalities.

This paper builds on previous work. Epidemiologists have shown that under random matching, increased variance in the number of partners is likely to spur the spread of sexually transmitted diseases, and that prevention efforts are likely to be most effective if targeted to high activity groups [Hethcote and Yorke, 1984, Over and Piot, 1993]. Anderson, et al., [1991] consider an example based on an African country, in which a reduction in the number of partners by low activity women causes men to switch to prostitutes and thus leads to an increase in the number of partners of high activity women. In their example, this temporarily increases prevalence but reduces it in the long run. This paper makes the stronger claim that reductions in the number of partners by low activity people may increase prevalence even holding constant everyone else's number of partners, and even in the long-run.

This paper is most closely related to independent, but prior, work by Whittaker and

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[2]I neglect externalities that AIDS imposes on people who are not infected, such as increased insurance premiums and risk of tuberculosis. In an ideal world people would take these costs into account, but they are likely to be minor relative to the risk of death from AIDS.

Rentin. They show that in a two group example with random matching, an increase in activity by the low activity group may reduce steady state prevalence. This paper differs from Whittaker and Rentin [1992] by analytically approximating the cutoff level of activity below which these perverse effects arise with an arbitrary distribution of activity, in estimating the proportion of the population below this cutoff level using empirical data, and showing that the effects are likely to be important in low and moderate prevalence environments, but not in high prevalence environments. Moreover, unlike the previous paper, this paper shows that prevalence can be increased even if all members of the population reduce their activity, demonstrates that the results are robust in the case of preferred mixing, makes clear that prevalence will temporarily increase in response to reduced activity, and interprets the results using the economic concepts of externalities and asymmetric information.

The paper is organized as follows. Section I examines incentives for sexual behavior in a hypothetical benchmark case in which the sexual history of potential partners is publicly observable. (Epidemiologists may wish to skip this section.) Section II examines the distortions which arise when people lack information about potential partners' sexual history and match randomly. It uses the standard SI epidemiological model and a survey of sexual activity to estimate the proportion of the population for which reductions in the rate of partner change will increase prevalence among others, or among the population as a whole. Section III extends the analysis to the case in which people have some information about how many previous partners others have had, so that high activity people are more likely to match together. Section IV examines the time-path of prevalence instead of focussing on steady

states. Section V argues that condom use is likely to create positive externalities, and discusses implications for the design of public health messages.

For the most part, this paper takes the number of partners and the pattern of matching as given. Kremer [1994A] integrates an epidemiological analysis of the effect of behavior on HIV prevalence with an economic analysis of the effect of HIV prevalence on behavior. In particular, it examines how the choice of how many partners to have may be affected by Akerlofian [1970] lemons problems and by the decreasing marginal risk of infection with the number of partners. Kremer [1994B] examines how the spread of the disease is affected when people evaluate potential partners using imperfect signals of the probability they are infected, such as their age, behavior, or ethnic or geographic group.

I. Symmetric Information

This section examines the pattern of matching between sexual partners in the benchmark case in which people can observe others' sexual history. This hypothetical case lays the groundwork for Section II, which examines the distortions that arise when people lack information about others' sexual history.

Philipson and Posner [1993] argue that people with similar observable probabilities of infection will tend to match together as sexual partners. Their model rules out the possibility that people will induce others to match with them through some monetary or non-monetary compensation. This section argues that Philipson and Posner's assortative matching result

extends to the case in which such compensation is possible only if everyone has a single partner. If some people expect to have many partners, they will offer to compensate people with few previous partners for matching with them. This will give people with few previous partners incentives to have additional partners. Surprisingly, this may reduce the total expected number of infections.

To see the intuition for Philipson and Posner's result, suppose that people differed only in the probability of being infected with HIV, and that there were an observable signal of this probability. If people had only one partner, those with similar observable probabilities of being infected would tend to match together in equilibrium. This is because those with a high probability of already being infected would place lower value on having a safe partner and hence would be outbid by others seeking safe partners.[3] In the limiting case, those who knew they were infected would place the least value on having a safe partner, and hence would not be willing to pay the premium safer people would demand in exchange for matching with them. HIV-positive personal advertisements constitute an extreme empirical example of this positive assortative matching by risk of infection.

More formally, the probability a person is infected after a match, denoted p, equals $S + \beta M - \beta MS$, where S (self) is the chance the person was initially infected, M (mate) is the

[3]If people could not compensate others for matching with them, everyone would prefer partners with a low risk of infection, so low-risk people would be able to pick the partners they preferred, and hence low-risk people would match together.

chance his or her partner was infected, and β is the transmission rate.[4] (The final negative term reflects the fact one cannot be infected twice.) Since $dp/dMdS = -\beta < 0$, low-risk people have greater marginal risk from a high-risk partner. Hence they will have an especially strong preference for low-risk partners and will "outbid" others hoping to match with them.[5] Thus in equilibrium, people will match with others of similar risk, just as marriage partners of similar quality match together in Becker [1981]. Assuming people have only a single partner, this positive assortative matching by initial risk will minimize the spread of the disease.

Note, however, that incentives for positive assortative matching are second order. If the difference in probability of infection between people is of order ε, the reduction in the expected total number of infections from matching people of similar risk together will be of order ε^2. Thus the tendency for people of similar risk to match together may be eclipsed by factors from which this analysis abstracts, such as wealth differences, search costs, opportunities to conceal information, and match-specific payoffs depending on personal compatibility.[6]

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[4]The transmission rate differs between men and women, but this does not affect the conclusions.

[5]Still more formally, if utility is linear or concave in money, of the von-Neumann Morgenstern expected utility type in health, and separable in the two, then $d^2E(u)/dMdS > 0$, so people of similar risk will match together.

[6]For example, there is anecdotal evidence from Africa that rich, middle-aged men with a high risk of infection, who presumably place a high value on safety relative to money, have been able to induce poor schoolgirls with a low risk of infection to match with them [Barnett and Blaikie, 1992]. This is likely to exacerbate the spread of the disease.

If some people have multiple partners, they will have an additional, first-order, incentive to make sure their early sexual partners are low risk. This is because if they have low-risk early partners, they will have a lower probability of infection entering subsequent periods. Hence, in equilibrium, they will have safer partners in the future. Someone who accepts a high-risk early partner therefore takes on extra risk not only with that partner, but with all future partners. Since this effect is first order, it dominates the second order tendency for positive assortative matching except in high prevalence populations or for people with very large numbers of partners.

To see this, consider a hypothetical example in which the government announced that all future sexual behavior would be monitored and the information would be made public. Assume that at the time of the announcement, everyone started with an initial probability of infection ε. For algebraic simplicity, assume the transmission rate equals one. Consider first the case in which ε is small, so that second order effects can be ignored.

As shown in the first row of Table I, under positive assortative matching, someone with three partners would have probability of infection 2ε after the first partner, 4ε after the second, and 8ε after the third.[7] On the other hand, if the person matched with someone of risk ε in the first two partnerships, and thereafter matched with people of equal risk, his risk would

[7]This excludes the possibility that people can find and match with the previous partners of their partners. If this were possible, then if in the first period A matched with B and C with D, and in the second period A matched with C, and B with D, it would be optimal for A to match with D in the third period, rather than with some other person E. The assumption of small search costs or variation in personal preferences would rule this out.

only be 3ε after the second partner and 6ε after the third, as shown in the second row of Table I. Matching with someone of risk ε rather than 2ε in the second partnership thus allows a reduction of 2ε in the final probability of infection for someone with three partners. On the other hand, accepting a partner of risk 2ε rather than ε only increases final risk by ε for someone who only has one partner, as shown in the final rows of Table I.

Thus there will be scope for Pareto improving trade if the person who plans to have three partners has a sufficient initial endowment to offer some monetary or non-monetary compensation in exchange for matching with him.[8] If this compensation induces someone who plans to have only one partner to be the second partner of the person who plans three partners, the total expected number of infections will be reduced by ε.

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[8]Suppose, for example, that people start out with an endowment of wealth E and that utility of the jth person is $U_j = u_j(i) - vw - p$, where i is the number of partners, w is wealth, p is the risk of infection, and u_j is person j's subutility function for the number of partners. Assume also that $u_j'' < 0$ and $u_j(4) - u_j(3) < 0$, so that there are diminishing returns to more partners, and no one wants more than three partners. The equilibrium price someone who plans to have three partners will pay will be in [ε/v, 2ε/v]. This is because those who plan to have only one partner will require a reservation price of ε/v to match with someone of risk 2ε, and those planning to match three times will be willing to pay 2ε/v to have someone of risk ε as their second partner. Hence excess demand is positive for prices less than ε/v and negative for prices greater than 2ε/v. A price in the interior of this range may balance supply and demand since the number of people who have three partners rather than two, or one partner rather than zero or two, will depend on the price.

Table I: Risk by Number of Partners: Different Matching Patterns (Second and Higher Order Terms Omitted)

	Initial Prob.	After 1 Partner	After 2 Partners	After 3 Partners	After 4 Partners	After n Partners
Person with Many Partners						
Equal Risk Partner	ε	2ε	4ε	8ε	16ε	$2^{n-2}4\varepsilon$
Low Risk 2nd Partner	ε	2ε	3ε	6ε	12ε	$2^{n-2}3\varepsilon$
Person with One Partner						
Equal Risk Partner	ε	2ε				
High Risk Partner	ε	3ε				

If people who expect to have many partners offer to compensate those with few previous partners for matching with them, they may induce some low activity people to have additional partners. To see that this may reduce the expected total number of infections, suppose, for example, that half the population preferred four partners, and that in the absence of compensation, half preferred zero partners. If compensation induced the people who would otherwise have had zero partners to become the second partners of people who planned to have four partners, the probability of infection for the person with four partners would fall by 4ε, as illustrated in the top two rows of Table I. On the other hand, the probability of infection for the person who changed from zero to one partner would increase from ε to 3ε. The total number of infections would thus fall by 2ε.

So far I have examined the case in which ε, the initial risk of infection, and i, the

number of partners, are small, so that second order terms can be neglected. If i or ε is large, the pattern of matching will differ because the second order effects will overwhelm the first order effects. Those who expect a high lifetime risk of infection will place lower weight on finding safe partners in the current period. People who expect to have a huge number of future partners will have less to lose from risky partners in the early periods, since they can be virtually sure of infection regardless of the risk of their early partners.[9] Thus for high enough ε or i, people who expect many future partners would accept payments to match with people with a higher current risk of infection.

In general, if a competitive equilibrium assignment of partners exists, it will be Pareto optimal. Appendix A provides a proof by Eric Maskin that an equilibrium will exist if everyone has a sufficiently large endowment and the marginal utility of money is bounded away from zero. If everyone has a sufficiently large endowment and the same tradeoff between money and risk of infection, Pareto optimality in turn implies minimization of the expected total number of infections given each person's number of partners. The assumption of a sufficiently large endowment in the existence proof is not innocuous, however, because the endowment must be larger than the payment required to induce people to accept matching

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[9]Neglecting third and higher order terms, the probability of infection for someone with three partners who matches with partners of the same risk in each stage is $8\varepsilon-(4\varepsilon)^2$. For someone who has a second period partner of risk ε, it is $6\varepsilon-(3\varepsilon)^2$. Hence the reduction in risk for the person with three partners from having a safer second period partner is $2\varepsilon-7\varepsilon^2$. The increase in risk for the person with one partner is $\varepsilon-\varepsilon^2$. Thus the gain from matching across risk categories is $\varepsilon-6\varepsilon^2$. Although this is positive for low ε, it is negative for high enough ε.

with anyone, including someone who was certainly infected. (In practice the necessary endowments are likely to be those which will induce people who plan to have few partners to match with those who have already had a moderate number of partners and expect to have more in the future.)

Note that under symmetric information, the more previous partners one has had, the less appealing a partner one will be. People with many previous partners who wish to have another partner must therefore either accept a more dangerous partner or pay more for a safer partner. Thus there will be a range of increasing marginal cost of partners under symmetric information. (Once someone has had enough partners that he or she is very likely to be infected, the marginal risk from additional partners will start to decline.) The increasing marginal cost of partners will create incentives for people to have a moderate number of partners, rather than a very small or large number.

To summarize, if people knew others' sexual history and initial prevalence were not too high, people who expected to have many partners in the future would have an incentive to compensate low-risk people for matching with them. This compensation would give people with few previous partners incentives to have more partners, and this could reduce the expected total number of infections. These incentives are absent when people lack information about others' sexual history.

II. Publicly Unobservable Sexual History

This section considers the case in which people lack information about others' sexual history and are otherwise identical, so they match randomly (or, in epidemiological terms, mix homogeneously.) In this case, those with many previous partners will be able to obtain safer partners than under symmetric information. Conversely, those with few previous partners will neither be compensated for having additional partners nor be able to obtain safer partners. This higher "price" for additional partners suggests that there may be some sense in which low activity people paradoxically "consume" too few sexual partnerships.

In fact, reductions in the number of partners by those with few previous partners make others worse off by worsening the quality of the pool of available partners. To see this, consider an example in which all sexual partners meet in a bar. Suppose that half the population goes to the bar and finds a partner eight times a year, and the other half goes twice a year. The high activity people will be over-represented on any given night. Someone who picks a partner at random from the pool of available partners in the bar will get someone with eight partners a year with probability 8/10, and someone with two partners a year with probability 2/10. The average number of partners per year of someone picked at random, weighted by the probability of picking that person at random, will therefore be (8 x 8/10) + (2 x 2/10) = 6.8 partners per year. However, if the people who have two partners a year reduce to one partner a year, a randomly picked person will be someone with one partner per year with probability 1/9, and eight partners per year with probability 8/9, so the weighted average will <u>increase</u> to (8 x 8/9) + (1 x 1/9) = 65/9 = 7.33 partners per year. If the low activity people drop out of the pool completely, someone who goes to the bar will be certain of

meeting someone with 8 partners a year. Thus reductions in the rate of partner change by low activity people can increase the expected number of partners per year of a randomly picked person at the bar, and thus the risk to other people, and in particular, to the high activity people who continue to patronize the bar.

This section uses the standard SI (susceptible-infected) epidemiological model to argue that this increase in risk for high activity people has a disproportionate effect on the spread of the disease in future periods, and may raise steady-state prevalence in the population as a whole.

II.A. The SI Model

Under the SI model, people are born and die according to a Poisson process with hazard rate δ[10], and the transmission rate per partnership is denoted β.[11] Assuming everyone

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[10]The model abstracts from AIDS-induced mortality for the sake of tractability. Although AIDS-induced mortality affects projections of prevalence, it does not have a dramatic impact on the effects considered here. Section IV reports simulations with AIDS-induced mortality.

[11]The model implicitly assumes that the transmission rate is independent of the number of sexual contacts with the partner. The prevailing view in the medical literature is that this is a reasonable approximation [Kaplan, 1990]. Peterman et al. [1988] find that the risk of infection for spouses of infected hemophiliacs is independent of their frequency of sexual contact. This may be due to heterogeneity in infectiousness and susceptibility between individuals. In particular, there is evidence that people are most infectious in the first few months after becoming infected and again when the disease develops into AIDS [Jacquez, et al., 1994]. To the extent transmission rates depend on the number of sexual contacts per partner, the results in this paper would presumably be somewhat weakened.

has i partners per period, and the population is normalized to one, the change in Y, the number of people infected, equals the number of uninfected people who match with infected people and become infected, minus the number of infected people who die:

$$\dot{Y} = i\beta Y(1-Y) - \delta Y. \tag{1}$$

There is one steady state at Y = 0, since if no one has the disease, no one else can be infected, and hence prevalence will remain equal to zero. To see whether this steady state is stable to the introduction of a few infected individuals, divide by Y and take the limit as Y goes to zero, to obtain $\dot{Y}/Y = i\beta - \delta$. Thus at prevalence close to zero, the growth rate of prevalence will be positive if and only if $i\beta > \delta$.

To understand the epidemiological intuition, note that the expected number of secondary infections a single infected person causes in an otherwise uninfected population before dying, denoted R_0, equals the number of partners per period, i, times the transmission rate per partner, β, times the expected life span, $1/\delta$. If R_0 is less than one, each infected person will transmit the infection to less than one person on average before dying, and prevalence will therefore fall to zero from any initial value. On the other hand, if R_0 is greater than one, then once the disease is introduced, prevalence will grow until so many people are infected that each infected person transmits the disease to just one uninfected person on average before dying. Setting dY/dt = 0 and dividing by Y shows that for $R_0 > 1$, the unique stable steady state level of prevalence, denoted Y^*, is $1-\delta/i\beta$.

To see that an increase in activity can lead to the eradication of the disease, suppose that a weighted average of 7 partners per year were required for each infected person to infect one uninfected person on average before dying. If a small minority of the population had eight partners a year, and the majority had no partners at all, the disease would persist among the active minority. If the inactive majority decided to have one partner each over their lifetime, and if the groups matched randomly, then the high activity people might match four times a year on average with the low activity people and four times a year on average with other high activity people. In this case, the disease would not be sustained because half the new infections would be "wasted" (from the point of view of the disease) on low activity people who would not pass it on.

Recall that under the symmetric information case examined in Section I, the negative externalities from low activity people having fewer partners would be internalized, since people who expected many future partners would compensate low activity people for matching with them. This is impossible if people cannot observe the sexual history of potential partners.

This example can be generalized using Anderson and May's [1991] extension of the SI model to the case in which people differ in their rate of partner change. They show that in this case, R_0 equals β/δ times a weighted average number of partners per period. This weighted average number of partners, denoted c, equals $\mu + \sigma^2/\mu$, where μ and σ^2 are the mean and variance of the number of partners per period in the population. To see the intuition, consider a population composed of K groups. Denote the proportion of the population in the kth group as α_k and the number of partners per period in the kth group as i_k. The probability

that a randomly picked partner will be in the kth group equals the number of partners of people in the kth group divided by the total number of partners of people in all groups, or $i_k\alpha_k/\sum i_L\alpha_L$. Denoting prevalence within the kth group as Y_k, the probability that a randomly picked partner will be infected, denoted $\bar{Y}$, is thus a weighted average of prevalence in all groups

$$\bar{Y} = \sum_{k=1}^{K} Y_k \frac{i_k\alpha_k}{\sum_{l=1}^{K} i_l\alpha_l}. \tag{2}$$

The differential equation for prevalence in the jth group is $dY_j/dt = (1-Y_j)\beta i_j\bar{Y} - \delta Y_j$. Zero is a stable steady-state prevalence only if the growth rate of the epidemic is negative at prevalence close to zero. As prevalence approaches zero, the growth rate of prevalence in the jth group approaches

$$\lim_{\bar{Y}\to 0} \frac{\dot{Y}_j}{Y_j} = \beta i_j \sum_{k=1}^{K} \frac{Y_k}{Y_j}\left(\frac{i_k\alpha_k}{\sum_{l=1}^{K} i_l\alpha_l}\right) - \delta. \tag{3}$$

Moreover, as prevalence approaches zero, the chance that someone is infected becomes proportional to their number of partners, so Y_k/Y_j approaches i_k/i_j. Thus the asymptotic growth

rate of prevalence is positive only if

$$\beta\left(\frac{\sum_{k=1}^{K} i_k^2\alpha_k}{\sum_{k=1}^{K} i_k\alpha_k}\right) - \delta > 0. \qquad (4)$$

Since $\Sigma i_k^2\alpha_k = \sigma^2+\mu^2$, and $\Sigma i_k\alpha_k = \mu$, the term in parentheses equals $\mu+\sigma^2/\mu$, which is the weighted average number of partners, denoted c. For the disease to spread c must therefore be greater than δ/β, the death rate over the transmission rate.

To see when an increase in the number of partners by a group with few partners could lead to the eradication of the disease,

note that

$$\frac{dc}{di_k} = \frac{2i_k\alpha_k\sum_{l=1}^{K} i_l\alpha_l - \alpha_k\sum_{l=1}^{K} i_l^2\alpha_{lk}}{\left(\sum_{l=1}^{K} i_l\alpha_l\right)^2}. \qquad (5)$$

Since the denominator is positive, the weighted average number of partners per period will fall in response to increased activity if and only if

$$i_k < \frac{1}{2}\left[\frac{\sum_{l=1}^{K}\alpha_l i_l^2}{\sum_{l=1}^{K}\alpha_l i_l}\right] = \frac{1}{2}\left[\mu + \frac{\sigma^2}{\mu}\right]. \qquad (6)$$

If increases in activity by those with fewer than $.5(\mu + \sigma^2/\mu)$ partners per period cause c to fall

below β/δ, and thus R_0 to fall below one, the disease will be eradicated.

If R_0 cannot be reduced below one, so the disease cannot be eradicated through increases in activity, there will still be a cutoff number of partners per period, denoted j_e, below which having fewer partners will create negative externalities in steady-state by increasing prevalence among others. There may also be another, lower, cutoff level of activity, denoted j_t, below which reductions in the number of partners will increase total steady-state prevalence in the population as a whole, even allowing for the possible reduction in steady-state prevalence among the group which reduces its number of partners. In general, j_e and j_t will be less than c/2, the cutoff number of partners for reducing R_0. However, if steady-state prevalence is low, so the probability people are infected is close to proportional to their number of partners, j_e and j_t will be well approximated by c/2.

A simple fixed point argument shows that there will be a unique stable steady-state prevalence level for any distribution of rates of partner change. To see this, recall that just as $\bar{Y}^*$ is a linear function of prevalence in the K groups, the vector of steady-state prevalence in the K groups, $(Y_1^*, Y_2^*, \ldots Y_K^*)$, denoted Λ, is also a function of $\bar{Y}^*$, the steady-state weighted average prevalence. In particular, since $\dot{Y}_k = i_k\beta(1-Y_k)\bar{Y} - \delta Y_k$, steady-state prevalence in the

kth group is

$$Y_k^* = \frac{\bar{Y}^* i_k \beta}{\bar{Y}^* i_k \beta + \delta}. \tag{7}$$

Thus it is possible to define a function $\bar{Y}^*(\Lambda(\bar{Y}^*))$ mapping [0,1] onto itself. Since $\bar{Y}^*$ increases linearly with the elements of Λ, but the elements of Λ increase less than linearly with $\bar{Y}^*$, this function has a unique fixed point, and can be approximated numerically with arbitrary precision by iteration. In the special case in which there are only two groups, this approach yields Hethcote and Yorke's [1984] quadratic equation for Y_1^*,

$$\begin{aligned} Y_1^{*2}\left[(i_2 - i_1)\alpha_1 i_1\right] + Y_1^*\left[2\alpha_1 i_1^2 + \alpha_2 i_2^2 - \alpha_1 i_1 i_2 + (\alpha_1 i_1 + \alpha_2 i_2)\frac{\delta}{\beta}\left(\frac{i_2 - i_1}{i_1}\right)\right] \\ + \left[(\alpha_1 i_1 + \alpha_2 i_2)\frac{\delta}{\beta} - \alpha_2 i_2^2 - \alpha_1 i_1^2\right] = 0. \end{aligned} \tag{8}$$

and thus allows the unique stable steady-state prevalence to be obtained analytically.[12]

In general there is no analytic solution for j_e and j_t as a function of the distribution of number of partners in the population. However, it is possible to solve for j_e given steady-state prevalence in the pool of available partners, $\bar{Y}^*$, and to solve for both j_e and j_t in the special

[12]I thank Ted Miguel for assistance with these calculations.

case in which all but a negligible fraction of the population have the same rate of partner change.

To solve for j_e given $\bar{Y}^*$, suppose that a small group has j partners per period, and denote their prevalence as Y_j. For someone who matches with a member of the group, rather than with someone from the general pool, the probability of encountering an infected partner in steady state is reduced from $\bar{Y}^*$ to Y_j^*, which by (8) is $\bar{Y}^* j\beta/(\delta + \bar{Y}^* j\beta)$.. Each person's chance of matching with a member of the small group is approximately proportional to j. Thus the steady-state reduction in risk to other members of the population is

$$j\left[\bar{Y}^* - \frac{\bar{Y}^* j\beta}{\delta + \bar{Y}^* j\beta}\right]. \tag{9}$$

j_e is defined as the number of partners which maximizes this reduction in risk. Taking the first order condition, simplifying, and applying the quadratic formula yields the unique positive solution for j_e:

$$j_e = \frac{\delta}{\beta \bar{Y}^*}\left(\frac{1}{\sqrt{1-\bar{Y}^*}} - 1\right). \tag{10}$$

In the special case in which the entire population has i partners, j_e can be expressed as

a function of i, since in this case $\bar{Y}^* = 1-\delta/(i\beta)$. Substituting and simplifying,

$$j_e = \frac{i}{1 + \sqrt{\frac{i\beta}{\delta}}} = \frac{i}{1 + \sqrt{\frac{1}{1 - Y^*}}}. \qquad (11)$$

Note, that in the limit, as β, the transmission rate, falls to δ/i, so that prevalence approaches zero, the probability someone is infected becomes linear in their number of partners, and j_e approaches one-half the weighted average number of partners. (In a population in which everyone has i partners, c = i.) As β, and thus prevalence, increase, the probability someone is infected becomes more concave in the number of partners. Hence someone with half the average probability of infection in the population has fewer than half the weighted average number of partners, and j_e falls below i/2. However, in a population in which everyone has the same number of partners, this effect becomes strong only at high prevalence. The first row of Table II shows j_e/i as a function of Y* in the case of a uniform activity level in the rest of the population. At low prevalence, j_e is well approximated by c/2.

Groups with fewer than j_e partners will raise <u>others'</u> steady state probability of infection by having fewer partners. However, since they may reduce their own steady-state probability

of infection, total steady-state prevalence may decline. There also may exist a cutoff number of partners, j_t, below which groups will raise *total* prevalence by reducing their activity. In the special case in which all except a negligible fraction of the population have the same rate of partner change, j_t can be calculated analytically.[13]

Suppose that a small group has j partners per period, and let the prevalence of this group be denoted as Y_j. Total prevalence in the two-group case is given by $Y = (1 - \varepsilon)Y_i + \varepsilon Y_j$, where ε denotes the proportion of the total population in the small group (the group with j partners), and Y_i denotes prevalence in the large group (the main population, which has i partners per period). By Equation (8), we know that in steady state,

$$Y_i^2((j-i)(1-\epsilon)i)+Y_i\left(2(1-\epsilon)i^2+\epsilon j^2-(1-\epsilon)ij+\frac{\delta}{\beta}((1-\epsilon)i+\epsilon j)\frac{j-i}{i}\right) \\ +\left(((1-\epsilon)i+\epsilon j)\frac{\delta}{\beta}-\epsilon j^2-(1-\epsilon)i^2\right)=0 \tag{12}$$

and

$$Y_j^2((i-j)\epsilon j)+Y_j\left(2\epsilon j^2+(1-\epsilon)i^2-\epsilon ij+\frac{\delta}{\beta}((1-\epsilon)i+\epsilon j)\frac{i-j}{j}\right) \\ +\left(((1-\epsilon)i+\epsilon j)\frac{\delta}{\beta}+\epsilon j)\frac{\delta}{\beta}-\epsilon j^2-(1-\epsilon)i^2\right)=0 \tag{13}$$

Finding the positive solutions for Y_i and Y_j, and taking the first order condition for minimizing Y with respect to j, yields a quintic equation, which cannot be solved analytically.

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[13]I thank Ed Drodz for assistance with these calculations.

However, taking the limit as the size of the small group approaches zero, gives a quadratic equation which implies

$$j_t = \frac{i\delta^2 - i^2\beta\sqrt{2\delta^2 - i\delta\beta}}{\delta^2 - i\delta\beta}. \tag{14}$$

Since $Y^* = 1 - \delta/(i\beta)$, j_t can be expressed as

$$j_t = i\frac{\sqrt{(1-Y^*)(1-2Y^*)} - (1-Y^*)^2}{Y^*(1-Y^*)}. \tag{15}$$

Note that $j_e \geq j_t$, since it is only possible to increase prevalence by reducing activity if one creates negative externalities by doing so. Moreover, j_t is positive and decreasing in Y* over the interval $[0, \frac{1}{2}(3-\sqrt{5}))$, zero at $\frac{1}{2}(3-\sqrt{5}) \approx .37$, and is undefined at higher prevalence. Thus, in homogenous populations with prevalence greater than $\frac{1}{2}(3-\sqrt{5})$, reductions in the number of partners can never increase steady-state prevalence. I conjecture that j_t will be maximized for any value of Y^* if the population is homogenous at in its level of activity. The more heterogenous is the population in its level of activity for any Y^*, the lower will be j_t. If this conjecture is correct, reductions in the number of partners will never increase steady state prevalence in <u>any</u> population in which prevalence is greater than $\frac{1}{2}(3-\sqrt{5})$. Table II shows representative values of j_e/i and j_t/i for various prevalence levels, and Figure 1 plots j_e/i and j_t/i

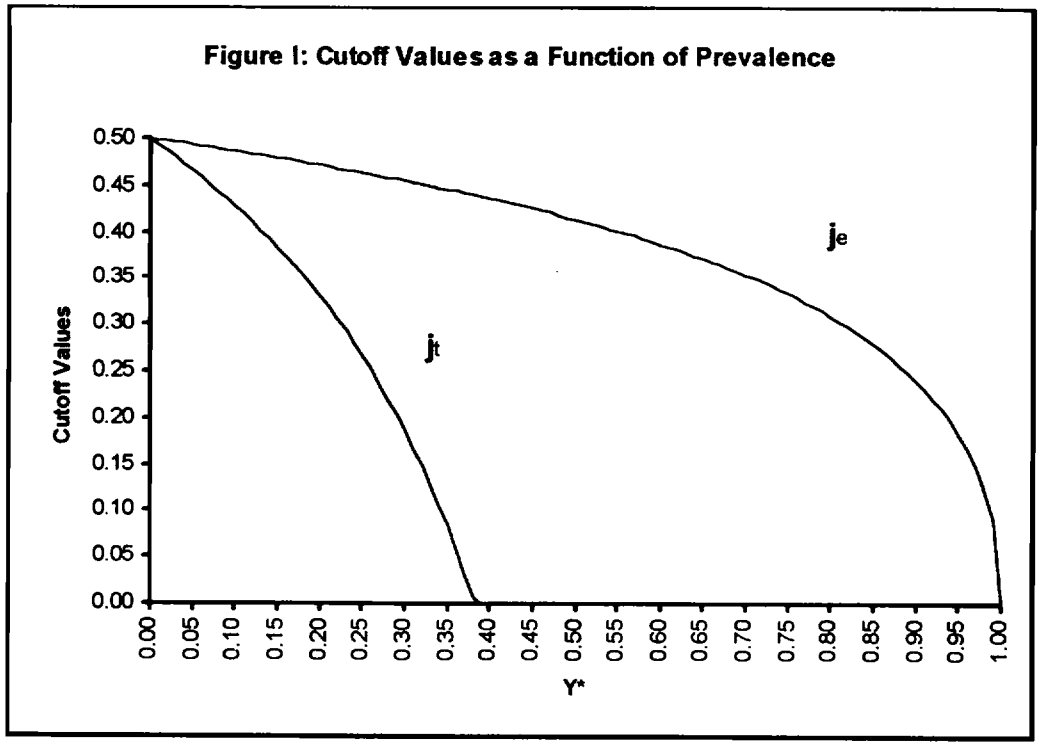
Figure I: Cutoff Values as a Function of Prevalence
Cutoff Values
0.50
0.45
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
0.00
0.05
0.10
0.15
0.20
0.25
0.30
0.35
0.40
0.45
0.50
0.55
0.60
0.65
0.70
0.75
0.80
0.85
0.90
0.95
1.00
Y*
je
jt

as functions of Y^*.

Table II: j_e/i and j_t/i as a Function of Y^* in a Population with i Partners/Period.

Y^*	0.00	0.01	0.10	0.25	0.50	0.75
j_e/i	0.500	0.499	0.487	0.464	0.414	0.333
j_t/i	0.500	0.494	0.428	0.266	0.00	0.000

II.B. Calibration to the Data

A first-pass analysis of survey data on sexual activity suggests that in low prevalence populations, a large proportion of the population may create negative externalities and increase steady-state prevalence by having fewer partners.

The simulations in this paper are based on data from the *National Survey of Sexual Attitudes and Lifestyles* (NSSAL), a comprehensive survey of sexual behavior in Britain encompassing 18,000 people.[14] The mean numbers of heterosexual partners in the last five years was 1.98[15] and the variance was 19.03 [Johnson, et al, 1993] Table III shows the distribution of numbers or heterosexual partners in the last five years. In the simulations, I

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[14]I am grateful to Cesaltina Pires for conducting the simulations reported in this paper.

[15]I confine attention to the exclusively heterosexual population, because the sample size was too small to make reliable inferences about homosexuals. I use British data because the comparable U.S. survey was not available when this paper was written.

assume that the annual rate of partner change is one-fifth the number of heterosexual partners over the last five years.[16]

Because the distribution of sexual activity has high variance and is extremely skewed, the vast majority of the population has fewer than c/2 partners. As shown in Table III, approximately 93.9% of the sample had fewer than c/2 = .5* (1.98 + 19.03/1.98) = 5.79 partners over the five year period. c/2 corresponds to 1.157 partners per year over the five year period. Although measurement error could artificially inflate the variance, nearly 90% of the population remains below this cutoff even if the high activity observations are assumed to be misreported.[17]

Given that English heterosexuals have extremely low prevalence, the risk of infection will be close to linear in the number of partners over the relevant range, and j_e and j_t will be

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[16]There is no unproblematic way of moving from the theoretical concept of the rate of partner change to empirical observations of the number of partners per period, since people may have several partners simultaneously, and may reestablish old partnerships [Johnson, 1993]. I assume that those people who have one partner over a five year period have an average of 0.2 partners per year. If the people listed as having one partner every five years actually changed partners less frequently, as seems plausible, the variance of sexual activity would be even larger. This would exacerbate some of the perverse effects discussed in this paper.

[17] If the highest-activity person - with one hundred partners per year - is dropped from the sample, the total variance of annual partners falls from 0.761 to 0.514, c/2 falls from 1.157 to 0.849, and the percentage of the population below the cutoff falls to 91.9%. However, it does not seem implausible that one person out of a sample of 18,000 had an average of 100 partners per year over a five year period, so it may well be a mistake to delete this from the sample. Dropping further observations has a much smaller impact. For example, if the two people reporting 20 partners per year are dropped from the sample, c/2 falls to 0.820. Even if all those reporting more than eight partners a year are assumed to have actually had 8 partners a year, c/2 would be 0.71 and 88.4% of the population would still be below this cutoff.

close to c/2. Hence under the assumption of random matching, more than 90% of the population would increase others' risk of infection and the long-run prevalence of AIDS by having fewer partners.

Table III: Distribution of Numbers of Partners per Year over the Last Five Years[18]

Partners/yr	%age	Partners/yr	%age	Partners/yr	%age	Partners/yr	%age
0	8.95	2.2	0.128	4.4	0.033	8	0.061
0.2	62.56	2.4	0.261	4.6	0.010	9	0.010
0.4	10.56	2.6	0.069	4.8	0.023	10	0.037
0.6	6.39	2.8	0.033	5	0.061	12	0.026
0.8	3.44	3	0.347	5.2	0.008	13	0.003
1	2.05	3.2	0.026	5.4	0.008	14	0.018
1.2	1.64	3.4	0.063	6	0.149	15	0.015
1.4	0.90	3.6	0.031	6.4	0.005	16	0.003
1.6	0.70	3.8	0.027	7	0.027	18	0.003
1.8	0.24	4	0.263	7.6	0.005	20	0.006
2	0.83	4.2	0.003	7.8	0.005	100	0.002

Under the SI model, an increase in activity by low-activity groups could even reduce R_0 below one and lead to the eradication of the disease. In general, this could occur if c_{min}, the lowest possible level of c that can be achieved through increases in the number of partners by low activity people, is less than δ/β. Given the distribution of rates of partner change in Table III, $c_{min} = 1.58$ partners per year, so the disease could be eradicated purely through increased

[18] In order to reduce the number of state variables, the simulations group together people with 1.8-2.2, 2.4-3.8, 4-5.8, 6-7.8, 8-9.8, 10-13.8, 14-15.8, and 16-20 average annual partners/year, assigning each group its average number of partners. Further details of simulations are available from the author.

activity if $\delta/\beta > 1.58$.[19]

Calibrating the SI model to match observed prevalence suggests that this may be the case. Since prevalence among heterosexuals in the U.K. is not growing rapidly, it is likely to be near its steady-state level. Steady-state prevalence of 0.005 would correspond to δ/β of 1.81, and even steady-state prevalence of 0.01 would correspond to δ/β of 1.62. Thus within the context of the SI model and the NSSAL estimates of activity, it seems quite possible that the disease could be eradicated through increased activity.[20] Since the data is imperfect, the population is not closed, and the SI model abstracts from important features of the disease, this calibration should not be taken too literally. However, it does suggest that increases in activity by low activity people are likely to cause large reductions in steady-state prevalence and might lead to the eradication of the disease from low activity populations.

The weighted average number of partners, and thus R_0, may fall even if all members of the population increase their frequency of partner change by the same absolute amount, so that

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[19] c_{min} attains this level when everyone with fewer than 0.79 partners per year increases their rate of partner change to 0.79 partners per year.

[20] Estimates of the transmission rate based on medical evidence, such as studies of spouses of infected people, suggest δ/β is substantially lower. The discrepancy arises because the SI model abstracts from several crucial aspects of the epidemic, such as the higher death rate of infected people, and the tendency for people to match with others of similar age. Both these factors will substantially lower the steady state prevalence associated with any transmission rate. Future research could examine whether the disease could be eradicated through increased activity in a more complex model incorporating these factors. However, there is no reason to think that the results would change under more complicated models which generate lower values of δ/β. Since prevalence among English heterosexuals is low and is not growing rapidly, the disease is likely to be close to eradication under a variety of models.

μ increases and σ^2 is unchanged. Since $c = \mu + \sigma^2/\mu$,

$$\frac{dc}{d\mu} = 1 - \frac{\sigma^2}{\mu^2}. \qquad (16)$$

Thus if $\mu < \sigma$, increases in μ will reduce c. Holding σ^2 constant, c reaches its minimum level of 2σ when $\mu = \sigma$. Thus in the NSSAL sample, with $\mu = 0.397$ partners per year and $\sigma = 0.761$ partners per year, the weighted average number of partners would be minimized if everyone had 0.364 more partners per year. In this case, c would fall to approximately 1.52 - quite possibly low enough to cause the eradication of the disease under the SI model. The intuition is that if everyone increased their number of partners by the same absolute amount, the chance of meeting low activity people would increase, reducing the weighted average risk in the pool, and thus cutting prevalence among the high activity groups which disproportionately influence the future spread of the disease.

In order to keep the analysis simple, I have not distinguished between prevalence among men and women, but a fuller model would do so. Anderson and May [1991] show that in a two sex model, R_0 is proportional to $(c_m c_f)^{1/2}$, where c_m and c_f are the weighted average number or partners among males and females respectively. By definition, in a heterosexual population, men and women must have the same total number of partners. At least in societies with a significant amount of prostitution, it seems likely that women have a higher variance in the of number of partners, so that c_f is greater than c_m. I would therefore conjecture that the perverse effects discussed in this paper are stronger for women than for men.

While perverse effects may arise among British heterosexuals, they are much less likely

to arise in high prevalence populations. In these populations, j_e and j_t are significantly less than c/2, so a much smaller percentage of the population will create negative externalities or increase total steady-state prevalence by reducing activity. Table IV shows j_e and j_t for values of δ/β consistent with various levels of steady-state prevalence, assuming the same distribution of rates of partner change as found in the NSSAL sample.[21] In sub-Saharan Africa as a whole, prevalence among adults is approximately 2.5% and in Thailand it is 2% [World Bank, 1993], so a substantial portion of the population may generate negative externalities and increase steady-state prevalence by reducing activity in these populations. However, in the highest risk areas of Africa, where prevalence among adults is as high as one third, low activity people probably will not increase total steady-state prevalence by having fewer partners. Similarly, prevalence among IV-drug users in the U.S. is high enough that reductions in needle-sharing by infrequent users are unlikely to have perverse effects on total prevalence.

Table IV: j_e and j_t in Partners/yr by Y^*
(Percentage of the population below cutoff in parentheses.)

Y^*	.005	0.01	0.02	0.05	0.1	0.2	0.3	0.5
j_e	0.92 (92%)	0.83 (92%)	0.74 (88%)	0.59 (82%)	0.47 (82%)	0.35 (72%)	0.28 (72%)	.197 (9%)
j_t	0.79 (88%)	0.67 (88%)	0.54 (82%)	0.33 (72%)	0.13 (9%)	0	0	0

[21]Note that j_e/i and j_t/i in these simulation with heterogenous populations are smaller than j_e/i and j_t/i in homogenous populations.

III. Partially Observed Rates of Partner Change

Section I examined the case of symmetric information and Section II examined the case in which people lack information about their potential partners' sexual history, and therefore match randomly. This section examines the case studied by Kaplan, Cramton and Paltiel, [1989], in which people observe an imperfect signal of potential partners' frequency of partner change, which indicates the true rate of partner change with probability γ and contains no information with probability $1 - \gamma$. This gives rise to preferred mixing, as in Jacquez et al., [1988], in which people match with others who have the same frequency of partner change with probability γ and match randomly with probability $1-\gamma$.

This section argues that the perverse effects of reductions in the frequency of partner change by low activity people are not necessarily ameliorated - and are in some ways exacerbated - by information about others' rate of partner change. In low prevalence populations, the proportion of the population that generates negative externalities by having fewer partners increases with γ. The proportion of the population that increases total steady-state prevalence by having fewer partners does not change monotonically with γ. In order to internalize the externalities from having more partners, people must be able to observe the sexual history not only of potential partners, but also of potential partners' previous partners.

In general, simulations are necessary to examine the effect of changes in the numbers of partners under preferred matching. However, it is possible to solve for j_e in the limiting case as γ approaches one, so that people match almost exclusively with others of the same activity

level. As γ approaches one, steady-state prevalence in a group with i partners approaches 1 - $\delta/i\beta$ if $i > \delta/\beta$ and approaches zero if $i < \delta/\beta$. Thus the steady-state weighted average prevalence in the pool approaches

$$\lim_{\gamma \to 1} \bar{Y}^* = \frac{\sum_{k=1}^{K} i_k \alpha_k \max\left(1 - \frac{\delta}{i_k \beta}, 0\right)}{\sum_{k=1}^{K} i_k \alpha_k}, \tag{17}$$

Taking the derivative with respect to i_k shows that the cutoff below which reducing activity increases weighted-average prevalence in steady state is δ/β partners per year. Under the preferred matching model, $\delta/\beta = 2.57$ would generate steady-state prevalence of 0.005 for $\gamma = 1$. In this case $j_e = 2.57$ partners per year, compared to only 0.92 partners per year under random matching. Thus preferred matching increases the proportion of the population that generates negative externalities by having fewer partners. However, these negative externalities become smaller as people match outside their own groups less often. (As γ approaches one, people will have less and less influence on others' prevalence.) Moreover, prevalence among those with fewer than δ/β partners per year will approach zero, and thus will also become insensitive to marginal changes in their own activity.

Outside of the limiting case as γ approaches one, it is necessary to calculate j_e and j_t numerically. The first entry in each cell of Table V shows an estimate of j_e for particular values of steady-state prevalence and γ, the degree of assortativeness in matching. The

transmission rate, β, is chosen so as to match the target prevalence given γ.[22] The figures in parentheses show the proportion of the population with less than the cutoff frequency of partner change. At low prevalence, j_e increases with γ. The second entry in each cell of Table V shows an estimate of j_t, the cutoff number of partners below which reductions in the number of partners will increase steady-state prevalence in the population as a whole. Note that at low prevalence, such as that found among British heterosexuals, j_t does not change monotonically with γ, but that for all values of γ in the table, j_t is greater than the number of partners of 80% of the population.[23] At high prevalence, j_t and j_e decline with γ. In summary, reductions in the rate of partner change are likely to have perverse effects in low prevalence populations, but not in high prevalence populations, whether matching is random or partially assortative.

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[22]Thus different values of β are used in each cell, so β is not held constant moving across a row of Table II.

[23]In these simulations, j_t is higher for high or low γ than for moderate γ. This is not true for a general distribution of rates of partner change, however. In simulations using data from Anderson and Medley's now-superseded study of sexual behavior [reported in Anderson and May, 1991], j_t was often higher for moderate than for extreme values of γ.

Table V: j_e and j_t by Steady State Prevalence and Assortativeness in Matching.
(j_e in first entry, j_t in second entry, percentage of population below cutoff value in parentheses)

	γ=0	γ=0.25	γ=0.5	γ=0.75	γ=0.99	lim $\gamma \to 1$
Y*=.005	0.92(92%) 0.79(88%)	1.42(96%) 0.75(88%)	1.80(97%) 0.67(88%)	2.07(98%) 0.59(82%)	2.39(98%) 0.68(88%)	2.57(99%)
Y*= .01	0.83(92%) 0.67(88%)	1.16(94%) 0.60(88%)	1.38(96%) 0.50(82%)	1.53(96%) 0.43(82%)	1.65(97%) 0.49(82%)	1.75(97%)
Y*=.02	0.74(88%) 0.54(82%)	0.93(92%) 0.44(82%)	1.05(94%) 0.34(72%)	1.11(94%) 0.28(72%)	1.12(94%) 0.37(72%)	1.17(94%)
Y*=.05	0.59(82%) 0.33(72%)	0.66(88%) 0.22(72%)	0.70(88%) 0.15(9%)	0.71(88%) 0.11(9%)	0.66(88%) 0.17(9%)	0.67(88%)
Y*= 0.1	0.47(82%) 0.13(9%)	0.50(82%) 0.05(9%)	0.50(82%) .005(9%)	0.48(82%) .001(9%)	0.41(82%) 0.09(9%)	0.39(72%)
Y*= 0.2	0.35(72%) 0	0.35(72%) 0	0.34(72%) 0	0.27(72%) 0	0.23(72%) 0	0.197(9%)
Y*= 0.3	0.28(72%) 0	0.28(72%) 0	0.26(72%) 0	0.24(72%) 0	0.18(9%) 0.02	0.17(9%)
Y*= 0.5	.197(9%) 0	.19(9%) 0	0.17(9%) 0	0.16(9%) 0	0.12(9%) 0	0.11(9%)

To understand why j_e and j_t can increase with γ, recall from Section I that under symmetric information, people who expected to have many partners in the future would compensate low activity people for matching with them in order to obtain safer partners in future periods. For this mechanism to operate, it is essential that people be able to observe not only how many partners other people have had, but also how risky those partners were. If people can observe others' frequency of partner change, but cannot observe the riskiness of others' previous partners, there is no incentive for people who expect to have many partners in the future to choose safer early partners. Thus the positive externalities from low activity people having more partners will not be internalized.

To understand the intuition epidemiologically, note that as γ rises, prevalence falls

among low activity people. Thus when low activity people match in the general pool they cause greater reductions in the weighted average prevalence. Moreover, as γ rises, prevalence becomes flatter in the number of partners for those with few partners. Thus increases in activity by low activity people increase their own risk of infection by a smaller amount. These effects can cause increases in γ to increase j_e and j_t. (Of course, as γ rises, people are less and less likely to match with the general pool, and this effect will cause j_t to fall with γ.)

Recall that as prevalence approaches zero, so the risk of infection becomes linear in the number of partners, j_e approaches c/2. As prevalence rises, and the risk of infection becomes more concave in the number of partners, j_e falls further below c/2. Preferred mixing makes the risk of infection convex in the number of partners, except for those with very high numbers of partners, and tends to make j_e greater than c/2.

IV. Dynamics

Previous sections have concentrated on steady-state analysis. This section examines the time-path of prevalence in response to reductions in the frequency of partner change. In the short-run, a greater percentage of the population will increase prevalence among <u>others</u> by having fewer partners. However, having fewer partners cannot increase <u>total</u> prevalence immediately, but only after a transition period that may last several years.

To see why more people will generate negative externalities by having fewer partners in the short-run than in the long-run, note that in the short-run, reductions in the rate of partner

change by people with less than the weighted average probability of infection, $\bar{Y}$, will increase others' risk of infection. Thus if the average risk of infection in the pool of available partners is 10%, someone with a 9% risk of infection who increases his rate of partner change to one thousand a year will reduce the average risk in the pool for the first few days, until his risk of infection reaches 10%. In low prevalence populations, the risk of infection is approximately proportional to the number of partners under random matching.[24] Thus those with fewer than approximately the weighted average number of partners per period, c, will create short-run negative externalities by reducing their rate of partner change. In contrast, as shown in Section II, only those with fewer than c/2 partners per period will generate long-run negative externalities.

However, reductions in the frequency of partner change always reduce <u>total</u> prevalence in the short-run. This is because immediately after a decrease in the rate of partner change by low activity people, some infected people will have fewer partners, and the partners they do have will be more likely to have already been infected. (Prevalence eventually rises because the new infections will be concentrated on high activity people who are more likely to spread the disease.) Public health officials, who may wish to base policy on total prevalence, rather than externalities, may wish to know how long a transition period is required before prevalence

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[24]Similarly, under preferred mixing, as γ approaches one, the risk of infection approaches $1 - \delta/i\beta$, so those with fewer than $\delta/(\beta(1- \bar{Y})$ partners per period will generate short-run negative externalities by having fewer partners. This is higher than the long-run cutoff, δ/β, and in low prevalence populations it will encompass virtually the entire population.

increases in response to a reduction in activity.

The transition period is fairly long under the simple SI model, but much shorter under more realistic models. In a model that incorporates a mortality effect of the disease, prevalence decreases faster following an increase in activity, because the people who are infected immediately after the increase in activity do not continue to infect new people for as long a period. Moreover, the transition time is even shorter when allowance is made for the likelihood that people are most infective immediately after becoming infected and again after developing AIDS.

Table VI shows the dynamics of prevalence in response to reduced activity under the basic SI model, a more realistic model with AIDS induced mortality[25], and a still more realistic model in which infectiousness varies with the stage of the disease, as in Jacquez, et al [1994].[26] In all three simulations, initial prevalence is assumed to be one half of one percent. The simulations examine the impact of a reduction in activity to 0.16 partners per year by all people with 0.2 partner per year. The first row shows that steady-state prevalence increases in

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[25] The death rate from causes other than AIDS is assumed to be 0.03, and the death rate from AIDS is assumed to be 0.1. The model requires a higher transmission rate to match any given steady-state prevalence. It also requires that a higher proportion of the population be born into highly active groups in order to match the observed proportion of high activity groups in the population. It does not allow for the effect of AIDS-induced mortality on the birth rate, as would be appropriate in studies of aggregate population dynamics in some high prevalence African countries.

[26]Transition probabilities are taken from Jacquez et al. [1994], and converted to Poisson hazard rates. The hazard rates for progression into the next stage are 0.96976 and 0.118775 in the first and second stage respectively. The death rate in the final stage of infection is 0.53. The transmission probability is set at 0.01 in the final stage, and 0.001 in the second stage. In the first stage, it is calibrated to match the desired prevalence, given γ.

response to the reduction in activity under all three models, but the effect is greatest under the basic model. The second row shows that prevalence takes 185 years to fall half-way from its initial level to its new steady-state level under the basic model, thirty years under a model with AIDS-induced mortality, and only five years if infectiousness varies with the stage of the disease.[27] The third row shows the initial percentage change in the number of new infections (or *incidence* in medical terminology), in response to a reduction in activity by those with one partner a year. Note that in all three models the initial reduction in incidence is small compared to the steady-state increase. The final row shows that incidence returns to its original level after 5.4 years in the basic model, after 1.4 years in a model that incorporates the effect of the disease on mortality, and after only two months if infectiousness depends on the stage of infection. (Prevalence, the stock of infected people as a fraction of the population, takes approximately twice as long to return to its original level.)

[27]If infectiousness varies with the stage of infection, prevalence initially declines in response to a reduction in activity and then overshoots its steady-state value, before declining to a new steady-state value above its original level. The time until 50% of the steady state change is attained therefore underemphasizes the costs of reductions in activity.

Table VI: Dynamics under Different Models[28]
(In all three models, prevalence is set at 0.005 , and the group with 1 partner/5 yrs. reduces its activity to 0.8 partners/5yrs.)

	Basic	AIDS Mortality	Varying β
% increase in Y^*	30.9	16.5	15.0
50% of change (yrs)	185	30	5
% Initial Δ in Incidence	-0.81	-0.74	-0.74
Yrs Until Incidence = Initial Incidence	5.4	1.4	0.14

Although the dynamics are affected most strongly by AIDS induced mortality and by varying infectiousness with the stage of the disease, dynamics are also affected by γ, the degree of assortativeness in matching, Y^*, the steady-state prevalence, and the how close the number of partners of the group changing its activity is to j_t. Table VII shows more months are required for incidence to rise to its initial level in response to a reduction in activity for higher Y^*, higher γ, and higher i. j_e and j_t seem to be lower under models that allow for mortality effects of the disease and for infectiousness to vary with the stage of infection.

[28] The simulation with varying β requires three times as many state variables as groups. In order to keep the number of state variables manageable, the simulations in this table therefore aggregated everyone with more than 8 partners per year into a single group. The number of partners in this group was chosen not to be the average in the group, but to keep c in the simulation equal to c in the original data.

Table VII: Months Until Incidence Rises to Its Original Level in Response to a Reduction in Activity

Top entry in each cell assumes a reduction from 0.4 partner/yr to 0.3 partners/yr: bottom entry in each cell assumes a reduction from 0.2 partners/yr to 0.1 partners/yr. Dashes indicate j_t is less than the number of partners. Infectiousness is assumed to vary with the stage of infection as in Jacquez, et al [1994].

	$\gamma = 0$	$\gamma = 0.5$	$\gamma = 0.9$
$Y^* = 0.005$	4.2 1.8	10.4 5.2	23.8 9.6
$Y^* = 0.01$	4.7 2.2	- 6.6	- 12.7
$Y^* = 0.02$	7.3 3.2	- 9.1	- 18.7
$Y^* = 0.05$	- 6.0	- -	- -

V. Policy Implications and Directions for Future Research

This paper has argued that, in low prevalence populations, reductions in the frequency of partner change by low activity people may create negative externalities and increase the long-run prevalence of AIDS. The results should be considered preliminary, given the limitations of the data and the simplifying assumptions of the SI and preferred mixing models. However, to the extent they are confirmed in future research, they suggest that public health messages are more effective in reducing the steady state prevalence and creating positive

externalities if they stress condom use rather than abstinence.[29] Furthermore, while others have argued that public health messages targeted to high activity individuals are more likely to reduce prevalence, this analysis would suggest that untargeted public health messages may backfire and actually increase prevalence.

Before adopting these conclusions, however, more research is needed both to examine the robustness of the results discussed here, and to evaluate the impact of condom use on the epidemic. If people in a particular geographic, age, or sexual preference group match mainly with each other, then c/2, j_e, and j_t should be calculated separately for the group. Further research is also necessary to see if these results are robust when differences between the sexes, the age-structure of matching, and the process of partnership-formation and dissolution are explicitly modeled. Finally, it would be useful to examine other ways of modeling partial information, in which the probability of two people matching declined continuously with the difference in their frequency of partner change. There is no reason, however, to presume that this would significantly change the results.

The case for emphasizing condom use, rather than abstinence, is strengthened by a preliminary analysis using the SI model. This suggests that condom use may create large positive externalities in steady state. Suppose that all people have i partners per period, but

[29] I am not suggesting that public health officials encourage people to have more partners, since anyone who followed such advice would face a higher risk of infection, and people have an expectation that public health officials will inform them about how to protect themselves from health risks.

that a proportion q insist on condom use.[30] (For simplicity, I will assume the disease is never transmitted when condoms are used.) If people who insist on condom use match only with each other, then the decision to use condoms will not affect others' risk of infection. However, if those who would not otherwise use a condom always agree to use a condom when they meet people who insist on it, the differential equation for prevalence will be

$$\dot{Y} = (1-q-Y)\beta iY-\delta Y \tag{18}$$

since only non-infected, non-condom users are susceptible. Setting the change in prevalence equal to zero implies $Y^* = 1 - q - \delta/\beta i$. Dividing by 1 - q shows steady-state prevalence among the group which does not insist on condoms is $1 - \delta/[(1-q)\beta i]$, so condom use affects prevalence in the rest of the population just as would a reduction in the transmission rate.

To see why condoms are likely to have large positive externalities in steady state, note that if an additional 1% of the population insists on condoms, those who do not insist on condoms will have to use condoms, contrary to their preferences, an additional 1% of the time. They may or may not be compensated by their partners for this. On the other hand, since $Y^* = 1 - q - \delta/\beta i$, steady-state prevalence in the pool of available partners will fall by 1%. Thus if people are willing to trade off a 1% increase in condom use against a 1% reduction in the chance that they will have unprotected sex with an infected person, increases in condom use

[30]Fausto Panunzi and Jeronimo Zettelmeyer have pointed out that an increase in the number of partners by those who use condoms will reduce steady-state prevalence.

will create positive externalities in steady state.[31] This will be the case under the weak condition that people prefer to use condoms with those they know are infected.[32] This paper has examined the consequences of changes in the frequency of partner change by low activity people holding constant the number of partners of others. In fact, reductions in the number of partners by low activity people may lead others to have fewer partners both by increasing the riskiness of the pool and by increasing the search costs needed to find partners. Incorporating these effects would change the positive analysis of the spread of the epidemic but would not change the direction of externalities. Abstracting from search costs, each person's welfare depends on the risk of infection in the pool, because it is this risk of infection which determines the trade-off between having a desired number of partners and risk of infection. If low activity people reduce their activity and thus increase prevalence in the pool of available partners, this reduces the welfare of high activity people - even if it induces high activity people to have fewer partners, and thus reduces their risk of infection. Similarly, under standard search models, reductions in the rate of search by some people make others worse off because they reduce their available opportunities.

A companion paper, [Kremer, 1994A], integrates an economic analysis of the effect of

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[31]Showing that condom use creates positive externalities in steady state does not imply that it creates positive externalities when transition costs are included. By insisting on using a condom with someone who would not use one otherwise, one may reduce the utility of one's current partner, but will reduce risk for that person's future partners.

[32]As discussed in Kremer [1994B], signalling could conceivably create negative externalities from condom use, but it seems likely that these negative externalities will be outweighed by the positive externalities discussed above.

HIV prevalence on individual behavior with an epidemiological analysis of the effect of behavior on HIV prevalence. If increases in prevalence in the pool of available partners spur equal absolute reductions in the number of partners by all people, then reductions in the number of partners by low activity people will increase steady-state prevalence, as long as $\mu < \sigma$, as is the case in the data. The paper identifies conditions under which increases in the number of partners by low activity people may improve the quality of the pool of available partners and thus spur further increases in activity. It also shows that the declining marginal risk of infection with the number of partners under asymmetric information may spur prostitution in high prevalence populations.

Kremer [1994b] examines cases in which people choose partners using imperfect signals of risk, such as age, ethnicity, or behavior. Use of such signals may be self-reinforcing because they may widen disparities between groups. In low prevalence societies, this segregation may increase overall prevalence.

The model predicts that societies with uniformly low activity would have the lowest prevalence of sexually transmitted diseases (STDs), but that among societies in which a segment of the population had high activity, those in which the rest of the population had moderate, rather than very low, activity might have lower prevalence. Testing this proposition is not likely to be possible. However, it is interesting to note that, despite claims that the AIDS epidemic is a product of the sexual revolution, STD prevalence is relatively low in

modern western societies.[33] In contrast, prevalence was high during the Victorian era, when social mores were such that many women were expected to have only a single partner over their lifetime. Men, however, were allowed to have more partners and the difference was accounted for by a small number of high activity women. A similar situation may hold in Thailand and in some parts of Africa, although hard evidence is scarce. It seems possible that changes in sexual mores that allow typical women to have more than one partner over their lifetime may actually reduce steady-state prevalence in societies in which men already have many partners.

[33]I thank Anne Johnson for suggesting this point.

Appendix: Existence of Equilibria Under Symmetric Information

Eric Maskin has supplied the following proof that an equilibrium will exist, assuming that people can have only a finite number of partners, that people have sufficient endowments of money, and that the marginal utility of money is bounded away from zero.

Suppose that each individual's utility can be written as a function of money and of his type and the type of a finite number of partners, where type refers to someone's history and preferences. Denote the number of possible types as N and the number of periods in which people can match as τ. For each initial type τ, there is a continuum of traders of measure I_τ. Let K be an amount of money such that a payment of K/N is sufficient to induce people to accept any vector of partnerships and such that they would not be willing to pay K/N for any vector of partnerships.

$$u(\theta, E-K/N) < u(\theta', E) \qquad \forall\theta,\ \theta' \tag{19}$$

and

$$u(\theta', E+K/N) < u(\theta, E) \qquad \forall\theta,\ \theta' \tag{20}$$

where E is the endowment of money, and θ and θ' are vectors of partners.

The problem is to find a set of prices for each match such that the market for each type of match clears. Normalize prices in period T so that they sum to K. Normalize those in T-1 so that they sum to 2KN. Normalize those in period T-2 so that they sum to $4N^2K$, etc.

Prices here are gross prices - if type A matches with type B, A will pay a price P_{AB}, and B will pay a gross price P_{BA}, so the net price will be $P_{AB} - P_{BA}$.

Suppose there is a game between an auctioneer and the agents, in which the auctioneer chooses prices period by period to maximize the value of excess demand, and each player chooses the matches given these prices. If the agents are indifferent, they randomize over all optimal choices. Thus for each player there will be a best reply set to any price vector announced by the auctioneer. The vector of randomizations for all the players determines excess demand for each match. For example, if the probability that a type τ trader demands a particular match m in some period t is q, and the probability that the reciprocal type τ' demand the reciprocal match m′ is q′, then the excess demand for m is $qI_{\tau} - q'I_{\tau}'$. Since the auctioneer chooses prices to maximize the value of excess demand, the auctioneer will put positive prices on matches for which there is excess demand, and zero prices on all other matches.

Define a mapping from each profile of price vectors $(p_1.....p_T)$ and each vector of strategies (one for each initial type) to the cross product of the best-reply sets for traders and the sets of excess-demand-value-maximizing prices (where the maximizations are done period by period). Given a finite pure strategy space in this game, it will have a fixed point.

To see that a fixed point of this mapping is an equilibrium, suppose to the contrary that there is non-zero excess demand for some match in, say, period T-1. If this excess demand is negative, then excess demand for the reciprocal match is positive. Consider the period T-1 matches for which excess demand is highest. The corresponding price for at least one of these

matches is at least 2KN/N = 2K. But then even if a trader choosing this match selects a period T match for which the net price is -K (the worst-case scenario), he still ends up paying K more for these two matches than if the choose no matches at all for periods T-1 and T, a contradiction.

By standard arguments, any competitive equilibrium of this game will be Pareto optimal. Note that there may be several vectors of equilibrium prices which will sustain the equilibrium allocation of partners to matches.

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