

# HIV BREAKTHROUGHS AND RISKY SEXUAL BEHAVIOR\*

DARIUS LAKDAWALLA<sup>†</sup>  
NEERAJ SOOD  
DANA GOLDMAN

Recent HIV treatment breakthroughs have lowered HIV mortality in the United States, but have also coincided with increased HIV incidence. We argue that these trends are causally linked, because new treatments have improved health and survival for the HIV+, increased their sexual activity, and thus facilitated HIV's spread. Using variation in state-level Medicaid eligibility rules as an instrument for HIV treatment, we find that treating HIV+ individuals more than doubles their number of sex partners. A change of this magnitude would increase infection risk by at least 44 percent for the HIV-negative and likely have lowered their expected welfare.

## I. INTRODUCTION

The diffusion of breakthrough HIV drugs in developed countries has transformed the disease from a death sentence into a manageable chronic condition. Since 1995, the overall U. S. AIDS death rate has fallen by almost 70 percent. Perhaps most crucial to this remarkable turn of events was the introduction of highly active antiretroviral therapy (HAART), the currently recommended treatment regimen for HIV [Carpenter et al. 2000].

Figure I illustrates the dramatic decline in the U. S. AIDS mortality rate. In the span of just six years, AIDS death rates fell from more than sixteen per 100,000 to around five. Progress was especially rapid during the mid-1990s, during which death rates fell by two-thirds in just three years. In spite of the encouraging declines in mortality, the rising HIV infection rate is cause for

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<sup>†</sup> Corresponding author. 1776 Main Street, Santa Monica, CA 90407. [darius@rand.org](mailto:darius@rand.org).

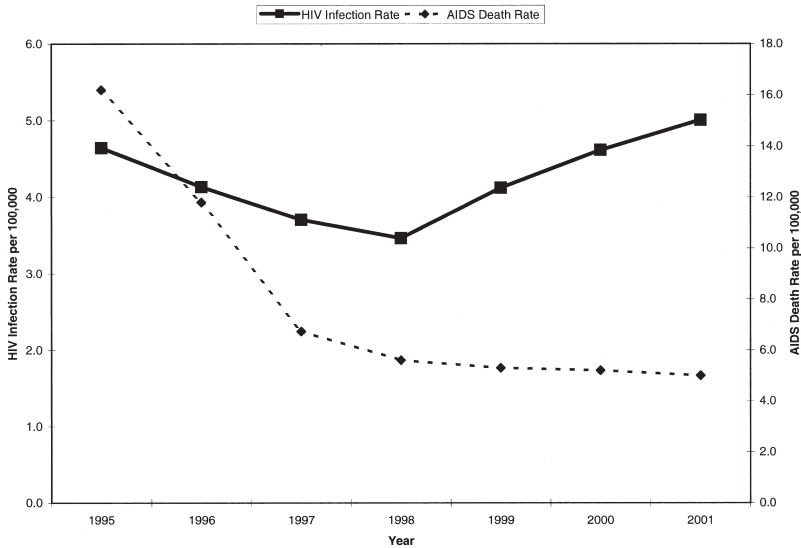


FIGURE I

## HIV Incidence and Deaths from AIDS in the United States

Data are from CDC HIV/AIDS Surveillance Report 1993–2001 (Year-end editions), and CDC HIV Mortality L285 Slide Series 2000. Also see Centers for Disease Control [1998]. Data on HIV infection rates are from the 26 states that collected confidential data on HIV infection rates throughout this period. The death rate is based on nationwide numbers.

concern. The number of incident HIV cases in the United States climbed by over 40 percent from 1998 to 2001.<sup>1</sup> Moreover, the increased rate of infection has occurred over a period during which sexually active Americans have become more vigilant about safe sex, rather than more complacent.<sup>2</sup> Are these trends in

1. The increased infection rate is almost certainly not due to better diagnosis, as the rate of HIV testing was stable over this period (based on authors' calculations from the General Social Surveys, 1998–2002). Similar growth in incidence after the diffusion of HAART treatment has been observed for gay men in San Francisco [Katz et al. 2002], heterosexuals in France [Gremy and Beltzer 2004], and gay men in Amsterdam [Dukers et al. 2002].

2. From 1998 to 2002, the rate of condom usage among unmarried Americans rose every year, and by 17 percent over the entire period; this is statistically different from zero. We compiled these statistics from the 1998 to 2002 General Social Surveys (discussed later), which asked respondents if they used a condom the last time they had sex. The sample is unmarried adults who had sex within the last year. Similar trends of flat or increasing precaution are apparent along various complementary dimensions, such as the number of different partners, frequency of casual sex, sex outside a committed relationship, and sex with a prostitute.

HIV infection and treatment breakthroughs linked? And how have infection rates risen in spite of increasing precaution in the population at large?

This paper posits that increases in HIV infection rates might themselves have resulted from improvements in HIV treatment and the accompanying declines in the deadliness of HIV. In particular, we argue that advances in treatment have improved the health of the infected, reinvigorated their sexual activity, and thus rekindled the spread of HIV.<sup>3</sup> While risky behavior and sexual promiscuity among the relatively small number of HIV+ people will not show up in nationwide prevention statistics, it is of first-order importance for HIV incidence, because the HIV+ represent society's vectors of infection.

Clinical trials and observational studies conclusively demonstrate that HAART lowers mortality and improves the physical health of HIV+ persons [Hammer et al. 1997; Staszewski et al. 1999; Jordan et al. 2002; Detels et al. 1998; Palella et al. 1998; Lucas, Chaisson, and Moore 1999; Vittinghoff et al. 1999; Lucas, Chaisson, and Moore 2003; Duggan and Evans 2005]. We argue that these health benefits have led HIV+ Americans to increase their sexual activity. Sexual activity by the HIV+ carries a significant risk of transmission, because many display a clear preference for HIV-negative sex partners: 45 percent of HIV+ individuals actively seek HIV-negative sexual partners at least some of the time, and 20 percent seek HIV-negative partners all the time.<sup>4</sup> This may be due in part to a general taste for healthy, high-quality partners, and possibly also to the unique incentive of individuals with impaired immune systems to seek out "clean" partners.

The epidemiological literature has downplayed the causal link between treatment and sexual activity, because simple correlations—both in our data and elsewhere—often find that treatment and sexual activity are unrelated or negatively related [Crepaz, Hart, and Marks 2004; Moatti et al. 2003]. However, these correlational analyses suffer from the problem that patients on HAART are intrinsically much sicker than other patients. This difference in health status masks the causal effect of treatment

3. Both treatment and risk-taking (or lack of preventive) behavior determine rates of infection [Auld 2003].

4. These figures are based on the authors' calculations from the HIV Cost and Services Utilization Study (HCSUS), which is described in Section III and Appendix 1.

on sexual behavior, because the poor health of the treated population leads to diminished sexual activity.

To overcome this problem, we exploit state-level variation in the availability of public insurance for HIV-sufferers. Since treatment with HAART is expensive—costing on average about \$13,000 per year—people with insurance are more likely to get HAART. We find that HIV-sufferers who get treatment because they live in states with more generous Medicaid eligibility rules are more likely to engage in sexually risky or promiscuous behavior. According to these estimates, plausibly exogenous treatment with HAART causes an HIV+ individual to have sex with more than twice as many partners in a given six-month period. A simple theoretical model suggests that these effects of HAART are likely to have increased the risk of infection faced by the uninfected by 44 percent or even as much as 100 percent.

This positive relationship between treatment and risky behavior has several important implications for welfare and behavior. Since HIV is an *incurable* infectious disease, improvements in HIV treatment may not have unambiguous benefits for the uninfected. While the cost of being infected falls, the risk of infection rises with the stock of HIV+ people, who now live longer and more sexually active lives. Breakthroughs can reduce expected welfare among the uninfected if the cost of higher infection risk more than offsets the welfare gain from the reduced cost of infection. Calibrations of our theoretical model suggest that welfare losses may indeed have occurred among uninfected Americans. Since the prevalence of HIV is relatively low in the United States (3 per 1000 in 2002), the increased risk faced by people when uninfected likely outweighs the new treatments' benefits, which accrue only in the (relatively unlikely) event of infection.

It may be appropriate to respond to these adverse welfare effects by increasing public subsidies for HIV-prevention. Treatment breakthroughs can raise the returns to HIV-prevention by increasing the external costs of risky behavior. This occurs because first, the increased disease prevalence means that risky behavior is more likely to result in an infection; and second, each new infection is likely to result in more secondary infections due to the increased longevity of the infected. Given these effects, we explore the conditions under which treatment breakthroughs call for greater prevention subsidies.

We begin by laying out the positive implications of HIV treatment breakthroughs for behavior and the prevalence of HIV.

Next, we estimate the effect of treatment on sexual activity among the HIV+ and use these estimates to quantify the impact of new treatments on infection risk in Section VI. Finally, in Section VII we quantify the likely impact on the welfare of the uninfected and then discuss whether public prevention spending can be used to mitigate welfare losses for the uninfected.

II. THE POSITIVE IMPLICATIONS OF HIV BREAKTHROUGHS

Treating HIV expands the pool of survivors able to spread the infection, and strengthens their physical ability to engage in transmissible behavior. The theoretically predicted result is an increase in the prevalence of the disease. In response, the precautionary behavior of the uninfected may rise or fall depending on two offsetting effects: the decline in the costliness of the newly treatable disease, versus the increased risk of exposure.<sup>5</sup>

Consider a population of sexually active individuals, with the HIV prevalence  $\pi$ . All individuals value risky sexual activity. For simplicity, suppose that HIV+ people engage in as much risky behavior  $r^+$  as their (exogenous) health allows and receive the corresponding level of per-period utility  $w(r^+)$ . With probability  $1 - s$ , they die or become too sick for sex.<sup>6</sup> The lifetime utility of a sexually active infected individual is  $w(r^+)/(1 - s\beta)$ , where  $\beta$  is the one-period discount factor.

Uninfected individuals derive concave, increasing utility  $u(r)$  from their risky behavior  $r$ . The probability of infection  $\phi$  rises in a person's own risky behavior  $r$ , the prevalence of HIV  $\pi$ , and the risky behavior of the HIV+  $r^+$ :  $\phi(r; r^+, \pi)$ . If  $r^*$  represents the equilibrium level of risky behavior among the uninfected and  $\pi'$  is next period's prevalence, the lifetime utility of the uninfected person can be written as<sup>7</sup>

$$(1) \quad v(\pi) = \max_r \left\{ u(r) + \beta \phi(r; r^+, \pi) \frac{w(r^+)}{1 - s\beta} + \beta(1 - \phi(r; r^+, \pi))v(\pi') \right\}$$

5. The economic theory of AIDS prevention and transmission was pioneered by Philipson and Posner [1993]; Over et al. [2004] explore the specific relationships between the new treatments, prevention investments, and risk-taking.

6. For our purposes, it is not important to distinguish between these two outcomes, because death and illness are absorbing, zero-utility states.

7. A single uninfected individual is assumed to have no control over  $r^*$ .

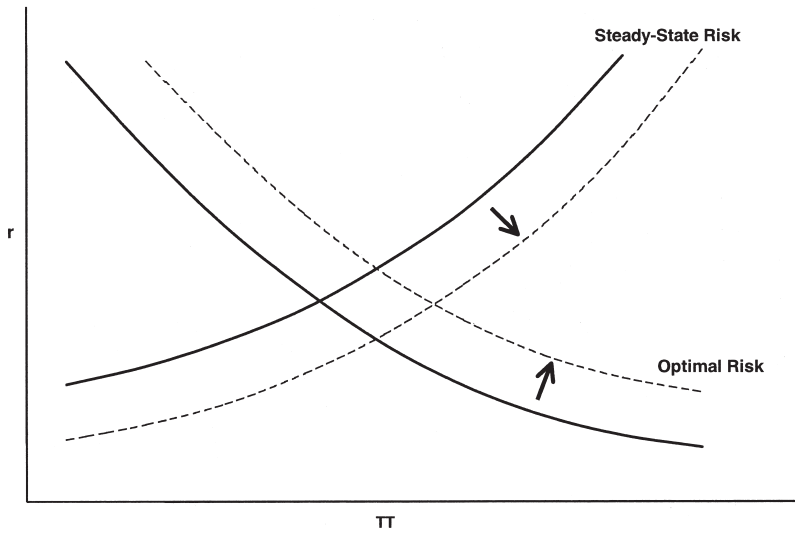


FIGURE II

Effect of Treatment Breakthroughs on Steady-State Risk-Taking and HIV Prevalence among Sexually Active

subject to

$$(2) \quad \pi' = \frac{\pi s + (1 - \pi)\phi(r^*; r^+, \pi)}{\pi s + (1 - \pi)}.$$

The unique pair of steady-state risk-taking and disease prevalence levels satisfies<sup>8</sup>

$$(3) \quad \frac{du}{dr} - \beta \frac{\partial \phi}{\partial r} \left( v^{ss} - \frac{w(r^+)}{1 - s\beta} \right) = 0;$$

$$(4) \quad \pi(1 - s) - \phi(r; r^+, \pi) = 0.$$

Each equation defines a relationship between  $r$  and  $\pi$ , indexed by the two correlates of HIV+ health,  $s$  and  $r^+$ .

Figure II demonstrates the intuition behind the steady-state

8. The unique steady state exists under assumptions best suited to low-prevalence developed countries, as discussed in Appendix 2. Kremer [1996] analyzes the more general possibility of multiple steady states, which are particularly important when people become "fatalistic" about the risk of the disease, so that increased prevalence primarily raises the cost of staying healthy. In the United States, where prevalence is relatively low, increased risk appears to lower risk-taking (cf. Ahituv, Hotz, and Philipson [1996]), at least for the average person.

dynamics that result from the pair of equations above. Equation (3) defines the “Optimal Risk” curve, which characterizes the *behavioral* relationship between prevalence and risk-taking: uninfected individuals are more cautious when faced with higher disease prevalence. Breakthroughs in treatment shift this curve up by improving the welfare of the HIV+ (i.e., raising  $(r^+, s)$ ), which makes the uninfected more willing to risk infection at a given level of exposure risk. On the other hand, the “Steady-State Risk” curve, defined by equation (4), characterizes the steady-state or *epidemiological* relationship between risk-taking and prevalence: more risk-taking results in higher prevalence. Breakthroughs shift this curve down, because a given level of prevalence can be supported by less risk-taking on the part of the uninfected. Increases in survival and sexual activity among the HIV+ multiply the effects of risky behavior among the uninfected, so that less of it is needed to sustain the disease at a given level.

The shifts in these two curves both combine to increase prevalence:<sup>9</sup> there are more sexually active HIV+ people who can spread infection; this is reinforced because the uninfected take more risk at any given level of prevalence. However, the effects on precaution are offsetting and ambiguous: the lower cost of infection encourages risk-taking, while higher prevalence discourages it.

This reasoning explains why precaution may have risen, as suggested by Figure I, in spite of the falling cost of HIV-infection. It also illustrates the way in which new treatments can increase risk for the uninfected, and reveals the importance of quantifying the impact of treatment breakthroughs on  $\phi(\cdot; r^+, \pi)$ , the risk schedule faced by the uninfected. In our empirical analysis, we recover the impact of treatment on  $r^+$  and combine this with the known clinical effects of treatment on survival ( $s$ ), to arrive at estimated increases in the risk faced by the uninfected. The increased risk is then used in conjunction with the theoretical model to assess whether or not the welfare of the uninfected declined in the United States.

9. In the most general models of rational epidemics, the change in long-run prevalence may be ambiguous. In Appendix 2 we provide support for the regularity conditions that lead to this unambiguous result, and we explain why they are appropriate for the recent history of HIV in the United States.

## III. DATA

We use data from a nationally representative study of HIV+ patients in care—the HIV Costs and Services Utilization Study (HCSUS). The HCSUS employed a multistage national probability sample design to identify HIV+ patients over eighteen years old, who made at least one visit for regular care in the contiguous United States in January or February of 1996. It does not include HIV+ patients whose only contact with the health care system was through military, prison, or emergency department facilities, or who have not made contact with the health care system for their HIV. HCSUS collected data between March 1996 and January 1998—a period when HAART entered clinical practice and disseminated widely. HCSUS is a panel data set with three waves of interviews, which we refer to as “Baseline,” “Follow-Up 1,” and “Follow-Up 2.” Questions about sexual activity were posed to a random sample of 1794 respondents in Follow-Up 2. Of these respondents, 1421 completed the interview (a completion rate of 79 percent, or 84 percent after adjusting for mortality).<sup>10</sup> We use this subsample of HCSUS respondents for our analysis of sexual behavior.<sup>11</sup>

As our outcome measure, we study the number of sex partners an individual reports over the past six-month period. The key explanatory variable of interest is treatment with HAART. This is derived from HCSUS questions about medications taken by each respondent. HAART is defined (both clinically, and by the survey) as a combination drug therapy involving three types of drugs: nucleoside reverse transcriptase inhibitors, protease inhibitors, and nonnucleoside reverse transcriptase inhibitors. All HAART regimens require at least three drugs, and the vast majority involve at least one protease inhibitor. A respondent is classified by HCSUS as being on HAART if he/she reports taking any of the recognized HAART drug combinations. Table I presents descriptive statistics for all model variables.

To characterize the health of treated and untreated individuals, we report both the prevalence of AIDS,<sup>12</sup> and the distribu-

10. This sample is representative of the 197,063 HIV+ U. S. adults who received care in 1996 and survived until 1998.

11. Of the 1421 respondents, 1396 answered the question about “number of sex partners in the last 6 months.”

12. The occurrence of AIDS in an individual is derived from her self-reported CD4 counts, and the self-reported occurrence of opportunistic infections. The HCSUS imputes the presence of AIDS according to these data, using CDC algorithms.

TABLE I  
WEIGHTED DESCRIPTIVE STATISTICS BY HAART STATUS (N = 1396)

Variable	No HAART <sup>a</sup> (N = 573)	HAART <sup>a</sup> (N = 823)	Overall (N = 1396)
<b>Demographics</b>			
Age (years)	39	39	39
Non-White (%)	65	56	60
Female (%)	33	25	28
<b>Education</b>			
Less than HS degree (%)	31	25	27
High school degree (%)	31	29	30
Some college or AA degree (%)	28	27	27
College degree (%)	11	19	16
<b>Lowest ever CD4 count (cells/<math>\mu</math>l)<sup>b</sup></b>			
>500 (%)	11	2	6
200–499 (%)	46	33	39
50–200 (%)	26	37	33
0–50 (%)	17	27	23
AIDS (%) <sup>c</sup>	33	46	41
<b>State instruments</b>			
Medically needy threshold (% of FPL)	47	49	49
SSI threshold > 65% of FPL (% respondents)	93	91	92
<b>Number of partners in the last 6 months</b>			
0–1 partner (%)	73	74	74
2 partners (%)	9	8	8
3 or more partners (%)	19	18	18
<b>Number of new partners in the last 6 months</b>			
No new partners (%)	68	70	69
1 new partner (%)	15	12	3
2 or more new partners (%)	17	18	18

State instruments were obtained from Westmoreland [1999]. All other variables come from HCSUS Follow-Up 2. All data are directly self-reported, except where noted.

a. HAART is defined as a combination drug therapy involving three types of drugs: nucleoside reverse transcriptase inhibitors, protease inhibitors, and nonnucleoside reverse transcriptase inhibitors. All regimens require at least three drugs, and the vast majority involve at least one protease inhibitor. A respondent is classified by the survey as being on HAART if he/she reported taking any one of the HAART combinations.

b. CD4 count, which is self-reported, is used to gauge the health of an HIV+ person. A higher count indicates better health.

c. The presence of AIDS is based on self-reported CD4 counts, and self-reported opportunistic infections. The HCSUS imputes the presence of AIDS using these data, according to CDC algorithms.

tion of lowest-ever CD4 counts. The CD4+ t-lymphocyte cell count is a critical measure of the function of a patient's immune system. A depletion in these cells correlates strongly with the

worsening of HIV disease and physical health [Fauci et al. 1998]. Lowest-ever CD4 count measures severity by gauging the farthest progression of the disease. Table I splits CD4+ counts into four categories. Patients with CD4+ lymphocyte counts below 50 have a very poor prognosis in general; while those with counts above 500 are considered reasonably healthy.

Table I corroborates the findings of other researchers in showing that, unconditionally, the treated are no more promiscuous than the untreated. However, it also demonstrates the difficulty of drawing causal inferences from this fact alone. Treated patients suffer from more advanced disease, as they are more likely to have CD4 counts that once dropped below 50, or below 200, and they are also more likely to have AIDS.

#### IV. EMPIRICAL MODEL

Table I illustrates how simple comparisons of sexual activity by treatment status can be misleading. As a result, we develop an identification strategy that isolates quasi-random variation in the provision of treatment. The strategy derives its power from HAART's high cost and from its extraordinary effectiveness. In 1998 the average HAART regimen cost Medicaid about \$13,000, but HAART cuts mortality (in clinical trials) by about 50 percent. Since only one-quarter of the HIV+ have private insurance,<sup>13</sup> obtaining health insurance through Medicaid can mean the difference between receiving or not receiving HAART, and in turn the difference between good health and rapid physical deterioration. This suggests the possibility of using Medicaid eligibility rules as instruments for HAART receipt that are arguably unrelated to other determinants of sexual activity.

Previous research has already documented the strong effect of public insurance on the receipt of HAART and correspondingly on health and survival. Bhattacharya, Goldman, and Sood [2003] use Medicaid eligibility rules (the same ones proposed as instruments here) to instrument for public insurance, as 90 percent of the publicly insured HIV+ have Medicaid coverage. They find that Medicaid eligibility rules are extremely strong predictors of public insurance (Wald Test = 38) with the medically needy

13. In HCSUS Follow-Up 2, 25 percent have private insurance, 17 percent are uninsured, and 58 percent have public insurance. Fifty-one percent of respondents have some Medicaid coverage.

threshold having the highest predictive power. Their results show that the receipt of public insurance reduces one-year mortality rates by 66 percent (also cf. Goldman [2001]). In addition, they find that controlling for receipt of HAART virtually wipes out the mortality gradient across the publicly insured and uninsured. Therefore, access to HAART can explain most of the mortality benefits of public insurance for the HIV+, if not all of them.

#### *IV.A. Treatment Effects Model of HAART and Sexual Behavior*

The first step in developing our identification strategy is to build a model that allows for the joint determination of treatment status and sexual activity. A simple way to do this is to use a standard “treatment effects” model with instrumental variables. Specifically, we use a two-equation model where the first-stage models the binary receipt of HAART treatment as a probit, and the second-stage is a linear model for the number of sexual partners. As instruments for the receipt of treatment, we rely on state Medicaid policy variables that, we argue, affect the availability of treatment, but not sexual activity.

Let  $T_i^*$  represent the latent index function that measures the treatment propensity for HIV+ patient  $i$ :

$$(5) \quad T_i^* = \beta_1 X_i + \beta_2 Z_i - \epsilon_{Ti}.$$

The vector  $X_i$  represents observed exogenous covariates that determine treatment propensity: age, gender, and education, along with state-level social and economic factors. (The full list of variables appears in Section V.)  $Z_i$  is our vector of state Medicaid eligibility instruments. Treatment is also assumed to depend on a random error component  $\epsilon_{Ti}$  that is uncorrelated with  $X_i$  and  $Z_i$ . Define  $T_i$  as the indicator variable for whether individual  $i$  actually received HAART; it equals unity if and only if the latent index  $T_i^*$  exceeds zero.

Let  $S_i$  represent the number of sex partners for HIV+ patient  $i$ :

$$(6) \quad S_i = \zeta_1 T_i + \zeta_2 X_i - \epsilon_{Si}.$$

For simplicity, we assume that  $S_i$  depends linearly on  $X_i$  and  $T_i$ . The parameter of interest  $\zeta_1$  measures the causal effect of treatment on the number of partners. To complete the model and allow for correlation between treatment and sexual activity, we assume that the errors  $\epsilon_{Si}$  and  $\epsilon_{Ti}$  are jointly distributed as bivariate normal with correlation coefficient  $\rho$ . It is useful to think of this

correlation  $\rho$  as unobserved health. That is, patients with poor unobserved health are more likely to get treatment, and they are also less likely to be sexually active. We estimate this joint model via maximum likelihood.<sup>14</sup>

#### *IV.B. A Count Data Model of Sex Partners*

While the simple treatment effects approach is standard, its limitation is the modeling of sex partners as a continuously distributed variable, instead of an integer-valued count. Therefore, we present an alternative “count data” approach where the number of partners follows a negative binomial distribution, but HAART receipt continues to be modeled simply as a probit (see Deb and Trivedi [2004] for a detailed exposition of the model).

Suppose that there is some common unobserved component in the treatment and sex partner equation. Concretely, one can think of this as health status. This component  $\eta$  is assumed to be distributed as a standard normal random variable, so that the latent treatment equation reads as

$$(7) \quad T_i^* = \delta_0 + \delta_1 X_i + \delta_2 Z_i - v_{T_i} - \eta_i.$$

We assume that  $v_{T_i}$  and  $\eta_i$  are distributed standard normal, and that  $T_i$  continues to follow the latent index  $T_i^*$ . Therefore, conditional on  $\eta$ ,  $T_i$  follows a probit as before. Effectively, unobserved heterogeneity in the treatment equation is decomposed into one component that is not correlated with sexual behavior ( $v_{T_i}$ ) and another one that is ( $\eta_i$ ).

The number of sex partners is distributed as a Poisson process with mean/variance parameter  $\exp(\theta_0 + \theta_1 X_i + \theta_2 T_i - \lambda \eta_i - v_{S_i})$ . Just as in the treatment equation, heterogeneity in the number of sex partners is decomposed into a component that is not correlated with treatment,  $v_{S_i}$ , and one that is,  $\eta_i$ . Notice that  $\lambda$  is the covariance between the correlated errors in the two equations. A negative value of  $\lambda$  implies that people with high treatment propensity have fewer sex partners, controlling for all observables. If  $v_{S_i}$  is assumed to follow a  $\Lambda(\alpha)$  distribution,  $S_i$  is distributed as a negative binomial with mean  $\mu \equiv \exp(\theta_0 + \theta_1 X_i + \theta_2 T_i - \lambda \eta_i)$ . The coefficient  $\theta_2$  is the parameter of interest, which estimates the causal impact of treatment on sex part-

14. Maddala [1986] derives the maximum likelihood estimator. Computationally, we use the standard “treatreg” command (with weights and robust standard errors clustered at the state level) in STATA version 8.2.

ners. We estimate the parameters of this model via Maximum Likelihood; details of the likelihood function appear in Goldman, Lakdawalla, and Sood [2004].

#### *IV.C. Identification*

We use state Medicaid policies as the instrumental variables  $Z_i$  assumed to affect treatment status but not sexual activity directly. Medicaid is the most common form of insurance for the HIV+ population in care, covering 51 percent of the HIV+ and 61 percent of the insured HIV+. HIV+ patients can qualify for Medicaid through at least two pathways.<sup>15</sup> First, Supplemental Security Income (SSI) beneficiaries are automatically eligible for Medicaid in 38 states. The other states have different standards for eligibility either as a 209(b) state or a waiver state. Section 209(b) of the Social Security Amendments Act of 1972 allows States to include more restrictive definitions of “disability” and lower income and assets standards for Medicaid eligibility. Second, Medicaid eligibility is also available through a “medically needy” program for individuals who meet Medicaid’s disability criteria but have incomes that exceed the financial eligibility limit. The program allows individuals to “spend-down” to Medicaid eligibility by deducting medical-related expenses from their reported income. States can choose whether or not to establish medically needy programs, and they can also choose the income eligibility level.

As our instruments, we use the following two variables:

- “Medically Needy Threshold” is the state’s income eligibility threshold for the medically needy program expressed as a percentage of the federal poverty line.<sup>16</sup>
- “SSI > 65 percent of Federal Poverty Line” is an indicator variable for whether the state’s income eligibility threshold for Medicaid eligibility through the “SSI” category was

15. The third pathway is Aid to Families with Dependent Children (AFDC): patients who meet the state’s income eligibility and family composition requirements for AFDC as they existed on July 16, 1996, qualify for Medicaid coverage. We do not use AFDC eligibility as an instrument, since this could affect incentives for family formation and thus sexual activity.

16. States without medically needy programs are coded with an income threshold of zero percent. Specifying the state’s medically needy threshold as a continuous variable was a better predictor of treatment (in the sense of higher partial chi-squared) than various nonlinear alternatives. The main results for the effect of HAART on sexual activity are highly robust across these different specifications.

less than 10 percentage points lower than the federal guideline of 75 percent of the federal poverty line.<sup>17</sup>

Our state policy instruments could fail if they are correlated with unobserved state-level determinants of sexual activity.<sup>18</sup> To address this issue, we develop evidence that our instruments are related to state-specific differences in sexual activity only through their effects on HIV treatment receipt, and not otherwise.

Valid instruments would affect sexual activity only through their impact on HAART. Before the advent of HAART around 1996, there should have been no relationship between Medicaid eligibility policies and sexual activity. A nonzero pre-HAART relationship would suggest the presence of unobserved state-specific determinants of sexual activity, correlated with Medicaid policies. After HAART, generous Medicaid rules would increase the survival of HIV+ patients; according to theory, this would promote precaution among the uninfected.<sup>19</sup> Among the *uninfected*, therefore, valid instruments generate a negative post-HAART relationship between Medicaid generosity and sexual activity, but uncorrelatedness before HAART.

Motivated by these observations, we examine the relationship between 1996 state Medicaid rules and sexual activity among the uninfected, both before and after the advent of HAART. To construct the most relevant test, we examine subpopulations at the highest risk for HIV infection: their sexual behavior patterns are likely to be most similar to the HIV+, and the post-HAART effects of HIV treatment on their behavior are likely to be more direct and pronounced.

The GSS collect data from a nationally representative sample of individuals on social attitudes, beliefs, and behaviors, including sexual behavior. These include questions about lifetime sexual history: number of partners since age eighteen, by gender; and ever having paid for or having been paid for sex. Respondents

17. We coded the SSI threshold as a dummy variable, because there were only four states that implemented a significantly more restrictive eligibility standard than the federal guideline of 75 percent of the federal poverty line.

18. If respondents with more severe disease moved to states with more generous Medicaid policies, our instruments would be correlated with unobserved severity of disease. However, less than 3 percent of the HCSUS sample migrated across states between Baseline and the Second Follow-Up interviews, despite dramatic improvement in HIV treatment during this period.

19. There may be an offsetting force if uninfected people in more generous states face a lower cost of disease, but this presumes that uninfected people heavily discount the possibility of cross-state migration before or at the future date of infection.

are also asked more specific questions about their sexual experiences during the last twelve months: the number and gender of sex partners; and the type of sex partner, e.g., prostitute, acquaintance, neighbor, friend, stranger, spouse, long-term lover, and so forth. From these questions, we measure the recent and long-term riskiness of each respondent's behavior.

For the pre-HAART period, we use all GSS survey years from 1988 to 1994;<sup>20</sup> these include 1988–1991 annually, 1992, and 1994. For the post-HAART period, we use all years from 1998 to the present; these include 1998, 2000, and 2002. These choices end up providing about as much power in the pre-HAART period as post-HAART, a desirable property since we are testing for a zero effect pre-HAART and nonzero post-HAART.

We construct high-risk samples from the GSS in three ways. Our simplest approach is to isolate males who report ever having had sex with someone previously unknown to them, i.e., respondents who report (1) Sex with a prostitute in the last 12 months, or (2) Sex with a casual date or "pick-up" in the last 12 months, or (3) Ever having paid for or ever having been paid for sex.<sup>21</sup> We pick males here, because the HIV+ population is three-quarters male. We call this the "Risky Males" sample.

While the simplicity of the above approach is attractive, there are differences in observable characteristics (race, age, gender, and sexual orientation) between risky males and the HIV+. To complement it, therefore, we reweight the sample of all risky (male and female) respondents to match the age, gender, race, and sexual orientation composition of the HIV+ population.<sup>22</sup> Specifically, we reweight the sample so that it matches HCSUS proportions in terms of age category (18–29, 30–39, 40–49, and 50+), race (White and non-White), gender, and sexual orientation (straight male or nonstraight male).<sup>23</sup> Henceforth, this is the "Weighted Risky" sample.

Finally, we also reweight the entire universe of adult GSS respondents, according to the characteristics above. We call this the "Weighted Adults" sample. This provides us with a larger,

20. The sexual behavior questions were redesigned beginning in 1988.

21. The latter question is asked from 1991 onwards.

22. More detailed weighting is not possible (e.g., adding education groups), because empty cells emerge among young, gay Black males.

23. Since very few (less than 2 percent) HIV+ females report being gay or bisexual, we do not explicitly consider the proportion of nonstraight females.

TABLE II  
 DESCRIPTIVE STATISTICS FOR HIGH-RISK AND LOW-RISK  
 UNINFECTED GSS SUBSAMPLES

Variable	Weighted risky <sup>a</sup>	Risky males <sup>b</sup> (N = 1,056)	Weighted adults <sup>c</sup> (N = 11,817)
Demographics			
Age (years)	39	43	39
Non-White (%)	60	19	60
Female (%)	28	0	28
Years of schooling			
Less than 12th grade (%)	19	16	18
12th grade (%)	17	29	33
1–3 years of college (%)	38	28	31
4+ years of college (%)	26	27	25
Number of partners in last 12 months			
0–1 partner (%)	35	50	74
2 partners (%)	26	14	11
3 or more partners (%)	39	36	15

Sample is GSS years 1988–1991 annually, 1992, 1994, 1998, 2000, and 2002.

a. “Weighted risky” sample consists of risky males and females (with “risky” defined as in note b) reweighted to match HCSUS Follow-Up 2 composition in terms of age (18–29, 30–39, 40–49, and 50+), race (White or non-White), gender, and sexual orientation (straight male or nonstraight male).

b. High-risk males are GSS (years given above) respondents who reported: (1) Sex with a prostitute in the last 12 months, (2) Sex with a casual date or “pick-up” in the last 12 months, or (3) Ever having paid or ever having been paid for sex.

c. HIV-weighted sample is the entire GSS sample reweighted to match HCSUS Follow-Up 2 composition in terms of age category (18–29, 30–39, 40–49, and 50+), race (White or non-White), gender, and male sexual orientation (straight male or nonstraight male).

more powerful sample that still matches the HCSUS on key observables.

Summary statistics for the three GSS subsamples are shown in Table II. By construction, the age, race, and gender composition of the reweighted samples is the same as the HIV+ population in HCSUS Follow-Up 2. Risky males are older and more likely to be White. All groups are somewhat more educated than the HIV+. <sup>24</sup>

Comparing Table II to the characteristics of the HIV+ population in Table I suggests that the sexual activity of the HIV+ is likely to be similar. Direct comparison is difficult, because the HCSUS asks respondents to report partners over the last six

24. This has ambiguous and likely small impacts on the sexual risk-taking of these groups, as education has ambiguous and nonmonotonic effects on the number of sexual partners reported in the GSS.

months, while the GSS asks about the last year. However, reconciling the numbers suggests that mean partners among the HIV+ are comparable, particularly to the weighted risky sample. When we consistently topcode the number of partners at 5 in both data sets, mean partners are 1.36 in six months for HCSUS, and 2.46 in twelve months for the weighted risky GSS sample.<sup>25</sup> We can overstate the true twelve-month mean in HCSUS by doubling the six-month mean, to obtain 2.72.<sup>26</sup> The mean of the weighted risky sample is just 10 percent below the overstated HCSUS mean. Moreover, under a range of assumptions, the distribution of partners for the HIV+ likely lies somewhere between our subsamples.<sup>27</sup>

Table III presents the effect of Medicaid eligibility rules on the sexual activity of high-risk uninfected groups, pre- and post-HAART. For each sample and subperiod, we run an OLS regression and a negative binomial regression (whose coefficients can be interpreted as percent changes in the number of partners) of sexual activity on 1996 Medically Needy Thresholds; whether the 1996 SSI threshold was above or below 65 percent of the poverty line; year dummies; gender; Black; annual income decile; age category (18–24, 25–29, 30–34, 35–39, 40–44, 45–54, 55–64, 65+); and years of schooling group (less than 12th grade, 12th grade, 1–3 years of college, or 4+ years of college). The number of partners is topcoded at 5 (less than 2 percent of the sample is topcoded). Due to the three subsamples, two subperiods, and two models, the table reports the results of twelve different models. To conserve space, we report only the coefficients on the state policy variables. All standard errors are clustered by state.

Without exception, there is no statistically significant relationship between 1996 state policies and sexual activity pre-

25. The means are 2.14 for unweighted risky males, and 1.42 for the full reweighted GSS sample.

26. This is an overstatement, because it topcodes the variable at 10 rather than 5, and because some people will remain monogamous or not acquire additional partners in the extra six months.

27. Consider two opposite scenarios: (1) HCSUS respondents with zero or one partner in the last six months remained abstinent or monogamous for the last twelve months, but other respondents ended up with twice as many partners over twelve months as six months; or (2) HCSUS respondents abstinent over six months remained abstinent or monogamous over twelve months, but all others had twice as many partners over twelve months. These alternative approaches produce HCSUS twelve-month partner distributions [0–1 partners, 2 partners, and 3+ partners] of [74 percent, 0 percent, and 26 percent] and [34 percent, 40 percent, and 26 percent], respectively. Both of these would be less risky than the distribution for the weighted risky sample, but more risky than the weighted adults sample.

TABLE III  
STATE POLICY AND THE SEXUAL ACTIVITY OF HIGH-RISK UNINFECTED GROUPS,  
BEFORE AND AFTER THE ADVENT OF HAART

	Weighted risky <sup>a</sup>		Risky males <sup>b</sup>		Weighted adults <sup>c</sup>	
	Pre- <sup>d</sup>	Post- <sup>e</sup>	Pre- <sup>d</sup>	Post- <sup>e</sup>	Pre- <sup>d</sup>	Post- <sup>e</sup>
OLS on number of partners <sup>f</sup>						
Med. needy thresh.	-0.0033 (0.0046)	-0.0135*** (0.0035)	-0.0022 (0.0023)	-0.0042** (0.0018)	-0.0012 (0.0016)	-0.0065*** (0.0023)
SSI thresh. > 65% FPL	-0.3564 (0.3002)	-0.2521 (0.2428)	-0.0204 (0.0908)	-0.0473 (0.1247)	0.0688 (0.2020)	-0.1632 (0.1954)
R <sup>2</sup>	0.37	0.29	0.40	0.29	0.12	0.14
Negative binomial on number of partners <sup>f</sup>						
Med. needy thresh.	-0.0010 (0.0019)	-0.0058*** (0.0013)	-0.0011 (0.0010)	-0.0020** (0.0008)	-0.0009 (0.0011)	-0.0043*** (0.0014)
SSI thresh. > 65% FPL	-0.1550 (0.1219)	-0.1124 (0.0933)	-0.0104 (0.0379)	-0.0279 (0.0570)	0.0490 (0.1519)	-0.0156 (0.1149)
Sample sizes	506	640	497	559	6338	5479

Robust standard errors, clustered by state, appear in parentheses, underneath coefficients.

\* significant at 10 percent; \*\* significant at 5 percent; \*\*\* significant at 1 percent.

All models include dummies for survey year, gender, Black, (annual) income decile, age (18–24, 25–29, 30–34, 35–39, 40–44, 45–54, 55–64, 65+), and years of schooling group (less than 12, exactly 12, 13–16, and 16+).

a. “Weighted risky” sample consists of risky males and females (with “risky” defined as in note b) reweighted to match HCSUS Follow-Up 2 composition in terms of age (18–29, 30–39, 40–49, and 50+), race (White or non-White), gender, and sexual orientation (straight male or nonstraight male).

b. “Risky males” are defined as GSS males who reported (1) Sex with a prostitute in the last 12 months, (2) Sex with a casual date or “pick-up” in the last 12 months, or (3) Ever having paid for or having been paid for sex (available from 1991 onwards).

c. “HIV-weighted adults” sample reweights GSS so that it matches the composition of the HIV+ population in terms of age categories (18–29, 30–39, 40–49, 50+), race (White or non-White), and gender risk group (gay male, straight male, or female). HIV+ population proportions are estimated from the HCSUS Follow-Up 2 sample.

d. Defined as GSS survey years 1988, 1989, 1990, and 1991.

e. Defined as GSS survey years 1998, 2000, and 2002.

f. Number of partners in the past 12 months, which we topcode at 5. Above this level (1.92 percent of the sample), the GSS reports partners categorically not continuously.

HAART. The majority of the point estimates lie at or below their standard errors. Post-HAART, on the other hand, more generous medically needy thresholds are always associated with less sexual activity in these high-risk uninfected groups. The insignificant coefficients on the SSI variable are consistent with our later finding that the medically needy threshold is by far the more individually powerful predictor of HAART receipt.

Moreover, with the one exception of the risky males subsample, the significant post-HAART effects survive (at the 1

percent level) the full battery of region dummies and state-specific controls we use in our later HCSUS analysis.<sup>28</sup> The pre- and post-HAART effects of the medically needy threshold are statistically different (at 5 percent) across the board for the weighted adults sample, and statistically different across the board (at 10 percent) for the weighted risky sample. We cannot statistically reject equality across periods for the smaller risky male subsample.

While one can in principle always find a statistically insignificant relationship in a small enough sample, it is important to note that post-HAART samples of comparable size and power<sup>29</sup> consistently yield significant relationships between the medically needy threshold and the number of sex partners among high-risk groups. This is true regardless of the model or sample used, and these relationships are fairly robust to a range of state- and region-level controls. If a correlation between policies and unobserved state-level sexual preferences existed, it would likely appear in both the pre- and post-HAART periods, but a causal effect appears only post-HAART. Therefore, our findings are consistent with the contention that state Medicaid policies have causal effects rather than correlations with unobserved determinants of sexual activity.

As a final check on the plausibility of our results, we compared our estimates for risky groups with estimates for the population at-large, where one would expect much smaller post-HAART effects. We do in fact find little to no post-HAART effect in the sample of all adult males. OLS regressions for all adult males produce a preperiod coefficient on the medically needy threshold of  $-0.0004$  with a standard error of  $0.0005$ , and a postperiod coefficient of  $-0.0013$  with a standard error of  $0.0010$ . The coefficients on the SSI threshold were likewise insignificant

28. These include region dummies (Northeast, Midwest, South, and West), state per capita income, state-level frequency of church attendance, state level attitudes about whether homosexuality is wrong, percent living in urban areas, and abortion rates. Details regarding these variables can be found in Section V (also cf. Goldman, Lakdawalla, and Sood [2004]).

29. In all six specifications, the pre-HAART data would have had enough power to identify the estimated post-HAART effect: Of the six post-HAART coefficients on the Medically Needy Threshold, five would be significantly different from zero at 95 percent using the standard error in the corresponding pre-HAART model, and the last would pass a 90 percent test. Conversely, in all six specifications, the post-HAART data would have failed to reject equality with zero (at 10 percent) for a coefficient of the size estimated in the corresponding pre-HAART data.

in the pre- and postperiods (sample sizes are 3096 and 2554, respectively).

## V. RESULTS

### V.A. HAART and the Number of Partners

Table IV reports the results from the treatment effects model where HAART receipt is modeled as a probit and number of partners is modeled linearly. In this table and throughout the balance of this section, we investigate the impact of HAART treatment on the sexual activity of the HIV+. To illustrate the consequences of selection bias, we first estimate a naïve model (left-hand column) where we do not include our state policy instruments and treat HAART as an exogenous variable. We then estimate instrumental variables models with progressively larger numbers of controls for state-specific heterogeneity.

Comparing the naïve model with our core instrumental variables specification (Instrumental Variables Model 1) again suggests that unobserved health generates a spurious negative correlation between HAART treatment and sexual activity. The naïve model associates HAART with a statistically significant reduction in sexual activity. Instrumenting for HAART, however, reveals a statistically significant increase in sexual activity for the treated population. A treated individual has 4.4 additional partners, which corresponds to a more than 100 percent increase. The instrumented model provides further evidence of the role played by unobserved health, since it estimates a significantly negative correlation ( $\rho = -0.13$ ) between unexplained treatment and unexplained sex partners. There is some unobserved factor (e.g., poor health) that raises the probability of treatment but reduces the number of sex partners.

Jointly, the instruments produce a Chi-2 statistic near 12 ( $p = 0.003$ ). The medically needy threshold is the more powerful instrument,<sup>30</sup> but the addition of the SSI threshold improves joint significance. Not surprisingly, the first-stage power of the instruments declines as we add more state-level controls: Instrumental Variables Model 2 adds 5 additional state-level variables, and Instrumental Variables Model 3 adds 3 more. At the end of this

30. This is likely to be because only four states had SSI thresholds below the federal maximum of 75 percent of the poverty line.

TABLE IV  
EFFECT OF HAART ON THE NUMBER OF SEX PARTNERS FOR THE HIV+, ESTIMATED FROM JOINT TREATMENT EFFECTS MODEL

	Naive Model		IV Model 1		IV Model 2		IV Model 3	
	Partners	HAART	Partners	HAART	Partners	HAART	Partners	HAART
Age	-0.080*** [0.016]	-0.003 [0.006]	-0.076*** [0.009]	-0.002 [0.005]	-0.078*** [0.011]	-0.001 [0.006]	-0.076*** [0.011]	-0.002 [0.006]
Non-White	-0.543** [0.227]	-0.229** [0.107]	0.117 [0.278]	-0.228* [0.124]	-0.056 [0.212]	-0.175 [0.111]	-0.075 [0.212]	-0.167 [0.110]
Female	-1.743*** [0.429]	-0.164** [0.072]	-1.064*** [0.180]	-0.140** [0.065]	-0.980*** [0.204]	-0.151** [0.070]	-0.946*** [0.208]	-0.168** [0.069]
Less than HS degree	-2.548*** [0.527]	-0.310** [0.132]	-1.509*** [0.432]	-0.319** [0.145]	-1.364*** [0.421]	-0.353** [0.145]	-1.200*** [0.413]	-0.392*** [0.145]
High school degree	-2.825*** [0.579]	-0.262** [0.107]	-1.866*** [0.500]	-0.262** [0.117]	-1.803*** [0.504]	-0.268** [0.108]	-1.719*** [0.511]	-0.291*** [0.109]
Some college or AA degree	-1.965*** [0.341]	-0.228** [0.098]	-1.010*** [0.291]	-0.271** [0.107]	-0.989*** [0.294]	-0.283*** [0.104]	-0.924*** [0.288]	-0.303*** [0.104]
<b>HAART</b>	<b>-5.290***</b> <b>[0.624]</b>		<b>4.384***</b> <b>[0.962]</b>		<b>4.237***</b> <b>[0.998]</b>		<b>4.184***</b> <b>[1.038]</b>	

TABLE IV  
(CONTINUED)

	Naive Model		IV Model 1		IV Model 2		IV Model 3	
	Partners	HAART	Partners	HAART	Partners	HAART	Partners	HAART
Instruments <sup>a</sup>								
Medically needy threshold				0.003** [0.001]		0.004 [0.003]		0.002 [0.003]
SSI threshold > 65% of FPL				0.082 [0.076]		0.274 [0.176]		0.197 [0.179]
Chi-2 test for joint significance of instruments				11.91		5.79		1.75
<i>p</i> -value of Chi-SQ test				0.003		0.0553		0.417
Correlation (Rho) <sup>b</sup>		0.14***		-0.13***		-0.13***		-0.13***
State controls <sup>c</sup>	NO		NO		YES		YES	
Region fixed effects	NO		NO		NO		YES	
Observations	1396	1396	1396	1396	1396	1396	1396	1396

State-clustered standard errors in brackets. Estimation used STATA 8.2 “treatreg” with weights and state clusters.

\* significant at 10 percent; \*\* significant at 5 percent; \*\*\* significant at 1 percent.

a. State instruments were obtained from Westmoreland [1999].

b. Correlation coefficient between error terms: a negative sign means someone with a positive shock to treatment probability has a negative shock to his number of partners. State controls include state per capita income in 1998, percent living in urban areas, abortion rate, proportion of GSS respondents (1988–2002) in state who think sexual relations between persons of the same sex are always wrong or almost always wrong, proportion of GSS respondents (1988–2002) in state who pray several times a week. Data on state per capita income in 1998 are based on data from the Bureau of Economic Analysis available online at <http://www.bea.gov/bea/regional/spi/>. Urbanization data are based on the 2000 Census as reported in the *Statistical Abstract of the United States: 2004–05*. Data on abortion rates (abortions per 1000 females aged 15–44) in 1999 are based on the survey by the Alan Guttmacher Institute [Finer and Henshaw 2003], and population estimates from the U. S. Census Bureau. We thank Theodore Joyce for the abortion rates data.

subsection, we show that first-stage power is sufficient in all specifications to make meaningful statistical inferences.

Instrumental Variables Models 2 and 3 check the robustness of our results to the inclusion, in both the first- and second-stage equations, of state controls and region dummies. In Model 2, we add 1998 state per capita income, from Bureau of Economic Analysis Data; the 1999 state-specific abortion rate, from a survey by the Alan Guttmacher Institute;<sup>31</sup> the percent of the state population living in urban areas, based on the 2000 Census and as reported in the U. S. Census Bureau [2005]; and measures of state-specific church attendance and tolerance of homosexual activity, both of which are estimated from 1988–2002 GSS respondents.<sup>32</sup> The results from Instrumental Variables Model 2, with state controls, are extremely similar to the results from Model 1. The coefficient on HAART falls to 4.24 from 4.38; this decline represents less than one-sixth of a standard deviation.

In Instrumental Variables Model 3 we add region dummies to all the state-level controls mentioned above. It is encouraging that the estimated effect of HAART is qualitatively and quantitatively robust to this inclusion.<sup>33</sup>

As discussed earlier, an alternative approach that accounts for the integer-valued nature of the sex partner distribution is negative binomial regression. Table V displays the coefficients and standard errors estimated from the negative binomial sexual partner equation, estimated jointly with a probit for HAART receipt. The first column shows the naïve model estimated without instruments. As before, HAART is naïvely associated with a significant reduction in the number of partners. Instrumenting for HAART once again flips the sign of this effect. Since negative binomial coefficients have the natural interpretation of percentage changes, Instrumental Variables Model 1 implies that HAART increases the number of partners by 134 percent. This is similar (though slightly larger) in magnitude to the treatment effects estimates, since the mean number of partners is approxi-

31. We thank Theodore Joyce for providing us with the abortion data.

32. Further details regarding variable construction are found in Goldman, Lakdawalla, and Sood [2004].

33. Separately, we also studied the impact of HAART on reported safe sex practices. We found that HAART provision (instrumented by Medicaid eligibility rules) had no significant effect on the frequency with which individuals practiced safe sex. This implies that treatment increases the number of partners but does not induce more safe sex practices that might offset some of the resulting increase in the risk of transmission.

TABLE V  
EFFECT OF HAART ON THE NUMBER OF SEX PARTNERS FOR THE HIV+, ESTIMATED  
FROM JOINT NEGATIVE BINOMIAL COUNT DATA MODEL

	Naïve Model		IV Model 1		IV Model 2	
	Partners	HAART	Partners	HAART	Partners	HAART
Age	-0.048*** [0.007]	-0.002 [0.007]	-0.048*** [0.005]	-0.002 [0.007]	-0.048*** [0.005]	-0.002 [0.007]
Non-White	-0.160** [0.068]	-0.290* [0.157]	0.055 [0.089]	-0.292* [0.163]	-0.031 [0.074]	-0.199 [0.147]
Female	-0.764*** [0.167]	-0.239** [0.099]	-0.587*** [0.136]	-0.214** [0.084]	-0.498*** [0.130]	-0.262*** [0.095]
Less than HS degree	-0.998*** [0.202]	-0.400** [0.196]	-0.710*** [0.130]	-0.430** [0.211]	-0.596*** [0.141]	-0.522** [0.206]
High school degree	-0.960*** [0.180]	-0.360** [0.151]	-0.652*** [0.189]	-0.383** [0.159]	-0.618*** [0.170]	-0.403*** [0.146]
Some college or AA degree	-0.693*** [0.112]	-0.322** [0.148]	-0.384*** [0.094]	-0.404** [0.147]	-0.393*** [0.101]	-0.423*** [0.146]
<b>HAART</b>	<b>-1.639***</b> <b>[0.168]</b>		<b>1.339***</b> <b>[0.071]</b>		<b>1.327***</b> <b>[0.076]</b>	
Instruments <sup>a</sup>						
Medically needy threshold				0.004** [0.002]		0.003 [0.005]
SSI threshold > 65% of FPL				0.066 [0.144]		0.356 [0.290]
Chi-2 Test for joint significance of instruments				7.62		1.67
p-value of test				0.02		0.43
Lambda <sup>b</sup>		1.319*** [0.161]		-1.278*** [0.060]		-1.269*** [0.055]
State controls <sup>c</sup>		NO		NO		YES
Region fixed-effects		NO		NO		YES
Observations		1396		1396		1396

Standard errors clustered by state appear in brackets underneath coefficients.

\* significant at 10 percent; \*\* significant at 5 percent; \*\*\* significant at 1 percent.

Except where noted, all data are from HCSUS follow-Up 2 sample. Number of partners is top-coded at 24 (1.9 percent of the sample). All estimates were obtained using ml maximize (with weights and state clusters) in STATA 9.0.

a. State instruments were obtained from Westmoreland [1999].

b. Loading factor on common unobserved heterogeneity. The negative sign of this coefficient implies that errors of Partner and HAART equations are negatively correlated.

c. State controls include state per capita income in 1998, percent living in urban areas, abortion rate, proportion of GSS respondents (1988–2002) in state who think sexual relations between persons of the same sex are always wrong or almost always wrong, proportion of GSS respondents (1988–2002) in state who pray several times a week. Data on state per capita income in 1998 are based on data from the Bureau of Economic Analysis available online at <http://www.bea.gov/bea/regional/spi/>. Urbanization data are based on the 2000 Census as reported in the *Statistical Abstract of the United States: 2004–2005*. Data on abortion rates (abortions per 1000 females aged 15–44) in 1999 are based on the survey by the Alan Guttmacher Institute [Finer and Henshaw 2003], and population estimates from the U. S. Census Bureau. We thank Theodore Joyce for the abortion rates data.

mately 3.8. The estimated effect is robust to the inclusion of region dummies and state controls in Model 2, where the estimated effect is 133 percent.

We now turn to the instruments' strength. In our core treatment effects model (Instrumental Variables Model 1), the first-stage Chi-2 statistic exceeds the Staiger and Stock [1997] "rule of thumb" for strong instruments, namely a test statistic of 10.0 or higher. However, the core negative binomial model shows a Chi-2 statistic of 7.62, somewhat below the recommendation of 10.

Weak instruments can create bias toward the naïve estimates and can result in confidence intervals that are too narrow [Staiger and Stock 1997]. The first issue is not a major qualitative concern here, since our instruments flip the sign of the estimate. Moreover, the relevance of the second problem is limited by our use of maximum likelihood, which is less vulnerable to this problem [Staiger and Stock 1997; Stock and Yogo 2002]. For example, Staiger and Stock find that (for linear models with correlation  $\rho$  under 0.5) maximum likelihood produces appropriately sized confidence intervals even when instruments are arbitrarily weak.<sup>34</sup> From Monte Carlo simulations for our nonlinear models, we reached similar conclusions, suggesting that weak instruments are not causing spurious statistical significance.<sup>35</sup>

### *V.B. External Validity Tests*

The magnitude of our estimated effects seems consistent with external clinical evidence on the effects of HAART. Evidence from clinical trials of HAART's effect on CD4 cell counts—a proven marker of health status and immune function for the HIV+—suggests that HAART increases CD4 cell counts by 220

34. In their Monte Carlo analysis, Staiger and Stock analyze a linear model with correlation between the error terms equal to 0.2 (ours is 0.13). They find that maximum likelihood estimates with one or two instruments produce very little distortion: 95 percent Wald tests are rejected roughly 10 percent of the time or less, even when power approaches zero. As a point of reference, Stock and Yogo [2002] suggest that rejection rates at or below 10 percent may be taken as evidence that instruments are not weak.

35. For the negative binomial model, confidence intervals around the treatment effect were appropriately sized even with uncorrelated instruments. In the treatment effects model, inflating standard errors by a factor of 2.08 would guarantee appropriately sized confidence intervals around the treatment effect, even with totally uncorrelated instruments. Applying this conservative inflation factor would still leave us with significant effects of HAART across the board in Table IV. Detailed results and methods for our Monte Carlo simulations are available in Goldman, Lakdawalla, and Sood [2004]. These issues are discussed more generally in Deb, Lakdawalla, and Sood [2005].

for the median patient [Hammer et al. 1997; Gulick et al. 1997].<sup>36</sup> To estimate the likely impact of a 220-point CD4 increase in sex partners, we used our entire HCSUS Follow-Up 2 sample, and regressed number of partners on categorical measures of current (self-reported) CD4 cell count (the categories were 0–50, 51–200, 201–500, and 500+). Based on this regression, we calculated the increase in partners that would occur if every member of the HCSUS population had his CD4 count increased by 220. The result was an estimated increase of 4.1 partners in six months, or a roughly 110 percent increase. Since these are quite close to the estimates of the treatment effects and negative binomial models, respectively, improved health alone is a plausible mechanism through which HAART could achieve the effects we estimate.

In addition, the effects of Medicaid seem to be operating as theory predicts they should. Most persons with HIV/AIDS gain Medicaid coverage as a result of illness-related disability [Westmoreland 1999]. Therefore, Medicaid policies should have a larger impact on receipt of HAART for the disabled population. To test this, we reestimated our first-stage probit model for HAART receipt, but this time allowed the effect of the medically needy threshold to vary with AIDS diagnosis.<sup>37</sup> The results suggest that increasing the medically needy threshold from 0 percent to 100 percent of the Federal Poverty Line increases the probability of receiving HAART by 26 percentage points for those with AIDS, but by only 6 percentage points for those whose disease has not yet progressed that far. This difference is statistically significant at the 1 percent level.

## VI. TREATMENT BREAKTHROUGHS AND INFECTION RISK

HAART increases the sexual activity and the longevity of HIV+ individuals. Both these effects will increase the average risk of encountering an HIV+ sex partner. However, HAART also decreases the concentration of the HIV virus in an infected individual. This lowers infectivity, or the risk of transmission condi-

36. This is the median change in CD4 after 52 weeks of treatment with HAART. The twenty-fifth percentile change was about 140, and the seventy-fifth percentile was just over 230 [Gulick et al. 1997].

37. This model included exactly the same demographic and state controls as Instrumental Variables model 2 in Table IV, which is our “middling” specification.

tional on sexual contact. In this section we present some simple calculations that weigh these various effects.

VI.A. *Calculating the Impact of Treatment on Risk*

The first-order impact of HAART on infection risk is the change in risk, holding precautionary behavior constant. To estimate this, we begin with the fact that infection risk is the probability that one’s partner is HIV+, multiplied by the probability of HIV transmission conditional on contact with an HIV+ partner. Accounting for the clinical evidence that the risk of transmission differs by treatment status, we can represent infection risk as

$$\begin{aligned}
 (8) \quad \phi &\equiv \Pr(\text{Infection}) \\
 &= \Pr(\text{Untreated HIV+ Partner})\Pr(\text{Transmission} | \\
 &\quad \text{Untreated HIV+ Partner}) \\
 &\quad + \Pr(\text{Treated HIV+ Partner})\Pr(\text{Transmission} | \\
 &\quad \text{Treated HIV+ Partner}).
 \end{aligned}$$

Recall from the theory that  $\pi$  is prevalence,  $r^+$  is the risky behavior of the HIV+, and  $r$  the risky behavior of the uninfected. For the purposes of this discussion, we define risky behavior as the number of sex partners. Define  $\tau$  as the proportion of HIV+ on HAART,  $r_T^+$  the number of sex partners for a treated HIV+ person,  $r_U^+$  the number of partners for an untreated HIV+ person, and  $r$  the number of partners for an uninfected person. Given these quantities, the probability of encountering a treated HIV+ partner is  $\Pr(\text{Treated HIV+ Partner}) = (\pi\tau r_T^+) / [\pi\tau r_T^+ + \pi(1 - \tau)r_U^+ + (1 - \pi)r]$ . The denominator represents the average number of sex partners population-wide, while the numerator is the portion of the average accounted for by the treated HIV+.  $\Pr(\text{Untreated HIV+ Partner})$  is computed similarly.

Since U. S. prevalence is quite close to zero (2002 prevalence was 3 per 1000 U. S. adults) and  $r$  is held constant in this first-order welfare calculation, HIV treatment will have little impact on the average number of sex partners in the American population. Therefore, the key parameters to consider are the proportion treated ( $\tau$ ), prevalence ( $\pi$ ), sex partners among the

HIV+ ( $r_U^+$  and  $r_T^+$ ), and infectivity ( $\text{Pr}(\text{Transmission})$ ) due to treatment. The percentage impact of treatment on the risk of infection in equation (8) can thus be approximated using these quantities as<sup>38</sup>

$$(9) \quad \frac{\text{Pr}(\text{Infection}|\text{HAART})}{\text{Pr}(\text{Infection}|\text{Post-HAART})} \approx (1 + g_\pi)[(1 - \tau) + \tau((1 + g_{r^+})(1 - g_I))].$$

$g_I$  is the percentage decline in infectivity due to treatment,  $g_{r^+}$  is the percentage increase in the number of sex partners among the HIV+ due to treatment, and  $g_\pi$  is the percentage increase in HIV prevalence associated with the new treatment (i.e., the increase in prevalence that occurs simply because treatments reduce mortality among the HIV+). Intuitively, the effect of HAART is the growth in prevalence plus the growth in new infections per HIV+ person. The latter depends on the growth in HIV+ sexual activity, minus the reduction in infectivity.

#### VI.B. *Estimated Parameters*

To estimate the impact of HAART on the infection risk of the uninfected, we need to know (1) the proportion of HIV+ individuals treated ( $\tau$ ); (2) HAART's percentage impact on sex partners ( $g_{r^+}$ ); (3) HAART's percentage impact on prevalence ( $g_\pi$ ); and (4) HAART's percentage impact on infectivity ( $g_I$ ).

According to HCSUS Follow-Up 2, about 60 percent of HIV+ individuals were receiving HAART. The percentage change in sex partners can be read directly from the negative binomial coefficients on HAART, which indicates a 133 percent increase.

The term  $g_\pi$  reflects how much prevalence would rise if HAART were the only thing that changed. We calculate that the introduction of HAART by itself would have increased the long-run prevalence of HIV by at least 50 percent. Long-run prevalence is approximately equal to the incidence of the disease

38. To be precise,

$$\frac{\text{Pr}(\text{Infection}|\text{HAART})}{\text{Pr}(\text{Infection}|\text{Post-HAART})} = (1 + g_\pi) \frac{[(1 - \tau) + \tau((1 + g_{r^+})(1 - g_I))] \frac{\pi r^+ + (1 - \pi)r}{\pi \tau r_T^+ + \pi(1 - \tau)r_U^+ + (1 - \pi)r}}{1}$$

If  $\pi$  is close to zero, the last term approaches unity.

multiplied by the longevity of an HIV+ individual.<sup>39</sup> Holding sexual activity constant, incidence will certainly not fall. Moreover, clinical trials show that HAART increases the life-span of treated individuals by 50 percent to as much as 80 percent [Freedberg et al. 2001]. Therefore, the decline in mortality on its own increases prevalence by at least 50 percent.

Our estimate of  $g_I$  acknowledges the medical evidence that HAART reduces the infectivity of a treated HIV+ individual [Barroso et al. 2000; Gray et al. 2001; Fiore et al. 2003; Porco et al. 2004]. The clinical reduction in infectivity is tempered by a selection effect, which can cause HAART to increase mean viral load in the sexually active population: reducing viral loads among the healthiest sexually inactive patients can move them into the sexually active population, where they may raise mean viral loads. The question, therefore, is what the *empirical* viral load distribution looks like with and without HAART, not just what HAART does to viral load in a randomized trial.

We estimate  $g_I$  in two steps. We begin with the empirical viral load distributions of the sexually active HIV+ people in HCSUS, by HAART status. We combine these with estimates of infectivity by viral load from Gray et al. [2001] and Quinn et al. [2000] to arrive at an estimate of average infectivity by HAART status (that is, infectivity weighted by the empirical viral load distributions by HAART status). The Gray et al. and Quinn et al. papers are based on a study of 415 heterosexual, monogamous, and discordant (one HIV+ and one HIV- partner) couples in Uganda. The investigators measured viral load for each HIV+ respondent, tested each initially HIV- respondent regularly for HIV seroconversion, and calculated the probability of transmission based on observed coital frequency. The result was a table of infectivity risk by viral load; these are the only such estimates in the literature. Combining this with the empirical effect of HAART on viral loads in the HCSUS, we calculate that the sexually active treated population would exhibit infectivity about 13 percent lower than the sexually active untreated population.

39. Annual steady-state HIV prevalence is equal to  $\phi * L$ , where  $\phi$  is incidence and  $L$  is the expected longevity in years of the HIV+. Defining the survival rate of HIV+ persons as  $s$ , and the overall population as  $Pop$ , the evolution of the HIV+ population is governed by  $HIV_t = sHIV_{t-1} + \phi Pop$ . The steady-state expression comes out of this equation, coupled with a constant mortality hazard.

This leads to an estimated 142 percent increase in infection risk:

$$(10) \quad \frac{\Pr(\text{Infection}|\text{HAART})}{\Pr(\text{Infection}|\text{Post-HAART})} \approx (1 + 0.5) \\ \times [(1 - 0.6) + 0.6((1 + 1.33)(1 - 0.13))] \\ = (1.50)(1.62) = 2.42.$$

This is higher than the actual change in incidence (about 42 percent), which also includes the effect of rising precaution in the uninfected population. All else equal, the pure mortality reduction led to a 50 percent increase in risk, sexual activity contributed a 79 percent increase, and reduced transmissivity lowered risk by 8 percent.

The calculation above uses the lowest estimate of HAART's impact on mortality and the Ugandan study that is widely considered to be the best available analysis of HIV infectivity. However, our qualitative conclusions hold up even when we use the least generous available estimate of infectivity. Porco et al. [2004] calculate a 60 percent decline in infectivity by comparing empirical rates of transmission pre- and post-HAART. Using this number leads to an estimated 44 percent increase in the risk of infection. Later, we show that this lower number still suggests a decrease in welfare for uninfected Americans.

## VII. NORMATIVE ANALYSIS OF TREATMENT BREAKTHROUGHS

While new and improved treatments vastly raise the welfare of the infected, they can sometimes lower welfare for the uninfected by exposing them to a larger and more sexually active pool of HIV+ people. Therefore, new treatments can call for increases in government subsidies for HIV prevention.

### VII.A. *Welfare Effects of Treatment Breakthroughs*

Permanent breakthroughs in treatment obviously improve the lifetime welfare of the infected. Less clear, however, is the effect on the private welfare of the uninfected. In the long-run (steady state), the lifetime welfare of an uninfected person satisfies

$$(11) \quad v(\pi^{ss}) = \frac{u(r^{ss})}{1 - \beta(1 - \phi(r^{ss}, \pi^{ss}))} + \frac{\beta\phi}{1 - \beta(1 - \phi)} \frac{w(r^+)}{1 - s\beta}.$$

This consists of the expected utility an individual derives before falling ill [ $u(r^{ss})$ ]/ $[1 - \beta(1 - \phi(r^{ss}, \pi^{ss}))]$ , and the utility expected from the infected state  $[(\beta\phi)/(1 - \beta(1 - \phi))][w/(1 - s\beta)]$ .

Calibrating this expression suggests that treatment breakthroughs are most likely to have first-order lowered expected welfare for the uninfected. We can rewrite the lifetime welfare of the uninfected as

$$(12) \quad v(\pi^{ss}) = \frac{u(r^{ss})}{1 - \beta(1 - \phi)} \left[ 1 + \phi \frac{\beta}{1 - s\beta} \frac{w(r^+)}{u(r^{ss})} \right].$$

Consider the first-order welfare change that occurs when infection risk  $\phi$  rises. Take  $\beta$  to be 0.97,<sup>40</sup> and pre-HAART survival to be 0.8, based on a constant mortality hazard and an expected lifetime of five years from initial infection [Freedberg et al. 2001]. We calculate pre-HAART  $\phi$  to be approximately 0.08 percent.<sup>41</sup> Since a life-year spent with disability is commonly estimated to be about half as valuable as a healthy life-year, we assume that pre-HAART  $[w(r^+)]/[u(r^{ss})] = 0.5$  [Erickson, Wilson, and Shannon 1995]. For the post-HAART values, we use the maximum clinical estimate of an 80 percent increase in longevity from HAART, our most conservative estimate that first-order infection risk  $\phi$  rose by 44 percent, and conservatively assume that HIV+ individuals become perfectly healthy, so that  $w = u(r^{ss})$ . Even under these conservative assumptions, calibrating equation (12) reveals a first-order welfare decline for the uninfected.<sup>42</sup>

### VII.B. Optimal Public Prevention

Welfare decline for the uninfected can never in itself justify the end of treatment provision, whether from a Paretian, public health, or pragmatic point of view. The relevant question is how

40. This is based on a real interest rate of 3 percent, assuming that  $\beta$  is the reciprocal of unity plus the real rate.

41. In 1995 the incidence of HIV was about 4.75 per 100,000 people. In that year, about half the U. S. population was over age eighteen, and (according to the 2000–2002 GSS) approximately 13 percent of U. S. adults were unmarried with multiple sex partners in the last year. Assuming conservatively that only such people are at risk for HIV, the incidence is 5 per 6500 people at risk, or 0.08 percent.

42. This result is less likely to hold in developing countries where prevalence and infection risk are extremely high, or in extremely high-risk subpopulations. However, for the average sexually active, nonmonogamous American, expected utility is likely to have fallen.

the government can manage and ameliorate this effect. Prevention subsidies can be an efficient way to do so.

Prevention subsidies have value because a healthy uninfected person does not consider the impact of actions on tomorrow's rate of infection. In a long-run steady-state, the external costs of risk-taking by the uninfected can be written as

$$(13) \quad \underbrace{\phi_r(1 - \pi)}_{\text{Marginal Incident Cases}} * \underbrace{\left\{ \frac{\beta \phi_\pi}{1 - \beta(s + \phi_\pi(1 - \pi))} \right\}}_{\text{Incidence Multiplier}} * \underbrace{\left\{ \beta \left( \frac{w}{1 - s\beta} - v \right) \right\}}_{\text{Cost of becoming HIV+}}.$$

The long-run external cost of risk-taking consists of (1) the number of incident cases that result from a unit change in risky behavior; (2) the long-run increase in the number of cases that results from a single incident case, which we call the “incidence multiplier;” and (3) the cost to an individual of becoming infected. Since the external cost can be interpreted as the optimal per unit tax on risky behavior, understanding changes in external costs provides insight into the optimal subsidy for prevention. HIV breakthroughs always raise the first component of the external cost,<sup>43</sup> have ambiguous effects on the second, and always lower the third. The need for and degree of government prevention subsidies thus turn on the relative importance of these three effects.

A variety of subtle comparisons can be made based on the analysis above, but the broad outlines are straightforward. Increased prevention is called for when new treatments significantly raise the prevalence of the disease, relative to the cost of being infected. In some cases, sufficient prevention may be undertaken by the private sector alone. When this fails to be enough, prevention subsidies may be a tool to mitigate the erosion of welfare for the HIV-negative.

The case for public prevention subsidies is particularly

43. Formally,  $\phi_r$  rises with  $\pi$ . This could be offset if  $r$  rises, but this is not the empirical experience of the United States. In addition, if  $\pi$  is extremely large, it is possible that increases in prevalence shrink the pool of uninfected people to such an extent that there are fewer new cases of HIV for a given increase in risk-taking.

strong in the case of foreign aid to countries ravaged by HIV. Treatment aid may need to be systematically coupled with resources for prevention, because a one-time foreign aid gift does not reduce the long-run cost of infection, but may raise long-run prevalence.

### VIII. CONCLUSIONS

Treating an incurable infectious disease like HIV involves unique issues for individual behavior and social policy. While improvements in treatment clearly raise the welfare of infected individuals, societies must cope with the reality that treatment can fuel the further spread of the disease. We have presented empirical evidence consistent with this argument and have developed a few of its implications for private behavior and public policy.

HIV treatment more than doubles the number of sex partners of the HIV+. For HIV- people, this increases infection risk by at least 44 percent, and likely more than doubles it. Calibrating the theoretical model suggests that this increase in risk lowered the expected welfare of uninfected Americans on balance: in welfare terms, the increase in infection risk outweighs the expected welfare benefit from the reduction in the cost of the disease.

The government's best response to the welfare consequences may be to increase public spending on prevention. When treatment involves large negative external costs, it is efficient for the government to respond by boosting prevention incentives, which benefit the uninfected. An incurable infectious disease like HIV can generate a unique complementarity between treatment breakthroughs and prevention. Looking to the longer run, our results suggest that a cure for HIV would have extraordinary welfare benefits, because it would break the link between treatment and risky behavior by infected individuals.

Our research also suggests the importance of investigating the impact of treatment breakthroughs on the behavior of the uninfected. Elsewhere, we have found that the introduction of HAART coincided temporally with an increase in condom usage by unmarried and nonmonogamous people, but no change in condom usage by married or monogamous people [Lakdawalla and Sood 2004]. Further work is needed to determine whether HIV breakthroughs were the dominant force in generating this

behavior. These results will help further quantify the long-run welfare impacts of breakthroughs on the uninfected population.

#### APPENDIX 1: HCSUS DATA

In this appendix we describe the variable definitions and sample selection for the HCSUS data. Detailed technical descriptions of the HCSUS sample and weighting procedure appear in Duan et al. [1999]. Questionnaires and information about the survey are available on the web at <http://www.rand.org/health/projects/hcsus/index.html>.

We construct analytic weights to calibrate the sample to the reference population. A respondent's analytic weight, which may be interpreted as the number of people in the population represented by that respondent, is the product of three patient-specific quantities: the sampling weight, the multiplicity weight, and the nonresponse weight. The sampling weight adjusts for oversampling (of women, for example); the multiplicity weight adjusts for patients who could potentially enter the sample via multiple providers; and the nonresponse weight adjusts for differential cooperation [Duan et al. 1999]. All analyses presented in this paper use these weights.

**Number of partners** and **number of new partners** were self-reported. Respondents were asked, "how many different people have you had either oral, anal, or vaginal sex with in the last 6 months? Your best estimate is fine." Respondents who reported at least one sex partner were asked, "Of these people, how many of them were new partners, that is, people you had oral, anal, or vaginal sex with for the first or only time in the last 6 months?"

The indicator for **HAART** use was constructed by Andersen et al. [2000] and is based on recommendations published by the Centers for Disease Control and Prevention. It is based on detailed self-reports of prescription drug use. Specifically, HAART was defined as using a protease inhibitor (PI), a nonnucleoside reverse transcriptase inhibitor (NNRTI), or a nucleoside reverse transcriptase inhibitor (NRTI) in various combinations. For example, HAART include two or more NRTIs in combination with at least one PI or one NNRTI, or one NRTI in combination with at least one PI and at least one NNRTI. Combinations of older drugs such as zidovudine, which is an NRTI, with either a PI or NNRTI, were not considered HAART.

**Education** is reported by degree attainment and years com-

pleted. Respondents were asked, “How many years of regular school or college did you ever complete and get credit for?” Respondents were also asked about highest degree or diploma they have attained. Based on the responses to these questions, we classified respondents into four mutually exclusive education groups: (1) Less than high school degree—highest degree is “none/less than high school” and years of schooling is less than or equal to 9 years, (2) High school degree—highest degree is “high school diploma or G.E.D.” and years of school is less than or equal to 12 years, (3) Some college or AA degree—highest degree is high school diploma, AA degree or 2-year college degree, and years of schooling is less than or equal to 15 years, (4) College degree—highest degree is 4-year college degree, or professional/graduate degree, and years of schooling is 16 years or more.

We used two measures of health status from the HCSUS—**lowest-ever CD4 count** and **AIDS**. CD4 cells are immune cells and lower cell counts imply worse health. The data on lowest ever CD4 cell count are self-reports based on the question, “Of all your CD4 tests, what was your lowest count?” AIDS is based on self-reports of a detailed list of clinical conditions and symptoms. Respondents were coded as having AIDS if they had any of the “AIDS-defining or indicator conditions” outlined by the CDC definition of AIDS, or if their CD4 count was sufficiently low as to indicate AIDS according to CDC guidelines.

For the analysis described in note 33, we used a measure of **unsafe sex** from the HCSUS, based on the question, “When you have had sex since finding out you are HIV+, how often have you had unsafe sex? (1) Always, (2) Usually, (3) About half the time, (4) Sometimes, or (5) Never.” In our analysis, people who responded about half the time, sometimes, or never were coded as practicing unsafe sex. This information was combined with information on the number of partners in the last six months to classify respondents into four mutually exclusive categories: abstinent, monogamous, safe sex with multiple partners, or unsafe sex with multiple partners.

## APPENDIX 2: STEADY-STATE DYNAMICS

The value function  $v(\pi)$  is convex decreasing because its associated functional mapping satisfies Blackwell’s sufficient conditions for a contraction mapping and because that mapping takes a convex decreasing function and produces another convex

decreasing function: if  $v$  is convex decreasing, and  $\phi$  is concave increasing in  $\pi$ ,  $\beta\phi * [(w)/(1 - s\beta) - v(\pi')] + \beta v(\pi')$  is convex decreasing in  $\pi$ . Therefore, the fixed point of the mapping must also be convex decreasing.

To ensure the uniqueness and existence of a steady state, we make two assumptions:

ASSUMPTION 1.

$$\left(\frac{\partial}{\partial \pi}\right) \left[ \left(\frac{du}{dr}\right) - \beta \left(\frac{\partial \phi}{\partial r}\right) \left(v^{ss} - \frac{w}{1 - s\beta}\right) \right] < 0;$$

ASSUMPTION 2.  $1 - s - \phi_{\pi} > 0$ .

Assumption 1 means that the private marginal utility of risk-taking falls when prevalence rises. Assumption 2 implies that higher steady-state prevalence levels must be supported by higher levels of risk-taking by the uninfected.

Assumption 1 is nontrivial, because increased prevalence has offsetting effects on the marginal utility of risk-taking: it raises the impact of risk-taking on exposure ( $\phi_{r,\pi} > 0$ ), but it lowers the lifetime utility of a healthy person relative to a sick one. High prevalence could encourage risk-taking, because staying healthy becomes more costly. This point is emphasized by Kremer [1996]. For observed U. S. prevalence levels (lower than developing country levels), it appears that prevalence discourages risk-taking [Ahituv, Hotz, and Philipson 1996; Philipson 2000].

Assumption 2 is very likely to hold in the pre-HAART equilibrium, and thus is useful for assessing the effect of HAART. Note that, at any steady state, it must be true that  $1 - \phi_{\pi} > 0$ . Therefore, when survival rates  $s$  are low, the assumption condition is also likely to hold. Comparative dynamics that begin with the post-HAART equilibrium might be different, if rates of healthy survival increase dramatically so as to violate this assumption. This would lead to unstable equilibria.

Given Assumption 1, the optimal risk-taking level falls with prevalence (which lowers the marginal utility of risky behavior), and rises with breakthroughs in treatment  $s$  (which lower the cost of infection, holding prevalence fixed). Given Assumption 2, the steady-state risk level rises with steady-state prevalence, but falls with  $s$ . When  $s$  is higher, infected people live longer, and it requires less risk-taking to support a given level of prevalence. This leads to the two curves in Figure II that define a unique steady-state equilibrium.

Finally, we make an assumption analogous to Assumption 1.

ASSUMPTION 3.

$$\left( \frac{\partial}{\partial r^+} \right) \left[ \frac{du}{dr} - \beta \frac{\partial \phi}{\partial r} \left( v^{ss} - \left[ \frac{w}{1 - s\beta} \right] \right) \right] < 0.$$

This assumption implies that the increase in risky behavior by the HIV+ lowers risk-taking among the HIV-. Generally, there are two offsetting effects: increases in  $r^+$  lower the relative benefit of remaining healthy, but raise the risk of infection. Just as with prevalence, we assume that the latter effect dominates the former in developed countries.

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