

Safer Sex? The Effect of AIDS Risk on Birth Rates

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Abstract

Behavioral adjustments to mitigate increasing risk of STIs can have spillover effects that increase or decrease the likelihood of pregnancy. This paper measures the empirical effects of the arrival and spread of AIDS across U.S. cities in the 1980s on births and abortions. I show that the AIDS epidemic increased the birth rate by 0.6 percent and the abortion rate by 1.5 percent for a total of at least 391,553 additional pregnancies between 1981 and 1996. This effect is driven by women primarily using monogamy, not condoms, to protect from AIDS. These results suggest that monogamy is an important risk mitigation tool for women to avoid STIs, possibly because of women's limited control over condom use.

Keywords: HIV/AIDS, STI, Fertility, Birth Rate, Epidemic, Infectious Disease, Family Structure

JEL Classifications: I12, J13, J12, J16

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I. Introduction

Choices regarding sexual behavior and contraceptive use can mitigate the risks of acquiring a sexually transmitted infection (STI) and becoming pregnant. A key feature of these choices is that risk mitigation strategies entail trade-offs. While some choices, such as abstinence, reduce both STI and pregnancy risk, others decrease one risk but increase or leave the other unchanged. For example, condoms and monogamy both reduce STI risk, but can potentially increase the likelihood of pregnancy. This is especially true if condoms are used to substitute for more reliable forms of contraception, such as oral contraceptives, or if women with only one sexual partner have condomless sex more frequently.

I estimate the effect of increases in STI risk and resulting STI avoidance behaviors on birth rates. Because of the trade-offs between STI and pregnancy prevention strategies, the effects of increases in STI risk are theoretically ambiguous. I empirically examine the effect of STI avoidance on birth rates by exploiting variation in the spread of AIDS across U.S. cities in the 1980s and 1990s. The AIDS epidemic created a large and plausibly exogenous increase in the cost of contracting an STI. During this period, the spread of AIDS was largely driven by male same-sex contact and the average time between HIV infection and AIDS diagnosis was 10 years. Thus, within a city, the timing of AIDS arrival and the extent of the epidemic was unrelated to women's sexual behavior.

I find that local AIDS incidence has a positive and statistically significant effect on both birth rates and abortion rates. I estimate that the birth rate increased by 0.64 percent due to AIDS avoidance behavior, for a total of 221,821 additional births between 1981 and 1996. I show that the increase in births is not due to a broader cultural shock associated with the AIDS epidemic, nor is it due to increases in "risky" sexual behavior. Women adjust their behavior in response to their specific risk of infection, as measured by local AIDS incidence among those with only opposite sex partners. Further, there is no corresponding increase in gonorrhea incidence. Women adopted behaviors that decreased their likelihood of contracting AIDS and other STIs, but at the expense of heightened pregnancy likelihood.

I provide evidence that the increase in births is due to women entering monogamous partnerships to avoid AIDS. Using birth certificate information, I find that the overall increase in births is due to an increase in births to white, married and cohabiting women. This suggests that the overall increase in births is driven by women choosing to have only one sexual partner to protect themselves from AIDS. This mechanism is consistent with survey data showing that 16 percent of unmarried women decided to stop having sex with more than one man to avoid AIDS, and that this was the most commonly used AIDS avoidance strategy (Mosher and Pratt 1993).

Previous studies have shown that people adjusted their behavior in response to the AIDS epidemic and that these behavioral adjustments differed across men and women. In the United States, Francis (2008) shows that those who had a relative with AIDS adjusted their choice of sexual partners to lower their risk of infection: Men shifted to opposite-sex partners and women shifted to same-sex partners. Ahituv, Hotz, and Philipson (1996) show that men adopted condoms in response to local AIDS prevalence, but did not find a similar effect among women. This is consistent with my findings that women primarily used monogamy, not condoms, to protect themselves from AIDS. Similarly, in Brazil, Hakak and Pereda (2021) show that the AIDS epidemic increased women's marriage market value and increased the marriage rate.

I show that these behavioral changes had a spillover effects on birth rates. This is the first paper to examine the effect of increased STI risk on birth rates in an developed country. There are multiple studies examining the effects of the AIDS epidemic on fertility in countries in sub-Saharan Africa, with mixed results (Fortson 2009; Magadi and Agwanda 2010; Kalemli-Ozcan and Turan 2011; Karlsson and Pichler 2015; Duflo, Dupas, and Kremer 2015; Chin and Wilson 2018). Studies in developing countries also document the importance of bargaining power in women's ability to protect themselves from STIs. For example, Gertler, Shah, and Bertozzi (2005) show that sex workers with more bargaining power can charge a higher premium for condomless sex. Anderson (2018) shows that female HIV rates

are higher when women have less bargaining power and are less able to negotiate safe sex practices, such as condom use. Cassidy et al. (2021) also documents the role of women's bargaining power in determining condom use.

This paper highlights that decisions about number of partners are an important risk mitigation strategy for women, particularly given women's lesser control over condom use. In further support of this conclusion, I find that there is no effect of local AIDS incidence on births to Black women. This is consistent with results from Charles and Luoh (2010) and Johnson and Raphael (2009) showing that the high incarceration rate among Black men during the 1980s and 1990s decreased the marriage rate and increased AIDS incidence in Black women. Absent the option to mitigate STI risk via monogamy, women's ability to protect themselves from STIs is limited.

Understanding the effects of increases in STI risk is relevant to current trends in public health. The emergence of sexually transmitted monkeypox cases and the rapid rise of drug-resistant gonorrhea highlight the rising costs associated with STI risk and the continual importance of understanding how people respond to these risks (Kupferschmidt 2022, US DHHS 2017; Bodie et al. 2019). Understanding how people adjust their behaviors in response to STI risk can inform future public health interventions to combat the spread of disease. My research provides insights into the unique constraints women face in mitigating STI risk given the concurrent risk of pregnancy. Decisions about sexual partnerships can be an important risk mitigation strategy, but at the cost of increased pregnancy likelihood.

II. The AIDS Epidemic in the United States

The first cases of what would come to be known as Acquired Immunodeficiency Syndrome (AIDS) were identified in the United States in June of 1981. During the first year of the epidemic, the Centers for Disease Control and Prevention (CDC) tracked cases of rare pneumonia, cancer, and other opportunistic infections that occurred predominately in young men with same-sex partners in California and New York (Shilts 2007). By July of 1982, the

CDC had confirmed cases in hemophiliacs, intravenous (IV) drug users, and infants with the disease. Shortly thereafter, there were at least two documented cases of women who were exposed to the disease via opposite-sex contact (Heywood and Curran 1988).

AIDS is the result of advanced infection with human immunodeficiency virus (HIV), which is found in semen, blood, vaginal and anal fluids, and breast milk. The virus can be transmitted via sexual contact or shared needles, and from mother to child during pregnancy, childbirth, and breastfeeding (US DHHS 2020). Despite early misconceptions among the general public that AIDS was confined to men who have sex with men, scientists had identified the exposure categories and risk factors for AIDS infection as early as 1983 and were concerned that AIDS would spread quickly via opposite-sex contact. Indeed, the early concern that AIDS would reach epidemic levels in ostensibly low-risk groups influenced public health efforts and knowledge of the disease. Early responses to AIDS emphasized the idea of “universal vulnerability,” or that everyone is at risk of contracting AIDS (De Cock, Jaffe, and Curran 2011). Public health departments produced posters targeted at women that focused on both the risk of AIDS and pregnancy with messages such as, “My boyfriend gave me AIDS. I was only worried about getting pregnant,” and “A man who shoots up can be very giving. He can give you and your baby AIDS” (US NIH 1987, 1989).

Evidence suggests that public health fears of AIDS spreading rapidly via opposite-sex contact influenced knowledge and behavior regarding AIDS. In 1987, *The New York Times* ran an article describing fears of AIDS infection among women. At one clinic in New York, over 40 percent of those requesting HIV tests were women considered low risk (i.e., no history of drug use, sex with drug users, or sex with men who have sex with men), none of whom were found to have AIDS (Sullivan 1987).

Further contributing to fear of AIDS was the volatile nature of the disease’s spread across the United States (Mann 1992). The sudden and unstable spread of AIDS led to a large number of epidemiological studies seeking to track and predict the prevalence of the disease (Taylor 1989; Lam, Fan, and Liu 1996; Steinberg and Fleming 2000). The epidemiology

literature identifies the following pattern: Initially, AIDS spread from city to city, with cases concentrated among men with same-sex partners and IV drug users. As AIDS became more prevalent in a city, it began to spread outward from urban areas. Further contributing to the unpredictable nature of the epidemic was the lengthy incubation period between HIV infection and the presentation of AIDS symptoms. In the 1980s and early 1990s, the average time between infection with HIV and an AIDS diagnosis was 10 years (Osmond 1998). As a result, outbreaks in cities were not being driven by current behaviors, but behaviors from as much as 10 years prior.

The AIDS epidemic is ideal for studying the relationship between STI risk and birth rates for two reasons. First, by focusing on cities, I can exploit the spread of AIDS, which was plausibly exogenous with respect to birth rates; As noted above, AIDS spread from city to city via same-sex male contact that had occurred years prior. Second, the emergence of AIDS created an economically large increase in the cost of contracting an STI. Results from the 1990 National Survey of Family Growth (NSFG) show that 22 percent of women reported changing their sexual behavior or using condoms to avoid AIDS (Mosher and Pratt 1993). Given that a substantial share of women changed their behavior, it is reasonable to expect that birth rates were affected by the AIDS epidemic.

III. Expected Effect of AIDS Risk on Births

It is unclear how women would respond to an increase in AIDS risk given the concurrent risk of pregnancy. Condoms are the only method of contraception that can protect from AIDS transmission, but are not very effective in preventing pregnancy.¹ Condoms have a typical use failure rate of 18 percent, meaning that 18 out of 100 women will become pregnant within the first year of using condoms as their primary contraceptive method. Prescription contraceptive methods such as the oral contraceptive pill are more effective in preventing

1. In recent years, pre-exposure prophylaxis (PrEP) has offered another method of protection from HIV transmission. However, in the 1980s and early 1990s condoms were the only technological option (McManus et al. 2020).

pregnancy: The Pill has a typical use failure rate of 9 percent (Trussell 2004). If many women mitigated the risk of AIDS by switching from the Pill to condoms, then the birth rate in the population would increase. If women switched from not using contraceptives to using condoms, or adopted condoms in addition to their current method, the birth rate would decrease.²

It is also possible that women responded to AIDS risk by adjusting their sexual behavior. At the extensive margin, women can choose to abstain from any sexual activity. At the intensive margin, they can limit their number of sexual partners or choose lower-risk male partners (i.e., those who do not use IV drugs and do not have concurrent partners). In this case the birth rate would likely increase, since there is less incentive to use consistent contraception with a low-risk or monogamous partner and the frequency of sexual activity may increase with one partner. Furthermore, women may be more likely to continue a pregnancy with a low-risk partner or a sole, committed partner. However, the birth rate would decrease if women switch from male partners to female partners. Francis (2008) shows that women who had a relative with AIDS shifted from opposite-sex partners to same-sex partners to lower risk of infection.³

We expect that there are heterogeneous responses to the AIDS epidemic in the population: Women have different preferences over pregnancy, sexual behavior, and contraception. Using data on birth rates and AIDS risk at the city level, it is only possible to determine the average effect of AIDS risk on birth rates. The average effect depends on both which risk mitigation strategy is most prevalent in the population and how much each strategy affects pregnancy and birth probabilities. For example, 5 percent of women choosing to abstain from sexual activity would have a much larger effect on birth rates than 5 percent of women choosing to add condoms to their current method of contraception.

An ideal data set for this analysis would include the sexual behavior and contraceptive

2. Appendix Figure A1 summarizes the possible effects of these contraception changes on pregnancy likelihood.

3. Appendix Figure A2 summarizes the possible effects of these behavioral changes on pregnancy likelihood.

choices of individual women over time. Unfortunately, such a data set does not exist for this time period.⁴ However, we can still gain important insights by estimating the average effect. For example, a positive average effect indicates that some women are switching to condoms or limiting the number of sexual partners. A negative average effect indicates that some women are abstaining, adopting condoms in addition to other contraceptive methods, or shifting to same-sex partners. Additional analyses and survey data can then be used to assess possible mechanisms.

IV. Data Sources and Sample Construction

I estimate the theorized relationship between birth rates and AIDS risk using data from the CDC. The AIDS Public Information Data Set (APIDS) contains information on the timing of AIDS diagnosis and demographics of the patient (US DHHS 2005). All data are aggregated within Metropolitan Statistical Areas (MSAs) and are available to the public for MSAs with more than 500,000 people.

Since an individual's risk of contracting AIDS is unobservable, I use AIDS incidence in the MSA of residence as a proxy for average AIDS risk in the population. AIDS incidence is defined as the number of new AIDS diagnoses per year per 100,000 people. To demonstrate that this definition is a reasonable proxy for AIDS risk, I use regional data from National Health Interview Surveys that asked respondents to rate their chances of getting AIDS (US DHHS 1997). I regress respondents' perceived risk of getting AIDS on regional AIDS incidence. Results are presented in Table 1.

I find that AIDS incidence has a positive and statistically significant effect on the probability that unmarried women rate their own chance of contracting AIDS as high. I find no effect of AIDS incidence on unmarried men's perceptions. Consistent with the aforemen-

4. The NLSY79 would not work for this analysis for several reasons: (1) it lacks information on number of sexual partners, (2) responses to questions about sex and contraception are subject to substantial refusals and reporting bias, and (3) the sample size of women who consistently respond to sexual behavior questions is very small. The NSFG would also not work because key questions about sexual behavior, such as number of partners, were not included until 1988 so there is no pre-treatment data to serve as a comparison group.

tioned anecdotal evidence from *The New York Times*, these results suggest that women may have been uniquely risk averse to AIDS, despite the higher incidence of AIDS among men. Table 1 further shows that AIDS incidence not only affected unmarried women's perception of risk, but also their perception of AIDS avoidance strategies. Living in a region with a higher AIDS incidence increased the likelihood that unmarried women reported monogamy or condom use as a very effective strategy in preventing the transmission of AIDS. Also notable is that both men and women, on average, view monogamy as more effective than condoms in preventing AIDS transmission. 71.6 percent of unmarried women reported monogamy as very effective, compared to 36.5 percent reporting condoms as very effective. However, relative to women, men are more optimistic about both options. 75 percent and 41.2 percent of men reported monogamy and condoms, respectively, as very effective.

Using APIDS data, I create a panel of AIDS incidence among women aged 20-44 in the previous year across 102 MSAs from 1981 to 1996.⁵ I merge this data with birth records obtained from the CDC's restricted-access Natality Detail file (US DHHS, 2002). I aggregate data by MSA-year to create a panel of birth rates from 1981 to 1996. Due to the very low incidence of AIDS in adolescent women during this period, I limit my sample to the population of adult women aged 20-44.⁶

V. Empirical Approach

The primary results presented in this paper come from the following fixed effects specification:

$$y_{m,t} = \beta_0 + \beta_1 Z_{m,t-1} + \alpha \mathbf{X}_{m,t} + \gamma_m + \delta_t + \epsilon_{m,t}, \quad (1)$$

where $y_{m,t}$ is the birth rate in MSA m in year t , $Z_{m,t-1}$ is AIDS incidence in the prior year in

5. APIDS data are available through 2002. I stop my panel at 1996 prior to the nationwide introduction of Highly Active Antiretroviral Therapy (HAART), the first effective treatment for HIV. With the introduction of HAART, it is no longer plausible that AIDS incidence is exogenous with respect to birth rates. Access to HAART may be highly correlated with access to healthcare and especially reproductive healthcare. I use previous year's AIDS incidence to account for risk at time of conception.

6. Descriptive statistics for the panel are presented in Appendix Table A1

each MSA, $\mathbf{X}_{m,t}$ is a set of controls described below, and γ_m and δ_t are MSA and year fixed effects, respectively. β_1 is the coefficient of interest. In order to interpret β_1 as the causal effect of AIDS risk on birth rates, it must be that AIDS risk is independent of the error term conditional on controls and MSA and year fixed effects. In this setting, the key identifying assumption for interpreting β_1 as the causal effect of AIDS risk on birth rates is that current behaviors are not predictive of AIDS incidence due to the 10 year average incubation period between HIV infection and AIDS diagnosis. I evaluate this assumption in subsection 6C.

Despite the 10 year incubation period and the inclusion of fixed effects, we might be concerned that there is some omitted factor that varies within MSAs over time and affects both birth rates and AIDS incidence. For example, the crack cocaine epidemic of the 1980s and 1990s may have resulted in risky behaviors that increased both AIDS risk and birth rates. I address these concerns by including the following sets of controls in $\mathbf{X}_{m,t}$. All regressions control for the share of the female population in an MSA-year that is Black and the share that is neither Black nor white.⁷ I define a set of additional controls that include prior year demographic characteristics and state policy changes that likely affect birth rates. These include state-level data on the unemployment rate (US BLS 2022), the incarceration rate among men and women (US BJS 2020), the maximum welfare benefit for a family of four (Moffitt, Phelan, and Winkler 2020), and whether the state Medicaid plan covers abortion (Myers and Ladd 2020).⁸ At the MSA-level, I control for the poverty rate (US Census 2010) and the share of the population with a high school degree and a college degree (USDA 2022). I also control for the crack cocaine epidemic using a crack index developed by Fryer et al. (2005).⁹ Finally, I compare results from an unweighted and population-weighted version of equation (1). Differing coefficients between an unweighted and a weighted version

7. Prior to 1989, SEER population data and NCHS natality data are only available by three race groups: white, Black, and other.

8. Given that many control variables are observed at the state-level and some MSAs in my sample cross state boundaries, I also show in Appendix Table A4 that results are robust to limiting the sample to MSAs contained within a single state.

9. The index is calculated at the city level and proxies the spatial and temporal patterns in the crack epidemic using a variety of measures that include arrests, emergency room visits, overdose deaths, and news coverage. Further information on control variables is available in Appendix B.

could be evidence of model misspecification or heterogeneous effects across location (Solon, Haider, and Wooldridge 2015).

Though I control for a wide range of time-varying controls, we might still be concerned that heterogeneity in the effects of AIDS over time are biasing estimates. Specifically, recent advancements in the econometric literature highlight problems with the two-way fixed effects approach used in this paper. In the presence of heterogeneous treatment effects, the two-way fixed effects estimator is a weighted sum of all average treatment effects across groups and time (Goodman-Bacon 2021; De Chaisemartin and d'Haultfoeuille 2020). Results may be biased if heavily weighted by later-treated units that are compared to earlier-treated units. However, there are no estimators currently available to address these concerns in the case of a continuous treatment such as AIDS incidence.¹⁰ Given this limitation, I use three alternative approaches to indirectly address concerns about the two-way fixed effects approach.

First, I address the concern of heterogeneity over time by allowing the estimated effect of AIDS risk to vary by year. Though there is no clear “later-treated” group in the case of a continuous treatment, to the extent that later-treated groups could bias results, we might be concerned if the overall estimate is driven by the later years of the AIDS epidemic. Such a finding would also raise concerns about omitted variable bias given the large number of policy changes in the mid-1990s that could affect AIDS incidence and birth rates, such as welfare reform, Medicaid family planning waivers, changes to abortion restrictions following *Planned Parenthood v. Casey*, additional funding for AIDS prevention from the Ryan White CARE Act, and the possible early roll-out of HAART in some markets.¹¹ However, because AIDS incidence is increasing over time, heterogeneous time effects could also be attributable

10. Specifically, there are no estimators available for the case of a continuous, non-staggered treatment with dynamic effects and without stayers (De Chaisemartin and d'Haultfoeuille 2022). AIDS incidence is non-staggered as it can increase or decrease over time and changes value in every year within MSAs, also indicating there are no stayers. The effect is likely dynamic given the results from an event study specification shown in Figure 3 and given the serial correlation in AIDS incidence due to disease contagion.

11. Zavodny and Bitler (2010) find that Medicaid expansions for pregnant women in the 1980s and 1990s had no effect on birth rates. Dillender (2021) finds that funding from the Ryan White CARES Act beginning in 1991 decreased the number of deaths due to HIV/AIDS. Myers and Ladd (2020) shows that abortion restrictions such as parental involvement laws did not affect birth rates until after *Planned Parenthood v. Casey* in 1992.

to non-linearity in the effects of AIDS incidence on births. As such, I also test the robustness of results to the inclusion of a quadratic term in AIDS incidence and a Poisson specification.

Second, I attempt to directly control for differences over time by including an MSA-specific linear time trend, with the sample expanded to begin in 1970. Finally, I discretize AIDS incidence in order to test the robustness of results to recent advances in the difference-in-differences econometric literature. I create two thresholds: the first year that female AIDS incidence in an MSA reaches 1 case per 100,000 women and 2 cases per 100,000.¹² Using these thresholds, I analyze the corresponding difference-in-differences regression using a Goodman-Bacon (2021) decomposition. I also use these thresholds to analyze the effects of AIDS risk with an event study design.

Additional robustness tests exploit characteristics specific to the setting of the AIDS epidemic. I show that the relationship between AIDS risk and birth rates is driven by AIDS diagnoses in women and heterosexual men, and that birth rates are unaffected by AIDS diagnoses in homosexual and bisexual men. I analyze the effect of AIDS risk on abortion rates to show that changes in birth rates are not driven by changes in abortion likelihood. I also analyze the effect of AIDS risk on gonorrhea incidence to show that estimates are not driven by unobserved sexual behavior. Finally, I exploit the 10-year average latency period between HIV infection and AIDS diagnosis to test for a spurious relationship.

VI. Results

A. Effect of AIDS Risk on Birth Rates

Empirical results for equation (1) are presented in Table 2, columns (1) through (3). For this specification and all subsequent regressions, I estimate robust standard errors that are clustered at the MSA level. I find that AIDS incidence in women in the previous year has a positive and statistically significant effect on birth rates across all three specifications. I find

12. These thresholds are arbitrary as it is not obvious that there is an optimal way to discretize AIDS incidence. However, Appendix Figure A4 shows that these two thresholds produce a roughly normal distribution of treatment years in the sample.

that estimates are similar in magnitude but more conservative when weighting by female population. When including the full set of controls, I estimate that every additional AIDS diagnosis in women 20-44 per 100,000 women led to an increase in births of 0.0393 per 1,000 women.

To interpret the magnitude of this result, I predict the counterfactual birth rate had AIDS incidence been zero in every year. I use the fully estimated regression model from equation (1), corresponding to the most conservative estimate (column (3) in Table 2), and find the difference between actual birth rates and a linear prediction with AIDS incidence set to zero. I find that the birth rate was 0.64 percent higher than the predicted birth rate in the absence of AIDS. I estimate a cumulative effect of 221,821 additional births between 1981 and 1996.

I next evaluate whether estimates are biased due to concerns about heterogeneity over time in the two-way fixed effects specification. When allowing the estimated effect of AIDS to vary by year, I find that the main results likely underestimate the effect of AIDS risk on birth rates. Figure 1 shows the estimated coefficients from regressing birth rates on AIDS incidence interacted with year dummies, including the full set of controls described in Section 5. The effect of AIDS risk is largest and most significant between 1986 and 1992, with the effect attenuating over time.¹³ However, though the effect of each *additional* AIDS case on births decreases over time, it could still be that the net effect of AIDS on births is increasing due to rising AIDS incidence. This could be because the marginal effect of one more AIDS case is small when behavior has already changed substantially in response to AIDS. To evaluate this possibility, I first add a quadratic term in AIDS incidence to my main specification. Results are presented in Table 2, panel B. Though the quadratic term is negative, the net effect on birth rates remains positive at all observed values of AIDS

13. Estimates are large and imprecise between 1982 and 1985. See regression coefficients in Appendix Table A2. It could be that there was more heterogeneity in behavioral response to AIDS early on in the epidemic when people were still learning about the disease. This would be consistent with low media coverage of AIDS prior to 1985 (see Appendix Figure A3) and misperceptions about the disease in the early years of the epidemic.

incidence. The marginal effect of one more AIDS case is negative for only one percent of the sample.¹⁴ Second, I repeat the same counterfactual prediction exercise as above for both the linear-by-year specification and the quadratic specification. The results of this exercise are shown in Figure 2.

The downward bias created by later years cannot solely be attributed to a mechanical effect of rising AIDS incidence. When using the linear-by-year specification, Figure 2 shows that the net effect of AIDS incidence begins to level off in 1989, despite the fact that AIDS incidence in women continues increasing through the early 1990s. This trend closely mirrors that of national news media coverage of the AIDS epidemic: Appendix Figure A3 shows that national broadcast news coverage of both the AIDS epidemic and the risk of AIDS to women peaked in 1987 and declined steadily beginning in 1992. Similarly, I find that effects are not driven by MSA-year observations occurring later in the sample with very high AIDS in women. Figure 2 shows that adding a quadratic term in AIDS incidence increases the predicted net effect of AIDS on births. These findings suggest that the main estimates are a lower bound of the true effect. While I predict a cumulative effect of 221,821 additional births with my main specification, I predict 426,967 additional births in the linear-by-year specification and 540,289 additional births in the quadratic specification.

I use two additional approaches to address the two-way fixed effects concerns. First, I attempt to directly control for differences over time by including an MSA-specific linear time trend, with the sample expanded to cover 1970-1996. Results are presented in Appendix Table A3. Results are robust to the time trend inclusion, provided the year 1996 is excluded from the sample. This is consistent with the heterogeneous time effects shown in Figure 1, and with policy changes in 1996, such as welfare reform, creating a downward bias on estimates. Second, I discretize AIDS incidence into two thresholds, the first year that female AIDS incidence in an MSA reaches 1 case and 2 cases per 100,000 women, respectively, and analyze the corresponding difference-in-differences regression using a Goodman-Bacon

14. I also find that results are robust to using a Poisson specification to allow for a nonlinear relationship between AIDS incidence and birth rates. Results are shown in Appendix Table A4.

decomposition. Results are shown Figure 3, Panel A. I find that the positive and statistically significant effect of AIDS arrival and spread on birth rates is attributable to earlier treated groups being compared to not yet treated groups. Once again, this further shows that any concerns regarding the comparison between later treated and already treated groups is leading to an underestimate of the true effect. Finally, I also use these thresholds to analyze the effects of AIDS risk with an event study design. I find that the specific year in which AIDS incidence reaches a given threshold is unrelated to pre-trends in birth rates, with statistically significant, increasing effects on birth rates in post-treatment years as AIDS continues to spread.

To the extent we are concerned that later treated units are biasing results, these finding suggests that the inclusion of later treated units is downward biasing results. The downward bias created by later years in the sample also addresses any concerns that health policy changes in the later years of the sample are confounding results. To the extent that the Ryan White CARE Act, HAART roll out or welfare, Medicaid, and reproductive health policy changes in the mid-1990s are affecting birth rates, it is unlikely that these changes are driving the estimated effects of AIDS incidence on birth rates. Finally, these results also demonstrate that any bias created by heterogeneous treatment effects in levels of AIDS incidence is also downward biasing results, such that the main estimates are likely a lower bound of the true effect.

While these tests have shown the econometric robustness of results, I now turn to a more intuitive discussion of threats to identification. I evaluate and reject alternative hypotheses that could explain the positive relationship between AIDS risk and birth rates.

B. The AIDS Epidemic as a Cultural Shock

Despite the inclusion of fixed effects and controls, we might still be concerned that unobservable changes within MSAs are violating the identifying assumption by shifting both AIDS risk and birth rates. Specifically, we might be worried that the AIDS epidemic as a

whole affected cultural attitudes (Fortin 2015; Mansour and Reeves 2022). If this were the case, we would expect to find a positive relationship between birth rates and AIDS incidence in men with same-sex partners, given that the majority of AIDS cases were among this group. In contrast, if women are adjusting their sexual behavior in response to their true risk of infection, we would expect most of the effect to come from incidence of diagnoses in women or heterosexual men.¹⁵ To test this alternative hypothesis, I analyze the effect of AIDS incidence among people of different sexual orientations on birth rates.¹⁶

Results are presented in Table 3. I find that AIDS diagnoses in both women and heterosexual men result in an increase in births. In comparison, I find there is no effect of AIDS diagnoses in homosexual and bisexual men on births. Thus, instead of adjusting behavior in response to the AIDS epidemic as a whole, women are adjusting their behavior in response to their specific risk of infection, as proxied by the incidence of AIDS in potential sexual partners in their area.

Another concern related to the idea of the AIDS epidemic as a cultural shock is that the birth rate could be increasing because the abortion rate is decreasing. In other words, holding the rate of pregnancy fixed, AIDS risk results in fewer women choosing to terminate unintended pregnancies. This could be true if, as argued by Fortin (2015), the AIDS epidemic as a cultural shock led women to hold more conservative values towards marriage and family. To test this hypothesis, I use data from the Guttmacher Institute on the number of abortions by state each year per 1,000 women aged 15-44 (Jones and Kooistra 2011).¹⁷

Results showing the effect of AIDS risk on abortion rates are presented in Table 4, Panel

15. Analyzing data separately by sexual orientation is also important due to differential trends across these groups. As the APIDS manual notes, “Because men who have sex with men comprise such a large proportion of the total number of AIDS cases, trends in this subgroup will overshadow those in other groups unless the data are examined separately. Analysis of data, without regard to specific subgroups, may conceal information or lead to misinterpretation of the data.”

16. I use categories of sex and sexual orientation as provided in the APIDS data: women, heterosexual men, bisexual men, and homosexual men

17. Abortion rates are calculated by state of occurrence. MSA-level data on abortions and annual data on abortions by age group are unavailable. I use state-level abortion rates for women 15-44. Data on abortion rates are only available for 10 years between 1981 and 1996.

A. I find that AIDS risk has a positive and statistically significant effect on abortion rates.¹⁸. This result rejects the alternative hypothesis that birth rates are increasing because abortion rates are decreasing. In contrast, I find that the overall increase in pregnancies is larger than the increase in births. I estimate that there were 0.0694 additional pregnancies per 1,000 women in response to each additional AIDS case, and approximately 43 percent of these pregnancies resulted in abortion.¹⁹ This share is on par with recent literature estimating that 42 percent of unintended pregnancies result in abortion (Finer and Zolna 2016). The positive effect of AIDS risk on abortion rates also indicates that at least some of the additional pregnancies that result from AIDS avoidance behaviors are unintended pregnancies.

C. Confounding Differences in Sexual Behavior

Another alternative hypothesis argues that women are engaging in more condomless sex, which increases both AIDS incidence and birth rates. If the increase in AIDS incidence and birth rates are both driven by an increase in condomless sex, then the incidence of other STIs would also increase. I analyze the effect of AIDS incidence on other STIs using state-level data on gonorrhea incidence from 1984 to 1996. These data are publicly available via the CDC (US DHHS 2015).²⁰ Results are presented in Table 4, Panel B. I find no evidence that gonorrhea incidence increases in response to AIDS incidence. Across all specifications, AIDS risk has a negative though statistically insignificant effect on gonorrhea incidence. This result contradicts the hypothesis that higher births and AIDS incidence are both due

18. Results are robust to a Poisson specification and limiting the sample to MSAs contained within a single state, as shown in Appendix Table A4.

19. Using the estimated coefficients from Table 2, column (3) and Table 4, column (3): $0.0393 + 0.0310 = 0.0694$. $0.0301/0.0694 = 0.434$. Using the same counterfactual prediction exercise as above, I also estimate that the abortion rate increased by 1.57 percent in the 10 observed years between 1981 and 1996, for a total of 169,732 additional abortions. Combining this result with my counterfactual prediction of 221,821 additional births, I predict 319,553 additional pregnancies due to AIDS avoidance behaviors. Note that this prediction is an underestimate of the true effect, given the 6 years of missing abortion data.

20. Unfortunately, MSA-level gonorrhea data are only available after 1995. Data on syphilis are also available at the state level during the time period. I use gonorrhea as a measure of other STIs because it is much more common than syphilis and less concentrated geographically (Chesson, Harrison, and Kassler 2000) Chlamydia data is also publicly available via the CDC. However, chlamydia diagnoses were not required to be reported to the CDC until 1988 (Worboys 2019).

to unobserved increases in condomless sex among women. If births and AIDS are increasing due to increases in risky sexual activity, we would also expect gonorrhea to increase. In contrast, results are suggestive of a decrease in gonorrhea in women.

As a final robustness check, I exploit the incubation period of HIV infection. In the 1980s and 1990s, the average time between infection with HIV and an AIDS diagnosis was 10 years (Osmond 1998). Thus, AIDS incidence in a given year is not driven by current sexual behaviors but by sexual behaviors from 10 years prior. However, we might be concerned that sexual behaviors 10 years prior are highly correlated with current sexual behaviors within MSAs, driving both the increase in AIDS incidence and the increase in birth rates. To reject this alternative explanation, I first show that AIDS in women 10 years in the future is not predictive of birth rates from 1971 to 1986 (Table 5, column 1). Second, I show that birth rates 10 years prior are actually negatively correlated with birth rates from 1981 to 1996 (Table 5, column 2). In combination, these two results suggest that the positive effect of AIDS risk on birth rates found in the main analysis is not driven by unobservable differences in levels of sexual activity among women.

Finally, I add a variable for birth rates 10 years prior to my main regression specification shown in equation (1). This inclusion captures the characteristics of sexual behaviors at the time of HIV infection. To the extent that sexual behavior 10 years prior is correlated with current sexual behavior, controlling for prior birth rates removes the unobservable relationship between sexual behavior, AIDS, and births. Results are presented in Table 5, column (3). I find that AIDS risk has a positive and statistically significant effect on current birth rates, even when controlling for birth rates 10 years prior.

The analyses presented in this section argue that there is a causal effect of AIDS risk on birth rates. The AIDS epidemic led women to adjust their behavior to mitigate the risk of AIDS exposure. My results show that an unintended consequence of these AIDS avoidance behaviors was an increase in birth rates and abortion rates, with no corresponding increase in gonorrhea incidence.

VII. Underlying Mechanisms

What types of behavior changes could decrease the likelihood of AIDS infection but increase the likelihood of pregnancy and birth? One possibility is that women adopt condoms in favor of more effective contraceptives such as the Pill. Switching from effective contraception to condoms would decrease infection likelihood but increase pregnancy likelihood. Another possibility is that women respond to AIDS risk by limiting the number of sexual partners or choosing less risky, male partners (i.e., based on drug use, number of other partners). The likelihood of a birth may increase in this case if women with only one partner have sex more frequently, are less likely to use contraception, or are more likely to continue a pregnancy. Both mechanisms are consistent with the decrease in gonorrhea incidence: More condom use or fewer sexual partners would both limit the spread of STI.

To evaluate which mechanism is driving results, I make use of information from birth certificate records on mother's marital status and father's age. If the increase in births is primarily due to women opting into monogamous partnership, we might expect an increase in births to married women (either because of marriage following an unintended pregnancy, or because of women endogenously selecting into both marriage and childbearing in response to AIDS risk). If the increase in births is primarily due to women switching to condoms, we might expect an increase in births to single women. However, in evaluating potential mechanisms, it is necessary to distinguish between unmarried women who are single (i.e., without a long-term partner) and those who are unmarried but could still be using monogamy to avoid AIDS. The latter group is more appropriately grouped with married women in evaluating these mechanisms.

Assume that the father's information is more likely to be recorded on a birth record if he was the mother's only partner. Following this assumption, I split the sample into two groups. *Partnered* are those who are married as well as those who are unmarried but the birth record includes the father's age. *Single* women are those who are unmarried and the birth record is missing information on the father's age. Results showing the effect of AIDS

by the mother’s partnership status are presented in Table 6. I further disaggregate the data by race to evaluate the possibility of heterogeneity in AIDS avoidance behavior across the population.

Among partnered white women, I estimate a positive and statistically significant effect of 0.0397. This magnitude is similar to the main results presented in Table 2 and is driving the overall estimate, given that women in this group represent 73.8 percent of birth records. There is also evidence of an increase in births to partnered Black women, though the effect is small and insignificant, and a large increase in births to partnered women who are neither Black nor white. I interpret the increase in births to partnered women as evidence that a large share of women are responding to the risk of AIDS by having only one sexual partner. A similar pattern has been found in Brazil, where Hakak and Pereda (2021) show that women used marriage as a strategy to avoid AIDS between 1984 and 1991. Survey data further confirms that women limited the number of sexual partners in response to AIDS risk. According to the 1988 National Survey of Family Growth (NSFG), 5.3 percent of married women and 37.5 percent of unmarried women reported changing their behavior to avoid AIDS. Furthermore, 16 percent of sexually active, unmarried women specifically reported they “stopped having sex with more than one man” to avoid AIDS. Ceasing to have multiple partners was the most common way women reported adjusting their behavior to avoid AIDS (Mosher and Pratt 1993).

I find evidence of heterogeneous effects in single women. Among single white women, I find an increase in birth rates. This could be evidence of single white women switching to condoms. However, this effect is unlikely to explain the overall increase in births given the smaller magnitude of the estimated effect (0.0198), the small share of births to single white women, and the fact that this positive effect is fully offset by a negative effect on births to single Black women. Though insignificant, among single Black women I estimate an effect of -0.0261.

The insignificant effects among Black women could be due to fewer men available for

partnership given high incarceration rates of Black men. This would be consistent with the findings of Charles and Luoh (2010) showing that high incarceration rates among men decrease the likelihood of marriage. Similarly, Johnson and Raphael (2009) show that the high incarceration rate among Black men explained the majority of the disparity in AIDS incidence across Black and white women between 1982 and 1996. In combination with the findings of this paper, these results suggest that women primarily avoided AIDS by entering monogamous partnerships. However, in the absence of this option, women's ability to avoid AIDS was limited. This is evidenced by the results of Johnson and Raphael and the lack of a significant effect on birth rates for Black women as shown in Table 6. If Black women could effectively avoid AIDS using the other avoidance options described in Section 3, we would expect a negative effect on birth rates and smaller effect of male incarceration on AIDS in women. Instead, these results point to the relative importance of partnership decisions in women's choice set. While decisions about condoms and other contraceptive options could also be used to mitigate the combined risk of STI and pregnancy, these options could be less effective or inaccessible. For example, lower bargaining power can limit women's ability to convince a male partner to use a condom (Anderson 2018; Cassidy et al. 2021). Financial constraints and legal restrictions could limit access to effective contraceptives and abortion (Kearney and Levine 2009; Lindo and Pineda-Torres 2021). In contrast, women may have more autonomy and fewer constraints over partnership choices.

VIII. Conclusions and Policy Implication

Economic theory predicts that rational individuals will shift to “safer” sexual behaviors in response to increases in the cost of fertility (Becker and Lewis 1973; Willis 1973) and increases in the risk of STI (Posner 1993). When responding to STI risk, those safer choices could increase or decrease birth rates.

I show that the risk of AIDS led to an increase in pregnancy rates and birth rates. While prior research has shown that individual AIDS avoidance behaviors can affect AIDS

rates in the population, this is the first paper in the US context to relate AIDS avoidance behaviors and birth rates. My results are consistent with two possible behavioral changes: Adopting condoms in place of more effective contraception or decreasing the number of sexual partners. My analysis shows that the latter behavioral change is driving results. The majority of women who change their behavior opt to have only one sexual partner to protect from AIDS. I find that women mitigate the risk of AIDS at the expense of higher pregnancy likelihood. As a result, an unintended consequence of AIDS avoidance behaviors is an increase in birth rates.

By documenting an increase in birth rates, this paper contributes to the economics literature on both the effects of AIDS and STIs and the determinants of fertility. Empirical studies have validated the importance of economic cost considerations in determining sexual behavior, contraceptive choices, and fertility outcomes (Michael and Willis 1973; Bailey, Hershbein, and Miller 2012; Bailey, Guldi, and Hershbein 2013; Burke, Gong, and Jones 2015; Myers 2017). Within the STI literature, this work is closest to studies that examine behavioral changes in response to STI risk, such as Ahituv, Hotz, and Philipson (1996), Lakdawalla, Sood, and Goldman (2006), Francis (2008), Gertler, Shah, and Bertozzi (2005), Fortin (2015), Greenwood et al. (2019) and Hakak and Pereda (2021). Similar to this work are studies that examine the spillovers between fertility and STI risk, for example, Sen (2003), Klick and Stratmann (2008), Durrance (2013), Mulligan (2016), Buckles and Hungerman (2018), Mallatt (2019), and Willage (2020). In comparison with this paper, those studies focus on the reverse relationship by studying the effect of changes in the cost of pregnancy prevention on STI rates. I find that women adopt STI avoidance behaviors at the cost of increased pregnancy likelihood, and that as a result, STI risk has a positive effect on birth rates.

This paper also highlights that decisions about number of sexual partners are an important risk mitigation strategy for women. Number of partners interacts with both STI risk and pregnancy risk, and is likely endogenously chosen with contraceptive method and

frequency of sexual activity. However, despite the far-reaching consequences of this decision on birth rates, abortion rates, and the spread of STIs, women’s number of sexual partners is relatively understudied in economics.

Further research is needed to understand how partnership choices interact with STI-pregnancy trade-offs in a modern context. For example, expanded access to long-acting reversible contraceptives and emergency contraception could have spillover effects on both partnership decisions and STI incidence. Similarly, continuing restrictions on abortion access increase the health risks associated with pregnancy. In measuring the effects of these changes on behavior and health outcomes, the mediating choice of number of partners could lead to surprising findings. For example, my research suggests that decreases in the desirability of pregnancy due to abortion restrictions could also decrease the desirability of monogamous partnership. If this were the case, decreasing abortion access could lead to less monogamy and higher incidence of STIs.

The trade-offs between pregnancy prevention and STI protection are of particular importance for the development of screening guidelines for healthcare providers. Current screening guidelines promote an emphasis on condoms as “safe sex” that fails to account for women’s unique burden in avoiding both STI and pregnancy. For example, when health providers screen patients for risk factors, having only one sexual partner is viewed as an indicator for low risk of STI (Lee et al. 2016). However, my results suggest that these patients are at higher risk for unintended pregnancy. Health providers may want to target these patients for discussions of contraceptive options that are effective in preventing pregnancy with a high frequency of sexual activity. Similarly, health providers who prescribe highly effective methods of contraception for a patient may want to emphasize the importance of combining the method with condoms for continued STI protection. An approach to reproductive health that takes into account the trade-offs between STI protection and pregnancy prevention is able to address both of these potential spillover effects.

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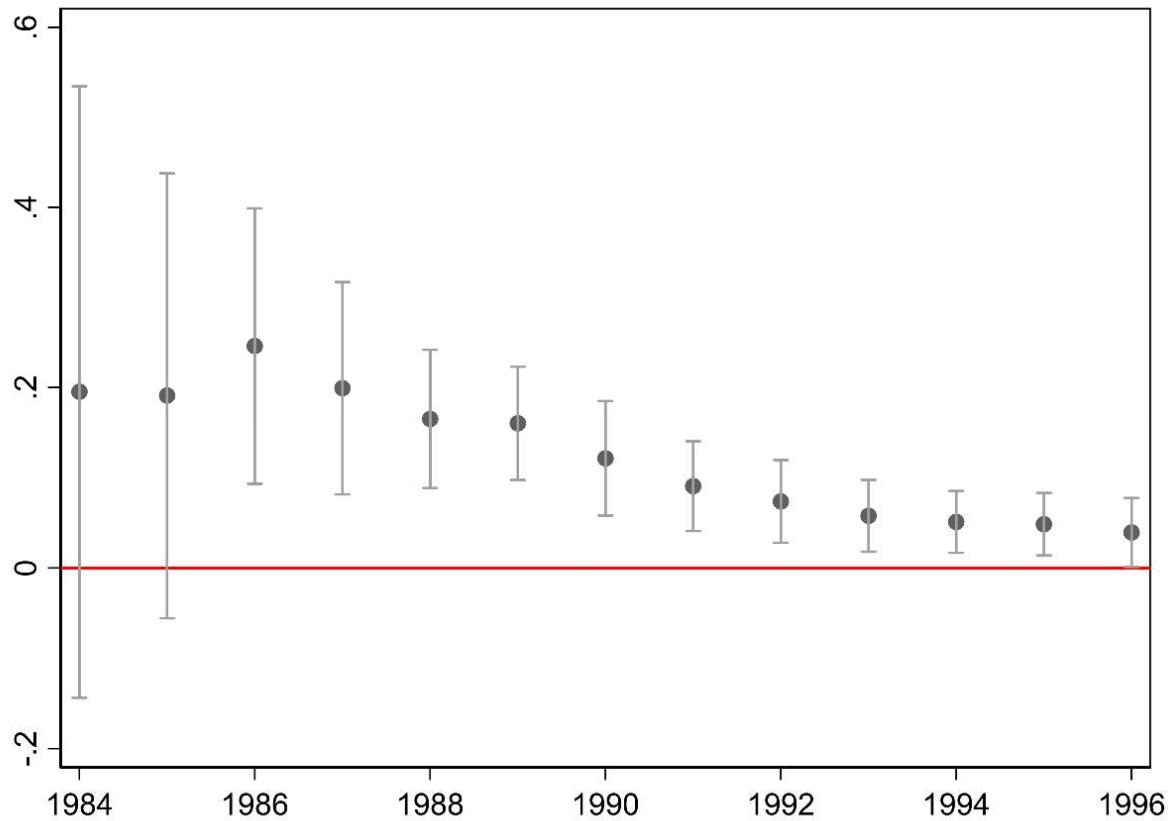


FIGURE 1: HETEROGENEOUS EFFECTS OVER TIME

Notes: This figure directly estimates heterogeneous effects over time using the primary specification described in equation (1) but allowing the effect of AIDS incidence in women to vary by year. All regressions are weighted by female population and include MSA and year fixed effects, as well as controls for the share of the female population of each race and the full set of additional controls described in section 5. Estimates for 1982 and 1983 are large but insignificant and are excluded from the figure but shown in Appendix Table A2. Robust standard errors are clustered at the MSA level. 95 percent confidence intervals are shown.

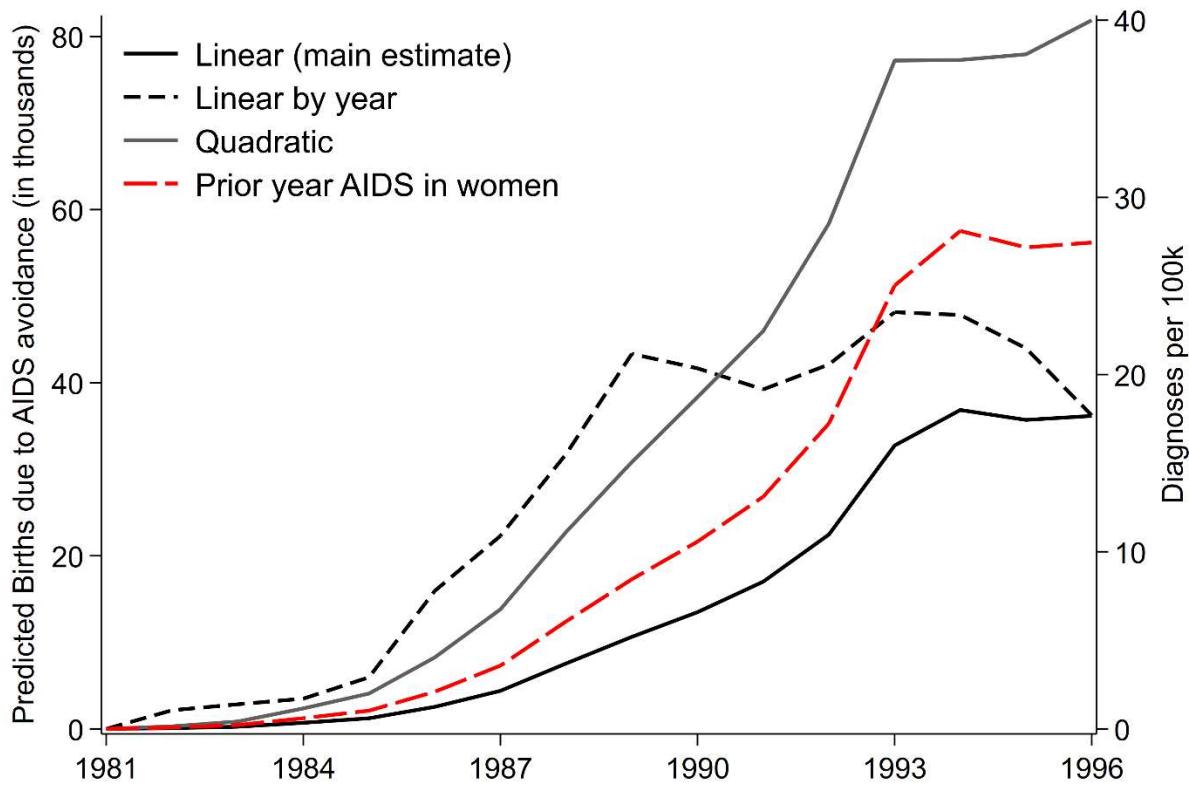


FIGURE 2: PREDICTED NET EFFECT OF AIDS ON BIRTH RATES BY YEAR

Notes: Using coefficient estimates from Table 1 column (3), Figure 1, and Table 1 column (6), I predict the net effect of AIDS incidence on birth rates in every year for the linear, linear-by-year, and quadratic specifications, respectively. To estimate the number of additional births due to AIDS avoidance, I compare the predicted number of births to a counterfactual prediction with AIDS in women equal to zero in every year.

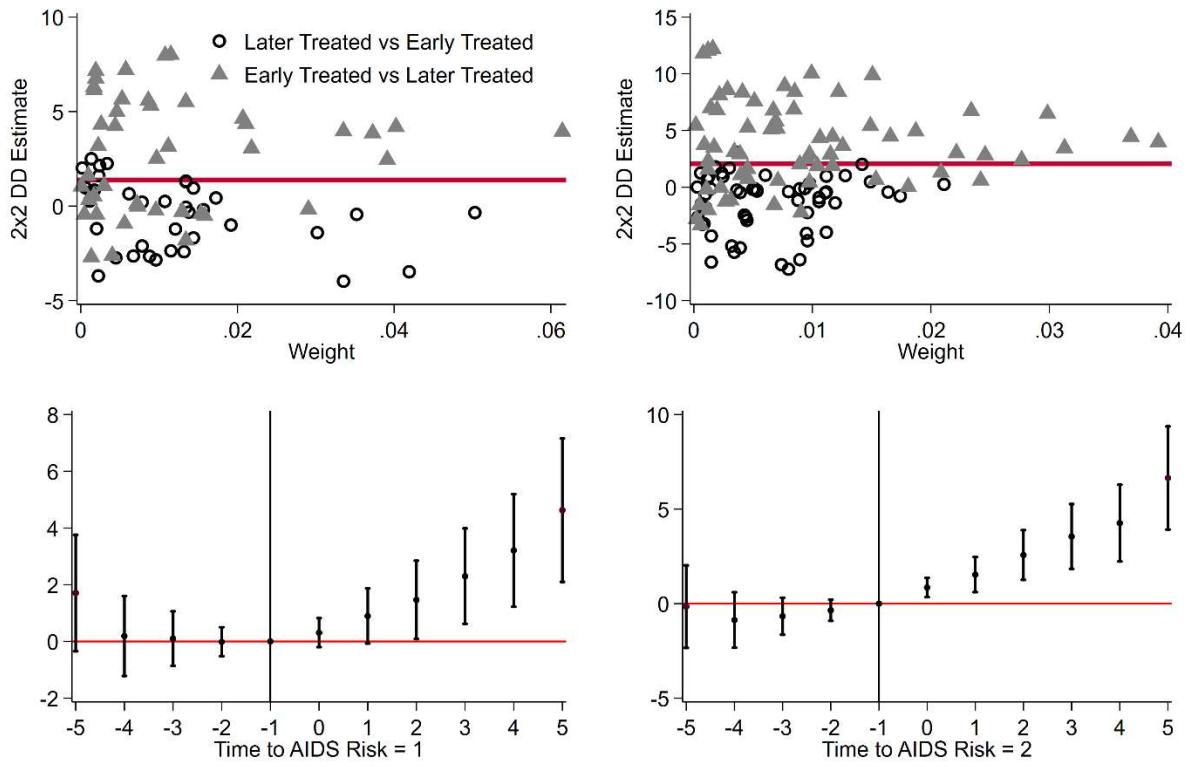


FIGURE 3: ROBUSTNESS TO DISCRETIZED AIDS INCIDENCE

Notes: This figure shows that results are robust to a discretized version of AIDS incidence, specifically whether AIDS incidence has reached 1 case per 100,000 women (column 1) or 2 cases per 100,000 women (column 2). The distribution of treatment years for these thresholds are shown in Appendix Figure A3. Row 1 shows the results of a Goodman-Bacon decomposition without controls and with the sample extended to 1975 to 1996 for more pre-treatment years. Row 2 shows the results of an event study specification with controls for female population shares by race and the sample extended to 1975 to 1996 for more pre-treatment years. 95 percent confidence intervals are shown. Standard errors are robust and clustered at the MSA level.

Table 1: Relationship Between AIDS Incidence, Perceived Risk, and Avoidance Opinions

Panel A: Unmarried Women Sample						
	Own Risk High		Monogamy		Condoms	
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS incidence	0.0202** (0.00887)	0.0200** (0.00782)	0.00827* (0.00460)	0.00856** (0.00356)	0.00372* (0.00202)	0.00381** (0.00179)
N	8,373	8,373	8,373	8,373	8,373	8,373
Outcome mean	0.0111		0.716		0.365	

Panel B: Unmarried Men Sample						
	Own Risk High		Monogamy		Condoms	
	(7)	(8)	(9)	(10)	(11)	(12)
AIDS incidence	0.0272 (0.0175)	0.0266 (0.0179)	-0.00341* (0.00202)	-0.00309 (0.00209)	-0.00419 (0.00348)	-0.00409 (0.00343)
N	8,300	8,300	8,300	8,300	8,300	8,300
Outcome mean	0.0151		0.750		0.412	
Region & year FE	X	X	X	X	X	X
Individual controls		X		X		X

Notes: This table shows the effect of regional AIDS incidence on perception of AIDS risk and mitigation strategies using data from NHIS supplement surveys in 1987, 1988, 1989, and 1993. Outcome variables include binary indicators for whether respondents rated their own risk of contracting AIDS as high and whether they perceived monogamy and condoms as very effective in preventing the transmission of AIDS. All regressions are logit and include region and year fixed effects. Individual controls include poverty level, race, and college education. Robust standard errors are clustered at the region level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 2: Effect of AIDS on Birth Rates

	Births per 1,000 women 20-44		
Panel A: Linear	(1)	(2)	(3)
AIDS in women	0.0629*** (0.0181)	0.0449** (0.0196)	0.0393** (0.0177)
Panel B: Quadratic	(4)	(5)	(6)
AIDS in women	0.146*** (0.0347)	0.153*** (0.0351)	0.135*** (0.0324)
AIDS in women ²	-0.000715*** (0.000239)	-0.000788*** (0.000201)	-0.000706*** (0.000213)
Share of N w/ marginal effect<0	0.008	0.010	0.010
N	1,632	1,632	1,632
Population weighted		X	X
Additional controls			X

Notes: This table shows the effect of prior year AIDS incidence on birth rates per 1,000 women aged 20-44. AIDS incidence is defined as AIDS diagnoses per 100,000 women 20-44. All regressions include MSA and year fixed effects, as well as controls for the share of the female population of each race. Panels A and B show results from linear and quadratic specifications, respectively. Panel B also indicates the share of observations in the sample for which the predicted marginal effect of one more AIDS case in women is negative. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 3: Effect of AIDS by Sexual Orientation

Births per 1,000 women 20-44	(1)	(2)	(3)
AIDS in women	0.0629*** (0.0181)	0.0449** (0.0196)	0.0393** (0.0177)
	(4)	(5)	(6)
AIDS in heterosexual men	0.0508*** (0.0134)	0.0344** (0.0139)	0.0322** (0.0126)
	(7)	(8)	(9)
AIDS in homosexual/bisexual men	-0.00311 (0.00894)	-0.00247 (0.0122)	-0.00906 (0.0131)
N	1,632	1,632	1,632
Population weighted		X	X
Additional controls			X

Notes: This table shows the effect of prior year AIDS incidence by sexual orientation on birth rates per 1,000 women aged 20-44. AIDS incidence is defined as AIDS diagnoses per 100,000 women or men 20-44. All regressions include MSA and year fixed effects, as well as controls for the share of the female population of each race. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 4: Effect of AIDS on Abortions and Gonorrhea

	Panel A: Abortions per 1,000 women age 15-44		
	(1)	(2)	(3)
AIDS in women	0.0338*** (0.00719)	0.0359*** (0.00717)	0.0301** (0.0134)
N	1,020	1,020	1,020
	Panel B: Gonorrhea diagnoses per 100,000 women		
	(4)	(5)	(6)
AIDS in women	-0.545 (0.360)	-0.213 (0.331)	-0.347 (0.327)
N	1,326	1,326	1,326
Population weighted		X	X
Additional controls			X

Notes: This table shows the effect of prior year AIDS incidence on abortion rates (Panel A) and gonorrhea incidence (Panel B). All regressions include MSA and year fixed effects, as well as controls for the share of the female population of each race. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 5: Robustness Test: 10-year lag between HIV Infection and AIDS diagnosis

	1971-1986 (1)	1981-1996 (2)	1981-1996 (3)
Births per 1,000 women 20-44			
AIDS in women 10 years future	0.00257 (0.0211)		
Birth rates 10 years past		-0.128* (0.0723)	-0.147* (0.0763)
AIDS in women prior year			0.0434** (0.0193)
N	1,632	1,632	1,632
Population weighted	X	X	X
Additional controls		X	X

Notes: This table evaluates whether the positive relationship between AIDS risk and birth rates is driven by higher level of sexual activity in some MSAs by exploiting the 10-year incubation period of HIV infection. Column 1 shows that AIDS in women 10 years in the future is not predictive of birth rates between 1971 and 1986. Column 2 shows that birth rates 10 years prior are negatively associated with birth rates between 1981 and 1996. Controlling for sexual behavior at the time of HIV exposure using birth rates ten years prior, I further find that the relationship between prior year's AIDS in women and birth rates remains positive (Column 3). All regressions include MSA and year fixed effects, as well as controls for the share of the female population of each race. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 6: Heterogeneous Effects of AIDS on Births by Partnership Status

	Births per 1,000 women 20-44					
	White		Black		Other	
	Partnered	Single	Partnered	Single	Partnered	Single
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS in women	0.0397** (0.0191)	0.0198*** (0.00618)	0.00153 (0.0403)	-0.0261 (0.0266)	0.199** (0.0816)	0.00260 (0.00976)
N	1,632	1,632	1,632	1,632	1,632	1,632
Share of total births	0.738	0.0500	0.107	0.0556	0.0457	0.00313
Outcome mean	63.79	4.344	50.68	23.58	67.76	5.161

Notes: This table shows the effect of AIDS risk on births by mother's partnership status. Partnered women are those who are married or for whom the birth record includes information on the father's age. Single women are those who are unmarried and the birth record lacks information on the father's age. All regressions are weighted by female population and include MSA and year fixed effects, as well as controls for the share of the female population of each race and the full set of additional controls described in section 5. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Appendix A: Additional Figures and Tables

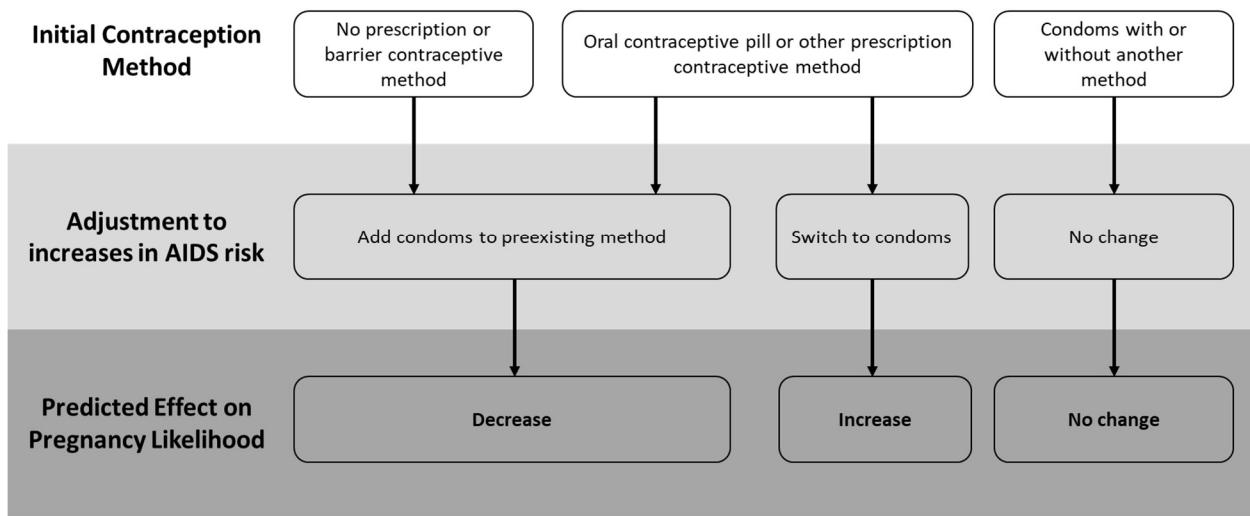


FIGURE A1: THEORETICAL EFFECTS OF CONTRACEPTIVE CHANGE

Notes: This figure summarizes the possible theoretical effects of contraceptive change to avoid AIDS, holding constant other choices about sexual behavior. For women not currently using condoms, adding condoms to existing methods should improve protection from pregnancy and decrease pregnancy likelihood. However, switching from a more effective method to condoms would increase the chance of pregnancy. See Figure A2 for a summary of theoretical effects of changes in other sexual behaviors.

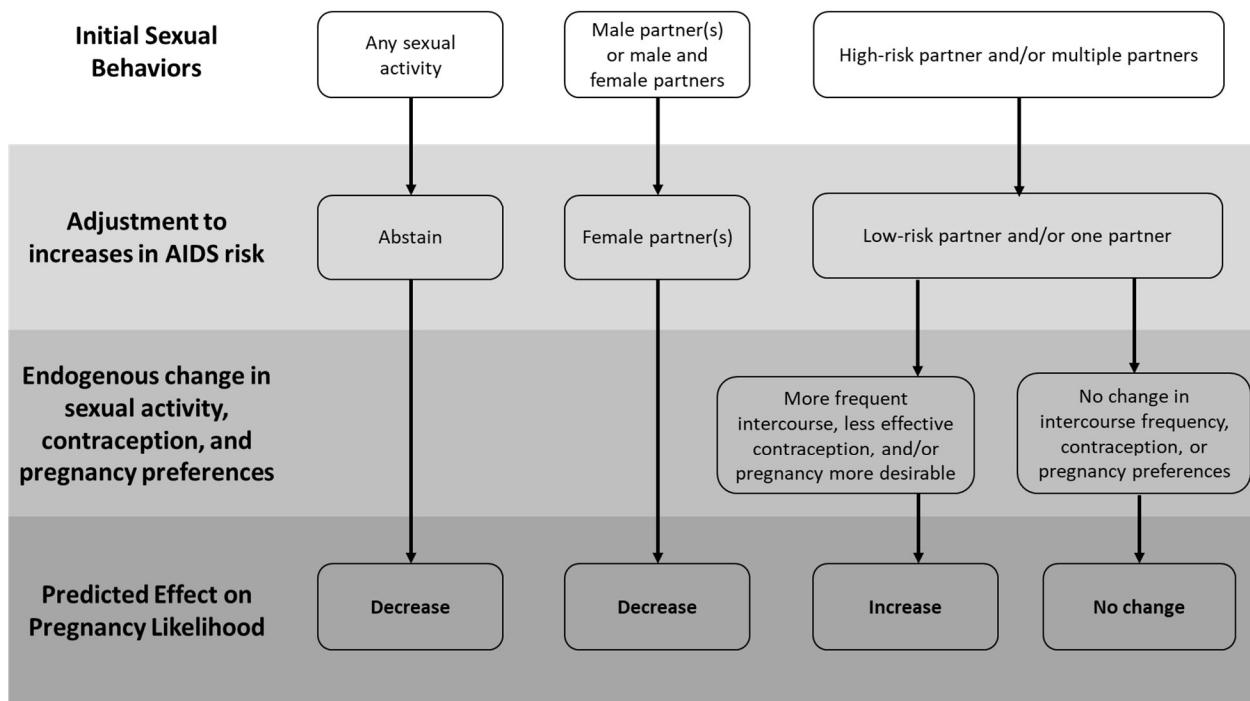


FIGURE A2: THEORETICAL EFFECTS OF BEHAVIOR CHANGE

Notes: This figure summarizes the possible theoretical effects of changes in sexual behavior to avoid AIDS. At the extensive margin, abstaining from any sexual activity to avoid AIDS eliminates any chance of pregnancy. Among women with any male partners, substituting toward female partners would decrease the likelihood of pregnancy. Among women with partners at high risk of AIDS infection or with multiple partners, switching to lower risk partners or to a monogamous partnership will increase pregnancy likelihood if this decision is endogenous to other choices about sexual behaviors. Specifically, we might expect that switching to a lower risk partner or a monogamous partner might also result in more frequent sexual intercourse, less effective or less consistent contraceptive use, or a higher desirability of pregnancy and childbirth.

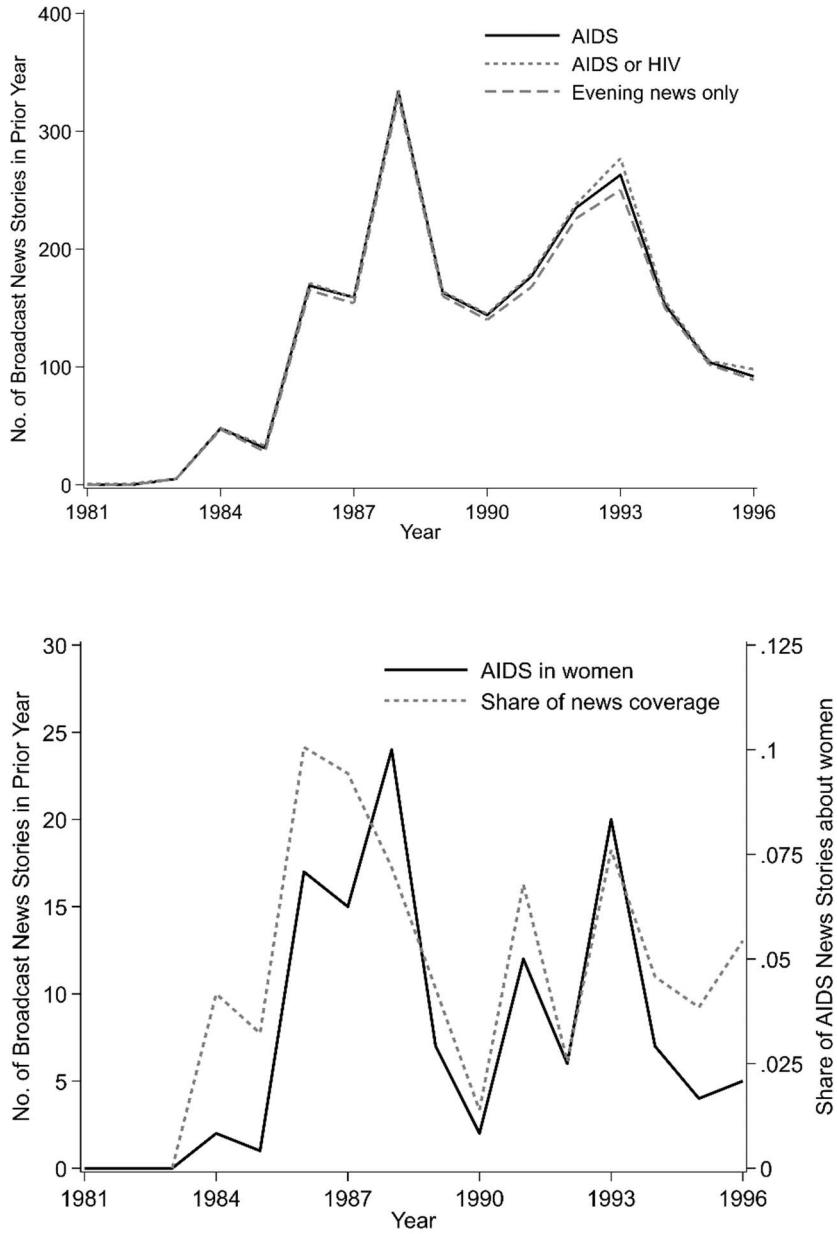


FIGURE A3: NATIONAL BROADCAST NEWS COVERAGE OF THE AIDS EPIDEMIC

Notes: This figure shows trends in national broadcast news coverage of the AIDS epidemic. I use data from the Vanderbilt TV News Abstracts as compiled by Sood and Laohaprapanon (2020). Panel A shows the number of news broadcasts per year for which the abstract includes “AIDS” or “HIV”, and for which the coverage was the typical evening news segment. Panel B shows the number and share of these broadcast segments for which the abstract also mentions “female”, “women”, “straight”, or “heterosexual.” I lag counts by one year to mirror the definition of primary treatment variable as prior year AIDS incidence.

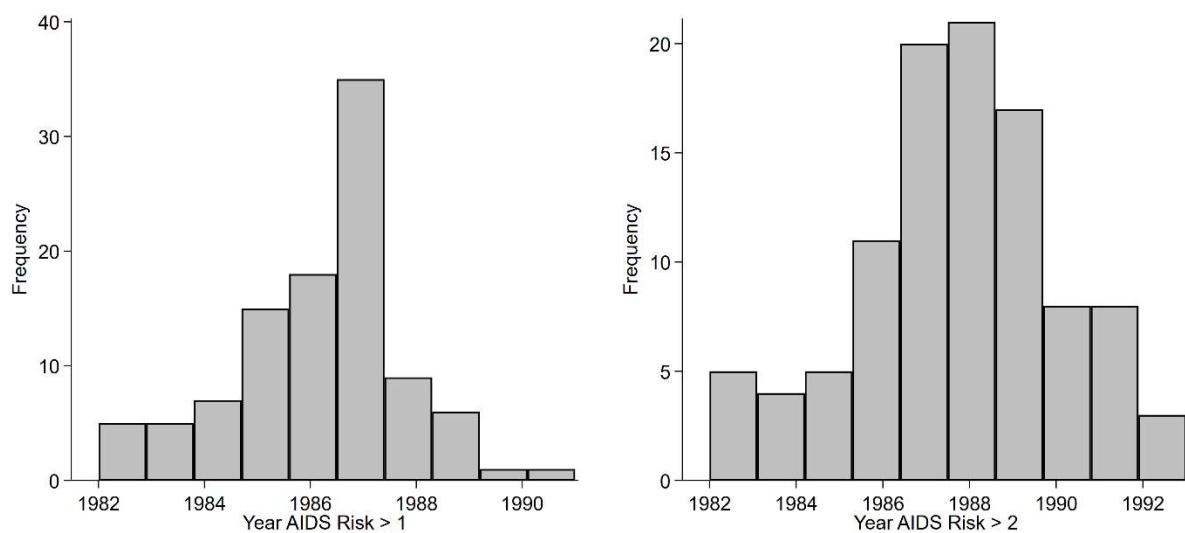


FIGURE A4: DISTRIBUTION OF TREATMENT YEARS FOR DISCRETIZED AIDS INCIDENCE

Notes: This figure shows the distribution of treatment years when AIDS incidence has reached 1 case per 100,000 women (column 1) or 2 cases per 100,000 women (column 2). These thresholds are used for the analysis in Figure 3.

Table A1: Summary Statistics

	Mean	SD	Min	Max	N
Births per 1,000 women age 20-44	69.21	8.77	48.78	125.85	1,632
Gonorrhea diagnoses per 100,000 women	216.92	119.27	9.17	664.71	1,326
Abortions per 1,000 women age 15-44	29.38	10.65	7.50	56.57	1,020
<i>AIDS incidence in previous year...</i>					
Total 20-44	37.15	47.32	0	344.38	1,632
Women 20-44	11.19	22.30	0	159.25	1,632
Heterosexual Men 20-44	18.86	35.49	0	218.06	1,632
Homosexual and Bisexual Men 20-44	44.49	51.73	0	575.99	1,632

Notes: This table presents descriptive statistics for the primary dependent and explanatory variables. The unit of observation is at the MSA-year level. Birth rates are calculated as live births per 1,000 women. AIDS incidence is defined as number of new AIDS diagnoses per year per 100,000 people. Summary statistics are weighted by MSA-year female population size.

Table A2: Heterogeneity in Effects Over Time**Births per 1,000 Women 20-44**

AIDS in women*	(1)	
1982	0.961	(1.286)
1983	0.448	(0.518)
1984	0.195	(0.171)
1985	0.191	(0.124)
1986	0.246***	(0.0770)
1987	0.199***	(0.0593)
1988	0.165***	(0.0387)
1989	0.160***	(0.0317)
1990	0.121***	(0.0320)
1991	0.0907***	(0.0250)
1992	0.0737***	(0.0231)
1993	0.0578***	(0.0201)
1994	0.0511***	(0.0172)
1995	0.0484***	(0.0174)
1996	0.0394**	(0.0192)
N	1,632	
Population Weighted	X	
Additional Controls	X	

Notes: This table shows coefficient estimates for the regression analysis presented in Figure 1. I directly estimate heterogeneous effects over time using the primary specification described in equation (1) but allowing the effect of AIDS incidence in women to vary by year. All regressions are weighted by female population and include MSA and year fixed effects, as well as controls for the share of the female population of each race and the full set of additional controls described in section 5. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A3: Robustness to Inclusion of Linear Time Trends

	Births per 1,000 women 20-44				
	(1) 70-96	(2) 70-95	(3) 70-94	(4) 70-93	(5) 70-92
AIDS in women	0.0294 (0.0200)	0.0366* (0.0192)	0.0437** (0.0193)	0.0611** (0.0236)	0.0957*** (0.0283)
Observations	2,754	2,652	2,550	2,448	2,346

Notes: This table shows that results from equation (1) and Table (2) are robust to the inclusion of MSA-specific linear time trends. All regressions are weighted by female population and include MSA and year fixed effects, as well as controls for the share of the female population of each race. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A4: Robustness to Sample Definition and Specification

Panel A: Births per 1,000 women 20-44					
	(1)	(2)	(3)	(4)	(5)
AIDS in women	0.0622*** (0.0187)	0.0422** (0.0191)	0.0339* (0.0183)	0.000694** (0.000270)	0.000528** (0.000254)
N	1,392	1,392	1,392	1,632	1,632

Panel B: Abortions per 1,000 women 15-44					
	(6)	(7)	(8)	(9)	(10)
AIDS in women	0.0372*** (0.00728)	0.0404*** (0.00709)	0.0413*** (0.00909)	0.00159*** (0.000178)	0.00139*** (0.000342)
N	870	870	870	1,020	1,020

Panel C: Gonorrhea Diagnoses per 100,000 women					
	(11)	(12)	(13)	(14)	(15)
AIDS in women	-0.476 (0.358)	-0.132 (0.304)	-0.0608 (0.266)	0.000282 (0.00159)	-0.000133 (0.000793)
N	1,131	1,131	1,131	1,326	1,326
Single state	X	X	X		
Population weighted		X	X	X	X
Additional controls			X		X
Poisson				X	X

Notes: This table shows that the primary estimates presented in Tables 2 and 3 are robust to limiting the sample of MSAs to those included within a single state and to using a Poisson regression as opposed to an OLS regression. Given that many of the control variables, abortion rates, and gonorrhea rates are measured at the state level, limiting the sample to MSAs that do not cross state boundaries diminishes concerns about measurement error in the construction of MSA-level variables. All regressions include MSA and year fixed effects, as well as controls for the share of the female population of each race. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance is denoted by * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Appendix B: Data Sources and Sample Construction

AIDS Public Information Data Set

The AIDS Public Information Data Set (APIDS) contains MSA-level annual data from 1981 to 2002 and is publicly available on CDC Wonder. I use APIDS for counts of AIDS diagnoses by year diagnosed among women aged 20-44, as well as heterosexual men aged 20-44, and homosexual and bisexual men aged 20-44. As the AIDS epidemic developed, the CDC expanded the criteria for an AIDS diagnosis. I use AIDS cases diagnoses under any criteria for my main analysis.

US Department of Health and Human Services (US DHHS), Centers for Disease Control and Prevention (CDC), National Center for HIV, STD and TB Prevention (NCHSTP). 2005. "AIDS Public Information Data Set (APIDS) US Surveillance Data for 1981-2002." CDC WONDER On-line Database. <https://wonder.cdc.gov/aidspublic.html>

Natality Detail File

I use restricted geographic data from the National Vital Statistics System to create annual counts of births by MSA. I also make use of information on birth certificates to create counts of births by mother's age, mother's race, and mother's marital status.

US Department of Health and Human Services (US DHHS). National Center for Health Statistics (NCHS). 2002. 'Restricted-Use Natality Detail File, 1969-2001.'

Population Data

To create measures of AIDS incidence and birth rates, I use population data from the Survey of Epidemiology and End Results (SEER) as made available online by the National Bureau of Economic Research. Specifically, I use adjusted, county-level data disaggregated by 19 age groups and white, Black, or other races.

Survey of Epidemiology and End Results. 2020. 'U.S. County-Level Population Data , 1969-2018.' National Bureau of Economics Research Data [distributor], <https://data.nber.org/seer-pop/uswbo19agesadj.dta.zip>.

Gonorrhea Surveillance Data

Information on gonorrhea incidence among women is publicly available via CDC WONDER's Sexually Transmitted Disease Morbidity Data. These data are available at the state level for the years 1984-2014. I merge this data with AIDS incidence data. For MSAs that cross state boundaries, I calculate the share of the MSA population in each state using SEER data, and then calculate the corresponding weighted average of gonorrhea incidence in each MSA.

US Department of Health and Human Services (US DHHS), Centers for Disease Control and Prevention (CDC), National Center for HIV, STD and TB Prevention (NCHSTP). 2015. 'Sexually Transmitted Disease Morbidity 1984 - 2014 by Gender.' CDC WONDER On-line Database. <https://wonder.cdc.gov/std.html>

Crack Cocaine Index

I use the crack cocaine index developed by Fryer et al. 2005. The crack index is calculated at the city and state level and proxies the spatial and temporal patterns in the crack epidemic using a variety of measures including arrests, emergency room visits, overdose deaths, and news coverage. For each MSA, I use the value for the largest city in that MSA. For MSAs that lack city-level crack index data, I use state values.

Fryer, Roland G, Paul S Heaton, Steven D Levitt, and Kevin Murphy. 2005. Measuring the Impact of Crack Cocaine. Working Paper, Working Paper Series 11318. National Bureau of Economic Research.<https://scholar.harvard.edu/fryer/publications/measuring-crack-cocaine-and-its-impact>

National Prison Statistics

The National Prison Statistics (NPS) data, available on ICPSR, details counts of persons incarcerated in state and federal prisons in each year by state, race, and sex. I merge this data with MSA data on birth rates and AIDS risk. For MSAs that cross state boundaries, I calculate the share of the MSA population in each state using SEER data, and then calculate the corresponding weighted average of incarceration rates in each MSA. Incarceration rates are calculated per 1,000 people.

US Bureau of Justice Statistics (US BJS). 2020. ‘National Prisoner Statistics, [United States], 1978-2018.’ ICPSR - Inter-University Consortium for Political and Social Research [distributor]. Ann Arbor, MI. <https://doi.org/10.3886/ICPSR37639.v1>

National Health Interview Survey AIDS Supplement

The National Health Interview (NHIS) AIDS Supplement was conducted every year between 1987 and 1995 and asks respondents about their own perceived risk of getting AIDS, as well as their AIDS knowledge. The publicly available data includes demographic information as well as geographic information at the census region level. Questions about the effectiveness of monogamy in preventing AIDS transmission were asked in 1987, 1988, 1989, and 1993. I restrict my sample to these years and to unmarried men and women under age 45.

US Department of Health and Human Services (US DHHS). Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS). 1997. ‘National Health Interview Survey, 1987-1996: AIDS Knowledge and Attitudes Supplement.’ ICPSR - Inter-University Consortium for Political and Social Research [distributor]. Ann Arbor, MI. <http://doi.org/10.3886/ICPSR09271.v1>

Abortion Rate

Data on abortions per 1,000 women aged 15-44 is from the Guttmacher Institute's Data Center. Data are state level (based on state of occurrence) and available for the years 1981, 1982, 1984, 1985, 1987, 1988, 1991, 1992, 1995, and 1996. For MSAs that cross state boundaries, I calculate the share of the MSA population in each state using SEER data, and then calculate the corresponding weighted average of abortion rates in each MSA.

Jones, Rachel K, and Kathryn Kooistra. 2011. "Abortion Incidence and Access to Services in the United States, 2008." *Perspectives on Sexual and Reproductive Health* 43 (1): 41–50.
<https://data.guttmacher.org/states/>

Unemployment Rates

State-level unemployment rate data is from the US Bureau of Labor Statistics, Local Area Unemployment Statistics. For MSAs that cross state boundaries, I calculate the share of the MSA population in each state using SEER data, and then calculate the corresponding weighted average of unemployment rates in each MSA.

US Bureau of Labor Statistics. 2022. "Local Area Unemployment Statistics 1980-1995."
<https://www.bls.gov/lau/>

Welfare Benefits

Welfare benefits are from Moffit et al. (2020) and are calculated as the maximum welfare benefit for a family of 4 in 1996 dollars. I calculate the share of the MSA population in each state using SEER data, and then calculate the corresponding weighted average of monthly welfare benefits in each MSA.

Moffitt, Robert A, Brian J Phelan, and Anne E Winkler. 2020. "Welfare rules, incentives, and family structure." *Journal of Human Resources* 55 (1): 1–42.

<http://www.econ2.jhu.edu/people/moffitt/datasets.html>

Medicaid Coverage of Abortion

Data on whether the state Medicaid program covers abortion is from Myers and Ladd (2020), as compiled from two sources:

Guttmacher Institute. 2016. "State funding of abortion under Medicaid, as of September 1, 2016" State policies in brief.

Jon Merz, Catherine Jackson, and Jacob Klerman. 1995. "A review of abortion policy: Legality, Medicaid funding, and parental involvement laws, 1967-1994." *Women's Rights Law Reporter* 17(1).

Myers, Caitlin, and Daniel Ladd. 2020. "Did parental involvement laws grow teeth? The effects of state restrictions on minors' access to abortion." *Journal of Health Economics*. 71:102302.
<https://www.openicpsr.org/openicpsr/project/136601/>

Poverty Rates

County level poverty rates are computed by the US Census Bureau using decennial census data. I aggregate county level rates to the MSA level and interpolate between decennial years.

US Census Bureau. 2010. "Historical County Level Poverty Estimates 1960-2000."
<https://www.census.gov/library/visualizations/time-series/demo/census-poverty-tool.html>

Education Levels

County level education levels are computed by the Economic Research Service at the United States Department of Agriculture using decennial census data. I aggregate county level rates to the MSA level and interpolate between decennial years.

USDA. 2022. US Department of Agriculture, Economic Research Service. ‘Educational attainment for adults age 25 and older for the U.S., States, and counties, 1970–2020.’
<https://www.ers.usda.gov/data-products/county-level-data-sets/county-level-data-sets-download-data/>