

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 and 1990s on births and abortions. I show that the AIDS epidemic increased the birth rate by 0.55 percent and the abortion rate by 1.77 percent. Two underlying mechanisms explain the increase in pregnancies. Some women opted into monogamous partnerships in response to the AIDS epidemic, with a corresponding increase in the marriage rate and improvement in infant health. Others switched from prescription contraceptive methods to condoms. These behavior changes lowered the incidence of other sexually transmitted infections, but increased both planned and unplanned pregnancies.

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 or view pregnancy as more desirable.

I estimate the effect of increases in STI risk and resulting behavioral changes 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 risk 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 pre-existing trends in birth rates.

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 0.55 percent due to women adopting behaviors associated with lower risk of contracting HIV/AIDS, for a total of 191,776 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 a corresponding decrease in the incidence of other STIs. 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 in part due to women entering monogamous partnerships to lower their risk of contracting HIV/AIDS. The increase in births is accompanied by an increase in marriages and an improvement in infant health as measured by birth weight and prenatal care. These results suggest that some women endogenously choose both monogamy and pregnancy in response to the AIDS epidemic. This mechanism is consistent with survey data showing that 16 percent of unmarried women decided to stop having sex with more than one man in response to the AIDS epidemic, and that this was the most commonly reported behavioral change (Mosher and Pratt, 1993). The increase in abortions may be the result of unplanned pregnancies in monogamous partnerships or of women switching from more effective contraceptives to condoms to lower their risk of contracting HIV/AIDS. There is also evidence of this in survey data – between 1988 and 1995 decreases in use of the oral contraceptive pill were fully offset by increases in condom use (Piccinino and Mosher, 1998).

Previous studies have shown that people adjusted their behavior in response to the AIDS epidemic using both contraceptive technology and choices about sexual partners. 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. 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 a 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).¹ These studies are in part motivated by studying the effects of higher child mortality rates on fertility. This mechanism is unlikely to explain findings in the U.S. context, given the much lower child mortality rate overall and the very low incidence of AIDS in children in the U.S. this time period. However, my results do correspond to those of Duflo, Dupas, and Kremer (2015), which find that pregnancy and STI in adolescent girls in Kenya are not determined by unprotected sex, but by choices over casual versus committed relationships.

Studies in developing countries also document the importance of bargaining power in women's ability to lower their risk of contracting 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 shows that condom use is lower in households with lower female bargaining power. It is unclear if these results would apply in the U.S. context, particularly given differences in the accessibility of contraceptives other than condoms. However, there is existing evidence that bargaining power in sexual partnerships affects women in the U.S.: Akerlof, Yellen, and Katz (1996) argue that greater availability of contraception and abortion decreased women's bargaining power in marriage decisions.

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 overall results are driven by an increase in births to white women and 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

1. Fortson (2009) uses data from 12 countries in sub-Saharan Africa and finds that regional HIV prevalence has little effect on total fertility rates. Karlsson and Pichler (2015) uses data on three countries in sub-Saharan Africa and also finds insignificant effects on birth rates. In contrast, Chin and Wilson (2018) relies on an instrumental variable approach to estimate the effect of disease risk on fertility, relying on distance to the origin of the pandemic as an instrument, and finds an increase in fertility. Kalemli-Ozcan and Turan (2011) also finds a positive effect of HIV prevalence on fertility when focusing on South Africa.

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 may have few other accessible options available to lower their risk of contracting an STI.

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 address 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. Background

This paper focuses theoretically and empirically on women's decisions in the context of an STI-pregnancy trade-off. This is a simplification, since consensual sexual contact requires bilateral decision-making and results may also be informative on men's behavior. However, there are a number of theoretical and empirical reasons for focusing on women's decisions in this context. The economics literature documents women's greater burden and costs associated with pregnancy and raising children (Miller, 2011; Angelov, Johansson, and Lindahl, 2016), such that the risk and cost of pregnancy may be more salient to women. The risk of STI might also be higher for straight women than straight men: Women are more likely than men to contract HIV from an opposite sex partner (Nicolosi et al., 1994). The choice set of options to lower both STI risk and likelihood of pregnancy may also differ across men and women. For example, prescription contraceptives such as the oral contraceptive pill are in women's choice set, but not directly in men's choice set. Similarly, the costs and

benefits associated with male condoms might differ across men and women, such that women have lesser control over condom use. Empirically, this paper relies primarily on data sources that are more informative on women's behavior than men's, such as birth rates, abortion rates, and data from surveys that exclusively interviewed women. Finally, as I describe in the next section, public health messaging from the 1980s that was targeted at women often had a dual focus on HIV/AIDS and pregnancy.

A. Women and 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 and 1989).

Evidence suggests that public health fears of AIDS spreading rapidly via opposite-sex contact influenced knowledge and behavior in response to the AIDS epidemic. 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). I provide empirical evidence (in Section 3 and Table 1) that the risk of AIDS was salient to women. Women who lived in areas with higher AIDS incidence were more likely to report that they were at high risk of contracting AIDS.

The AIDS epidemic created variation that can be used to study the relationship between STI risk and birth rates. The nature in which HIV/AIDS spread and was initially diagnosed across the United States was plausibly exogenous with respect to pre-existing trends in birth rates. Initially, HIV/AIDS spread from city to city, with cases concentrated among men with same-sex partners and IV drug users (Taylor, 1989; Lam, Fan, and Liu, 1996; Steinberg and Fleming, 2000). Further, newly diagnosed AIDS cases were not reflective of the current rate at which HIV was spreading. This is due to the 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 and perceptions of risk were not driven by current behaviors, but behaviors from as much as 10 years prior. The emergence of HIV/AIDS created a large increase in the cost of contracting an STI: As of January 1991, over half of AIDS patients died within two years of diagnosis (CDC, 1991). In response to this new health risk, 22 percent of women reported changing their sexual behavior or using condoms to lower their risk of contracting HIV/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. Data

I estimate the 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.

A. AIDS Incidence as a Proxy for Risk

Since an individual's risk of contracting AIDS is unobservable, I use AIDS incidence in the MSA of residence as a proxy for perception of AIDS risk in the population. AIDS incidence is defined as the number of new AIDS diagnoses in people aged 20-44 per year per 100,000 people of the same age group. By using AIDS incidence as a proxy for perception of risk, I capture both increases in perceived risk due to higher underlying disease incidence as well as increases in perceived risk due to greater awareness and testing rates. For example, Cardazzi, Martin, and Rodriguez (2021) show that Magic Johnson's November 1991 announcement that he contracted HIV resulted in 800 new AIDS diagnoses in men due to increases in testing. Though underlying disease incidence is unchanged in this case, the higher AIDS incidence still reflects an increase in awareness and perception of risk that could affect future behavior, yet remains unrelated to recent sexual behavior. Further, as a measure of the risk of encountering a sexual partner with HIV/AIDS, AIDS incidence is a closer proxy than AIDS mortality. While HIV incidence would be the most accurate measure of risk, data is unavailable for this time period.²

2. Though HIV testing becomes available beginning in 1985, the CDC HIV/AIDS Surveillance Reports did not include information on HIV incidence until 1993. HIV incidence data is also not available on CDC Wonder.

To demonstrate that AIDS incidence is a reasonable proxy for AIDS risk, I use regional data from National Health Interview Surveys (NHIS) that asked respondents to rate their chances of contracting HIV/AIDS (US DHHS, 1997). I regress respondents' perceived risk of contracting AIDS on regional AIDS incidence. Given the much higher incidence of AIDS in men, it could be that local AIDS incidence is only predictive of men's perception of risk and not women's. To test this, I disaggregate the sample by sex. 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. This is consistent with the aforementioned anecdotal evidence from *The New York Times* that suggests women were risk averse to contracting HIV/AIDS, despite the higher incidence of AIDS among men. I find no statistically significant effect of AIDS incidence on unmarried men's perceptions; however, the point estimate is similar in magnitude to estimates for women. Given the lower incidence of AIDS in women (see Appendix Table A1), it might seem unexpected that local AIDS incidence is predictive of women's perception of risk. However, women are more likely than men to contract HIV from an opposite sex partner (Nicolosi et al., 1994). Further, the high case-fatality rate suggests that the expected cost of contracting HIV is high even if the probability of infection is low. However, this result could also indicate that women overestimated their risk of contracting HIV/AIDS. This would be consistent with existing literature showing people tend to overestimate small risks and that women are more likely than men to adopt behaviors that lower health risks (Hakes and Viscusi, 2004; Hersch, 1996). Regardless, results from Table 1 indicate that local AIDS incidence was salient to women and related to perceptions of risk.

B. Sample Construction

Using APIDS data, I create a panel of AIDS incidence across 102 MSAs from 1981 to 1996.³ I define AIDS risk for women as new AIDS diagnoses in the previous year in women aged 20-44 per 100,000 women aged 20-44 in the population. 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 20-44.⁴

I summarize this data in Figure 1 by showing each MSA's change in birth rates from 1981 to 1996 versus the average AIDS incidence in women in each MSA over the sample period. There is a positive relationship between birth rates and AIDS incidence. This positive relationship is not driven by large MSAs, nor is it sensitive to excluding MSAs with very high AIDS incidence.⁵

IV. Theoretical Effect of AIDS Risk on Births

This section describes the underlying mechanisms that could produce a positive relationship between local AIDS incidence and birth rates, as indicated in Figure 1. In response to increases in AIDS risk, women may choose to change their sexual behavior or adopt condoms as a contraceptive method. Data from the National Survey of Family Growth (NSFG) in 1988 and 1990 show that behavior change was the more common choice. 37.5 percent of unmarried women said they had changed their sexual behavior in response to the AIDS epidemic, compared to only 15.6 percent who said they always used condoms to lower their

3. 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.

4. Descriptive statistics for the panel are presented in Appendix Table A1. Geographic variation over time in AIDS incidence in women and birth rates is visualized in Appendix Figure A1.

5. Figure 1 shows that New York City MSA is unique both in its high AIDS incidence and large population. Throughout the paper, I test the sensitivity of results to excluding New York City from the sample.

risk of contracting HIV/AIDS. The most commonly reported sexual behavior change was to "stop having sex with more than one man," and was reported by 16 percent of unmarried women (Mosher and Pratt, 1993).

NSFG data on reported behaviors are consistent with data from the NHIS on perceptions about strategies to avoid the transmission of HIV/AIDS. Table 1 shows that both men and women view monogamy as more effective than condoms in preventing the transmission of HIV/AIDS: 71.6 percent of women and 75 percent of men believe monogamy is very effective in preventing transmission, compared to 36.5 percent and 41.2 percent of women and men, respectively, who view condoms as very effective. However, though both women and men view monogamy as more effective than condoms on average, it could be that increasing awareness of the AIDS epidemic shifted perceptions in favor of condoms. This might occur if public health campaigns in response to rising incidence focused on increasing condom use. If this were the case, then increases in condom use or perceived effectiveness could be driven by MSAs with higher AIDS incidence while MSAs with lower AIDS incidence experienced increases in monogamy. However, I find that higher regional AIDS incidence is associated with higher perceived effectiveness of both monogamy and condoms among unmarried women. However, in men I find that regional AIDS incidence is negative but insignificantly related to perceptions of monogamy and condoms. It is unclear what mechanism would produce negative coefficients for men, but the differing effects on men and women further motivate this paper's focus on women's decisions, as men may respond differently to increases in STI risk.

Given evidence that both monogamy and condoms were reported as strategies to decrease the risk of contracting HIV/AIDS, though with a larger share the population preferring monogamy, how might these two behavioral changes produce an increase in birth rates? Upon entering a monogamous, opposite-sex partnership, likelihood of pregnancy could increase if there is less incentive to use consistent contraception due to the lower risk of STI, or if frequency of sexual activity increases. It could also be that having a child becomes

more desirable with a sole, committed partner, such that monogamy and pregnancy are endogenously chosen. In the latter case, additional births are more likely to be “wanted” or intended pregnancies. In the former, pregnancies may be unplanned.

Pregnancy rates could also increase with higher condom use due to differential rates of contraceptive failure across methods. 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 pregnancy: The Pill has a typical use failure rate of 9 percent (Trussell, 2004). If many women mitigated the risk of contracting HIV/AIDS by switching from the Pill to condoms, then the pregnancy rate in the population would increase. There is also evidence of this in survey data. Between 1988 and 1995, the share of women using the Pill decreased from 30.7 percent to 26.9 percent, while the share of women using condoms increased from 14.6 percent to 20.4 percent (Piccinino and Mosher, 1998).⁶

Though we might expect that increases in STI risk would lead women to adopt “safe sex” practices, such that pregnancy rates decrease, the above mechanisms show that increases in STI risk could result in higher pregnancy rates.⁷ In the remainder of this paper, I provide empirical evidence that the positive relationship between birth rates and AIDS incidence is causal and driven by these mechanisms. I further distinguish between the two possible mechanisms, monogamy and condoms, using additional data on abortion rates, marriage rates, and infant health.

6. Use of intrauterine devices (IUDs) also changed during this time period. From 1982 to 1988, use of IUDs decreased in the U.S. due to safety problems, including death and infertility, caused by the Dalkon Shield IUD (Hubacher, 2002). However, the decrease in IUD use was largely offset by an increase in female sterilization. Between 1988 and 1995, rates of IUD use and female sterilization were relatively constant (Piccinino and Mosher, 1998).

7. Note that there are also behavioral changes that could simultaneously lower both STI risk and pregnancy likelihood. For example, abstinence, adopting condoms in addition to existing contraceptive methods, or switching from opposite sex to same sex partners in women (see Francis (2008)). However, given the positive relationship in Figure 1 and the subsequent results in this paper, I focus on mechanisms which could increase pregnancy rates.

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 my measure of AIDS risk: local AIDS incidence in women aged 20-44 in the previous year. I use prior year to capture risk at time of conception as opposed to time of birth. $\mathbf{X}_{m,t}$ 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.⁸ 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

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

abortion (Myers and Ladd, 2020).⁹ 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).¹⁰

To further support the argument that the specific timing of initial AIDS diagnoses in an MSA and the subsequent extent of the epidemic is not driven by omitted factors, I also conduct a covariate balance test as proposed by Pei, Pischke, and Schwandt (2019).¹¹ Identification in this setting requires that changes in local AIDS risk are unrelated to changes in potential confounders. If this identifying assumption holds, then there should not be a statistically significant relationship between AIDS risk and covariates after controlling for fixed effects. To test this, I regress a standardized version of each covariate on standardized AIDS risk with and without fixed effects. Results are shown in Appendix Figure A2. Fixed effects are successful in absorbing differences in levels of covariates across MSAs with high and low AIDS. One notable exception is the unemployment rate, which increases in response to local AIDS incidence and may be an outcome of the AIDS epidemic if diagnosis with AIDS and subsequent disease progression prevents employment. As such, I also test whether results are sensitive to excluding the unemployment rate as a control variable.¹²

Finally, I compare results from an unweighted and population-weighted version of equation (1). Differing coefficients between an unweighted and a weighted version could be evidence of model misspecification or heterogeneous effects across locations with small and large populations (Solon, Haider, and Wooldridge, 2015). In addition, though Figure 1 sug-

9. In the case where an MSA crosses state boundaries, I compute covariates from state-level data using a weighted average of the states that compose the MSA, where weights are based on the share of the MSA's female population that lives in each state. Given that this approach may introduce measurement error, I also show in Appendix Figure A3 that results are robust to limiting the sample to MSAs that do not cross state boundaries.

10. 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.

11. Pei, Pischke, and Schwandt (2019) conclude that in the presence of measurement error in confounders, the covariate balance test is preferable to the coefficient comparison test to test identifying assumptions.

12. Angrist and Pischke (2009) show that outcomes of treatment variables are “bad controls” and should be excluded from the set of covariates.

gests that population size differences are not driving results, I also test whether results are robust to additional controls for population.

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.¹³ Given this limitation, I use alternative empirical approaches to indirectly address concerns about the two-way fixed effects approach.

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.¹⁴ However, because AIDS incidence is increasing over time, heterogeneous time effects could also be attributable

13. 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 4 and given the serial correlation in AIDS incidence due to disease contagion.

14. 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 CARE 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. I address this concern by first interacting year with MSA's average AIDS incidence over the entire sample period. This specification is also more flexible in that it does not require that perception of AIDS risk is directly tied to current AIDS incidence, but could instead be reflective of an MSA's overall exposure to the AIDS epidemic. I also test the robustness of results to the inclusion of a quadratic term in AIDS incidence, and attempt to directly control for differences over time by including an MSA-specific linear time trend, with the sample expanded to begin in 1970.

I next 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 of AIDS incidence that are defined using the median AIDS incidence in the sample, both unweighted and weighted. These medians are: the first year that female AIDS incidence in an MSA reaches 1.9 case per 100,000 women and 3.1 cases per 100,000.¹⁵ 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. Finally, though not directly related to concerns about heterogeneity over time, I also test the robustness of results to concerns about geographic spillovers that could bias results.

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 in men with only female partners, and that birth rates are unaffected by AIDS diagnoses in men with male partners. 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 syphilis and 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

15. Use of the medians avoids the problem of selecting arbitrary thresholds, as it is not obvious that there is an optimal way to discretize AIDS incidence. Appendix Figure A5 shows that these two thresholds produce a roughly normal distribution of treatment years in the sample.

relationship.

VI. Results

A. Effect of AIDS Risk on Birth Rates

Empirical results for equation (1) are presented in Table 2, columns (1) through (4). 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 four specifications. I find 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 0.034 per 1,000 women.¹⁶

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 (4) 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 0.55 percent higher than the predicted birth rate in the absence of AIDS. I estimate a cumulative effect of 191,776 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 risk to vary by year, I find that the main results likely underestimate the effect of AIDS risk on birth rates. Figure 2, Panel A shows the estimated coefficients from regressing birth rates on annual 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

16. These results are robust to alternative inclusion of control variables and different sample definitions (see Appendix Figure A3).

and 1992, with the effect attenuating over time.¹⁷ In Figure 2, Panel B, I also show that results are robust to instead interacting year dummies with average AIDS incidence within an MSA over the entire sample period. This specification relies less on the assumption that same-year AIDS incidence represents perceptions of risk. I again find that results are robust and that the main specification likely underestimates the true effect of the AIDS epidemic on birth rates.

However, though the effect of each *additional* AIDS diagnosis on births decreases over time, (as shown in Figure 2, Panel A), 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 the AIDS epidemic. 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 incidence. The marginal effect of one more AIDS case is negative for only one percent of the sample. 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 3.

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 3 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 A4 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

17. 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 A4) and misperceptions about the disease in the early years of the epidemic.

are not driven by MSA-year observations occurring later in the sample with very high AIDS in women. Figure 3 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.

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 2, and with policy changes in 1996, such as welfare reform, creating a downward bias on estimates. Second, I discretize AIDS incidence into two thresholds using sample medians, the first year that female AIDS incidence in an MSA reaches 1.9 and 3.1 cases per 100,000 women, respectively, and analyze the corresponding difference-in-differences regression using a Goodman-Bacon decomposition. Results are shown Figure 4, 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 the unweighted median of 1.9 cases per 100,000 women 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. The second threshold, 3.1 cases, also shows increasing birth rates in post years, with some limited evidence of small increases in births in the pre-treatment years. However, this is consistent with the fact that AIDS risk has already begun increasing in treated MSAs in the pre-period when using the higher threshold.

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.

As a final check of empirical bias in the two-way fixed effects specification, I also check for spillover effects. Butts (2021) shows that in a difference-in-differences specification, spatial spillovers in treatment effects can bias results. To test for spatial spillovers, within each census division I identify in each year the MSA with the highest level of AIDS incidence. I then assign this value as AIDS risk for all MSAs in the same census division and repeat my main analysis. Results are presented in Appendix Table A4. I find evidence of positive spillover effects. This result again indicates that the main estimates are biased towards zero and underestimate the true effect of the AIDS epidemic on birth rates.¹⁸

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

18. Butts (2021) shows that the difference-in-differences estimate is equal to the true treatment effect minus the spillover effect. Thus, if both treatment and spillover effect are positive then the difference-in-differences estimate is biased towards zero.

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 male 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 in men with only female partners.¹⁹ To test this alternative hypothesis, I analyze the effect of AIDS incidence among women and among men of different reported sexual behaviors on birth rates.²⁰

Results are presented in Table 3. I find that AIDS diagnoses in both women and in men with only female partners result in an increase in births. In comparison, I find there is no effect of AIDS diagnoses in men with male partners 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 created a cultural shock that led women to hold more conservative values towards marriage and family. To test this hypothesis, I analyze the effect of AIDS risk on abortion rates. I

19. Analyzing data separately by sexual behavior 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.”

20. Note that the APIDS data manual conflates sexual orientation with sexual behavior. The variable I use for this analysis is labeled, “Sex and Sexual Orientation” and defines the following categories: women, straight men, bisexual men, and gay men. However, earlier versions of the APIDS codebook indicate that this variable is created using information on patients’ sexual behavior (i.e., whether sexual partners are male, female, or both) and sexual orientation. As of 2003, the Adult HIV/AIDS Confidential Case Report Form records information about sexual behavior, not sexual orientation. For these reasons, I infer that this variable reflects sexual behavior, not sexual orientation. Further, for cases where information on sexual behavior was missing, men were assumed to have *only* male partners. Because of this, I also group men with only male partners and men with both male and female partners into one category: men with male partners.

use three data sources to measure abortion rates: Guttmacher Institute data on abortions by state of residence and by state of occurrence, and CDC data on abortions by state of occurrence.²¹

Results showing the effect of AIDS risk on abortion rates are presented in Table 4. I find that AIDS risk has a positive and statistically significant effect on abortion rates across all three measures.²² 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 at least 0.0597 additional pregnancies per 1,000 women in response to each additional AIDS case, and approximately 43 percent of these pregnancies resulted in abortion.²³ 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 some of the additional pregnancies are unintended.

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 and syphilis incidence in women from 1984 to 1996. These data are publicly available via the CDC (US DHHS, 2015).²⁴ Results are presented in

21. I use state-level abortion rates for women 15-44. Data on abortion rates are only available in select years and states between 1981 and 1996, and availability differs across the three measures.

22. Results are robust to limiting the sample to MSAs contained within a single state and to alternative inclusion of control variables (See Appendix Figure A6).

23. Using the estimated coefficients from Table 2, column (4) and Table 4, column (4): $0.034 + 0.0257 = 0.0597$. $0.0257/0.0597 = 0.43$. Using the same counterfactual prediction exercise as above, I also estimate that the abortion rate increased by 1.77 percent, for an additional 144,976 abortions in the 918 sampled MSA-year observations. Combining this result with my counterfactual prediction of 191,776 additional births, I predict 336,752 additional pregnancies due to women adjusting behaviors in response to the AIDS epidemic. Note that this prediction is an underestimate of the true effect, given the missing abortion data observations.

24. Unfortunately, MSA-level data are only available after 1995. Chlamydia data are also publicly available via the CDC. However, chlamydia data for this time period are unreliable as a measure of underlying disease

Table 5. I find no evidence that incidence of other STIs increase in response to local AIDS incidence. AIDS risk has a negative and statistically significant effect on syphilis incidence, and a negative, though statistically insignificant, effect on gonorrhea incidence.²⁵ This result contradicts the hypothesis that higher births and AIDS incidence are both due to unobserved increases in condomless sex with risky partners. If births and AIDS are increasing due to increases in risky sexual activity, we would also expect incidence of other STIs to increase. In contrast, I find that incidence of other STIs decrease in response to local AIDS incidence.

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 6, columns 1-2). Second, I show that birth rates 10 years prior are actually negatively correlated with birth rates from 1981 to 1996 (Table 6, columns 3-4). 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 6, columns 5-6. I find that AIDS risk has a positive and statistically significant effect on current

incidence due to increasing state efforts to record and report chlamydia cases to the CDC (Webster et al., 1993), which may have varied systematically with AIDS incidence.

25. These results are robust to limiting the sample to MSAs contained within a single state and alternative inclusion of control variables (See Appendix Figure A7).

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 adjusting behaviors to lower risk of HIV/AIDS infection was an increase in birth rates and abortion rates, with a corresponding decrease in incidence of other STIs.

VII. Underlying Mechanisms

As discussed in Section 4, there are two underlying mechanisms that could explain an increase in pregnancies: women could opt into monogamous partnership or could switch to condoms from contraceptive methods with lower failure rates in preventing pregnancy. Both mechanisms are consistent with a decrease in the incidence of other STIs. To evaluate which mechanisms are driving results, I make use of additional data sources and information from birth certificate records. I directly evaluate whether monogamy increased by measuring the rate of new marriages using state-level data on marriage certificates from 1981-1995.²⁶ Second, I evaluate the “wantedness” of additional births using data from birth certificates on infant health. If pregnancy becomes more desirable with a monogamous partner, then the monogamy mechanism may produce an increase in planned pregnancies, with associated improvements in prenatal health care usage and infant birth weight. If instead, the additional births are due to higher rates of contraceptive failure (either with or without a monogamous partner) then the additional births may be unplanned.

Results are presented in Table 7 and are supportive of the monogamy mechanism. Higher AIDS risk leads to an increase in the rate of new marriages, a decrease in the share of births with low birth weight (between 1500 and 2500 grams), and a decrease in the share of births with late (starting at 7 months gestation or later) or no prenatal care.

26. This data is publicly available from the National Center for Health Statistics via the NBER. See the Appendix B for further information on data sources.

This evidence supporting an increase in planned pregnancies contradicts the increase in abortions described in Section 6B. In combination, these two results suggest that there is an overall increase in pregnancies, some of which are planned and some of which are unplanned.²⁷ Among the unplanned pregnancies, it is unclear which mechanism is driving results - monogamy with inconsistent contraceptive use or switching from prescription contraceptives to condoms. Ideally, this question could be addressed using abortion data disaggregated by marital status. Unfortunately, state-level abortion-by-marriage data, typically available in CDC Abortion Surveillance Reports, is omitted from the reports between 1982 and 1988.

As a final step in evaluating underlying mechanisms, I evaluate the effects of AIDS risk on birth rates by race. Existing literature has documented differences in the marriage market for white and Black women in the 1980s and 1990s due to the high incarceration rate of Black men. Charles and Luoh (2010) use variation in the incarceration rate across race, age, and location groups to show that female marriage rates are lower within marriage markets with high male incarceration. Further, 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. One explanation for this finding is that male incarceration disrupts the continuity of relationships and increases the total number of lifetime sexual partners.

If opting into monogamy is driving the overall increase in births then we might expect larger effects among white women than Black women, given the high incarceration rate of Black men that constrains the market for monogamy or marriage for Black women. As with Charles and Luoh (2010) and Johnson and Raphael (2009), this prediction relies on data showing that the majority of sexual relationships and marriages are between people of the same race. I repeat my main analysis with births disaggregated by mother's race.²⁸ Results

27. I also find evidence of this using additional data from birth certificate records. In Appendix Table A5, I show that there is an increase in births both in plausibly monogamous women and in single women.

28. Prior to 1989, birth certificate data defines only three race groups: white, Black, and other.

are shown in Table 8. I find statistically significant increases in births to white women and to non-white, non-Black women. Among Black women, point estimates are negative but insignificant. Given the differences by race in the effects of AIDS risk on births, it would be informative to further disaggregate abortion data by race. It could be that the pregnancy rate is increasing similarly across groups, but differential abortions rates are driving the differences in birth rates. Unfortunately, state-level abortion-by-race data is also omitted from the CDC Abortion Surveillance reports between 1982 and 1988. However, the birth data alone are suggestive of monogamy as a mechanism in explaining the increase in births, which the overall effect due to an increase in births to white women. Though there may be other explanations underlying the differences in effects across white and Black women, the monogamy mechanism is consistent with the existing literature on male incarceration, marriage markets, and the spread of HIV/AIDS.

In combination with the existing literature, these results point to the relative importance of partnership decisions in women's choice set. White women opted into monogamy to lower the risk of contracting HIV/AIDS, even at the expense of increased pregnancy likelihood. Among Black women, births did not increase, but the limited availability of men for monogamous partnership led to an increase in AIDS incidence among Black women (as shown by Johnson and Raphael (2009)). While combining condoms and other contraceptive methods could in theory 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 (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 behavior changes can affect AIDS rates in the population, this is the first paper in the US context to relate the AIDS epidemic and birth rates. My results are consistent with two possible behavioral changes: Opting into monogamous partnership or adopting condoms in place of contraceptives with lower failure rates in preventing pregnancy. Data on marriages and further information from birth certificates on infant health and mother’s race are supportive of monogamy as the underlying mechanism. However, the increase in abortions may provide evidence of changes in contraceptive methods as the mechanism, or indicate that some pregnancies within monogamous partnerships were unplanned. Survey data suggests that both mechanisms occur in the population, though monogamy is the more commonly reported strategy used to lower STI risk.

By documenting an increase in birth rates, this paper contributes to the economics literature on both the effects of AIDS epidemic 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 behaviors to lower STI risk 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, 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. Future research could evaluate whether increases in the risk of pregnancy affect decisions about monogamy and marriage.

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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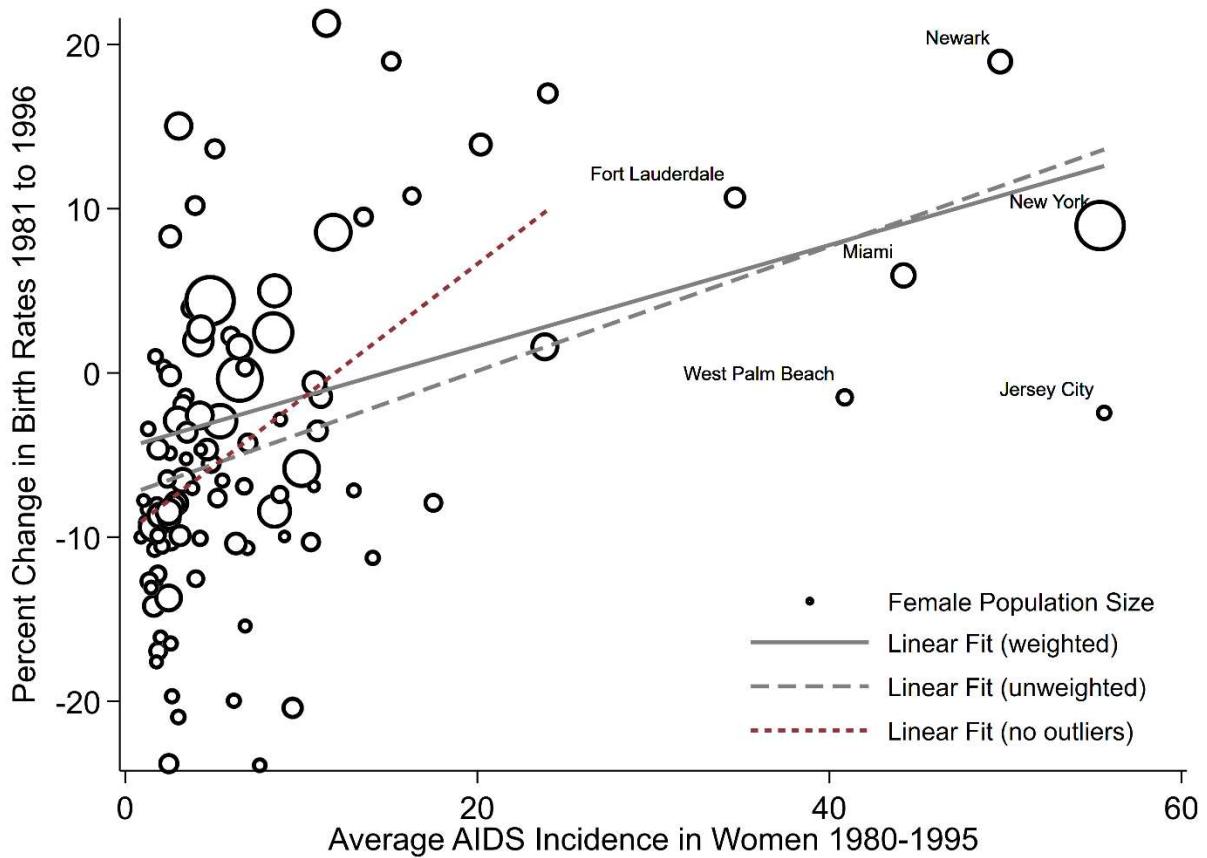


FIGURE 1: RELATIONSHIP BETWEEN AIDS RISK AND CHANGE IN BIRTH RATES

Notes: This figure shows the positive relationship between change in birth rates from 1981 and 1996 and average AIDS incidence in women for 102 MSAs in the United States. Markers are weighted by female 20-44 population size. The positive relationship is not sensitive to weighting by population, nor to excluding outliers in AIDS incidence.

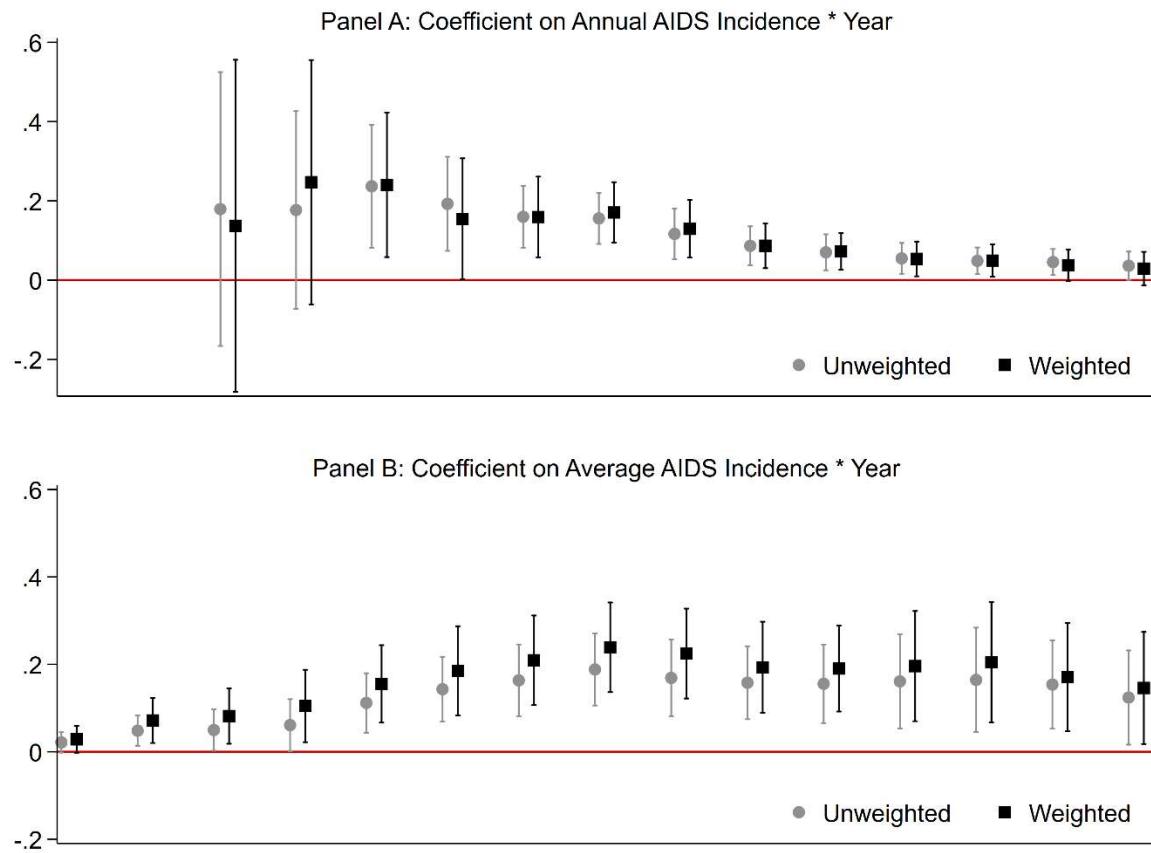


FIGURE 2: HETEROGENEOUS EFFECTS OVER TIME

Notes: This figure 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. Panel A shows estimated coefficients when interacting annual lagged AIDS incidence with year. Estimates for 1982 and 1983 are large but insignificant and are excluded from the figure but shown in Appendix Table A2. Panel B shows estimated coefficient when interacting average AIDS incidence in each MSA from 1980 to 1995 with year. All regressions 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. 95 percent confidence intervals are shown. Appendix Table A2 additionally shows that results are also robust to a specification with time-invariant controls by controlling for 1981 values of all control variables interacted with year.

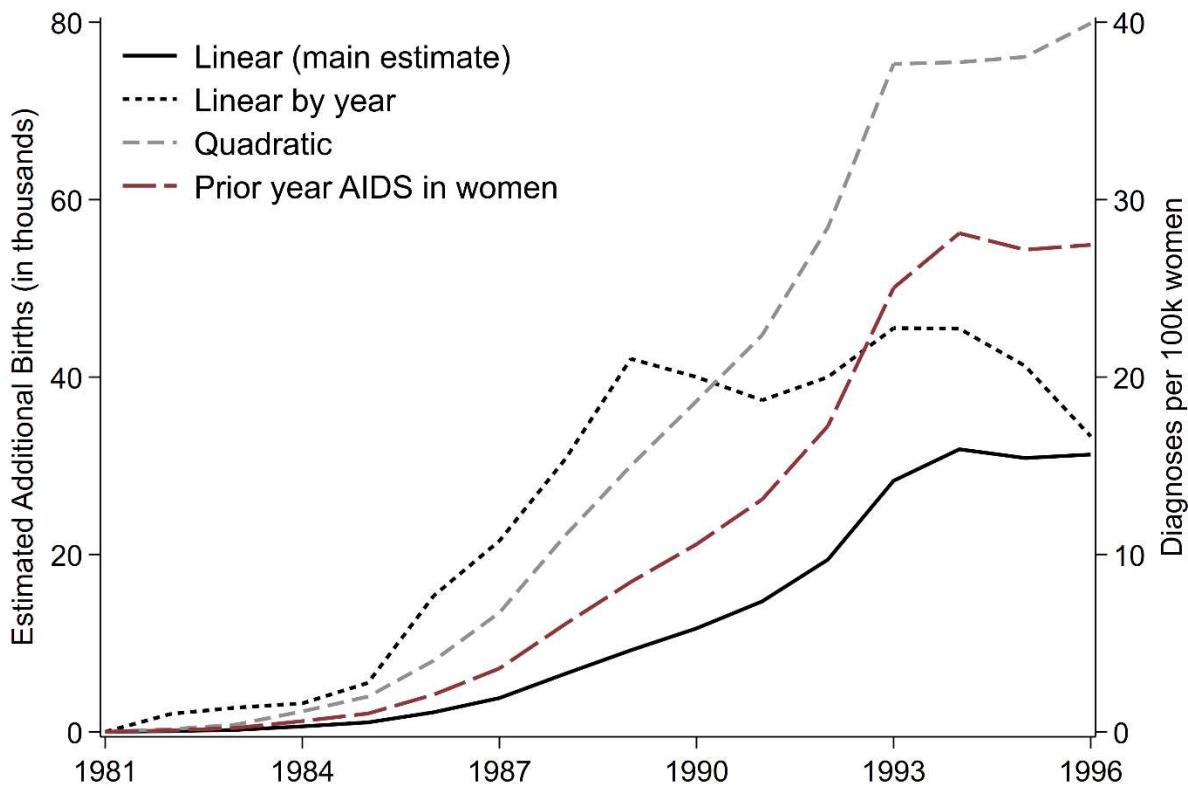


FIGURE 3: PREDICTED NET EFFECT OF AIDS RISK ON BIRTH RATES

Notes: Using coefficient estimates from Table 2 column 4, Figure 1 panel A, and Table 2 column 8, I predict the net effect of AIDS incidence in women 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 women adjusting behavior to lower their risk of contracting HIV/AIDS, I compare the predicted number of births to a counterfactual prediction with AIDS in women equal to zero in every year.

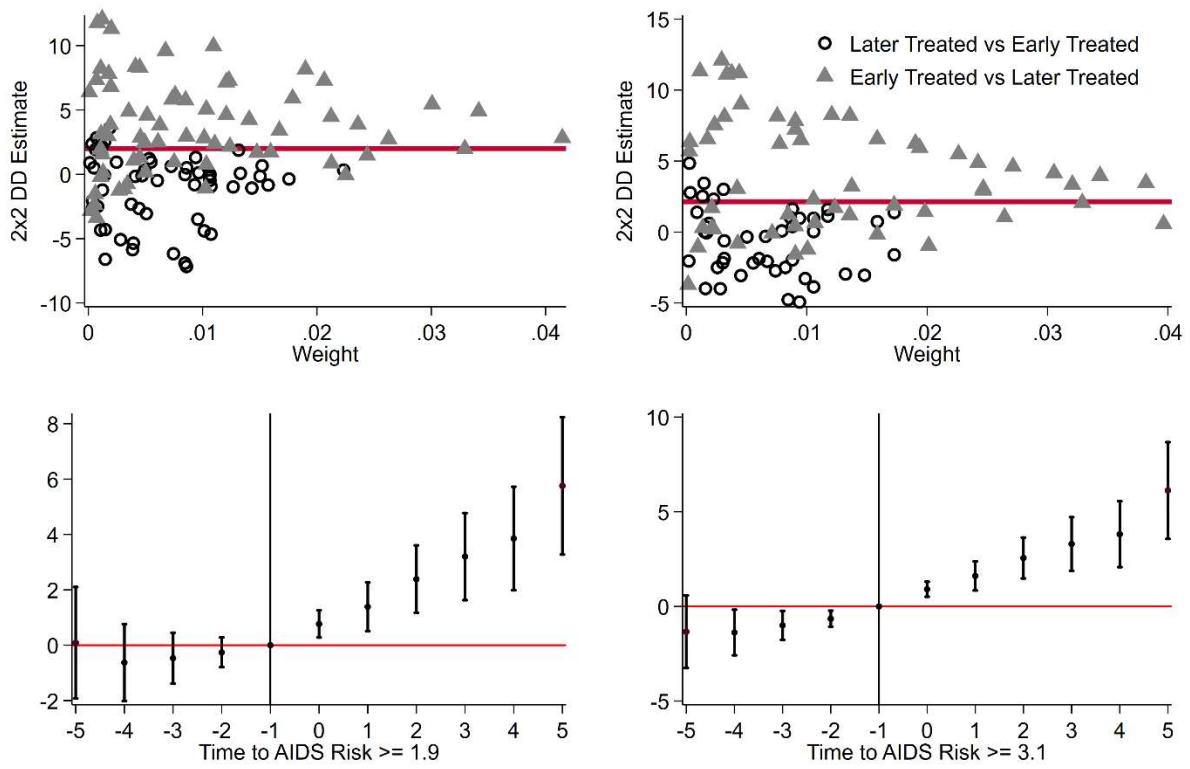


FIGURE 4: ROBUSTNESS TO DISCRETIZED AIDS INCIDENCE

Notes: This figure shows that results are robust to a discretized version of AIDS incidence, specifically whether AIDS incidence in women has reached 1.9 cases per 100,000 women (column 1) or 3.1 cases per 100,000 women (column 2). The distribution of treatment years for these thresholds are shown in Appendix Figure A5. 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 and Perceptions of Risk

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)	(4)
AIDS in women	0.0629*** (0.0181)	0.0449** (0.0196)	0.0383** (0.0187)	0.0340* (0.0186)
Panel B: Quadratic	(5)	(6)	(7)	(8)
AIDS in women	0.146*** (0.0347)	0.153*** (0.0351)	0.104*** (0.0349)	0.131*** (0.0327)
AIDS in women ²	-0.000715*** (0.000239)	-0.000788*** (0.000201)	-0.000530** (0.000242)	-0.000680*** (0.000210)
Share of N w/ marginal effect<0	0.008	0.010	0.009	0.010
N	1,632	1,632	1,632	1,632
Population weighted		X		X
Additional controls			X	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 Sex and Sexual Behavior

Births per 1,000 women 20-44	(1)	(2)	(3)	(4)
AIDS in women	0.0629*** (0.0181)	0.0449** (0.0196)	0.0383** (0.0187)	0.0340* (0.0186)
	(5)	(6)	(7)	(8)
AIDS in men with only female partners	0.0508*** (0.0134)	0.0344** (0.0139)	0.0324** (0.0142)	0.0274** (0.0132)
	(9)	(10)	(11)	(12)
AIDS in men with male partners	-0.00311 (0.00894)	-0.00247 (0.0122)	-0.0132 (0.00961)	-0.00975 (0.0133)
N	1,632	1,632	1,632	1,632
Population weighted		X		X
Additional controls			X	X

Notes: This table shows the effect of prior year AIDS incidence by sex and sexual behavior on birth rates per 1,000 women aged 20-44. Sex and sexual behavior are defined consistent with CDC categorizations in APIDS data. 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 Abortion Rates

Panel A: By State of Residence (Guttmacher)				
	(1)	(2)	(3)	(4)
AIDS in women	0.0281*** (0.00731)	0.0376*** (0.00816)	0.0188** (0.00783)	0.0257** (0.0104)
N	918	918	918	918
Panel B: By State of Occurrence (Guttmacher)				
	(5)	(6)	(7)	(8)
AIDS in women	0.0456*** (0.00727)	0.0458*** (0.00688)	0.0352*** (0.00766)	0.0319*** (0.00960)
N	1,020	1,020	1,020	1,020
Panel C: By State of Occurrence (CDC)				
	(9)	(10)	(11)	(12)
AIDS in women	0.0240** (0.0104)	0.0230*** (0.00861)	0.0218** (0.00992)	0.0172** (0.00858)
N	1,387	1,387	1,387	1,387
Population weighted	X			X
Additional controls		X	X	X

Notes: This table shows the effect of prior year AIDS incidence on abortions per 1,000 women aged 15-44 using three measures of abortions rates: Abortions by state of residence compiled by the Guttmacher Institute (Panel A), abortions by state of occurrence compiled by the Guttmacher Institute (Panel B), and abortions by state of occurrence compiled by the CDC (panel C). 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: Effect of AIDS on Syphilis and Gonorrhea Incidence

	Panel A: Syphilis diagnoses per 100,000 women			
	(1)	(2)	(3)	(4)
AIDS in women	-0.312** (0.132)	-0.0974 (0.116)	-0.260*** (0.0853)	-0.136** (0.0685)
N	1,326	1,326	1,326	1,326

	Panel B: Gonorrhea diagnoses per 100,000 women			
	(5)	(6)	(7)	(8)
AIDS in women	-0.545 (0.360)	-0.213 (0.331)	-0.429 (0.298)	-0.318 (0.284)
N	1,326	1,326	1,326	1,326

Population weighted	X		X
Additional controls		X	X

Notes: This table shows the effect of prior year AIDS incidence on syphilis incidence in women (Panel A) and gonorrhea incidence in women (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 6: 10-year lag between HIV Infection and AIDS diagnosis

Births per 1,000 women 20-44	1971-1986			1981-1996		
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS in women	0.0204	0.00257				
10 years future	(0.0291)	(0.0211)				
Birth rates 10 years past			-0.170*** (0.0621)	-0.124* (0.0712)	-0.187*** (0.0630)	-0.138* (0.0719)
AIDS in women prior year					0.0460** (0.0211)	0.0367* (0.0203)
N	1,632	1,632	1,632	1,632	1,632	1,632
Population weighted		X		X		X
Additional controls			X	X	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. Columns 1 and 2 shows that AIDS in women 10 years in the future is not predictive of birth rates between 1971 and 1986. Columns 3 and 4 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 5 and 6). 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 7: Effect of AIDS on Marriages and Infant Health

	Marriages per 1,000 women aged 20-44 Panel A: Unweighted	Share births very low birth weight (1)	Share births low birth weight (2)	Share births late/no prenatal care (4)
AIDS in women	0.0216** (0.0101)	0.000465 (0.000754)	-0.00291** (0.00126)	-0.0254** (0.0112)
Outcome mean	35.65	1.215	5.538	4.444
Panel B: Weighted	(5)	(6)	(7)	(8)
AIDS in women	0.0327** (0.0125)	-0.00426 (0.0120)	-0.00272** (0.00124)	-0.0359*** (0.0121)
Outcome mean	33.97	1.259	5.594	4.636
Observations	1,632	1,632	1,632	1,632

Notes: This table shows the effect of prior year AIDS incidence on the number of new marriages per year per 1,000 women aged 20-44 (Columns 1 and 5), the share of births that are very low birthweight (Columns 2 and 6), the share of births that are low birthweight (Columns 3 and 7), and the share of births with late or no prenatal care (Columns 4 and 8). All regressions 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. Panel A regressions are unweighted and Panel B regressions are weighted by female population. 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 8: Effect of AIDS on Births by Mothers' Race

	White		Black		Non-white, Non-Black	
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS in women	0.0498** (0.0221)	0.0569*** (0.0189)	-0.0262 (0.0249)	-0.0190 (0.0225)	0.175** (0.0709)	0.159** (0.0689)
N	1,632	1,632	1,632	1,632	1,632	1,632
Population weighted		X		X		X
Additional controls	X	X	X	X	X	X

Notes: This table shows the effect of prior year AIDS incidence on birth rates by mothers' race. All regressions include MSA and year fixed effects, as well as controls for the share of the female population of each race and the 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

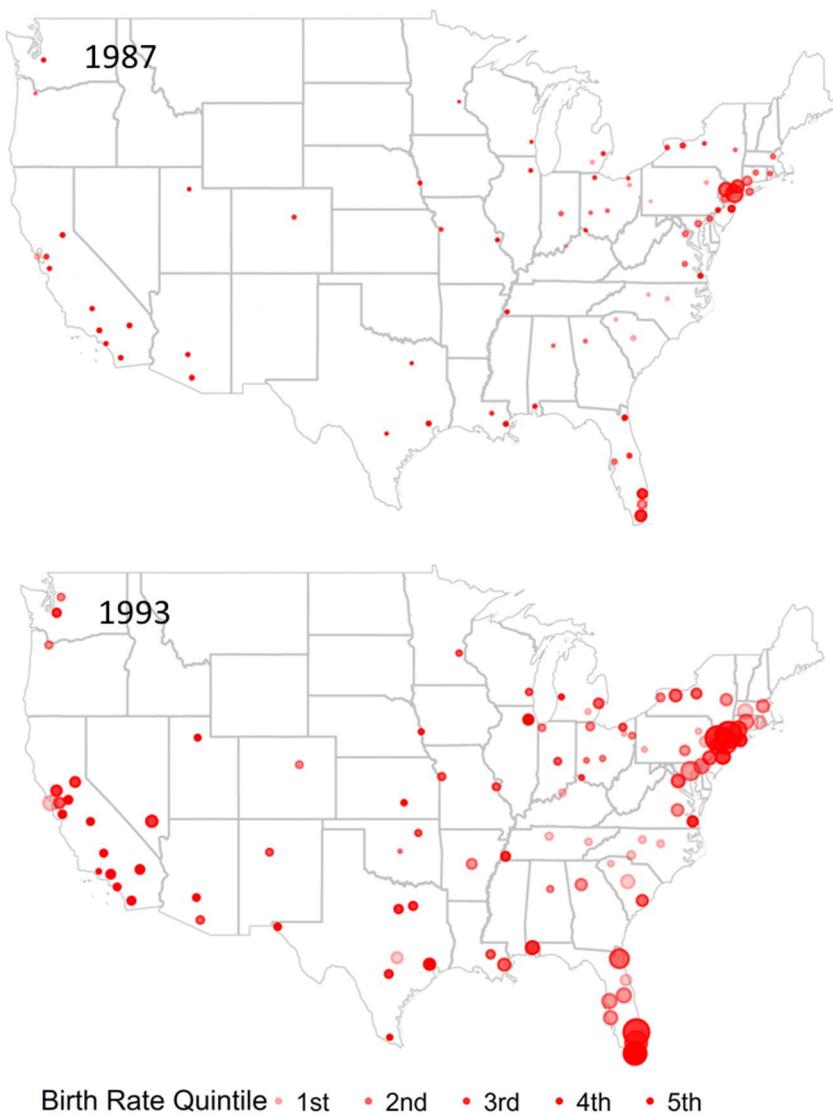


FIGURE A1: GEOGRAPHIC VARIATION IN AIDS RISK AND BIRTH RATES

Notes: This figure depicts AIDS incidence in women in each MSA sampled in 1987 and 1993. Larger markers indicate higher levels of AIDS incidence. Darker markers indicate a higher birth rate quintile.

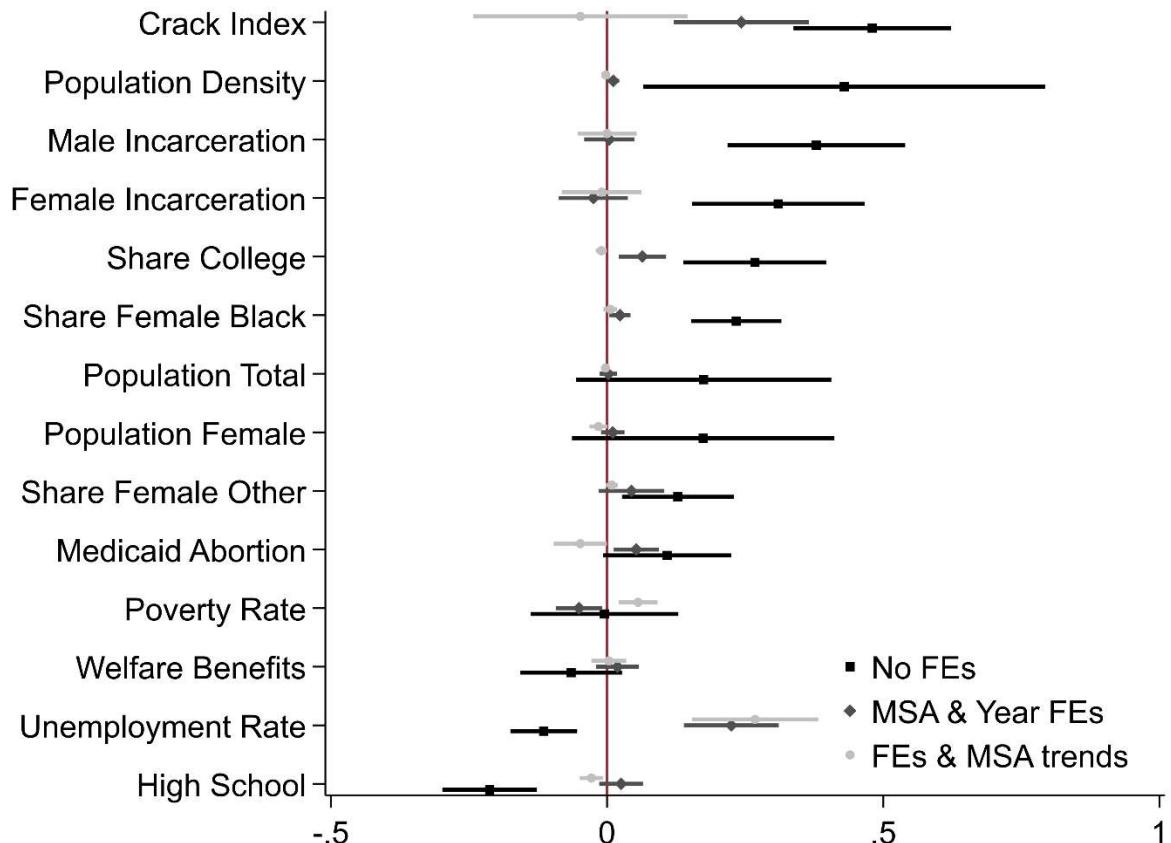


FIGURE A2: COVARIATE BALANCE TEST

Notes: Each covariate described in Section 5 is standardized and regressed on standardized AIDS incidence in women with and without MSA and year fixed effects and MSA-specific linear time trends. Without fixed effects, the figure shows that MSAs with higher levels of AIDS incidence are observably different than MSAs with lower levels of AIDS incidence. However, fixed effects absorb these differences in levels such that AIDS incidence is as-if randomly assigned across MSAs and years after fixed effects. One exception is the unemployment rate, which may itself be an outcome of local AIDS incidence and be a bad control.

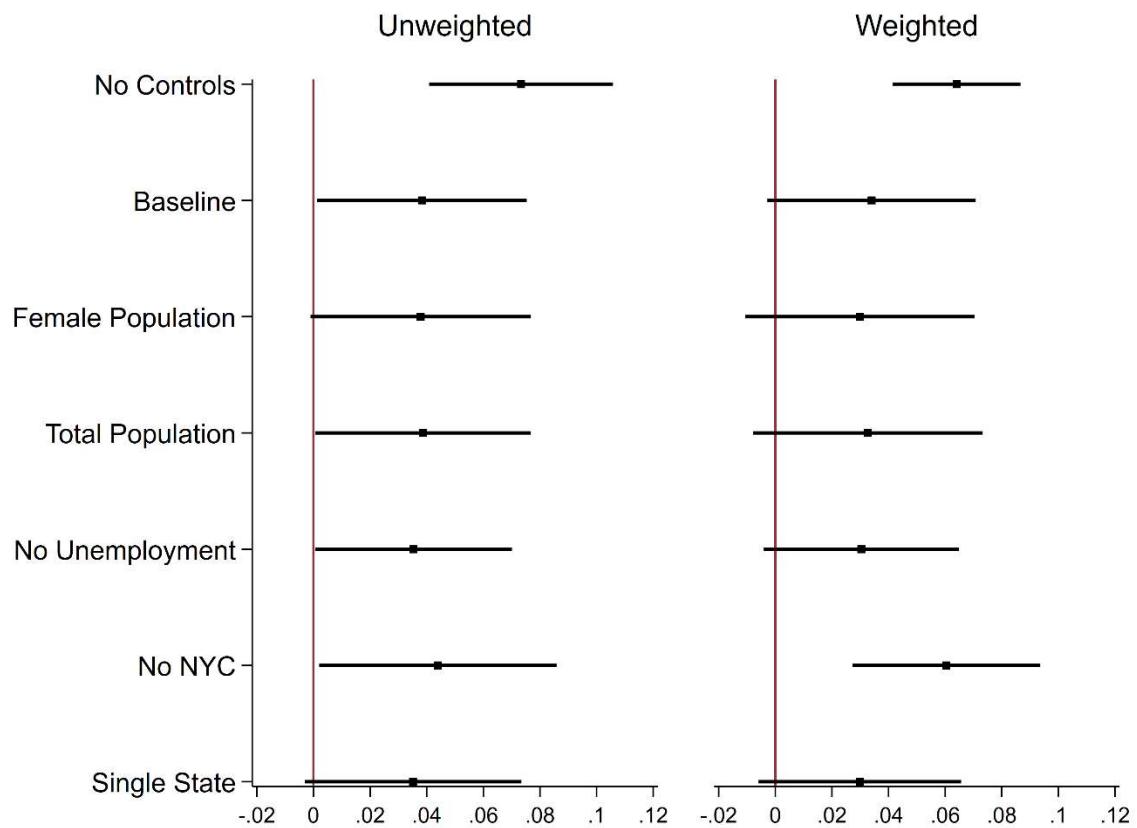


FIGURE A3: ROBUSTNESS OF THE EFFECT OF AIDS RISK ON BIRTH RATES

Notes: This figure shows that the main results presented in Table 2, columns 3 and 4 are robust to alternative inclusion of controls and alternative sample definitions. “No controls” shows the effect of AIDS risk on birth rates including MSA and year fixed effects and no other controls. “Baseline” shows the coefficients in Table 2, columns 3 and 4 for the unweighted and weighted specification, respectively. These results are robust to (from top to bottom): additionally controlling for female population, additionally controlling for total population, excluding the unemployment rate as a control variable, excluding New York City MSA from the sample, and limiting the sample to MSAs that do not cross state boundaries. 95 percent confidence intervals are shown. Standard errors are robust and clustered at the MSA level.

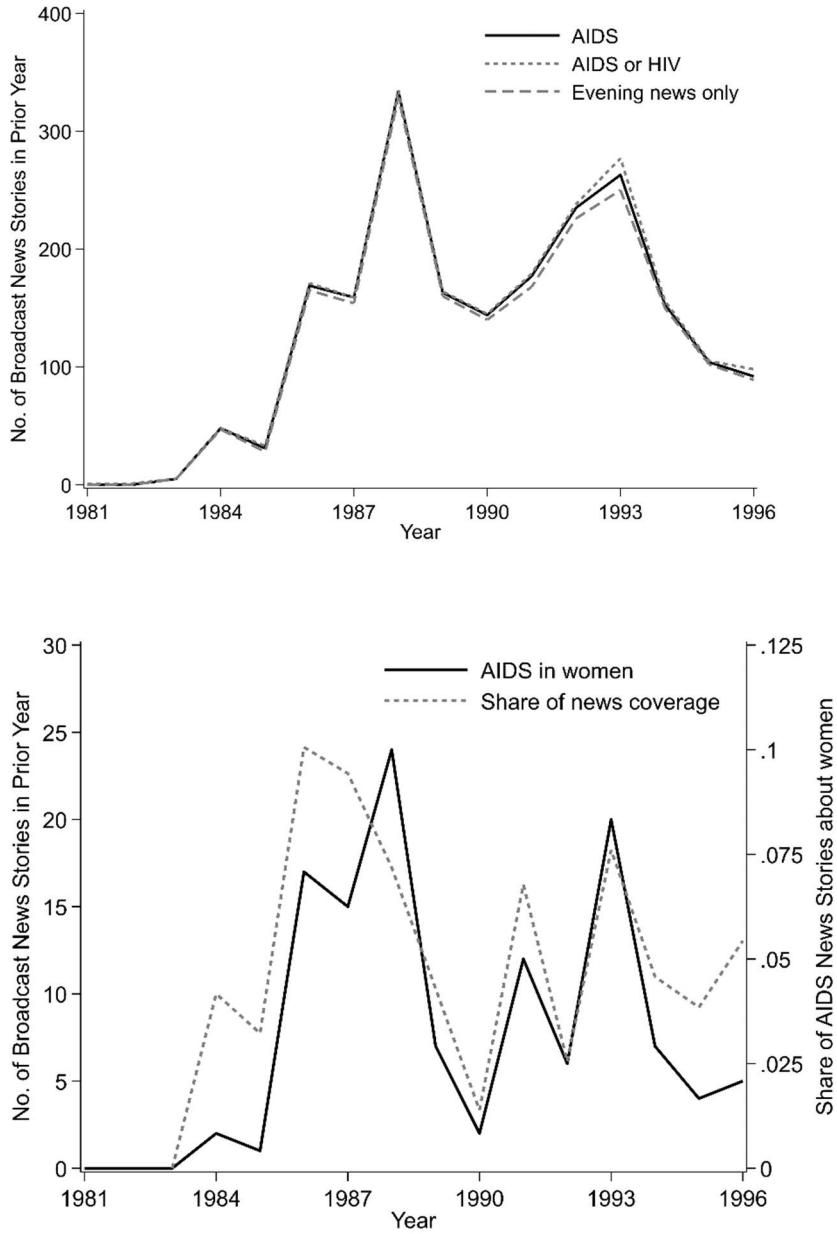


FIGURE A4: NATIONAL BROADCAST NEWS COVERAGE OF THE AIDS EPIDEMIC

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.

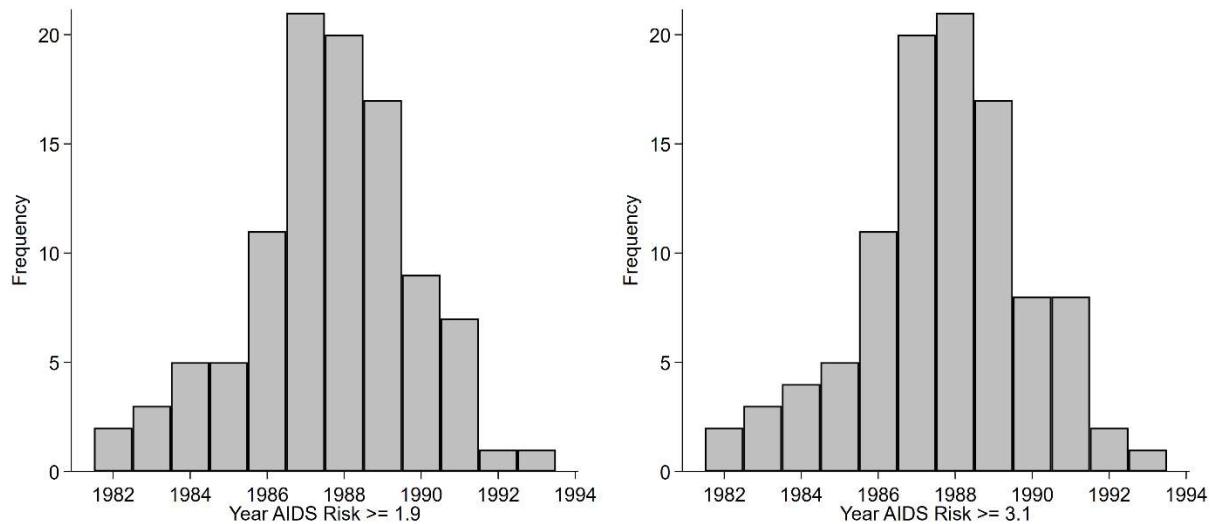


FIGURE A5: DISTRIBUTION OF TREATMENT YEARS FOR DISCRETIZED AIDS INCIDENCE

Notes: This figure shows the distribution of treatment years when AIDS incidence has reached 1.9 cases per 100,000 women (column 1) or 3.1 cases per 100,000 women (column 2). These thresholds are used for the analysis in Figure 4 and correspond to the unweighted and weighted median of AIDS incidence in the sample.

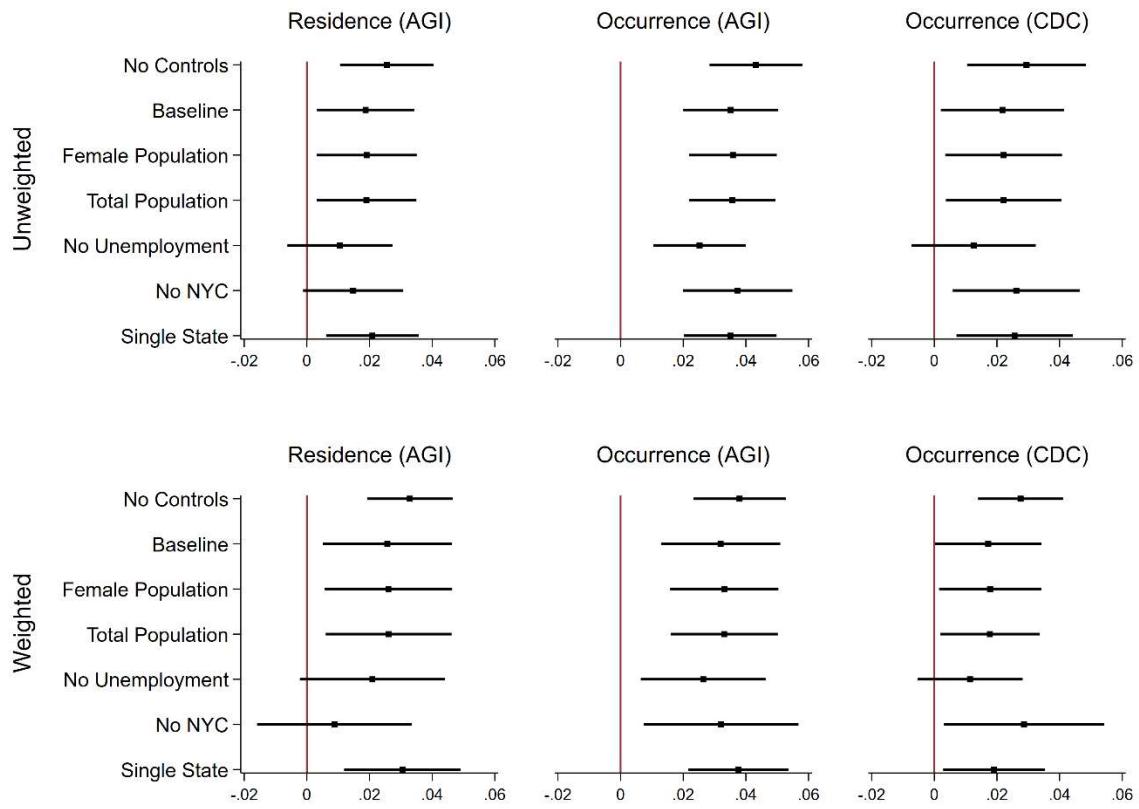


FIGURE A6: ROBUSTNESS OF THE EFFECT OF AIDS RISK ON ABORTION RATES

Notes: This figure shows that the results presented in Table 4 are robust to alternative inclusion of controls and alternative sample definitions. “No controls” shows the effect of AIDS risk on abortion rates including MSA and year fixed effects and no other controls. “Baseline” shows the coefficients in Table 4 including the full set of controls described in Section 5. These results are robust to (from top to bottom): additionally controlling for female population, additionally controlling for total population, excluding the unemployment rate as a control variable, excluding New York City MSA from the sample, and limiting the sample to MSAs that do not cross state boundaries. 95 percent confidence intervals are shown. Standard errors are robust and clustered at the MSA level.

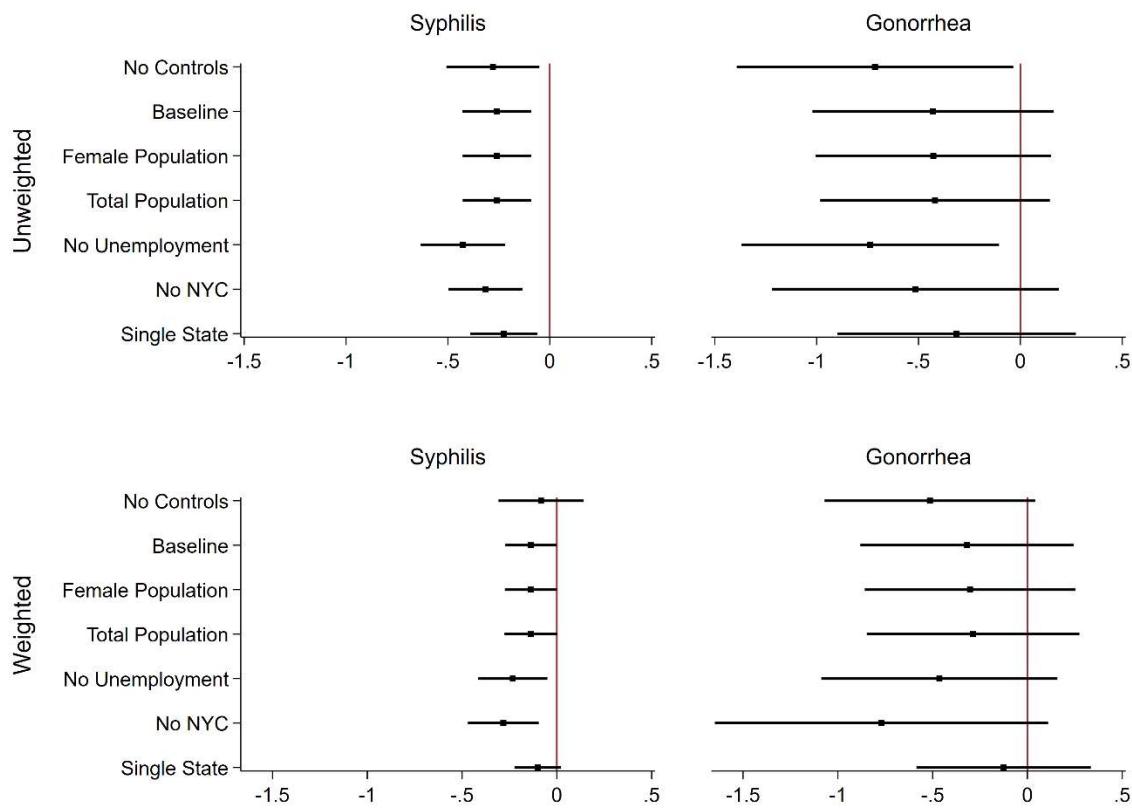


FIGURE A7: ROBUSTNESS OF THE EFFECT OF AIDS RISK ON STI INCIDENCE

Notes: This figure shows that the results presented in Table 5 are robust to alternative inclusion of controls and alternative sample definitions. “No controls” shows the effect of AIDS risk on incidence of other STIs including MSA and year fixed effects and no other controls. “Baseline” shows the coefficients in Table 5 including the full set of controls described in Section 5. These results are robust to (from top to bottom): additionally controlling for female population, additionally controlling for total population, excluding the unemployment rate as a control variable, excluding New York City MSA from the sample, and limiting the sample to MSAs that do not cross state boundaries. 95 percent confidence intervals are shown. Standard errors are robust and clustered at the MSA level.

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
<i>Abortions per 1,000 women age 15-44...</i>					
By state of residence (Guttmacher)	29.38	9.96	7.75	47.24	918
By state of occurrence (Guttmacher)	29.20	10.36	7.45	55.87	1,020
By state of occurrence (CDC)	25.26	11.25	5.38	50.12	1,387
Syphilis diagnoses per 100,000 women	34.51	27.31	0.32	171.68	1,326
Gonorrhea diagnoses per 100,000 women	216.92	119.27	9.17	664.71	1,326
<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
Men with only female partners 20-44	18.86	35.49	0	218.06	1,632
Men with male partners 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. 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

	Model 1		Model 2		Model 3	
	(1)	(2)	(3)	(4)	(5)	(6)
1982	0.300 (2.608)	0.900 (1.295)	0.0286* (0.0156)	0.0215* (0.0117)	0.0163 (0.0158)	0.0313** (0.0124)
1983	0.124 (0.568)	0.430 (0.517)	0.0716*** (0.0259)	0.0483*** (0.0174)	0.0665*** (0.0242)	0.0596*** (0.0216)
1984	0.137 (0.211)	0.179 (0.174)	0.0816** (0.0319)	0.0498** (0.0239)	0.0714*** (0.0226)	0.0594*** (0.0225)
1985	0.247 (0.155)	0.177 (0.126)	0.105** (0.0417)	0.0610** (0.0299)	0.104*** (0.0289)	0.0747*** (0.0280)
1986	0.240** (0.0919)	0.237*** (0.0781)	0.155*** (0.0447)	0.112*** (0.0343)	0.155*** (0.0324)	0.120*** (0.0314)
1987	0.154** (0.0770)	0.192*** (0.0599)	0.185*** (0.0515)	0.143*** (0.0373)	0.205*** (0.0397)	0.153*** (0.0338)
1988	0.159*** (0.0513)	0.160*** (0.0394)	0.209*** (0.0516)	0.163*** (0.0414)	0.236*** (0.0395)	0.178*** (0.0342)
1989	0.171*** (0.0386)	0.156*** (0.0325)	0.239*** (0.0517)	0.188*** (0.0416)	0.262*** (0.0426)	0.191*** (0.0385)
1990	0.129*** (0.0367)	0.117*** (0.0324)	0.225*** (0.0518)	0.169*** (0.0443)	0.246*** (0.0488)	0.159*** (0.0475)
1991	0.0864*** (0.0285)	0.0864*** (0.0248)	0.193*** (0.0525)	0.158*** (0.0421)	0.223*** (0.0498)	0.153*** (0.0422)
1992	0.0724*** (0.0234)	0.0700*** (0.0228)	0.190*** (0.0495)	0.156*** (0.0453)	0.224*** (0.0500)	0.155*** (0.0423)
1993	0.0533** (0.0221)	0.0547*** (0.0196)	0.196*** (0.0635)	0.161*** (0.0544)	0.236*** (0.0629)	0.177*** (0.0440)
1994	0.0491** (0.0206)	0.0485*** (0.0170)	0.205*** (0.0694)	0.165*** (0.0602)	0.267*** (0.0642)	0.212*** (0.0429)
1995	0.0375* (0.0200)	0.0454*** (0.0166)	0.171*** (0.0626)	0.154*** (0.0510)	0.268*** (0.0602)	0.225*** (0.0419)
1996	0.0289 (0.0213)	0.0362** (0.0179)	0.146** (0.0647)	0.124** (0.0544)	0.265*** (0.0568)	0.234*** (0.0384)
N	1,632	1,632	1,632	1,632	1,632	1,632
Population Weighted		X		X		X

Notes: This table shows coefficient estimates for the regression analysis presented in Figure 2. Model 1 interacts annual AIDS incidence in each MSA with year. Model 2 interacts average AIDS incidence in the MSA from 1980-1995 with year. Models 1 and 2 control for the share of the female population of each race and the full set of additional controls described in section 5. Model 3 repeats Model 2 but controls for 1981 values of all control variables interacted with year to demonstrate the robustness of results to time-invariant controls. All regressions include MSA and year fixed effects. 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				
	70-96	70-95	70-94	70-93	70-92
Panel A: Unweighted	(1)	(2)	(3)	(4)	(5)
AIDS in women	0.0370 (0.0268)	0.0465* (0.0252)	0.0566** (0.0249)	0.0809** (0.0329)	0.128*** (0.0368)
Panel B: Weighted	(6)	(7)	(8)	(9)	(10)
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 include MSA and year fixed effects, as well as controls for the share of the female population of each race. Panel A regressions are unweighted and Panel B regressions are weighted by female population. 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: Spillover Effects of High-AIDS MSAs on Birth Rates

	Births per 1,000 women 20-44			
	(1)	(2)	(3)	(4)
AIDS in women in division's highest incidence MSA	0.0444*** (0.0110)	0.0423*** (0.0122)	0.0336** (0.0140)	0.0455*** (0.0142)
N	1,632	1,632	1,632	1,632
Population weighted		X		X
Additional controls			X	X

Notes: This table estimates the spillover effects of high AIDS incidence MSAs on birth rates in other MSAs in the same census division. Treatment is defined as the highest incidence of any MSA in the same census division in the prior year. 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 A5: Effect of AIDS on Births by Partnership Status and Race

	Panel A: Births per 1,000 white women			
	Partnered (1)	Married (2)	Cohabiting (3)	Single (4)
AIDS in women	0.0353* (0.0207)	0.0152 (0.0244)	0.0201** (0.00820)	0.0216*** (0.00590)
Share of births	0.738	0.669	0.0688	0.0500
Outcome mean	63.79	57.46	6.332	4.344
	Panel B: Births per 1,000 Black women			
	(5)	(6)	(7)	(8)
AIDS in women	-0.0103 (0.0337)	-0.00190 (0.0156)	-0.00838 (0.0316)	-0.00869 (0.0261)
Share of births	0.107	0.0689	0.0384	0.0556
Outcome mean	50.68	32.55	18.14	23.58
	Panel C: Births per 1,000 non-white, non-Black women			
	(9)	(10)	(11)	(12)
AIDS in women	0.149** (0.0659)	0.112 (0.0738)	0.0378** (0.0156)	0.00914 (0.00785)
Share of births	0.0457	0.0425	0.00322	0.00313
Outcome mean	67.76	63.86	3.903	5.161

N	1,632	1,632	1,632	1,632
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Notes: This table shows the effect of prior year AIDS incidence on births by mothers' partnership status and race. Married women are identified using the marital status imputation on birth records. Cohabiting women are defined as those who are unmarried but the birth record includes the father's age. Single women are defined as those who are unmarried but the birth record lacks information on the father's age. Partnered women are those who are married or cohabiting. Results indicate that main estimates presented in section 5 are driven by an increase in births to white, cohabiting and single women. 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 * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

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, as well as information on infant birth weight and prenatal care usage.

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

Gonorrhea and Syphilis Surveillance Data

Information on gonorrhea and syphilis 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>

Abortion Rates

Data on abortion rates are from three sources: Guttmacher Institute data on abortions by state of residence; Guttmacher Institute data on abortions by state of occurrence, and CDC data on abortions by state of occurrence. Data for each measure are available for select states and years across the sample period. 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.

Henshaw, S.K. and Kost, K.L.. 2008. ‘Trends in the characteristics of women obtaining abortions, 1974 to 2004.’ New York: Guttmacher Institute.

US Department of Health and Human Services (US DHHS). Centers for Disease Control and Prevention (CDC). 1981 – 1996. Abortion surveillance—United States, Morbidity and Mortality Weekly Report: CDC Surveillance Summaries.

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>

Marriage Data

Data on marriage certificates from 1981-1995 are publicly available for select states via the NBER. 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 marriage rates in each MSA.

US Department of Health and Human Services (US DHHS). Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS). Marriage and Divorce Data 1981-1995. <https://www.nber.org/research/data/marriage-and-divorce-data-1968-1995>

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>.

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>

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/>