

# Safer Sex? The Effect of AIDS Risk on Birth Rates

Melissa K. Spencer\*

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## Abstract

Prior research shows that people responded to the AIDS epidemic by switching to sexual behaviors with lower likelihood of AIDS transmission. These behavioral adjustments also affect the likelihood of pregnancy. This paper shows that the AIDS epidemic in the United States increased the birth rate. I show that AIDS avoidance behaviors increased births among adult women by 0.32 births per 1,000 women per year for a total of 221,820 additional births between 1981 and 1996. My analysis suggests that the overall estimates are driven by women shifting to monogamous partnerships to avoid AIDS.

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

**JEL Classifications:** I12, J13, J12, J16

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\*University of Richmond, Robins School of Business, Department of Economics; mspencer@richmond.edu. I would like to thank Amalia Miller, Eric Chyn, and Sebastian Tello-Trillo, who guided me throughout this project. I would also like to thank many faculty and students at the University of Virginia for their advice and support, especially John Pepper, Eric Young, Lee Lockwood, Brett Fischer, and Haruka Takayama. Thank you also to Allison Luedtke, Jose Fernandez, Carmit Segal, and Barton Willage for their feedback on this project, as well as seminar and conference participants at the Virtual Economics of Risky Behavior seminar, Liberal Arts Labor and Public Conference, West Virginia University, St. Olaf College, and University of Toledo. I also wish to acknowledge the Jefferson Scholars Foundation and the Bankard Fund for Political Economy for their financial support. The results contained herein were derived in part from data provided by the National Center for Health Statistics, and specifically the natality detail data compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program.

## 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. In particular, condoms and monogamy both reduce STI risk, but can potentially increase the likelihood of pregnancy. This is especially true if condoms are used to substitute for more reliable forms of contraception, such as oral contraceptives, or if women with only one sexual partner have condomless sex more frequently.

I estimate the effect of increases in STI risk and resulting STI avoidance behaviors on birth rates. Because of the trade-offs between STI and pregnancy prevention strategies, the effects of increases in STI risk are theoretically ambiguous. 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. Empirical studies have validated the importance of economic cost considerations in determining sexual behavior, contraceptive choices, and fertility outcomes (Michael and Willis 1973; Goldin and Katz 2002; Francis 2008; Kearney and Levine 2009; Bailey 2010; Shah 2013; Myers 2017), but the effect of increased STI risk on fertility in developed countries has not previously been examined.

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

risk given the concurrent risk of pregnancy. Decisions about sexual partnerships can be an important risk mitigation strategy, but at the cost of increased pregnancy likelihood.

I empirically examine the effect of STI avoidance on birth rates by exploiting variation in the spread of AIDS across U.S. cities in the 1980s and 1990s. The AIDS epidemic created a large and plausibly exogenous increase in the cost of contracting an STI. During this period, the spread of AIDS was largely driven by male same-sex contact and the average time between HIV infection and AIDS diagnosis was 10 years. Thus, within a city, the timing of AIDS arrival and the extent of the epidemic was unrelated to women's sexual behavior. I analyze the effect of local AIDS incidence on birth rates using a fixed effects specification with controls for city and year.

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 on average by 0.32 births per 1,000 women per year due to AIDS avoidance behavior, for a total of 221,820 additional births between 1981 and 1996. I test whether the increase in birth rates is due to an increase in risky sexual behavior among women. I evaluate this alternative hypothesis using data on gonorrhea incidence in women. If women are having more condomless sex, we would expect increases in both pregnancy and gonorrhea. In contrast, I find evidence that AIDS risk leads to decreases in gonorrhea incidence. This suggests that women are adopting behaviors that decrease their likelihood of contracting AIDS and other STIs, but at the expense of heightened pregnancy likelihood. There are two AIDS avoidance behaviors that could result in an increase in births without increasing gonorrhea: Women with opposite-sex partners could switch from effective prescription contraceptives to condoms or could limit their number of sexual partners.

I provide evidence that the increase in births is due to women entering monogamous partnerships to avoid AIDS. Using birth certificate information, I find that the overall increase in births is due to an increase in births to partnered women. I also find an increase in births to white single women, however this effect is fully offset by a decrease in births to Black single

women. While these results confirm heterogeneous responses to AIDS in the population, survey data supports the conclusion that the overall increase in births is driven by women choosing to have only one sexual partner to protect themselves from AIDS. Survey data shows that 16 percent of unmarried women decided to stop having sex with more than one man to avoid AIDS, and that this was the most commonly used AIDS avoidance strategy (Mosher and Pratt 1993).

By documenting an increase in birth rates, this paper contributes to the economics literature on both the effects of AIDS and STIs and the determinants of fertility. 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). Within the STI literature, this work is closest to studies such as Ahituv, Hotz, and Philipson (1996), Lakdawalla, Sood, and Goldman (2006), Francis (2008), Thirumurthy et al. (2012), Shah (2013), Fortin (2015), Greenwood et al. (2019) and Hakak and Pereda (2021), which examine behavioral changes in response to STI risk. Similar to this work are studies that examine the spillovers between fertility and STI risk, for example, Sen (2003), Klick and Stratmann (2008), Durrance (2013), Mulligan (2016), Buckles and Hungerman (2018), Mallatt (2019), and Willage (2020). In comparison with this paper, those studies focus on the reverse relationship by studying the effect of changes in the cost of pregnancy prevention on STI rates. I find that women adopt STI avoidance behaviors at the cost of increased pregnancy likelihood, and that as a result, STI risk has a positive effect on birth rates.

## II. The AIDS Epidemic in the United States

The first cases of what would come to be known as Acquired Immunodeficiency Syndrome (AIDS) were identified in the United States in June of 1981. By January 1983, there were at least two documented cases of women who were exposed to the disease via opposite-sex

contact (Heywood and Curran 1988). 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).

Evidence suggests that public health fears of AIDS spreading rapidly via opposite-sex contact influenced knowledge and behavior regarding AIDS. In 1987, *The New York Times* ran an article describing fears of AIDS infection among women. At one clinic in New York, over 40 percent of those requesting HIV tests were women considered low risk (i.e., no history of drug use, sex with drug users, or sex with men who have sex with men), none of whom were found to have AIDS (Sullivan 1987). Further contributing to fear of AIDS was the volatile nature of the disease’s spread across the United States (Mann 1992). The sudden and unstable spread of AIDS led to a large number of epidemiological studies seeking to track and predict the prevalence of the disease (Taylor 1989; Lam, Fan, and Liu 1996; Steinberg and Fleming 2000). The epidemiology literature identifies the following pattern: Initially, AIDS spread from city to city, with cases concentrated among men with same-sex partners and IV drug users. As AIDS became more prevalent in a city, it began to spread outward from urban areas. Further contributing to the unpredictable nature of the epidemic was the lengthy incubation period between HIV infection and the presentation of AIDS symptoms. In the 1980s and early 1990s, the average time between infection with HIV and an AIDS diagnosis was 10 years (Osmond 1998). As a result, outbreaks in cities were not driven by current behaviors, but behaviors from as much as 10 years prior.

The AIDS epidemic is ideal for studying the relationship between STI risk and birth rates for two reasons. First, by focusing on cities, I can exploit the spread of AIDS, which

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

### **III. Expected Effect of AIDS Risk on Births**

It is unclear how women would respond to an increase in AIDS risk given the concurrent risk of pregnancy. Condoms are the only method of contraception that can protect from AIDS transmission, but are not very effective in preventing pregnancy.<sup>1</sup> 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 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 AIDS by switching from the Pill to condoms, then the birth rate in the population would increase. If women switched from not using contraceptives to using condoms, or adopted condoms in addition to their current method, the birth rate would decrease.<sup>2</sup>

It is also possible that women responded to AIDS risk by adjusting their sexual behavior. At the extensive margin, women can choose to abstain from any sexual activity. At the intensive margin, they can limit their number of sexual partners or choose lower-risk male partners (i.e., those who do not use IV drugs and do not have concurrent partners). In this case the birth rate would likely increase, since there is less incentive to use consistent

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

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

contraception with a low-risk or monogamous partner and the frequency of sexual activity may increase with one partner. Furthermore, women may be more likely to continue a pregnancy with a low-risk partner or a sole, committed partner. However, the birth rate would decrease if women switch from male partners to female partners. Francis (2008) shows that women who have a relative with AIDS shifted from heterosexual behavior to homosexual behavior to lower risk of infection.<sup>3</sup>

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

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

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

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

#### **IV. Data Sources and Sample Construction**

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

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

I find that AIDS incidence has a positive and statistically significant effect on the probability that unmarried women rate their own chance of contracting AIDS as high. I find no effect of AIDS incidence on unmarried men's perceptions. Consistent with the aforementioned anecdotal evidence from *The New York Times*, these results suggest that women may have been uniquely risk averse to AIDS, despite the higher incidence of AIDS among men. Table 1 further shows that AIDS incidence not only affected unmarried women's perception of risk, but also their perception of AIDS avoidance strategies. Living in a region with a higher AIDS incidence increased the likelihood that unmarried women reported monogamy or condom use as a very effective strategy in preventing the transmission of AIDS.

Using APIDS data, I create a panel of AIDS incidence among women aged 20-44 in the previous year across 102 MSAs from 1981 to 1996.<sup>5</sup> I merge this data with birth records

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

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.<sup>6</sup>

## V. Empirical Approach

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

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

where  $y_{m,t}$  is the birth rate in MSA  $m$  in year  $t$ ,  $Z_{m,t}$  is AIDS risk in the MSA,  $\mathbf{X}_{m,t}$  is a set of controls described below, and  $\gamma_m$  and  $\delta_t$  are MSA and year fixed effects, respectively.  $\beta_1$  is the coefficient of interest. In order to interpret  $\beta_1$  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  $\beta_1$  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 6E.<sup>7</sup>

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

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

7. 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 2018; De Chaisemartin and d'Haultfoeuille 2020). In Appendix B I show that heterogeneous effects overtime and the effects of later treated groups suggest my primary estimates are an underestimate of the true effect. I also show that results are robust to the inclusion of MSA-specific linear trends as well as event study analyses around AIDS incidence thresholds.

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.<sup>8</sup> I define a set of additional controls that include demographic characteristics and state policy changes that likely affect birth rates. These include state-level data on the unemployment rate (US BLS 2022), the incarceration rate among men and women (US BJS 2020), the maximum welfare benefit for a family of four (Moffitt, Phelan, and Winkler 2020), and whether the state Medicaid plan covers abortion (Myers and Ladd 2020). 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).<sup>9</sup> I also control for the crack cocaine epidemic using a crack index developed by Fryer et al. (2005).<sup>10</sup>

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 (Solon, Haider, and Wooldridge 2015).<sup>11</sup> Additional robustness tests exploit characteristics specific to the setting of the AIDS epidemic. I show that the relationship between AIDS risk and birth rates is driven by AIDS diagnoses in women and heterosexual men, and that birth rates are unaffected by AIDS diagnoses in homosexual and bisexual men. I analyze the effect of AIDS risk on gonorrhea incidence to show that estimates are not driven by unobserved sexual behavior. I also analyze the effect of AIDS risk on abortion rates to show that changes in birth rates are not driven by changes in abortion likelihood. Finally, I exploit the 10-year average latency period between HIV infection and AIDS diagnosis to test for a spurious relationship.

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

9. Further information on control variables is available in Appendix C. Estimates are robust to concerns about policy changes between 1993 and 1996, including welfare reform, Medicaid family planning waivers, changes to abortion restrictions following *Planned Parenthood v. Casey*, and the possible early roll-out of HAART in some markets. In Appendix B, I show that results are robust to ending the sample in 1992, prior to these changes.

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.

11. In Appendix Table A2, I also show that results are robust to a Poisson specification and to limiting the sample to MSAs contained within a single state.

## VI. Results

### *A. Effect of AIDS Risk on Birth Rates*

Empirical results for equation (1) are presented in Table 2, columns (1) through (3). For this specification and all subsequent regressions, I estimate robust standard errors that are clustered at the MSA level. I find that AIDS incidence in women in the previous year has a positive and statistically significant effect on birth rates across all three specifications. I find that estimates are similar in magnitude but more conservative when weighting by female population. When including the full set of controls, I estimate that every additional AIDS diagnosis in women 20-44 per 100,000 women led to an increase in births of 0.0393 per 1,000 women. I use this estimate to predict the difference in birth rates had AIDS incidence been zero in every year. I find that births among adult women increased on average by 0.32 births per 1,000 women per year for a total of 221,820 additional births between 1981 and 1996.

### *B. Effect of AIDS by Sexuality*

Despite the inclusion of fixed effects and controls, we might still be concerned that unobservable changes within MSAs are violating the identifying assumption by shifting both AIDS risk and birth rates. Specifically, we might be worried that the AIDS epidemic as a whole affected cultural attitudes (Fortin 2015; Mansour and Reeves 2022). If this were the case, we would expect to find a positive relationship between birth rates and AIDS incidence in homosexual and bisexual men, given that the majority of AIDS cases were among this group. In contrast, if women are adjusting their sexual behavior in response to their true risk of infection, we would expect most of the effect to come from incidence of diagnoses in women or heterosexual men.<sup>12</sup> To test this alternative hypothesis, I analyze the

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12. Analyzing data separately by sexual identity 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.”

effect of AIDS incidence among people of different sexual identities on birth rates.

Results are presented in columns (4) through (9) of Table 2. I find that AIDS diagnoses in both women and heterosexual men result in an increase in births. In comparison, I find there is no effect of AIDS diagnoses in homosexual and bisexual men on births. Thus, instead of adjusting behavior in response to the AIDS epidemic as a whole, women are adjusting their behavior in response to their specific risk of infection, as proxied by the incidence of AIDS in potential sexual partners in their area.

### *C. Effect of AIDS on Other STIs*

Another alternative hypothesis argues that women are engaging in more condomless sex, which increases both AIDS incidence and birth rates. If the increase in AIDS incidence and birth rates are both driven by an increase in condomless sex, then the incidence of other STIs would also increase. I analyze the effect of AIDS incidence on other STIs using state-level data on gonorrhea incidence from 1984 to 1996. These data are publicly available via CDC WONDER (US DHHS 2015). Unfortunately, MSA-level gonorrhea data are only available after 1995. Data on syphilis are also available at the state level during the time period. I use gonorrhea as a measure of other STIs because it is much more common than syphilis and less concentrated geographically (Chesson, Harrison, and Kassler 2000).<sup>13</sup>

I analyze the effect of AIDS risk on gonorrhea incidence in women. Results are presented in Table 3, Panel A. I find no evidence that gonorrhea incidence increases in response to AIDS incidence. Across all specifications, AIDS risk has a negative though statistically insignificant effect on gonorrhea incidence. This result contradicts the hypothesis that higher births and AIDS incidence are both due to unobserved increases in condomless sex among women. If births and AIDS are increasing due to increases in sexual activity, we would also expect gonorrhea to increase. In contrast, results are suggestive of a decrease in gonorrhea in women.

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13. Chlamydia data is also included in the CDC WONDER STD Morbidity Database. However, chlamydia diagnoses were not required to be reported to the CDC until 1988 (Worboys 2019).

#### *D. Effect of AIDS on Abortion Rates*

A final alternative hypothesis argues that the birth rate is increasing because the abortion rate is decreasing. In other words, holding the rate of pregnancy fixed, AIDS risk results in more 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 use data from the Guttmacher Institute on the number of abortions by state each year per 1,000 women aged 15-44 (Jones and Kooistra 2011).<sup>14</sup>

Results showing the effect of AIDS risk on abortion rates are presented in Table 3, Panel B. I find that AIDS risk has a positive and statistically significant effect on abortion rates.<sup>15</sup>. This result rejects the alternative hypothesis that birth rates are increasing because abortion rates are decreasing. In contrast, I find that the overall increase in pregnancies is larger than the increase in births. I estimate that there were 0.0694 additional pregnancies per 1,000 women in response to each additional AIDS case, and approximately 43 percent of these pregnancies resulted in abortion.<sup>16</sup> This share is on par with recent literature estimating that 42 percent of unintended pregnancies result in abortion (Finer and Zolna 2016) The positive effect of AIDS risk on abortion rates also indicates that at least some of the additional pregnancies that result from AIDS avoidance behaviors are unintended pregnancies (i.e., unwanted or mistimed).

#### *E. 10 Year Incubation Period*

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

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

15. Results are robust to a Poisson specification and limiting the sample to MSAs contained within a single state, as shown in Appendix Table A2.

16. Using the estimated coefficients from Table 2, column (3) and Table 3, column (6):  $0.0393 + 0.0310 = 0.0694$ .  $0.0301/0.0694 = 0.434$

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 birth rates from 1971 to 1986 are not predictive of AIDS incidence in women 10 years in the future (Table 4, column 1). Second, I show that birth rates 10 years prior are actually negatively correlated with birth rates from 1981 to 1996 (Table 4, column 2). In combination, these two results suggest that the positive effect of AIDS risk on birth rates found in the main analysis is not driven by unobservable differences in levels of sexual activity among women.

Finally, I add a variable for birth rates 10 years prior to my main regression specification shown in equation (1). This inclusion captures the characteristics of sexual behaviors at the time of HIV infection. To the extent that sexual behavior 10 years prior is correlated with current sexual behavior, controlling for prior birth rates removes the unobservable relationship between sexual behavior, AIDS, and births. Results are presented in Table 4, column (3). I find that AIDS risk has a positive and statistically significant effect on current birth rates, even when controlling for birth rates 10 years prior.

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

## VII. Underlying Mechanisms

What types of behavior changes could decrease the likelihood of AIDS infection but increase the likelihood of pregnancy and birth? One possibility is that women adopt condoms in favor of more effective contraceptives such as the Pill. Switching from effective contra-

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

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

Assume that the father's information is more likely to be recorded on a birth record if he was the mother's only partner. Following this assumption, I split the sample into two groups. *Partnered* are those who are married as well as those who are unmarried but the birth record includes the father's age. *Single* women are those who are unmarried and the birth record is missing information on the father's age. Results showing the effect of AIDS by the mother's partnership status are presented in Table 5. I further disaggregate the data by race to evaluate the possibility of heterogeneity in AIDS avoidance behavior across the population.

Among partnered white women, I estimate a positive and statistically significant effect

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

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

## VIII. Conclusion and Policy Implications

I show that the risk of AIDS led to an increase in birth rates for adult women. While prior research has shown that individual AIDS avoidance behaviors can affect AIDS rates in the population, this is the first paper in the US context to relate AIDS avoidance behaviors

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

This paper highlights that decisions about number of sexual partners are an important risk mitigation strategy for women. Number of partners interacts with both STI risk and pregnancy risk, and is likely endogenously chosen with contraceptive method and frequency of sexual activity. However, despite the far-reaching consequences of this decision on birth rates, abortion rates, and the spread of STIs, women's number of sexual partners is relatively understudied in economics. Furthermore, an emphasis on condoms as "safe sex" 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. An approach to reproductive health which takes into account the trade-offs between STI protection and pregnancy prevention is able to address this potential spillover effect.

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

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

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

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

**Table 2:** Effect of AIDS on Birth Rates

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

Notes: This table shows the effect of prior year AIDS incidence on birth rates per 1,000 women aged 20-44. AIDS incidence is defined as AIDS diagnoses per 100,000 women or men 20-44 in the previous 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 3:** Effect of AIDS on Gonorrhea and Abortions

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

Notes: This table shows the effect of prior year AIDS incidence on gonorrhea incidence (Panel A) and abortion rates (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 4:** Robustness Test: 10-year lag between HIV Infection and AIDS diagnosis

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

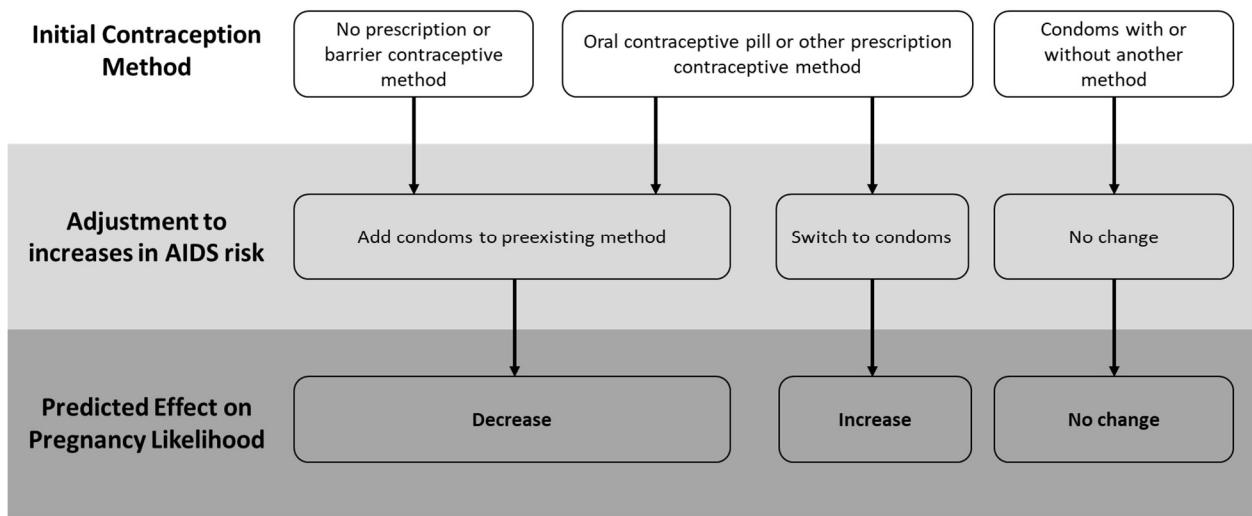
Notes: This table evaluates whether the positive relationship between AIDS risk and birth rates is driven by higher level of sexual activity in some MSAs by exploiting the 10-year incubation period of HIV infection. Column 1 shows that AIDS in women 10 years in the future is not predictive of birth rates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

**Table 5:** Heterogeneous Effects of AIDS on Births by Partnership Status

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

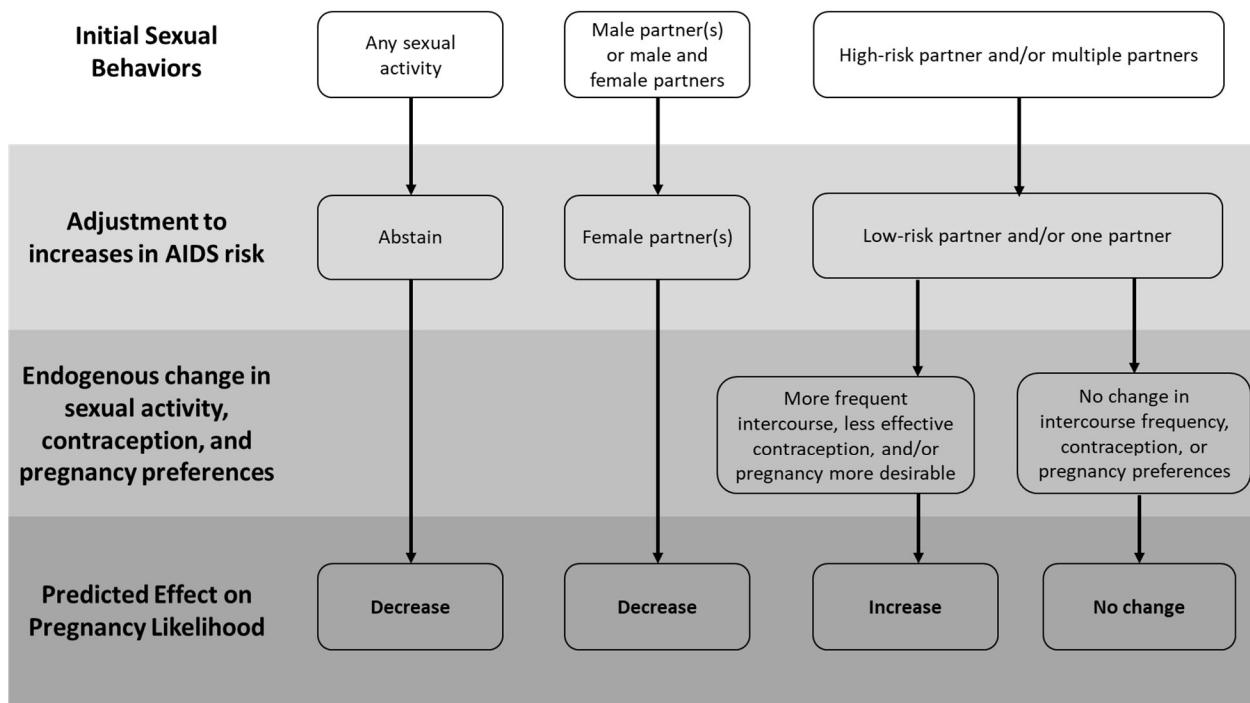
Notes: This table shows the effect of AIDS risk on births by mother's partnership status. Partnered women are those who are married or for whom the birth record includes information on the father's age. Single women are those who are unmarried and the birth record lacks information on the father's age.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

## Appendix A: Additional Figures and Tables



**FIGURE A1: THEORETICAL EFFECTS OF CONTRACEPTIVE CHANGE**

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



**FIGURE A2: THEORETICAL EFFECTS OF BEHAVIOR CHANGE**

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

**Table A1:** Summary Statistics

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

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

**Table A2:** Robustness to Sample Definition and Specification

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

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

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

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

## Appendix B: Robustness to Two-Way Fixed Effects Concerns

Recent advancements in the econometric literature highlight potential 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 (De Chaisemartin and d'Haultfoeuille 2020; Goodman-Bacon 2018). Specifically, heterogeneous effects overtime introduce the concern that results may be biased if later-treated units are compared to earlier-treated units. It is not obvious how to directly address these concerns in the case of a continuous treatment such as AIDS incidence. However, I use three alternative approaches to indirectly address concerns about the two-way fixed effects approach.

First, I address the concern of heterogeneous time effects by allowing the effect of AIDS incidence in women to vary by year. Results are presented in Figure B1. The effect of AIDS risk is largest and most significant between 1986 and 1992, with the effect attenuating overtime. To the extent we are concerned that later treated units are biasing results, this finding suggests that the inclusion of later treated units is downward biasing results and that the main results are a lower bound of the true effect. This time trend is also consistent with news coverage of AIDS in the U.S. which peaked between 1987 and 1991, with a downward trend beginning in 1992.<sup>1</sup> Additionally, this result addresses any concerns that health policy changes between 1993 and 1996 are upward biasing results.

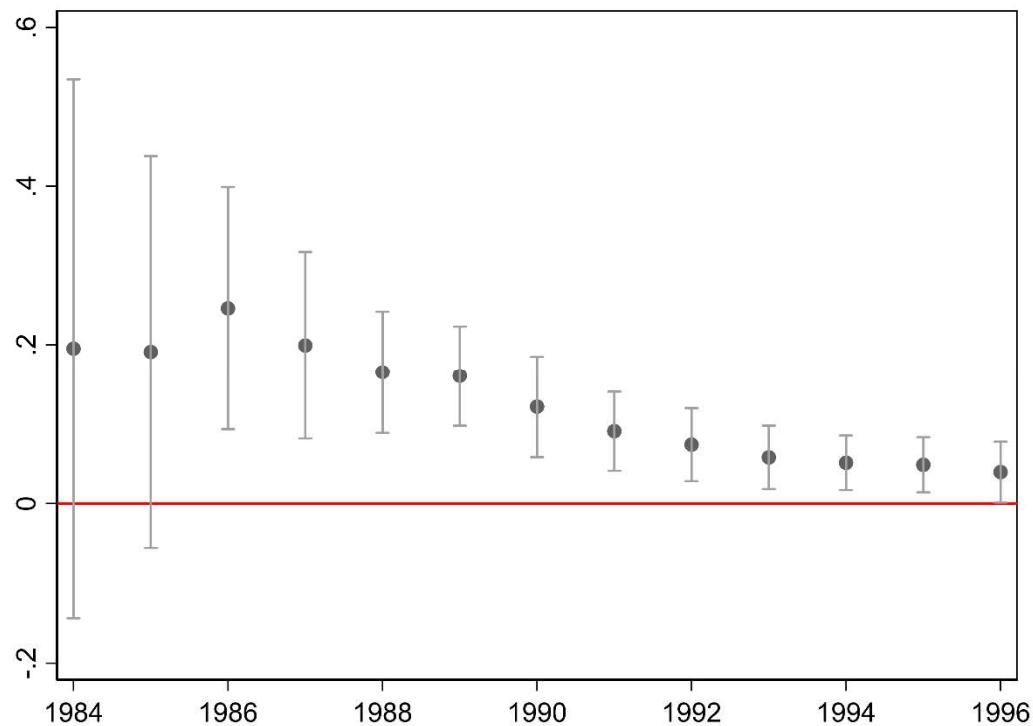
Second, I attempt to directly control for differences over time by including an MSA-specific linear time trend, with the sample expanded to cover 1970-1996. Results are presented in Table B1. Results are robust to the time trend inclusion, provided the year 1996 is excluded from the sample. This is consistent with the heterogeneous time effects shown in Figure B1 where effects are insignificant in 1996, and is consistent with policy changes in 1996, such as welfare reform, creating a downward bias on estimates.

Finally, I discretize AIDS incidence in order to test the robustness of results to recent advances in the difference-in-differences econometric literature. I create two thresholds: the first year that female AIDS incidence in an MSA reaches 1 case per 100,000 women, and the first year that female AIDS incidence in an MSA reaches 2 cases per 100,000 women. These thresholds are arbitrary, and it is not obvious that there is an optimal way to discretize AIDS incidence. However, panel 1 of Figure B2 demonstrate that these two thresholds produce a roughly normal distribution of treatment years in the sample.

Using these thresholds, I analyze the corresponding difference-in-differences regression using a Goodman-Bacon decomposition without controls and extending the sample to 1975-1996 to include more pre-treatment years. I find that the positive and statistically significant effect of AIDS arrival and spread on birth rates is attributable to earlier treated groups being compared to not yet treated groups. Once again, this further shows that any concerns regarding the comparison between later treated and already treated groups is leading to an underestimate of the true effect. Finally, I also use these thresholds to analyze the effects of AIDS risk with an event study design. I find that the specific year in which AIDS incidence reaches a given threshold is unrelated to pre-trends in birth rates, with positive and statistically significant effects on birth rates in post-treatment years as AIDS continues to spread.

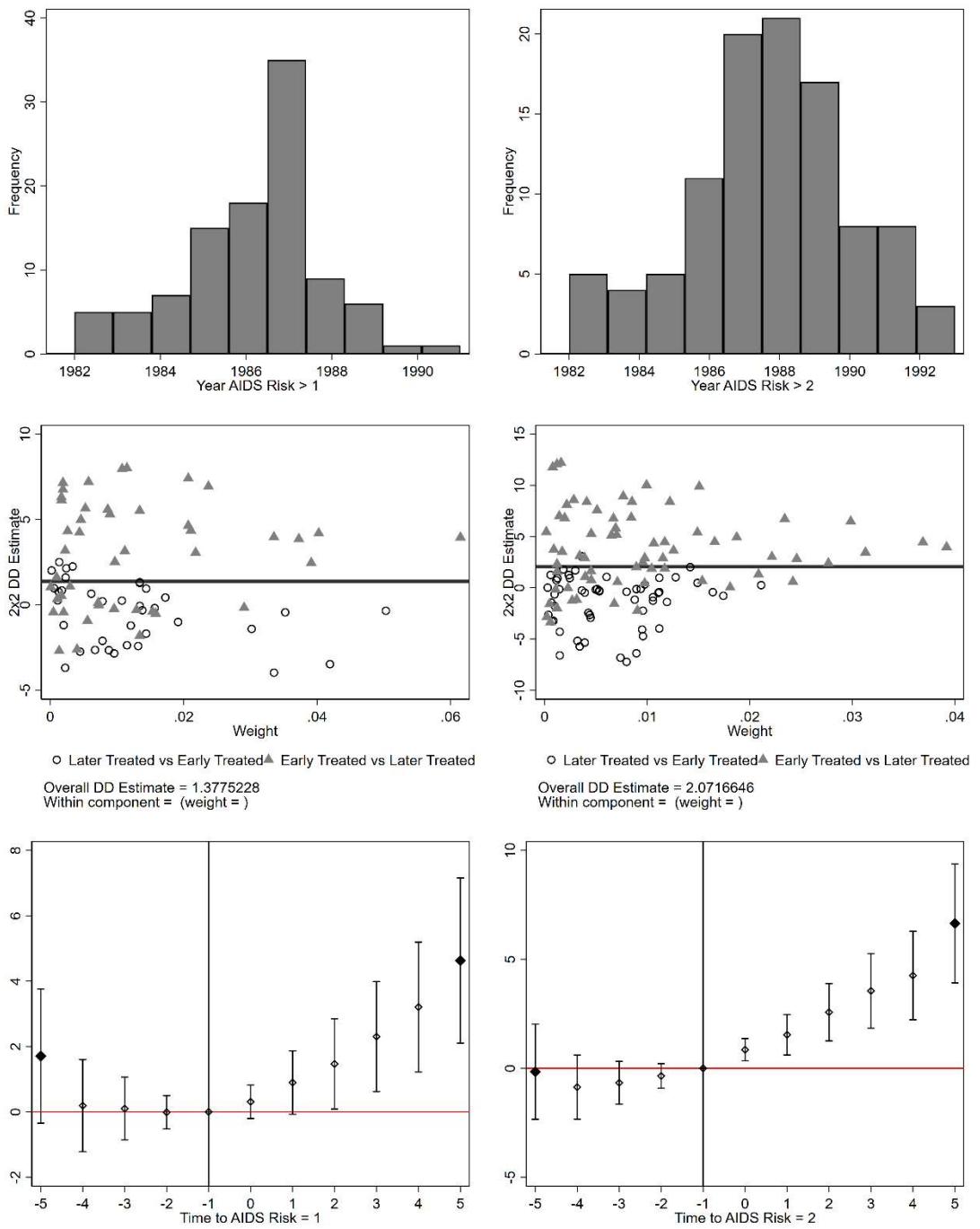
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<sup>1</sup> Brodie, M., Hamel, E., Brady, L. A., Kates, J., & Altman, D. E. (2004). AIDS at 21: Media Coverage of the HIV Epidemic 1981-2002. *Columbia Journalism Review*, 42(6), A1.



**FIGURE B1: HETEROGENEOUS EFFECTS OVERTIME**

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



**FIGURE B2: ROBUSTNESS TO BINARY TREATMENT DEFINITION**

Notes: This figure shows that results are robust to a discretized version of AIDS risk, specifically whether AIDS incidence has reached 1 or 2 cases per 100,000 women, respectively, and concerns about the two-way fixed effects approach.

**Table B1:** Robustness to Inclusion of Linear Time Trends

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

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

## **Appendix C: Data Sources and Sample Construction**

### **AIDS Public Information Data Set**

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

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

### **Natality Detail File**

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

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

### **Population Data**

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

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

### **Gonorrhea Surveillance Data**

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

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

### **Crack Cocaine Index**

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

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

### National Prison Statistics

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

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

### National Health Interview Survey AIDS Supplement

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

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

### Abortion Rate

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

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

### **Unemployment Rates**

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

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

### **Welfare Benefits**

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

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

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

### **Medicaid Coverage of Abortion**

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

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

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

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

### **Poverty Rates**

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

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

## **Education Levels**

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

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