Safer Sex?
The Effect of AIDS Risk on Birth Rates

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Abstract

The advent of the AIDS epidemic in the 1980s dramatically increased the cost of contracting a sexually transmitted infection (STI). Prior research shows that people responded to the AIDS epidemic by switching to sexual behaviors and contraceptive methods with lower likelihood of AIDS transmission. These behavioral adjustments also affect the likelihood of pregnancy and the incidence of other STIs. This paper provides the first evidence that the AIDS epidemic increased the birth rate and decreased the gonorrhea rate. I show that births among adult women increased on average by 0.5 births per 1000 women per year, for a total of 344,000 additional births between 1981 and 2001 due to AIDS avoidance behaviors. My analysis suggests that the overall estimates are driven by women avoiding AIDS by shifting to monogamous relationships.

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

JEL Classifications: I12, J13, J12, J16

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I. Introduction

Women face two potential health risks from sexual activity: acquiring a sexually transmitted infection (STI) and becoming pregnant. They can mitigate these risks through safer choices about sexual behavior and contraceptive use. 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, other choices 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 unprotected 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 in 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 (Kearney and Levine, 2009; Bailey, 2010; Goldin and Katz, 2002; Michael and Willis, 1976; Francis, 2008; Shah, 2013; Mulligan, 2016; Durrance, 2013), but the effect of increased STI risk on fertility has not previously been examined.

Understanding the effects of increased STI risk is particularly important given current trends in public health. The rapid rise of drug-resistant gonorrhea in the United States threatens an STI-driven epidemic similar to the AIDS epidemic in the 1980s (CDC, 2017; Bodie et al., 2019). The COVID pandemic of 2020 highlights both the cost and the challenges associated with containing infectious disease. The relationship between STI risk, sexual behaviors, and birth rates is a critical determinant of public health responses to drug-resistant

STIs. My research provides insight regarding how women might adjust their behavior in response to drug-resistant gonorrhea and can inform public health interventions to combat the spread of disease.

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 time period, the spread of AIDS was largely driven by male same-sex contact, and the average length of time between HIV infection and AIDS diagnoses 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 birth rates. At the height of the AIDS epidemic in 1993, the birth rate to adult women increased by 1.5 percent due to AIDS avoidance behaviors. Results are robust to the inclusion of city-specific year trends and controls for drug use, prostitution, poverty, education, and sex ratios. The magnitude of estimated effects is in line with the existing literature. I estimate the births increased by at most 1 birth per 1000 women. Similar in magnitude, Kearney and Levine (2009) find that expanded family planning coverage decreased the birth rate by 1.5 births per 1000 women.

I test whether the increase in birth rates is due to an increase in risky sex among women. I evaluate this alternative hypothesis using data on gonorrhea incidence among women. If women are having more unprotected sex, then we would expect increases in both birth rates and gonorrhea. In contrast, I find that AIDS risk leads to decreases in gonorrhea incidence. This result shows that women are successfully 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 a decrease in gonorrhea but an increase in births: women could switch from effective prescription contraceptives

to condoms, or women could choose to limit their number of sexual partners.

I provide evidence that the increase in births is due to women entering into monogamous partnerships to avoid AIDS. I make use of information from birth certificates on whether a father's age is recorded. For unmarried women, presence of a father's age on the birth record is a proxy for a cohabiting couple, while lack of a father's age suggests an absent father. I find that the increase in births is driven by an increase in births to unmarried, cohabiting women. There is no effect of AIDS on births with absent fathers. I supplement these results with survey data to argue that the increase in births is driven by women choosing to have only one sexual partner to protect from AIDS. Survey data shows 16% of unmarried women decided to stop having sex with more than one man (Mosher and Pratt, 1993). I estimate the birth rate would increase by 2 births per 1000 women if 16% of unmarried women switched from multiple partners to one partner. This is higher than my estimate of a 1 birth increase, but is consistent with heterogeneous responses in the population.

I analyze whether the effect of AIDS risk on birth rates varies across demographic groups. I find that the positive effect of AIDS risk on births is driven by women aged 30-44 years old and by white women. I find no effect of AIDS risk on births to Black women or women aged 20-29 years old. I provide a number of possible explanations for this heterogeneity, including differences in AIDS prevention knowledge and differences in the availability of potential partners.

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. Within the STI literature, this work is closest to studies such as Ahituv et al. (1996), Greenwood et al. (2019), Francis (2008), and Shah (2013) that examine behavioral changes in contraceptive use and sexual activity in response to STI risk. In the fertility literature, this paper is closest to studies that analyze the effects of changes in the costs of pregnancy prevention on fertility outcomes (Bailey, 2013; Bailey et al., 2012; Kearney and Levine, 2009; Levine, 2001).

Similar to this work are studies that examine the spillovers between fertility and STI risk,

for example, Sen (2003), Klick and Stratmann (2008), Colman et al. (2013), Mulligan (2016) Durrance (2013), Mallatt (2019), and Willage (2020). In comparison to this paper, these prior studies focus on the reverse relationship, 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 eventually 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 CDC tracked cases of rare pneumonia, cancer, and other opportunistic infections occurring predominately in young men with same sex partners in California and New York (Shilts, 2007). By July of 1982 the CDC had confirmed cases of hemophiliacs, intravenous (IV) drug users, and infants with the disease. Shortly thereafter there were at least two documented cases of women being exposed to the disease via opposite sex contact. (Heywood and Curran, 1988).

AIDS is the result of advanced infection with human immunodeficiency virus (HIV). HIV is found in semen, blood, vaginal and anal fluids, and breastmilk. The virus can be transmitted via sexual contact or sharing of needles, and from mother-to-child during pregnancy, childbirth, and breastfeeding (DHHS, 2020). Despite early misconceptions among the general public that AIDS was a disease 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 among ostensibly low-risk groups influenced public health efforts and knowledge of the disease. Early response to AIDS emphasized the idea of "universal vulnerability," or that everyone was at risk of contracting AIDS (De Cock et al., 2011).

Evidence suggests that public health fears of AIDS spreading rapidly via opposite sex contact influenced knowledge and behavior surrounding 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% of people 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 volatility with which the disease spread across the United States (Mann, 1992). The sudden and unstable spread of AIDS led to a large number of epidemiological articles attempting to track and predict prevalence of the disease (Steinberg and Fleming, 2000; Lam et al., 1996; Taylor, 1989). 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 in a contagious pattern, 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 1990s, the average time between infection with HIV and an AIDS diagnoses was 10 years (Osmond, 1998). As a result, outbreaks in cities were not being driven by current behaviors, but behaviors as much as ten 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 mentioned above, AIDS spread from city to city via same-sex male contact that occurred years prior. Second, the introduction 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% 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 unintended pregnancy. Condoms are the only method of contraception that can protect from AIDS transmission, but are not very effective in preventing pregnancy.[1] Condoms have a typical use failure rate of 18%, meaning that 18 out of 100 women will experience unintended pregnancy within the first year of using condoms as their primary contraceptive method. Prescription contraceptive methods such as the Pill and the IUD are more effective in preventing pregnancy: the Pill has a typical use failure rate of 9% and the IUD has a typical use failure rate as low as 0.2% (Trussell, 2004). If many women mitigated the risk of AIDS by switching from more effective methods 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.

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 low-risk partners (i.e. those who do not use IV drugs and do not have other concurrent partners). In this case the birth rate would likely increase as there is less incentive to use consistent contraception with a low-risk partner and frequency of sexual activity may increase with one partner. Furthermore, women may be more likely to keep a pregnancy with a low-risk partner or a sole, committed partner.

I model sexual decision making from the woman's perspective. This is a simplification, as consensual sexual contact requires bilateral decision making. There are three reasons for focusing on the women's decisions. First, the choice set of overlapping AIDS avoidance behaviors and pregnancy behaviors differs for men. Specifically, prescription contraceptives such as the Pill and the IUD are in the woman's choice set, but not directly in the man's choice

[1] In recent years, pre-exposure prophylaxis (PrEP) has offered another method of protecting from AIDS transmission. However, in the 1980s and 1990s condoms were the only option.

set. Similarly we might think that the costs and benefits associated with male condoms differ for men and women. Second, the biological HIV transmission probability via heterosexual contact differs for men and women. Women are much more likely than men to contract HIV from heterosexual contact (Nicolosi et al., 1994). Third, it is easier to draw inferences about individual behavior from birth rates when focusing on women - one birth closely approximates one mother. The same is not true for men, who could have multiple children in the same time period from different women.

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 both on which risk mitigation strategy is most prevalent in the population as well as 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 sexual behavior and contraceptive choices of individual women overtime. Unfortunately, such a data set does not exist for this time period.[2] However, we can still gain important insights from estimating the average effect. For example, a positive average effect indicates that some women are switching to condoms or limiting their number of sexual partners. A negative average effect indicates that some women are abstaining or adopting condoms in addition to their other contraceptive methods.[3] Additional analyses can then be used to disentangle possible mechanisms and to infer the share of women who adjusted their behavior accordingly.

[2]The NLSY79 would not work for this analysis for a number of 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 of women who consistently respond to sexual behavior questions is very small.

[3]One important caveat in using data on birth rates and STI incidence for this analysis is problems associated with ecological inference. When using group averages for regression analysis, omitted variable bias can be so severe as to reverse the direction of effects (King et al., 2004). I discuss this problem in more detail as it relates to this paper in Appendix C: The Ecological Inference Problem.

IV. Data Sources and Sample Construction

I estimate the theorized relationship between birth rates and AIDS risk using data from the Centers for Disease Control and Prevention (CDC). The AIDS Public Information Data Set (APIDS) contains information on the timing of AIDS diagnosis and the demographic of the patient. All data is aggregated within Metropolitan Statistical Areas (MSA) and 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. I further define *AIDS risk* for women of childbearing age as the AIDS incidence among women aged 20-44 in the previous year. This definition approximates both the risk at the time of conception as opposed to the time of birth, and the incidence of AIDS being spread by means other than same-sex contact.[4] To demonstrate that this definition is a reasonable proxy for AIDS risk, I use data from National Health Interview Surveys and ABC News polls that asked respondents to rate their chances of getting AIDS. I regress respondents' perceived risk of getting AIDS on local AIDS incidence. Results are presented in Appendix Tables A1 and A2. I find that local AIDS incidence is predictive of perceived risk. For women respondents, only AIDS incidence among women (my definition of AIDS risk) is predictive of perceived risk.

Using APIDS data, I create a panel of AIDS risk across 102 MSAs from 1981 to 2001.[5] I merge this data with birth records obtained from the CDC's restricted-access Natality Detail file, which includes information on birth date, as well as mother's county of residence, race, age, and marital status. I aggregate data by MSA-year to create a panel of birth rates from

[4] An alternative definition could use the previous year's AIDS incidence among men identifying as heterosexual. However, disclosing sexual preference is subject to reporting bias that may result in measurement error. As such, I choose to use AIDS incidence among women. See Table 3 for further discussion of different measures.

[5] APIDS data is available through 2002, however I stop my panel at 2001 in accordance with the availability of a key control variable - the crack cocaine index. See the data appendix for further information.

1969 to 2001. Due to the very low incidence of AIDS among adolescent women during this time period, I limit my sample to the population of adult women aged 20-44.[6] Birth rates are calculated as births per 1,000 women aged 20-44. The resulting panel of AIDS risk and birth rates contains 2,142 MSA-year observations across the years 1981 to 2001. Descriptive statistics for this panel are presented in Table 1. Figure 1 plots the difference in AIDS risk from 1981 to 2001 versus the difference in birth rates over the same time period. This figure indicates a positive relationship between birth rates and AIDS risk. I empirically examine this relationship by exploiting within-MSA, over-time variation in AIDS risk.

V. Empirical Approach

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

$$y_{m,t} = \beta_0 + \beta_1 Z_{m,t-1} + \gamma_m + \delta_t + \epsilon_{m,t} \tag{1}$$

where $y_{m,t}$ is the birth rate in MSA m in year t, $Z_{m,t-1}$ is AIDS risk in the MSA, and γ_m and δ_t are MSA and year fixed effects, respectively.[7] 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 MSA fixed effects. In other words, MSA fixed effects must be sufficient to capture any systematic differences in the cross-section that could be driving both trends in AIDS risk and trends in birth rates. I evaluate this identifying assumption using a series of empirical tests.

I test the robustness of estimates from equation (1) to a number of alternative empirical specifications. First, 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 et al., 2015). Second, I add a variety of controls which may plausibly affect both AIDS risk and birth rates, including

[6]For more information, see Appendix D: Births and AIDS Risk among Adolescent Women.

[7]Note that in all empirical analyses *AIDS risk* has the specific definition given in Section 4 as the AIDS incidence among women aged 20-44 in the previous year.

drug use, poverty, incarceration rates, prostitution, employment, sex ratios, and educational attainment. Third, I add MSA-specific linear trends to address concerns about within-MSA trends which could be affecting both AIDS risk and birth rates.

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 among women and heterosexual men, and that birth rates are unaffected by AIDS diagnoses among homosexual and bisexual men. I also analyze the effect of AIDS risk on gonorrhea incidence to show that estimates are not driven by unobserved sexual behavior. Finally, I exploit the 10 year average latency period between HIV infection and AIDS diagnosis to test for a spurious relationship.

VI. Results

A. Effect of AIDS Risk on Birth Rates

Empirical results for the fixed effects specification from equation (1) are presented in Table 2, column (1). For this specification and all subsequent regressions, I estimate robust standard errors that are clustered at the MSA level. I estimate that every additional AIDS diagnosis among women 20-44 in an MSA per 100,000 women led to an increase in births by 0.057 per 1000 women. Table 2, column (2) presents the results from a weighted version of equation (2). I weight regressions by the number of women aged 20-44 in the MSA each year. The estimated coefficient does not change when using a weighted specification.

Despite the inclusion of fixed effects, we might be concerned that there is some omitted factor that varies within MSAs over time and is driving results. 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. Other potentially confounding factors include changes to state Medicaid rules or differential trends in poverty rates or prostitution - all of which could plausibly influence both AIDS and birth rates. I address these concerns by adding a set of control variables to equation (1).

I control for the crack cocaine epidemic using a crack 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. To further control for use of IV drugs, I calculate MSA-level arrest rates for possession and sale of heroin and crack using data from the Uniform Crime Reports (UCR). I also use UCR data to calculate MSA-level arrest rates for prostitution, as well as total drug arrests.

During the 1980s and 1990s, many states changed Medicaid rules and expanded the population of Medicaid-eligible women (Dave et al., 2015). An ideal specification would capture these changes by adding a state-year fixed effect, however this is not possible due to sample size restrictions. On average, I observe only 2 MSAs per state, and, to the extent which AIDS incidence is correlated across MSAs in the same state, including state-year fixed effects would remove the majority of the variation in AIDS incidence. Instead, I use data from the Current Population Survey Annual Social and Economic Supplement (ASEC) to calculate the share of women 20-44 that are covered by Medicaid. I also use ASEC to calculate the race-specific weighted averages for: share of women living below the poverty line, the share of women who are married, the share of women with a high school degree, the share of women with a college degree, the share of women unemployed, the female labor force participation rate, and the share of men who are both employed and unmarried.[8] Lastly, I control for variation in the supply of sex partners using the female population share and Bureau of Justice Statistics on state-level male and female incarceration rates. Further information on control variables, including descriptive statistics (Table A3), can be found in the appendix.

Results with controls included are presented in Table 2, column (3). The estimated effect declines when adding controls, however I still find a positive and statistically significant effect

[8]To obtain representative averages, I drop MSAs for which I observe less than 10 women of the relevant demographic group. I fill in the missing MSA's using weighted averages by Census division. See the data appendix for further information.

of AIDS risk on birth rates. Estimated coefficients for the control variables can be found in Appendix Table A4, column (1).

As a final test of within-MSA trends that could be affecting both AIDS risk and birth rates, I add an MSA-year linear trend to equation (1). Results are presented in Table 2, column (4). For this regression, I use birth data from 1969 to 2001, resulting in a sample size of 3366. I once again find that there is a positive and statistically significant relationship between births and AIDS risk.[9]

To illustrate the relative magnitude of estimated effects, I compare actual birth rates to predicted birth rates under a counterfactual setting where AIDS risk is zero in every year. I use the specification with controls (Table 2, column (3)) for this analysis. Results are plotted in Figure 2. At the height of the AIDS epidemic in 1993, I estimate that the birth rate increased by about 1 birth per 1000 women, or 35,700 births, due to AIDS avoidance behaviors. In total, I estimate that there were 344,000 additional births between 1981 and 2001 due to the AIDS epidemic. Figure 3 also presents these results as a share of the actual birth rate. I estimate that the birth rate increased by as much as 1.5 percent in a given year due to the AIDS epidemic.

VII. Robustness Tests

A. Effect of AIDS by Sexuality

Despite the inclusion of MSA-specific linear trends and controls, we might still be concerned that unobservable changes in sexual behavior within MSAs are violating the identifying assumption by shifting both AIDS risk and birth rates. Specifically, we might worry that people are engaging in more sexual activity which is increasing both AIDS incidence and birth rates. I address this concern by exploiting characteristics specific to the AIDS epidemic.

[9]Results are also robust to using only AIDS cases diagnosed under the 1987 definition of AIDS to create a measure of AIDS Risk. See Appendix Table A5 for further information

One alternative hypothesis that could explain the results displayed in Table 2 is that AIDS risk is higher in areas that are "sexually liberal" (i.e. greater social acceptance of unprotected sex, multiple sex partners, same-sex partners, etc.), and, as a result, also have a higher birth rate. If this were the case, we would also expect to find a positive relationship between AIDS incidence among homosexual men and birth rates. 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 among women or heterosexual men.[10] To test this alternative hypothesis, I analyze the effect of AIDS incidence among people of different sexual identities on birth rates.

Results for this analysis are presented in Table 3. I find that AIDS diagnoses among adult women and heterosexual men both result in an increase in births. In comparison, I find there is no effect of AIDS diagnoses among 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 among potential sexual partners in their area.

B. Effect of AIDS on other STIs

Another alternative hypothesis argues that women are engaging in more unprotected sex, increasing both AIDS and birth rates. If the increase in AIDS incidence and birth rates are both driven by an increase in sexual activity, specifically sex without a condom, then incidence of other STIs would increase as well. I analyze the effect of AIDS incidence on other STIs using state-level data on gonorrhea incidence from 1984 to 2001. This data is publicly available via CDC WONDER. Unfortunately, MSA-level gonorrhea data is only available after 1995. Data on syphilis is also available at the state-level during the time

[10] 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."

period.[11] I use gonorrhea as a measure of other STIs because it is much more common than syphilis and less concentrated geographically (Chesson et al., 2000).

Using a fixed effects specification, I analyze the effect of AIDS risk on gonorrhea incidence among women. Results are presented in Table 4. I find that gonorrhea incidence decreases in response to AIDS incidence. Columns (2) and (3) repeat this analysis with a weighted specification and a regression with controls included, respectively. Across all specifications, AIDS risk has a negative and statistically significant effect on gonorrhea incidence. This result contradicts the hypothesis that higher births and AIDS incidence are both due to unobserved increases in sexual activity among women. If births and AIDS are increasing due to increases in sexual activity, we would also expect gonorrhea to increase. In contrast, I find that AIDS risk leads to a decrease in gonorrhea among women.

As a final robustness check, I exploit the incubation period of HIV infection. In the 1980s and 1990s, the average time between infection with HIV and an AIDS diagnoses was 10 years (Osmond, 1998). Thus, AIDS incidence in a given year is not driven by current sexual behaviors but by sexual behaviors ten years prior. I add to regressions a variable for birth rates ten years prior. This inclusion captures characteristics of sexual behaviors at the time of HIV infection. To the extent which 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 appendix Table A5. 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 and gonorrhea incidence. 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 the birth rate and a decline in gonorrhea incidence.

[11] 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).

VIII. Underlying Mechanisms

What types of behavior changes could decrease likelihood of AIDS infection but increase likelihood of pregnancy and birth? One possibility is that women adopt condoms in favor of more effective contraceptives like the oral contraceptive pill. Switching from effective contraception to condoms would decrease infection likelihood but increase pregnancy likelihood. Another possibility is that women respond to AIDS risk by limiting their number of sexual partners or choosing less risky partners (i.e. based on drug use, number of other partners). 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 keep 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.[12] I analyze the effect of AIDS risk on births to married and unmarried mothers per 1000 women. For each of these groups, I split the analysis by those for which the father's age is present in the birth record and those without a father's age recorded. I also split the sample of women by age group due to large differences in the rate of extramarital child-bearing by age. As with my main analysis, I use a fixed effects specification and control for the full set of control variables described in Appendix Table A8.

For both mechanisms (switch to condoms or limit number of partners) we expect that the birth rate to unmarried mothers would increase. However, the two mechanisms predict different results with regard to whether father's information is available. Assume that a father's information is more likely to be recorded on a birth record if he was the mother's only partner. Then, if many women are switching to only one partner, the number of births with father's age present would increase. In contrast, we would expect no change in records

[12]Beginning in 1980, 41 states directly asked for mother's marital status. For the remaining 9 states and District of Columbia, the CDC inferred marital status by comparing surnames across the mother, father and child.

with father's age if women are maintaining their current number of partners but switching to condoms.

Results showing the effect of AIDS by mother's marital status are presented in Table 5.[13] I find that AIDS risk increased the birth rate and birth share to unmarried women aged 30-44, but only for the population of birth records which contained information on father's age. There is limited evidence of an increase in births to married women, but the effect goes away when including control variables. I find no effect of AIDS on births which lack information on the father. This result further contributes to the causal interpretation of results. If the increase in births and AIDS was driven by IV drug users or more unprotected sex, we would expect an increase in births which lack information on the father. I interpret the increase in births to unmarried mothers with present fathers as evidence that a large share of women are responding to the risk of AIDS by having only one sexual partner.

My analysis showing that women are limiting their number of sexual partners in response to AIDS risk is consistent with survey data. According to the 1988 National Survey of Family Growth (NSFG), 5.3% of married women and 37.5% of unmarried women reported changing their behavior to avoid AIDS. Furthermore, 16% 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).

IX. Magnitude of Estimated Effects

The main results presented in Section 6 suggest that the birth rate to adult women increased by anywhere from 0.5 births per 1000 women to 1 birth per 1000 women as a result of the AIDS epidemic. I argue that this result is largely driven by women who would have otherwise had multiple partners choosing to have only one partner, at which point their likelihood of pregnancy increases. However, it is difficult to interpret the relative magnitude

[13]Note that AIDS Risk and all control variables are now defined using age group specific data.

of this effect due to heterogeneity in both behavioral changes and women's probability of pregnancy. While the results point to many women limiting their number of partners, other women may switch to condoms or even abstain from sexual activity. Though I am not able to precisely estimate what share of women changed their behavior and how, I can evaluate the extent to which estimates appear reasonable in magnitude.

I compare my estimates to those in the existing literature regarding changes in birth rates. For example, we would expect that the advent of the oral contraceptive pill had a much larger effect on birth rates than the AIDS epidemic. Bailey (2010) estimates that the legalization of the oral contraceptive pill in the 1960s decreased the rate of marital childbearing by 13 births per 1000 women. As expected, the effect of the Pill on birth rates is much larger than the estimated effect of AIDS risk.

Kearney and Levine (2009) analyze the effect of expanded Medicaid family planning on births. They find that expanded family planning coverage decreased the birth rate by 1.5 births per year per 1000 women. It seems implausible that the risk of AIDS infection would have as large an effect on birth rates as Medicaid coverage. However, Medicaid family planning waivers only increased the share of covered women by 5.3 percentage points. When we consider that potentially all women were "treated" by the heightened risk of AIDS infection, the estimated magnitudes seem more reasonable.

If we assume that all women are treated by the increased risk of AIDS, what share of women would need to change their behavior to justify the estimated effects (i.e. what share of women were "compliers" (Imbens and Angrist, 1994))? According to the NSFG, 16% of sexually-active, unmarried women reported they "stopped having sex with more than one man" to avoid AIDS. Is this share large enough to justify the estimated effects?

To answer this question, I use data from the National Longitudinal Surveys of Youth 1997 (NLSY97) for the years 1998 – 2011. Though this time period is later than the height of the AIDS epidemic, the NLSY97 is an ideal data set because it specifically asks about number of sexual partners. I create an indicator variable which describes whether a sexually-active

woman reports an additional biological child in the next survey round and then calculate the weighted mean for a variety of groups.

Across all 89,573 individual-year observations, the probability that a woman aged 20-30 will have an additional child by the next survey round is 0.0668, resulting in 66.8 births per 1000 women.[14] This estimate very closely approximates the actual population fertility rate during the time period. I then divide the sample into women with 1 partner and women with 2-5 partners. The birth rate among these two groups are 107.8 births per 1000 women and 63.5 births per 1000 women, respectively. The birth rate is 44.3 births higher among women with only one sexual partner. Thus, to increase the birth rate by 1 birth, 22.6 women per 1000 women (or 2.2%) must switch from multiple partners to 1 partner (1/44.3*1000=22.6).

I repeat this exercise for unmarried women and for women who are single and not cohabiting. Among unmarried women, 7.5% would need to switch from 2-5 partners to 1 partner in order to increase the birth rate by 1 birth. Among single women, 16.6% would need to switch for an increase of 1 birth. These numbers are similar to the NSFG survey responses. If 16% of unmarried women stopped having more than one partner as reported in the NSFG, then the birth rate would increase by a little more than 2 births per 1000 women. Due to sample selection, this is likely an overestimate of the true effect. Women who ex ante prefer multiple partners may be more likely to take steps to prevent pregnancy, even in monogamous partnership. In comparison, my results predict that the birth rate increased by at most 1 birth due to AIDS avoidance behaviors. My lower estimate is consistent with the aforementioned upward bias in predictions from the NLSY, as well as heterogenous responses in the population, i.e. in addition to the women who decrease number of partners, there are women who abstain or adopt condoms in addition to their current method of contraception, which pulls down the average effect on birth rates.

The available evidence suggests that the magnitude of estimated effects are reasonable and consistent with approximately 16% of unmarried women opting to have only one sexual

[14]Due to the age of participants observed in the NLSY, I can only conduct this analysis using women aged 20-30.

partner to decrease their chances of AIDS exposure.

X. Heterogeneity Analysis

For the final analysis presented in this paper, I evaluate whether there are heterogeneous effects of AIDS risk on birth rates across different demographic groups. Results presented in Table 5 show that there is heterogeneity across age groups. I also evaluate heterogeneity across race. The effect of AIDS risk by race and mother's marital status for women aged 30-44 is presented in Table 6. I use race-specific and age-specific data to calculate AIDS risk, as well as relevant values for control variables.[15]. Results for women aged 20-29 are in Appendix Table A9. I find no effect of AIDS risk on births to women ages 20-29 across all demographic groups.

Among women aged 30-44 years old, I find varying effects across white women and Black women. Among white women, I find that births increase to unmarried women with present fathers. This is consistent with white women switching to monogamous partnerships to avoid AIDS. Among Black women, I find a positive and statistically significant effect of AIDS risk on births to married women, but no effect among unmarried women. We might think that the increase in births to married women is due to marriages that happen as a result of pregnancy and birth, however I find no effect of AIDS risk on marriage rates for any demographic group (see Appendix Table A10).

There are a number of possible explanations for the heterogeneous effects found across age and race. One possibility is that, even within the more narrowly defined demographic groups, heterogeneous behavioral responses are moving birth rates in opposite directions and resulting in an overall null effect. For example, if women aged 20-29 are equally likely to adopt condoms or switch to monogamy in response to AIDS risk, overall birth rates might remain unchanged.

It is also possible that preferences and costs associated with sexual activity differ for

[15]Descriptive statistics for race and age specific variables are presented in Appendix Tables A7 and A8

women in their 20s versus women in their 30s. For example, the implicit costs of entering into monogamous partnerships may be higher for women in their 20s, or perhaps there are less men of the same age willing to be monogamous. Costs and preferences over childbirth may also differ. We only expect women to adjust their behavior at the expense of pregnancy risk if the perceived cost of AIDS outweighs the perceived cost of a birth. If the cost of a birth is higher for younger women, then there might not be any behavioral change.

The heterogeneity in results across white women and Black women are even more puzzling. AIDS incidence among Black women was more than 10 times as large as that of white women during the 1980s and 1990s. Yet, I find no effect of AIDS risk among unmarried Black women, and only a small effect of AIDS risk on births to married Black women. I propose two possible explanations for these results.

First, though AIDS risk was much higher among Black women, it is possible that knowledge about AIDS prevention was lower among Black women. Table 7 presents results from multiple rounds of the National Health Interview Survey AIDS Supplement in the 1980s and 1990s. Respondents were asked to rate the effectiveness of both condoms and monogamy in preventing AIDS transmission during sexual contact. Among women aged 20-44, Black women were less likely than white women to rate these methods as very effective, and more likely to report they were unsure of the effectiveness of these methods.

Second, given that white women responded to AIDS by entering into monogamous partnerships, it is also possible that the racial disparity in estimated effects is due to differences in the number of men available for monogamous partnership. White unmarried women largely responded to the risk of AIDS by choosing to have sex with only one man. A necessary condition for this behavioral response is that there are men available for monogamous partnership. Given that the majority of sexual relationships occur between people of the same race or ethnic group, it is possible that the high incarceration rate of Black men prevented this condition (Laumann et al., 2000). It is well-documented that the high incarceration rate of Black men affects partnerships for Black women (Cohen and Pepin, 2018; Charles and

Luoh, 2010; Cornwell and Cunningham, 2008). Furthermore, Johnson and Raphael (2009) find that the high male incarceration rate among Black men explains the majority of the differences in AIDS incidence across Black and white women.

I investigate whether differences in male incarceration rates can explain the racial gap in estimated effects. One way to study this problem empirically is to consider treatment effect heterogeneity. Specifically, does the effect of AIDS risk on birth rates depend on the number of men available for a monogamous partnership? To answer this question, I estimate the marginal treatment effect across values of the male incarceration rate.

I use Gaussian kernel reweighting to more flexibly estimate the effect of AIDS risk on birth rates across the distribution of own-race male incarceration rates (Hainmueller et al., 2019). Results are presented in Figure 4. There is a positive effect of AIDS risk on birth rates for white women across the range of male incarceration rates, but no effect for Black women. But, as Figure 4 illustrates, the distribution of male incarceration rates among Black people is drastically different from that among white people.

The results presented in Figure 4 indicate that the choice set available to Black women was different than that of white women. Entering into a monogamous partnership is not an option if there are no men available for such a partnership. The differences in the choice set provide one explanation for finding a positive effect for white unmarried women and a null effect for Black unmarried women. It is possible that AIDS risk would have had a positive effect on births to Black unmarried women had the male incarceration rate among Black people been different. However, the racial differences in these two factors are so drastic that it is impossible to estimate the counterfactual effect of AIDS risk had Black women faced white women's choice set without using out-of-sample extrapolation.

It is worth highlighting that both of the possible explanations presented here indicate that social inequality is an important contributing factor for racial disparities in AIDS avoidance behaviors. Both differences in knowledge about AIDS and differences in incarceration rates are directly related to institutional design and structural racism. These factors are

highlighted in a 2012 report on HIV/AIDS inequality stating that, "The racial HIV gap and the racial health gap in general, is strongly correlated with the racial wealth gap, which in turn is the direct outcome of both historical and contemporary processes of segregation in housing, education, employment, and health care as well as racially skewed mass incarceration. In this way, race—as it intersects with poverty, gender, and sexuality among other factors—becomes the embodiment of a multifaceted social exclusion and the rationalization for massive health inequities" (Robinson and Moodie-Mills, 2012, pg. 2).

Further research is needed in this area to better understand racial disparities in AIDS avoidance behaviors among women, as well as the Black-white gap in AIDS incidence among women. In the meantime, policies should reflect the constraints that many women, especially Black women, face in avoiding HIV/AIDS. For example, policies that promote comprehensive sex education in public schools and polices that ensure modern HIV preventatives, such as PrEP, are accessible and affordable so that people can protect themselves from HIV regardless of partnership constraints.

XI. Conclusion and Policy Implications

I show that the risk of AIDS led to an increase in birth rates among adult women. While prior research has shown that individual AIDS avoidance behaviors can affect AIDS rates in the population, this is the first paper 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 number of sexual partners. Results on the effect of AIDS on births to unmarried women and survey evidence from the NSFG shows that the latter behavioral change is likely driving results, with the majority of women who change their behavior opting to have only one sexual partner to protect from AIDS. I find that women mitigate their risk of AIDS at the expense of their pregnancy likelihood. As a result, an unintended consequence of AIDS avoidance behaviors is an increase in birth rates.

The trade-offs between pregnancy prevention and STI protection are of particular impor-

tance for the development of screening guidelines for healthcare providers. Current screening guidelines promote a unidimensional idea of "safe sex" (i.e. condoms) and fail to address the variety of margins along which women adjust their sexual behavior to avoid STIs 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. These two interventions could be particularly important given current trends in reproductive health. The rapid increase in antibiotic resistant gonorrhea suggests that more people may undertake STI avoidance behaviors such as decreasing number of partners. On the other hand, the increased uptake of effective contraceptive methods like IUDs could result in lower condom use. An approach to reproductive health which 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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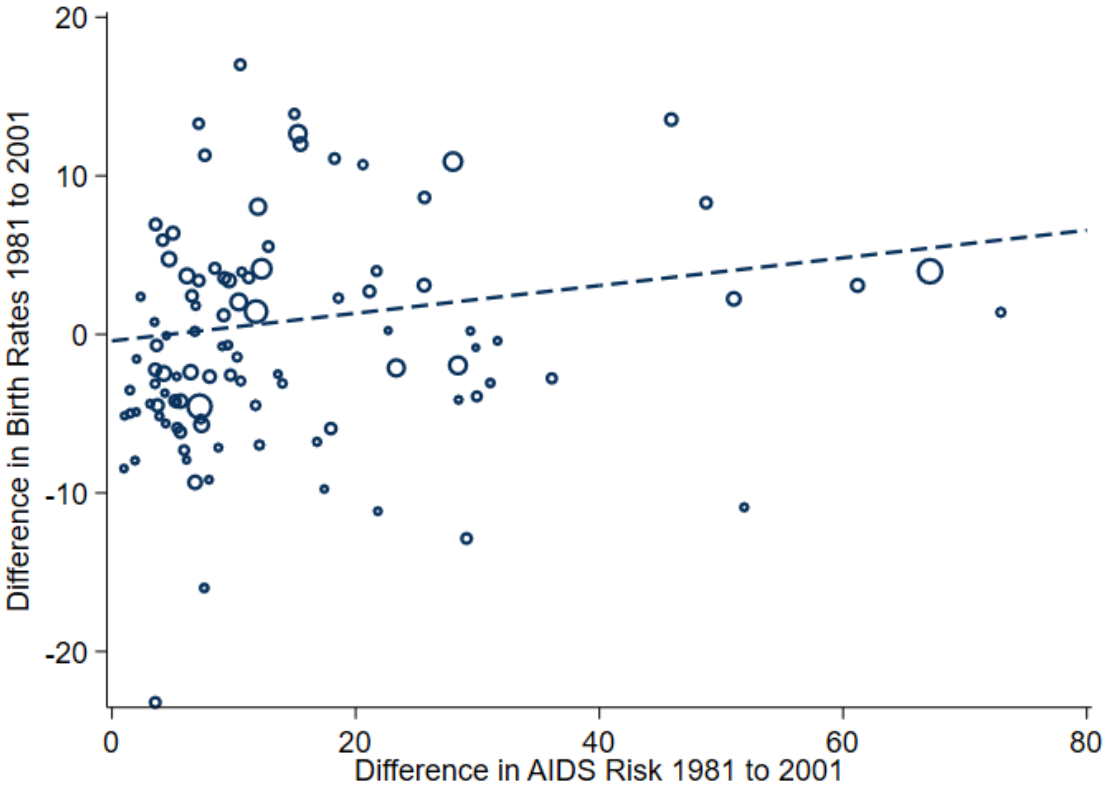

FIGURE 1: CHANGE IN BIRTH RATES VERSUS CHANGE IN AIDS INCIDENCE

Notes: This figure plots city level changes in birth rates from 1981 to 2001 versus cities' female AIDS incidence in 2000. Size of markers denotes relative female population size within a city in 2001. Raw trends indicate a positive relationship between birth rates and AIDS incidence. Note that female AIDS incidence in 2000 is equivalent to the difference in female AIDS risk between 1981 to 2001.

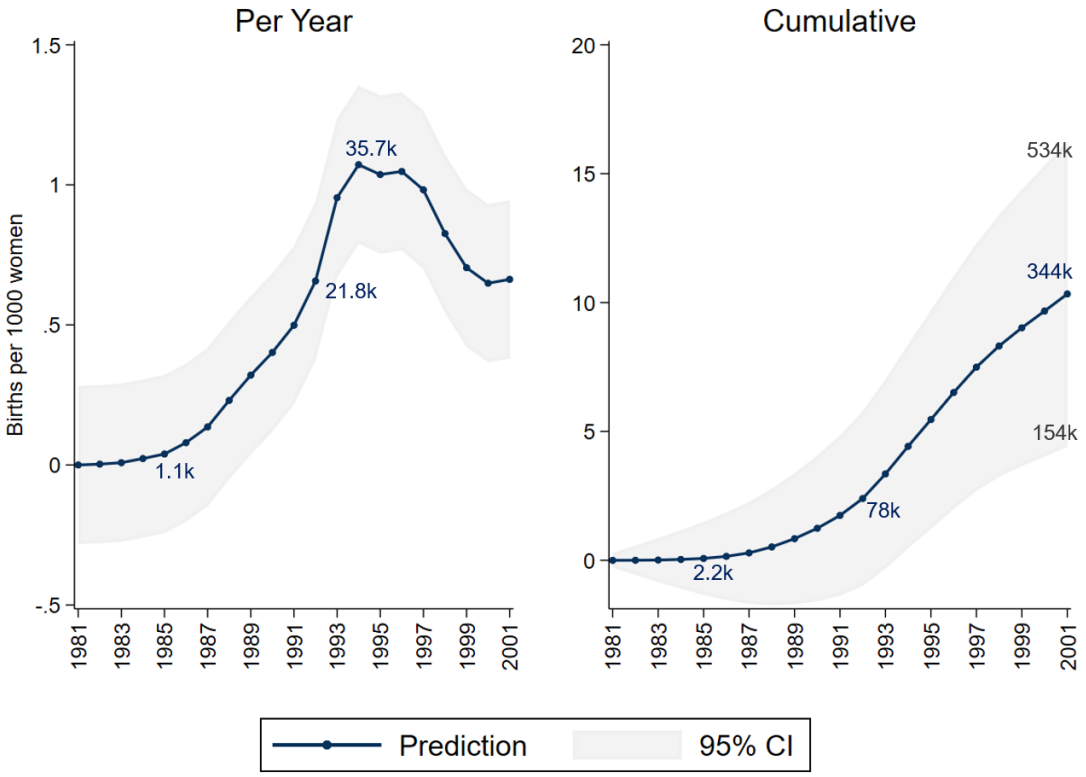

FIGURE 2: BIRTHS RESULTING FROM AIDS AVOIDANCE BEHAVIORS

Notes: This figure shows shows that additional births that result from AIDS avoidance behaviors based on the effect estimated in Table 1, Column 3.

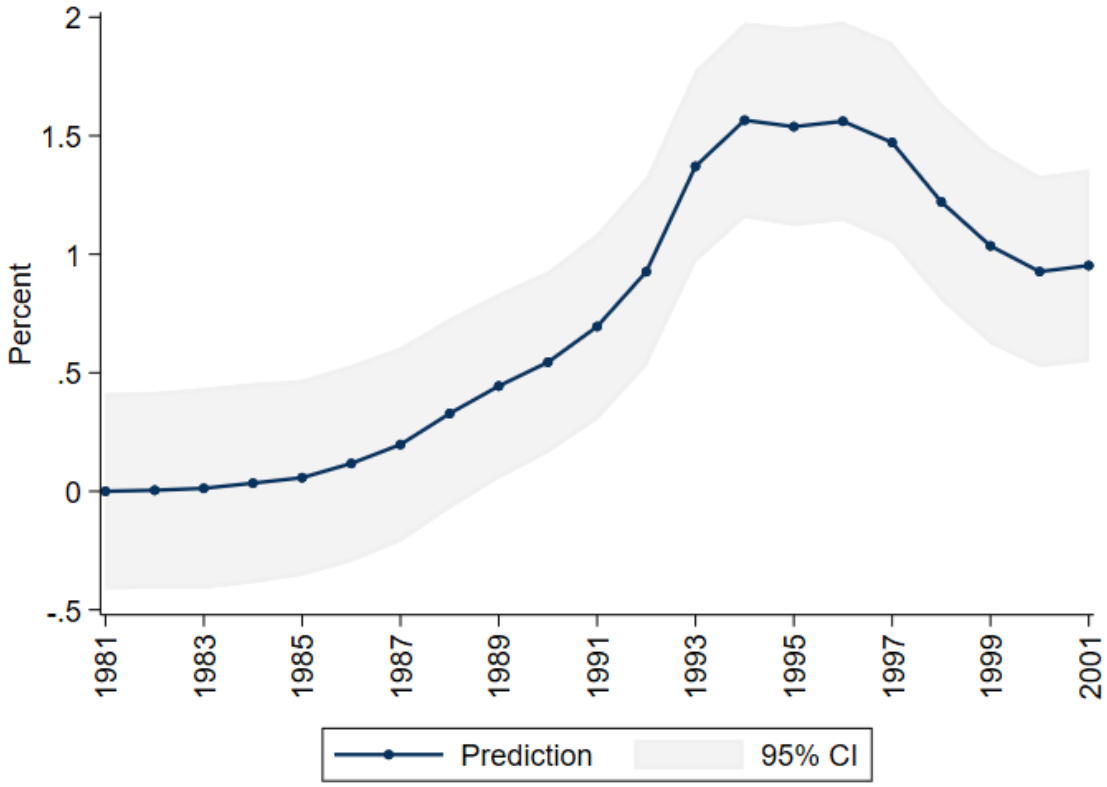

FIGURE 3: SHARE OF BIRTHS RESULTING FROM AIDS AVOIDANCE BEHAVIORS
Notes: This figure shows shows that additional births that result from AIDS avoidance behaviors as a percent of the total number of births in each year.

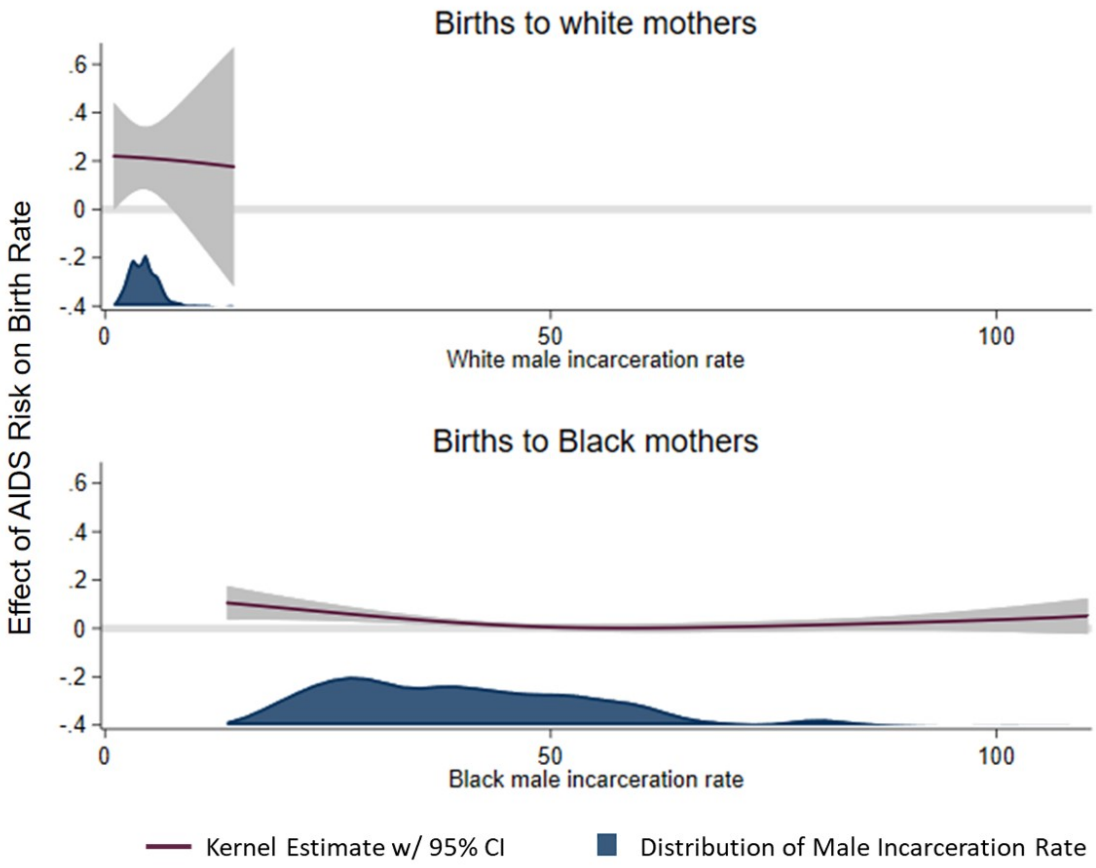

FIGURE 4: RACE DIFFERENCES AND MALE INCARCERATION RATE

Notes: This graph shows: (1) the effect of AIDS risk on birth rates by race across different levels of male incarceration rates, and (2) the distribution of male incarceration rates across white and Black men. For white women, AIDS risk has a positive and statistically significant effect on birth rates. I find that there is no effect of AIDS risk on births to Black women. However, Black women and white women faced very different constraints. Specifically, because male incarceration rates are drastically different across races, it is not possible to estimate how AIDS risk would have effected the birth rate among Black women had Black women faced male incarceration rates less than 20 (the range for white women) without making out of sample predictions.

Table 1: Descriptive Statistics

	Mean	Std. Dev.	Median	Min	Max
Births per 1000 women aged 20-44	68.51	9.88	66.73	48.78	125.85
AIDS Incidence in previous year...					
Total	14.65	9.60	18.98	0	203.11
Women 20-44	10.17	17.81	4.07	0	159.25
Homosexual/Bisexual Men 20-44	32.91	41.45	23.21	0	575.99
Heterosexual Men 20-44	15.25	24.96	6.95	0	218.06
N	2142				

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 1000 women. AIDS incidence is defined as number of new AIDS diagnoses per year per 100,000 people. I calculate the previous year's AIDS incidence among women aged 20-44 to create a measure of female AIDS risk.

Table 2: Effect of AIDS Risk on Birth Rates

	Primary Specification (1981-2001)			MSA Trends (1969-2001)
	(1)	(2)	(3)	(4)
AIDS Risk	0.057***	0.056***	0.038***	0.054***
	(0.015)	(0.010)	(0.014)	(0.017)
N	2142	2142	2142	3366
MSA and year FEs	X	X	X	X
Weighted		X		
Controls included			X	
MSA-specific linear trends				X

Notes: This table shows the effect of AIDS risk on birth rates. AIDS risk is defined as AIDS incidence in the previous year among women aged 20-44. There is a positive and statistically significant effect of AIDS risk on birth rates. Results are robust to a population-weighted specification (Column 2), controls for drug use, incarceration, prostitution, poverty, Medicaid coverage, sex ratio, and educational attainment (Column 3), and the inclusion of MSA-specific year trends (Column 4). When including MSA-specific year trends, the sample size increases because I include more pre-AIDS years. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 3: Effect of AIDS Incidence by Sexuality on Birth Rates

	Birth Rate among Women Aged 20-44					
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS incidence in previous year among...						
Women	0.057***	0.038***				
	(0.015)	(0.014)				
Heterosexual Men			0.047***	0.030***		
			(0.010)	(0.010)		
Homo/Bisexual Men					0.007	0.003
					(0.007)	(0.006)
N	2142	2142	2142	2142	2142	2142
MSA and year FEs	X	X	X	X	X	X
Controls included		X		X		X

Notes: This table presents the effect of AIDS incidence among people of different genders and sexuality on birth rates. AIDS incidence among women and heterosexual men leads to an increase in birth rates among women. There is no effect of AIDS incidence among homosexual and bisexual men on birth rates. This result is consistent with women adjusting their behavior in response to their true risk of AIDS, as opposed to unobservable factors such as attitudes towards sexual behavior or access to AIDS testing. All regressions include the full set controls as described in Appendix Table A2, as well as MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: Effect of AIDS Risk on Gonorrhea Incidence among Women

	Gonorrhea Incidence (1984-2001)		
	(1)	(2)	(3)
AIDS risk	-0.815**	-0.629*	-1.016***
	(0.315)	(0.335)	(0.293)
N	1835	1835	1835
MSA and year FEs	X	X	X
Weighted		X	
Controls Included			X

Notes: This table shows the effect of AIDS risk (previous year's female 20-44 AIDS incidence) on gonorrhea incidence among women. I find that AIDS risk has a negative and statistically significant effect on gonorrhea. This result rejects the alternative hypothesis that both AIDS risk and birth rates are increasing because women are having more unprotected sex, which would also result in higher gonorrhea incidence. Results are robust to weighting by female population size (column 2) and to the inclusion of controls described in appendix table A2 (column 3). All regressions include as MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 5: Effect of AIDS Risk on Birth Rates by Mother's Marital Status

	Married		Unmarried w/ Father		Unmarried no Father	
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Birth Rate Women Aged 30-44						
AIDS Risk	0.032**	0.019	0.011***	0.008***	0.004	0.004
	(0.015)	(0.015)	(0.004)	(0.003)	(0.003)	(0.002)
Mean of dependent variable	24.04	24.04	1.77	1.77	1.60	1.60
Effect as percent of mean	0.13%		0.62%	0.45%		
Panel B: Birth Rate Women Aged 20-29						
AIDS Risk	0.031	0.029	-0.003	-0.007	-0.011	-0.007
	(0.025)	(0.024)	(0.022)	(0.019)	(0.018)	(0.017)
Mean of dependent variable	53.71	53.71	10.17	10.17	10.02	10.02
N	2142	2142	2142	2142	2142	2142
MSA and year FEs	X	X	X	X	X	X
Controls included		X		X		X

Notes: Each cell in this table presents the results of a regression of birth rates by marital status and age group on AIDS risk. AIDS risk increases the birth rate to unmarried mothers with father present at birth, a proxy for cohabiting parents. This results confirms the mechanisms that women are responding to AIDS risk by entering into monogamous partnerships. The positive effect of AIDS risk on birth rates is limited to women aged 30-44 years old. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 6: Effect of AIDS Risk on Birth Rates by Race and Marital Status

	Married		Unmarried w/ Father		Unmarried no Father	
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Birth Rate White Women Aged 30-44						
AIDS Risk	0.107*	0.063	0.038***	0.026***	0.008	0.007
	(0.057)	(0.051)	(0.011)	(0.009)	(0.008)	(0.007)
Mean of dependent variable	25.19	25.19	1.51	1.51	1.01	1.01
Effect as percent of mean	0.42%		2.52%	1.66%		
Panel B: Birth Rate Black Women Aged 30-44						
AIDS Risk	0.011***	0.010***	0.002	0.000	0.000	0.000
	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)	(0.001)
Mean of dependent variable	14.05	14.05	3.91	3.91	4.97	4.97
Effect as percent of mean	0.078%	0.071%				
N	2142	2142	2142	2142	2142	2142
MSA and year FEs	X	X	X	X	X	X
Controls included		X		X		X

Notes: Each cell in this table presents the results of a regression of birth rates among women aged 30-44 years old by race and marital status on AIDS risk. AIDS risk increases the birth rate to unmarried mothers with father present at birth, a proxy for cohabiting parents, but only among white women. Among Black women, there is a positive effect to married women. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 7: Knowledge of AIDS Prevention among Women by Race

	Effectiveness of Condoms			Effectiveness of Monogamy		
	White	Black	Difference	White	Black	Difference
Very effective	0.290	0.280	0.010**	0.762	0.671	0.091***
	(0.454)	(0.449)	(2.27)	(0.426)	(0.470)	(14.19)
Somewhat effective	0.576	0.504	0.073***	0.182	0.212	-0.030***
	(0.494)	(0.500)	(15.34)	(0.386)	(0.408)	(-5.15)
Not at all effective	0.048	0.077	-0.029***	0.0226	0.0498	-0.027***
	(0.213)	(0.266)	(-13.49)	(0.149)	(0.218)	(-11.27)
DK how effective	0.067	0.110	-0.043***	0.0193	0.0423	-0.023***
	(0.251)	(0.313)	(-16.88)	(0.138)	(0.201)	(-10.27)
N	25,392	5,419		25,392	5,419	

Notes: This table shows NHIS respondents' perceptions of how effective condoms and monogamy are in preventing the transmission of AIDS. The sample is limited to women aged 20-44 year olds. White women are more likely than Black women to rate condoms and monogamy as very effective. Black women are more likely to rate condoms and monogamy as not effective, and are more likely to report they don't know how effective these methods are in preventing the transmission of AIDS.

Appendix A: Additional Tables

Table A1: Relationship between AIDS Incidence and Perceived Risk

	Full Sample		Unmarried Women Sample			
	Total AIDS		Female AIDS		Total AIDS	
	(1)	(2)	(3)	(4)	(5)	(6)
High	0.180*	0.204**	0.417**	0.424**	0.385	0.394
	(0.0991)	(0.101)	(0.198)	(0.199)	(0.242)	(0.243)
Medium	0.0379	0.0523	-0.0396	-0.0474	0.0387	0.0331
	(0.0457)	(0.0467)	(0.0954)	(0.0955)	(0.121)	(0.121)
Low	0.0298	0.0255	-0.0449	-0.0445	-0.0171	-0.0178
	(0.0182)	(0.0192)	(0.0414)	(0.0422)	(0.0537)	(0.0547)
None	0	0	0	0	0	0
	(.)	(.)	(.)	(.)	(.)	(.)
N	241,826	241,826	23,874	23,874	23,874	23,874
Region and year FEs	X	X	X	X	X	X
Controls		X		X		X
Share High	0.0059		0.01			

Notes: This table presents the effect of AIDS incidence on perception of AIDS risk using data from NHIS supplements. From 1987 - 1995, respondents were asked to rate their own chance of getting AIDS as high, medium, low, or none. This table presents the effects of a multinomial logit regression of responses on AIDS incidence. The unit of observation is the individual. AIDS incidence is calculated at the regional level. Regional AIDS incidence has a positive and statistically significant effect on the probability of rating own AIDS risk as high. Among unmarried women, only AIDS incidence among females is predictive of perceived risk. All regressions include region and year fixed effects. Columns (2), (3), and (4) include controls for educational level, race, and poverty status. Column (2) includes additional controls for sex and marital status. Standard errors shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A2: Relationship between AIDS Incidence and Perceived Risk

	Afraid AIDS for self ('85, '87, '90, '91)		AIDS risk for Public ('85, '87, '90)	
	linear	logit	linear	logit
	(1)	(2)	(3)	(4)
AIDS incidence	0.004*	0.022*	0.004*	0.026*
	(0.002)	(0.012)	(0.002)	(0.014)
Mean of dependent variable	0.25		0.83	
N	5206	5206	3946	3946
State and year FEs	X	X	X	X

Notes: This table presents the effect of AIDS incidence on perception of AIDS risk using data from ABC News polls. In certain years, polls asked the following yes or no questions: "Are you afraid that you make pick up the AIDS virus yourself?" and "Do you think AIDS is a threat to the general public in the United States?" This table presents the effect of AIDS incidence in the respondent's census division on answers to AIDS questions. Local AIDS incidence has a positive and statistically significant effect on perception of AIDS risk, both for one's self and for the general public. All regressions include state and year fixed effects. Robust standard errors are clustered at the state level and shown in parentheses. Columns (1) and (3) present results from a linear regression and columns (2) and (4) present results from a logit regression. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A3: Descriptive Statistics - Control Variables

	Mean	Std. Dev.	Min	Max
Gonorrhea diagnoses per 1000 women	195.47	115.61	6.46	664.71
Crack index	1.21	1.3	-1.01	8
Female incarceration rate per 1000 women	0.46	0.31	0.03	1.89
Male incarceration rate per 1000 women	8.29	3.8	1.5	22.45
Prostitution arrests per 100k people	142.56	284.15	6.91	4037.85
Heroin/Coke possession arrests per 100k people	193.31	244.01	3.56	3501.54
Heroin/Coke sale arrests per 100k people	120.52	129.15	3.56	2693.6
Total drug arrests per 100k people	638.52	1013.01	70.85	23042.59
Among women 20-44 years old...				
Female population share	0.5	0.01	0.46	0.54
Share below poverty level	0.13	0.06	0	0.52
Share married	0.58	0.07	0.21	0.92
Share on Medicaid	0.09	0.05	0	0.38
Labor force participation rate	0.74	0.07	0.44	0.96
Unemployment rate	0.06	0.04	0	0.37
Share with high school degree	0.66	0.21	0.17	1
Share with college degree	0.15	0.11	0	0.53
Share of men employed and unmarried	0.35	0.07	0.06	0.65
Among women 30-44 years old...				
Female population share	0.51	0.01	0.47	0.54
Share below poverty level	0.11	0.06	0	0.55
Share married	0.68	0.08	0.35	1
Share on Medicaid	0.07	0.05	0	0.43
Labor force participation rate	0.74	0.08	0.44	1
Unemployment rate	0.05	0.04	0	0.3
Share with high school degree	0.66	0.23	0.06	1
Share with college degree	0.17	0.11	0	0.62
Share of men employed and unmarried	0.25	0.08	0	0.62
Among women 20-29 years old...				
Female population share	0.5	0.02	0.44	0.54
Share below poverty level	0.16	0.08	0	0.63
Share married	0.42	0.11	0	0.9
Share on Medicaid	0.11	0.07	0	0.73
Labor force participation rate	0.73	0.09	0.12	1
Unemployment rate	0.08	0.06	0	1
Share with high school degree	0.67	0.21	0.05	1
Share with college degree	0.12	0.11	0	0.71
Share of men employed and unmarried	0.49	0.1	0	0.88
N	2142			

Notes: This table presents descriptive statistics for control variables used in Tables 2-5. See the data appendix for further information on data sources and sample creation.

Table A4: Control Coefficients

	Birth Rate Women 20-44 (1)	Gonorrhea rate among Women (2)	Birth Rate 30-44 Unmarried w/ father (3)
AIDS Risk	0.0382***	-1.016***	0.00835***
	(0.0141)	(0.293)	(0.00272)
Female population share	-367.8***	406.1	-61.74***
	(71.89)	(709.4)	(15.88)
Crack index	0.627***	12.72***	0.0960**
	(0.202)	(2.953)	(0.0403)
Share below poverty level	-1.819	20.29	0.376
	(2.995)	(37.37)	(0.694)
Share married	0.554	-16.89	0.438
	(1.811)	(23.85)	(0.439)
Share on Medicaid	-5.690	-87.99*	0.768
	(4.025)	(49.66)	(0.696)
Labor force participation rate	-2.149	48.98**	0.208
	(1.783)	(22.01)	(0.411)
Unemployment rate	-2.760	25.79	-1.006**
	(3.286)	(30.87)	(0.477)
Share with high school degree	-3.541*	33.34	-0.110
	(1.976)	(25.29)	(0.509)
Share with college degree	6.865***	-1.828	-0.955*
	(2.560)	(31.78)	(0.546)
Share of men employed & unmarried	0.647	-5.524	0.269
	(1.406)	(19.66)	(0.379)
Female incarceration rate	2.487	47.16*	0.685
	(2.429)	(27.88)	(0.504)
Male incarceration rate	-0.207	-2.170	0.0188
	(0.273)	(3.168)	(0.0512)
Prostitution arrests	-0.00123***	-0.00159	0.0000606
	(0.000430)	(0.0214)	(0.000272)
Heroin/Coke possession arrests	0.000466	-0.0127	0.0000277
	(0.000413)	(0.0103)	(0.000107)
Heroin/Coke sale arrestse	-0.0000500	0.0494***	0.000573**
	(0.00104)	(0.0128)	(0.000245)
Total drug arrests	0.0000688	-0.000767	-0.0000688**
	(0.0000579)	(0.00389)	(0.0000301)
N	2142	1835	2142

Notes: This table presents coefficients on control variables for regressions presented in the main analysis. See Appendix B for further information on control variable. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A5: Effect of AIDS Risk on Birth Rates

	Primary Specification (1981-2001)			MSA Trends (1969-2001)
	(1)	(2)	(3)	(4)
AIDS risk (1987 definition)	0.108***	0.108***	0.077***	0.101***
	-0.021	-0.023	-0.018	-0.018
N	2142	2142	2142	3366
MSA and year FEs	X	X	X	X
Weighted		X		
Controls included			X	
MSA-specific linear trends				X

Notes: This table shows the effect of AIDS risk on birth rates using the 1987 definition of an AIDS diagnosis. There is a positive and statistically significant effect of AIDS risk on birth rates. Results are robust to a population-weighted specification (Column 2), controls for drug use, incarceration, prostitution, poverty, Medicaid coverage, sex ratio, and educational attainment (Column 3), and the inclusion of MSA-specific year trends (Column 4). When including MSA-specific year trends, the sample size increases because I include more pre-AIDS years. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A6: Robustness Test: 10 year lag between HIV infection and AIDS diagnosis

	Birth Rate among Women Aged 20-44			
	(1)	(2)	(3)	(4)
Birth Rate 10 years prior	-0.083**	-0.090**	-0.101***	-0.105**
	(0.035)	(0.043)	(0.037)	(0.043)
AIDS Risk			0.062***	0.044***
			(0.015)	(0.014)
N	2142	2142	2142	2142
MSA and year FEs	X	X	X	X
Controls included		X		X

Notes: This table presents the results of an additional robustness check. I evaluate whether the positive relationship between AIDS risk and birth rates is driven by unobserved increases in sexual behavior by exploiting the 10 year incubation period of AIDS infection. I control for sexual behavior at the time of HIV exposure using birth rates ten years prior and find that the relationship between AIDS risk and birth rates among women remains positive. All regressions include the full set of race-specific controls as described in Appendix Table A2, as well as MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A7: Descriptive Statistics

	Mean	Std. Dev.	Median	Min	Max
AIDS Incidence in previous year...					
All women 20-44	10.17	17.81	4.07	0	159.25
All women 20-44 (1987 definition)	5.59	10.33	2.18	0	100.31
All women 30-44	11.79	21.67	4.31	0	220.89
All women 20-29	7.45	12.7	2.93	0	130.02
White women 20-44	3.06	4.51	1.56	0	61.75
White women 30-44	3.45	5.56	1.64	0	86.91
White women 20-29	2.37	3.63	1.01	0	30.06
Black women 20-44	41.02	66.49	15.51	0	595.95
Black women 30-44	51.43	85.49	17.43	0	833.33
Black women 20-29	25.93	46.91	7.00	0	554.17
N	2142				

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 1000 women. AIDS incidence is defined as number of new AIDS diagnoses per year per 100,000 people. I calculate the previous year's AIDS incidence among women aged 20-44 to create a measure of female AIDS risk.

Table A8: Descriptive Statistics - Race-specific Control Variables

	White Women				Black Women			
	Mean	Std. Dev.	Min	Max	Mean	Std. Dev.	Min	Max
Female incarceration rate per 1000 women	0.24	0.17	0.02	1.3	2.2	1.54	0.31	9.34
Male incarceration rate per 1000 women	4.27	1.85	0.99	14.45	38.7	15.68	13.7	110.24
Prostitution arrests per 100k people	78.7	122.2	0	2129.58	60.88	176.5	0	3062.09
Heroin/Coke possession arrests per 100k people	101.82	106.08	0	1370.42	89.93	181.87	0	2919.24
Heroin/Coke sale arrests per 100k people	55.23	59.7	0	1363.59	64.44	93.36	0	1321.29
Total drug arrests per 100k people	360.59	323.45	14.13	4514.84	269.88	764.33	0	18468.49
Among women 30-44 years old...								
Female population share	0.5	0.01	0.46	0.54	0.51	0.05	0.26	0.58
Share below poverty level	0.08	0.06	0	0.59	0.33	0.11	0	0.79
Share married	0.72	0.08	0.36	1	0.23	0.1	0	0.75
Share on Medicaid	0.05	0.04	0	0.35	0.29	0.11	0	0.73
Labor force participation rate	0.74	0.08	0.38	1	0.68	0.1	0.18	1
Unemployment rate	0.04	0.04	0	0.63	0.17	0.1	0	0.73
Share with high school degree	0.67	0.22	0	1	0.6	0.23	0.07	1
Share with college degree	0.18	0.12	0	0.68	0.06	0.06	0	0.55
Share of men employed and unmarried	0.24	0.08	0	0.62	0.46	0.1	0	0.93
Among women 20-29 years old...								
Female population share	0.49	0.01	0.43	0.54	0.52	0.05	0.22	0.58
Share below poverty level	0.13	0.07	0	0.59	0.26	0.1	0	0.7
Share married	0.46	0.11	0	0.91	0.42	0.1	0	0.87
Share on Medicaid	0.08	0.06	0	0.62	0.2	0.09	0	0.6
Labor force participation rate	0.74	0.09	0.26	1	0.76	0.09	0.35	1
Unemployment rate	0.06	0.05	0	0.56	0.09	0.06	0	0.45
Share with high school degree	0.68	0.21	0.06	1	0.6	0.26	0	1
Share with college degree	0.13	0.12	0	0.69	0.1	0.08	0	0.72
Share of men employed and unmarried	0.49	0.11	0	0.91	0.33	0.1	0	0.85

Notes: This table presents descriptive statistics for race specific control variables used for regressions in Table 6.

Table A9: Effect of AIDS Risk on Birth Rates by Race and Marital Status

	Married		Unmarried w/ Father		Unmarried no Father	
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Birth Rate White Women Aged 20-29						
AIDS Risk	0.046	-0.002	-0.024	-0.057*	0.025	0.019
	(0.063)	(0.061)	(0.035)	(0.033)	(0.028)	(0.025)
Mean of dependent variable	57.53	57.53	8.55	8.55	6.12	6.12
Effect as percent of mean				-0.67%		
N	2142	2142	2142	2142	2142	2142
Panel B: Birth Rate Black Women Aged 20-29						
AIDS Risk	0.015*	0.003	-0.017	-0.019	-0.009	-0.007
	(0.008)	(0.008)	(0.012)	(0.012)	(0.010)	(0.010)
Mean of dependent variable	31.40	31.40	21.60	21.60	29.71	29.71
Effect as percent of mean	0.048%					
N	2142	2107	2142	2107	2142	2107
MSA and year FEs	X	X	X	X	X	X
Controls included		X		X		X

Notes: Each cell in this table presents the results of a regression of birth rates among women aged 30-44 years old by race and marital status on AIDS risk. AIDS risk increases the birth rate to unmarried mothers with father present at birth, a proxy for cohabiting parents, but only among white women. Among Black women, there is a positive effect to married women. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A10: Effect of AIDS Risk on Marriage Rates

	Share of Women Married					
	All		White		Black	
	30-44	20-29	30-44	20-29	30-44	20-29
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS Risk	-0.00006	0.00012	-0.00047	0.00090	-0.00001	-0.00005
	(0.00020)	(0.00035)	(0.00052)	(0.00075)	(0.00005)	(0.00006)
AIDS Risk lagged	-0.00003	-0.00037	0.00014	-0.00071	0.00005	0.00005
	(0.00019)	(0.00026)	(0.00047)	(0.00088)	(0.00005)	(0.00009)
N	2142	2142	2142	2142	2142	2142
MSA and year FEs	X	X	X	X	X	X

Notes: This table shows the effect of AIDS risk on marriage rates using separate regressions of birth rates on current year's AIDS risk, previous year's AIDS risk, and the AIDS risk 2 years prior . I find no effect of AIDS risk on marriage rates.Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance 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. I also use APIDS for counts of AIDS diagnoses among women by age group (20-29 and 30-44) and race (white and Black). 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. As a robustness check, I also limit counts to AIDS cases diagnosed under the 1985 and 1987 criteria, and find similar results.

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.

Population Data

To create measures of AIDS incidence and birth rates, I use population data form 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.

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 the calculate the corresponding weighted average of gonorrhea incidence in each state.

Crack Cocaine Index

I use the crack 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. This data is available from 1980-2000. Since I lag all control variables by one year to account for the gestation period, including this control variable restricts the years in my final sample to 1981 to 2001.

Uniform Crime Reports Arrest Data

I use data from the Uniform Crime Reporting Program on arrests by race to calculate MSA-level arrest rates for drug-related offenses in every year. I take into account the number of months each agency reported to the UCR to calculate annual counts of arrests. In cases where I do not observe any agencies in a given MSA that report to the UCR, I use census division arrest rates to fill in missing observations. Arrest rates are calculated per 100,000 people.

Annual Social and Economic Supplement

I use the Current Population Survey (CPS) Annual Social and Economic Supplement (ASEC) to create multiple control variables. The ASEC is an annual household survey that asks respondents questions regarding employment, poverty, and program participation. I use survey data from 1980 - 2000 on respondents' marital status, poverty status, employment status, educational attainment, and Medicaid enrollment. I use sampling weights to calculate weighted averages of responses in each MSA in every year among women aged 20-44. In cases where

the sample size in a given MSA-year is less than 10 people, I use the corresponding average among metropolitan areas in the MSA's census division. I repeat this exercise to create control variables for all race and age specific sub-samples.

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 the calculate the corresponding weighted average of incarceration rates in each state. Incarceration rates are calculated per 1,000 people.

ABC News AIDS Public Opinion Poll

ABC News Opinion Polls are publicly available on ICPSR and contain demographic information and geographic information at the census division level. In 1985, 1987, 1990, and 1991, these polls asked respondents if they were afraid of contracting the AIDS virus and if they thought AIDS was a threat to the general public.

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.

Appendix C: The Ecological Inference Problem

One challenge in conducting analyses with data on birth rates and STI incidence is problems associate with ecological inference. When using group averages for regression analysis, omitted variable bias can be so severe as to reverse the direct of effects (King et al., 2004). For example, in this setting, we might be concerned that AIDS risk is affecting the size of the female population. Since female population is the denominator used to calculate birth rates, the primary dependent variable for this analysis, unobservable correlations between AIDS risk and female population could bias results. In my setting, the effect of AIDS risk on birth rates could be positive if AIDS risk is resulting in a decrease in the female population, either due to death or migration.

There are two ways to address concerns about ecological inference. The first approach is to use known information to bound effects. For example, consider that prior to the development of effective treatment in 1996, most women who contract AIDS die within just a few years. Thus, we know that AIDS risk does decrease the female population. However, we can check whether this decrease is large enough to justify effects. In the extreme case, if all women who contract AIDS die within one year, each additional AIDS case would increase the birth rate by no more than 0.01 births per 1000 women. We can think of 0.01 as a lower bound at which we cannot reject the possibility that deaths due to AIDS are driving results. I estimate that every additional AIDS case per 100,000 women results in 0.05 more births per 1000 women, much larger than the lower bound of 0.01

In my setting, because I separately observe information on birth counts and population levels, I can directly test whether there is a relationship between AIDS risk and female population. Using data at the MSA-year level and a fixed effects specification, I regress female population on AIDS risk by demographic group. Results are presented in Table C1. I find no effect of AIDS risk on female population. These results suggest that the positive effect of AIDS risk on birth rates is driven by an increase in births, and not a decrease in population.

Table C1: Effect of AIDS Risk on Female Population

	Female Population					
	All		White		Black	
	30-44	20-29	30-44	20-29	30-44	20-29
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS Risk	213.688	-51.981	-101.818	35.987	12.442	4.471
	(168.316)	(94.416)	(168.940)	(146.020)	(8.617)	(3.095)
N	2142	2142	2142	2142	2142	2142
MSA and year FEs	X	X	X	X	X	X

Notes: This table shows the effect of AIDS risk on female population. I find no evidence that AIDS risk affected female population size for any demographic group. This result rejects the hypothesis that women are migrating away from cities with high AIDS incidence. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Appendix D: Births and AIDS Risk among Adolescent Women

This appendix presents results on the effect of AIDS risk on births to adolescent women aged 15-19. For the majority of my sample, AIDS incidence among women aged 13-19 years old is zero (see Table D1). Repeating my primary analysis on the sample of adolescent women, I find limited evidence of a decline in birth rates due to AIDS risk (see Table D2). However, this effect disappears when controlling for MSA-specific trends. Thus, the effect of AIDS risk on births to adolescent mothers is not robust to the overall downward trend in teen births during this time period.

Table D1: Descriptive Statistics

	Mean	Std. Dev.	Median	Min	Max
Births per 1000 women aged 15-19	52.44	15.4	52.72	15.57	109.22
AIDS Incidence in previous year...					
Women 13-19	1.07	2.71	0	0	34.45
N	2142				

Notes: This table presents descriptive statistics for the birth rate and AIDS incidence among adolescent women. The unit of observation is at the MSA-year level. Birth rates are calculated as live births per 1000 women. AIDS incidence is defined as number of new AIDS diagnoses per year per 100,000 people.

Table D2: Effect of AIDS Risk on Birth Rates to Adolescents Aged 15-19

	Primary Specification (1981-2001)			MSA Trends (1969-2001)
	(1)	(2)	(3)	(4)
AIDS risk	-0.126*	-0.100*	-0.153**	0.046
	-0.064	-0.057	-0.063	-0.043
N	2142	2142	2142	3366
MSA and year FEs	X	X	X	X
Weighted		X		
Controls included			X	
MSA-specific linear trends				X

Notes: This table shows the effect of AIDS risk on birth rates to adolescents aged 15-19. Columns (1)-(3) suggest that births to adolescent mothers declined in response to AIDS risk, However, this effect disappears when controlling for MSA-specific linear trends. The overall decline in births to adolescent mothers during this time period appears to be driving results. Furthermore, the median AIDS risk for adolescent women is the sample is 0 and the mean is only 1.07, suggesting that adolescents are not at risk of AIDS during this time period. When including MSA-specific year trends, the sample size increases because I include more pre-AIDS years. All regressions include MSA and year fixed effects. Robust standard errors are clustered at the MSA level and shown in parentheses. Statistical significance denoted by * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.