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 increase by 1 birth per 1000 women as a result of AIDS avoidance behaviors. My analysis suggests that the overall estimates are driven by unmarried white 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). In the latter case, the effects of those safer choices could be higher or lower birth rates. Empirical studies have validated the importance of economic cost considerations in determining sexual behavior, contraceptive choices, 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.

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 current 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. A 1 standard deviation increase in AIDS incidence increases the birth rate among women 20-44 years old by 1 birth per 1000 women. Results are robust to the inclusion of city-specific year trends and controls for drug use, prostitution, poverty, education, and sex ratios. The positive effect of AIDS incidence on births is driven by an increase in the birth rate among white women. The magnitude of estimated effects is in line with the existing literature. I estimate the births to white women increased by 0.5 to 1.5 births 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 which 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.

In contrast to the positive effect of local AIDS incidence on birth rates to white women, there is no statistically significant effect of AIDS risk on births to Black women. I provide suggestive evidence that AIDS incidence might have led to an increase in births to Black women had the Black male incarceration rate been similar in level to the white male incarceration rate.

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), 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.

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 race-specific 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

[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.

rates from 1969 to 2001 by mother's race and age. 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. Birth rates are calculated as births per 1,000 women aged 20-44. I further divide birth rates into births to all women, births to white women, and births to Black women.[6] 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:

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

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

[6] I an unable to calculate births to Hispanic women, as most states did not ask about Hispanic ethnicity on birth certificates during the 1980s.

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

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 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 and reverse causality.

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. On the sample of all women, 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. For ease of interpretation, Table 2 also presents standardized coefficients. A 1 standard deviation increase in AIDS risk results in an increase of 1.024 births per 1000 women.

Panels B and C of Table 2 show that the positive relationship between AIDS risk and birth rates is primarily driven by an increase in births to white women. I find no evidence of a relationship between AIDS risk and births to Black women. I estimate that a 1 standard deviation increase in AIDS risk increases births to white women by 0.091 standard deviations,

or 0.96 births.

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 (or white women and Black women, respectively). Coefficients remain positive and statistically significant for the sample of all women and white women. For white women and Black women, the standard errors increase, suggesting that weighting by within-group population may actually be increasing the heteroskedasticity of the error term as opposed to correcting for it (Solon et al., 2015). As such, the remaining regressions are unweighted.

B.Inclusion of Controls

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 (as well as white women 20-44 and Black 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. Following Charles and Luoh (2010) and Johnson and Raphael (2009), I exploit the fact that most sexual relationships occur between people of the same race or ethnic group and use own-race estimates for control variables (Laumann et al., 2000). Further information on control variables, including descriptive statistics, can be found in the appendix.

Results with controls included are presented in Table 2, column (3). I find a positive and statistically significant effect of AIDS risk on birth rates to white women, but no effect for Black women. Coefficient estimates are similar in magnitude to those from regressions without controls included. Estimated coefficients for the control variables can be found in Appendix Table A4.

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 these regressions, 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

[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.

between births and AIDS risk which is driven by an increase in births to white women.

It is puzzling that I find significant results for white women, but no results for Black women - particularly given that AIDS incidence was much higher among Black woman than white women during the 1980s and 1990s. In the next sections, I evaluate the robustness of and the mechanisms behind the positive effect among white women. For completeness of information, I also present further results for Black women. Using information gleaned from the additional analyses, I then evaluate the racial differences in estimates in section 10.

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.

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.[9] To test this alternative hypothesis, I analyze the effect of AIDS incidence among people of

[9]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."

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 to white mothers. Furthermore, the magnitude of estimates is similar to those displayed in Table 2. 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 period.[10] 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

[10] 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).

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 the regression with controls 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. Further, I test whether birth rates 10 years prior are predictive of AIDS risk. Results are presented in appendix Table A5. I find no effect of birth rates 10 years prior on AIDS risk. Additionally, 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, specifically white 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.

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. 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. 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. In addition to birth rates, I compute each group's share of total births out of 100. As with my main analysis, I use a fixed effects specification and control for the full set of control variables described in Appendix Table A3.

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 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. I find that AIDS risk increased the birth rate and birth share to white unmarried women, 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 share of births

to married women declines. 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 white 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 white adult women increased by anywhere from 0.5 births per 1000 women to 1.5 births 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 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.[11] 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

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

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. I estimate that the birth rate increases by at most 1.5 births. My slightly lower estimate is consistent with 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 white women opting to have only one sexual partner to decrease their chances of AIDS exposure.

X. Racial Differences in the Effect of AIDS on Birth Rates

In contrast to the positive effect of AIDS risk on birth rates to white women, I find no effect of AIDS risk on births to Black women. It is possible that the effect of AIDS risk on births to Black women is positive, yet imprecisely estimated. See that in Table 2, the coefficients on the standardized AIDS risk are not statistically different across white women and Black women. However, given that AIDS risk is standardized within race groups, the magnitude of the effect estimated for Black women may not be economically significant. For white women, I find that a 1 standard deviation increase in AIDS risk (or approximately 18 AIDS diagnoses per 100,000 people) results in an increase of 1 birth for 1000 women. The

corresponding result for Black women shows that an increase in AIDS risk of approximately 66 diagnoses leads to an increase in 0.9 births. Further complicating the interpretation of results for Black women are the estimated effects of AIDS risk on births by mother's marital status. Table 5, Panel B provides evidence that births to married Black mothers increased in response to AIDS, and that births to unmarried, cohabiting mothers declined. It is possible these two effects are cancelling each other, resulting in the overall imprecise zero effect.

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 2. 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 2 illustrates, the distribution of male incarceration rates among Black people is drastically different from that among white people.

The results presented in Figure 2 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.

Further research is needed in this area to better understand the effects of incarceration on AIDS incidence in Black communities. In the meantime, policies should reflect the constraints that many women, especially Black women, face in avoiding HIV/AIDS. For example, policies 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 white 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 importance 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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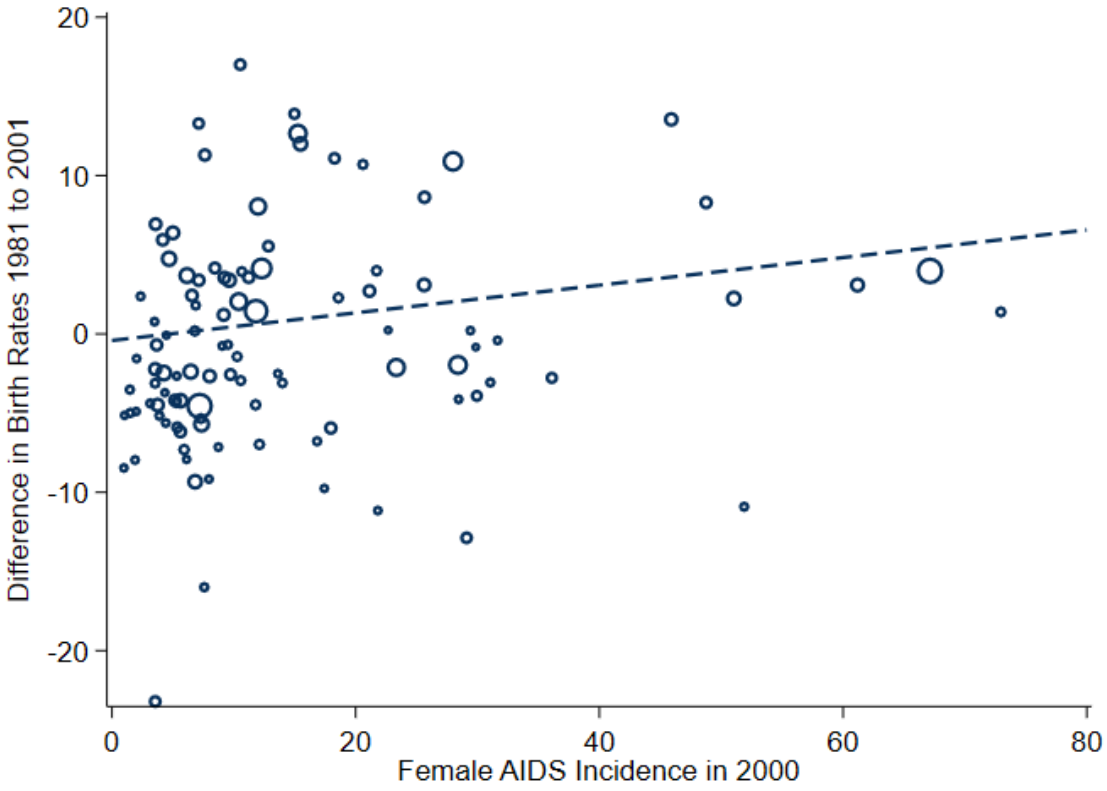

FIGURE 1: CHANGE IN BIRTH RATES VERSUS CHANGE IN AIDS INCIDENCE

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.

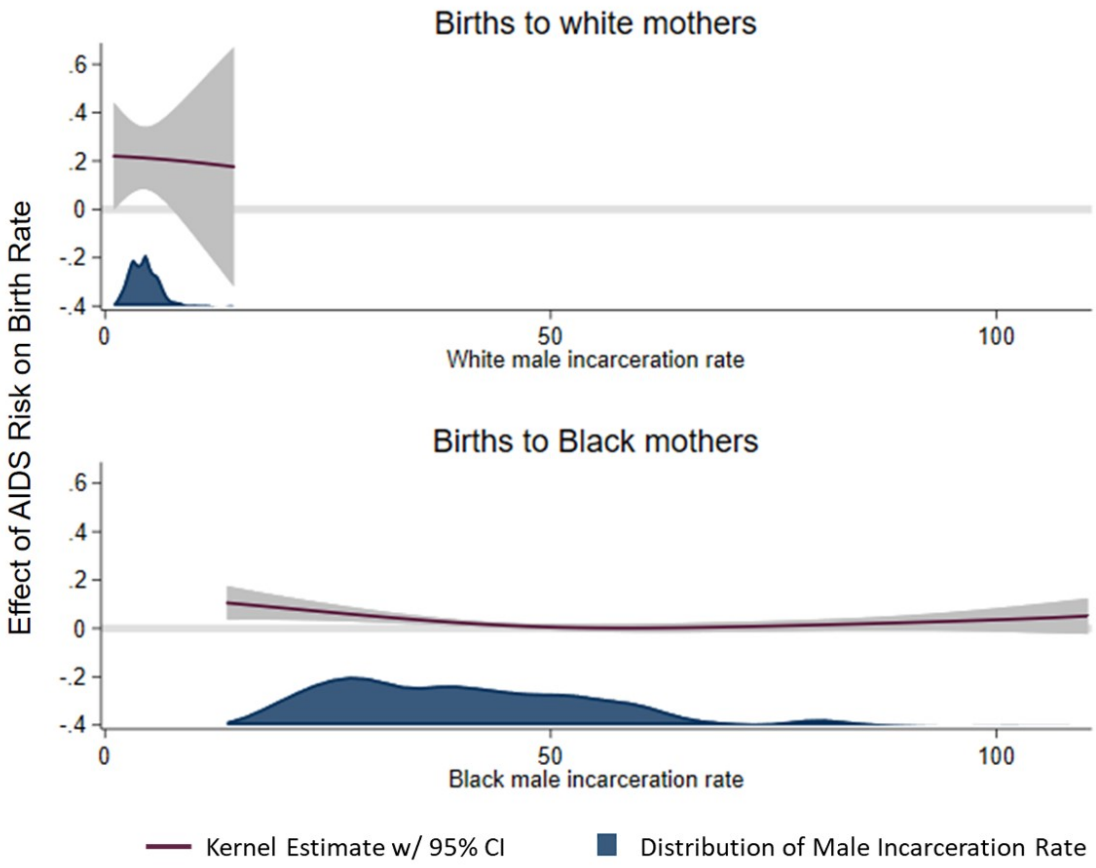

FIGURE 2: RACE DIFFERENCES AND MALE INCARCERATION RATE

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.	Min	Max
Births per 1000 women aged 20-44...				
All	68.51	9.88	48.78	125.85
White	67.39	10.51	46.59	126.59
Black	72.99	13.28	13.99	238.51
AIDS incidence in previous year...				
Total	14.65	18.98	0.00	203.11
Females 20-44	10.17	17.81	0.00	159.25
White Females 20-44	3.06	4.51	0.00	61.75
Black Females 20-44	41.02	66.49	0.00	595.95
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 females aged 20-44, white females aged 20-44, and Black females aged 20-44 to create a measure of own-race female AIDS risk. Both birth rates and AIDS risk are higher among Black women than white women.

Table 2: Effect of AIDS Risk on Birth Rates

	Primary Specification (1981-2001)			MSA Trends (1969-2001)
	(1)	(2)	(3)	(4)
Panel A: Birth Rate All Women 20-44				
AIDS risk	0.057***	0.056***	0.038***	0.054***
	(0.015)	(0.010)	(0.014)	(0.017)
Standardized AIDS risk	1.024***	1.003***	0.679***	0.812***
	(0.269)	(0.183)	(0.252)	(0.250)
Panel B: Birth Rate White Women 20-44				
AIDS risk	0.212***	0.331***	0.114**	0.179***
	(0.066)	(0.104)	(0.057)	(0.055)
Standardized AIDS risk	0.957***	1.489***	0.512**	0.695***
	(0.298)	(0.471)	(0.258)	(0.215)
Panel C: Birth Rate Black Women 20-44				
AIDS risk	0.014	0.007	0.001	0.013
	(0.009)	(0.011)	(0.006)	(0.009)
Standardized AIDS risk	0.903	0.439	0.034	0.711
	(0.596)	(0.764)	(0.422)	(0.489)
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 of the corresponding race. For each panel, row 1 shows the coefficient from a level-level regression. Row 2 scales the coefficient to show the effect of a 1 standard deviation increase in AIDS incidence on birth rates. There is a positive effect of AIDS incidence on the birth rate to white adult women. 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

	White Women			Black Women		
	(1)	(2)	(3)	(4)	(5)	(6)
AIDS incidence in previous year among...						
Women	0.114**			0.001		
	(0.057)			(0.006)		
Heterosexual Men		0.090**			0.005	
		(0.037)			(0.005)	
Homo/Bisexual Men			0.003			0.006
			(0.005)			(0.008)
N	2142	2142	2142	2142	2142	2142
MSA and year FEs	X	X	X	X	X	X
Controls included	X	X	X	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 white 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 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 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)
Standardized AIDS risk	-15.259**	-11.765*	-19.021***
	(5.891)	(6.268)	(5.490)
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

	Birth Rate		Share of Births	
	20-29 yrs	30-44 yrs	20-29 yrs	30-44 yrs
	(1)	(2)	(3)	(4)
Panel A: Birth Rate White Women 20-44				
Unmarried w/ father	0.010	0.033***	0.071***	0.055***
	(0.032)	(0.010)	(0.026)	(0.017)
Unmarried no father	-0.008	0.011	0.004	0.004
	(0.033)	(0.008)	(0.029)	(0.017)
Married	0.027	0.134**	-0.076**	-0.060**
	(0.065)	(0.052)	(0.030)	(0.024)
Panel B: Birth Rate Black Women 20-44				
Unmarried w/ father	-0.021*	0.000	-0.015*	-0.008
	(0.011)	(0.003)	(0.008)	(0.006)
Unmarried no father	-0.008	-0.000	-0.001	-0.009**
	(0.008)	(0.002)	(0.006)	(0.004)
Married	0.012**	0.014***	0.016***	0.018***
	(0.005)	(0.003)	(0.003)	(0.005)
N	2142	2142	2142	2142
MSA and year FEs	X	X	X	X
Controls included	X	X	X	X

Notes: Each cell in this table presents the results of a regression of birth rates or birth shares by race and marital status on own-race AIDS risk. Birth share is the percent (out of 100) of births with the corresponding marital status. Among white women, AIDS risk increases both the birth rate and birth share to unmarried mothers with father present at birth. 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 * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

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

	All Women 20-44				White Women 20-44				Black Women 20-44			
	Mean	Std.Dev.	Min	Max	Mean	Std.Dev.	Min	Max	Mean	Std.Dev.	Min	Max
Female population share	0.50	0.01	0.46	0.54	0.50	0.01	0.45	0.54	0.52	0.05	0.25	0.58
Share below poverty level	0.13	0.06	0.00	0.52	0.10	0.06	0.00	0.52	0.29	0.11	0.00	0.79
Share married	0.58	0.07	0.21	0.92	0.62	0.07	0.22	0.91	0.34	0.10	0.00	0.93
Share on Medicaid	0.09	0.05	0.00	0.38	0.06	0.05	0.00	0.33	0.24	0.11	0.00	0.75
Labor force participation rate	0.74	0.07	0.44	0.96	0.74	0.07	0.44	0.96	0.73	0.10	0.24	1.00
Unemployment rate	0.06	0.04	0.00	0.37	0.05	0.03	0.00	0.29	0.12	0.08	0.00	0.69
Share with high school degree	0.66	0.21	0.17	1.00	0.68	0.21	0.16	1.00	0.60	0.25	0.07	1.00
Share with college degree	0.15	0.11	0.00	0.53	0.16	0.12	0.00	0.62	0.08	0.08	0.00	0.50
Share men employed and unmarried	0.35	0.07	0.06	0.65	0.34	0.07	0.04	0.65	0.39	0.10	0.00	0.86
Female incarceration rate	0.46	0.31	0.03	1.89	0.24	0.17	0.02	1.30	2.20	1.54	0.31	9.34
Male incarceration rate	8.29	3.80	1.50	22.45	4.27	1.85	0.99	14.45	38.70	15.68	13.70	110.24
Prostitution arrests	142.56	284.15	6.91	4037.85	78.70	122.20	0.00	2129.58	60.88	176.50	0.00	3062.09
Heroin/Coke possession arrests	193.31	244.01	3.56	3501.54	101.82	106.08	0.00	1370.42	89.93	181.87	0.00	2919.24
Heroin/Coke sale arrests	120.52	129.15	3.56	2693.60	55.23	59.70	0.00	1363.59	64.44	93.36	0.00	1321.29
Total drug arrests	638.52	1013.01	70.85	23042.59	360.59	323.45	14.13	4514.84	269.88	764.33	0.00	18468.49
Crack Index	1.21	1.30	-1.01	8.00								
N	2142				2142				2142			
Gonorrhea incidence among women	195.47	115.61	6.46	664.71								
N	1835											

Notes: This table presents descriptive statistics for control variables used in Tables 3-7. See the data appendix for further information on data sources and sample creation.

Table A4: Effect of AIDS on Birth Rates - Control Coefficients

	All	White	Black
	(1)	(2)	(3)
AIDS Risk	0.0382***	0.114**	0.000507
	(0.0141)	(0.0572)	(0.00635)
In previous year...			
Crack index	0.627***	0.657***	1.539***
	(0.202)	(0.198)	(0.415)
Female population share	-367.8***	-437.4***	-126.7**
	(71.89)	(75.97)	(58.46)
Share below poverty level	-1.819	-1.900	-0.311
	(2.995)	(2.998)	(1.835)
Share married	0.554	0.963	0.256
	(1.811)	(1.683)	(1.854)
Share on Medicaid	-5.690	-1.733	-2.057
	(4.025)	(4.133)	(3.713)
Labor force participation rate	-2.149	-2.949	1.556
	(1.783)	(1.739)	(2.646)
Unemployment rate	-2.760	-3.691	-4.179
	(3.286)	(3.461)	(2.899)
Share with high school degree	-3.541	-5.913***	0.875
	(1.976)	(2.091)	(2.618)
Share with college degree	6.865***	6.586***	0.146
	(2.560)	(2.364)	(2.540)
Share men employed and unmarried	0.647	0.320	-2.268
	(1.406)	(1.389)	(1.544)
Female incarceration rate	2.487	6.475**	-1.130
	(2.429)	(3.129)	(0.697)
Male incarceration rate	-0.207	-0.330	0.0144
	(0.273)	(0.264)	(0.101)
Prostitution arrests	-0.00123***	-0.00286***	-0.000200
	(0.000430)	(0.000817)	(0.00163)
Heroin/Coke possession arrests	0.000466	0.00243	-0.000472
	(0.000413)	(0.00213)	(0.000985)
Heroin/Coke sale arrests	-0.0000500	-0.00121	0.00447
	(0.00104)	(0.00261)	(0.00320)
Total drug arrests	0.0000688	0.000383	0.0000198
	(0.0000579)	(0.000420)	(0.000146)
N	2142	2142	2142

Notes: This table expands the results presented in Table 2, column (3) to show the estimated coefficients on control variables. 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: Robustness Test: 10 year lag between HIV infection and AIDS diagnoses

	White		Black	
	Birth Rate	AIDS incidence	Birth Rate	AIDS incidence
	(1)	(2)	(3)	(4)
AIDS risk	0.164***		0.006	
	(0.053)		(0.006)	
Birth rate 10 years prior	-0.066*	0.036	-0.198***	0.274
	(0.039)	(0.035)	(0.031)	(0.234)
N	2142	2142	2142	2142
MSA and year FEs	X	X	X	
Controls included	X	X	X	

Notes: This table presents the results of two additional robustness checks. I evaluate whether the positive relationship between AIDS risk and birth rates is driven by unobserved increases 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 white women remains unchanged (Column 1). I check for reverse causality between AIDS and birth rates by verifying that there is no effect of birth rates 10 years prior on AIDs incidence (Column 2) Results for Black women are presented in columns 3 and 4. 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 A6: Effect of AIDS Risk on Marriage Rates

	All	White	Black
	(1)	(2)	(3)
AIDS risk in...			
Current year	-0.00010	-0.00022	0.00008
	(0.00017)	(0.00062)	(0.00006)
Previous year	-0.00011	-0.00015	0.00002
	(0.00016)	(0.00079)	(0.00006)
2 years prior	-0.00012	-0.00023	0.00005
	(0.00014)	(0.00080)	(0.00006)
N	2142	2142	2142
MSA and year FEs	X	X	X

Notes: This table shows the effect of AIDS risk on marriage rates using seperate 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 * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A7: Effect of AIDS Risk by 1987 definition on Birth Rates

	Primary Specification (1981-2001)			MSA Trends (1969-2001)
	(1)	(2)	(3)	(4)
Panel A: Birth Rate All Women 20-44				
AIDS risk (1987 def)	0.013***	0.008***	0.008**	0.010***
	(0.005)	(0.002)	(0.004)	(0.003)
Standardized AIDS risk	0.103***	0.619***	0.671**	0.677***
	(0.038)	(0.136)	(0.286)	(0.222)
Panel B: Birth Rate White Women 20-44				
AIDS risk (1987 def)	0.110***	0.081***	0.077***	0.088***
	(0.027)	(0.014)	(0.017)	(0.020)
Standardized AIDS risk	0.123***	0.956***	0.902***	0.853***
	(0.030)	(0.167)	(0.204)	(0.197)
Panel C: Birth Rate Black Women 20-44				
AIDS risk (1987 def)	0.016	0.002	0.005	0.008
	(0.012)	(0.004)	(0.010)	(0.008)
Standardized AIDS risk	0.056	0.073	0.238	0.292
	(0.042)	(0.196)	(0.487)	(0.286)
N	2142	2142	2142	3366
MSA and year FEs	X	X	X	X
Weighted		X		
Controls included			X	
MSA-specific linear trends				X

Notes: Each panel presents the effect of AIDS risk on birth rates using the 1987 definition of an AIDS diagnosis. There is a positive effect of AIDS incidence on the birth rate to white adult women, regardless of which case definition is used (see Table 3).. Results are robust to a 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 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 * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.