Safer Sex?
The Effect of AIDS Risk on Birth Rates

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Abstract

The advent of HIV/AIDS 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 evidence that the AIDS epidemic increased the birth rate among adult women by as much as 1.26 births per 1000 women and reduced gonorrhea incidence. The fertility results are driven by an increase in births to unmarried women, but only among births for which information on the father is available. I conclude that unmarried women responded to AIDS by entering monogamous relationships, which in turn led to an increase in the birth rate.

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.

This paper is motivated by the fact that 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. In those cases, a behavioral shift toward sex that is safer in terms of STI transmission may cause an increase in pregnancy risk. As a result, the theoretical direction of the relationship between STI risk and pregnancy risk is ambiguous.

I empirically examine the trade-offs between STI protection and pregnancy prevention using the AIDS epidemic in the United States of the 1980s and 1990s as a source of exogenous variation in STI risk. The advent of HIV/AIDS in the 1980s dramatically increased the economic and health costs of contracting a sexually transmitted infection. Prior to the development of effective treatment in 1996, a person diagnosed with AIDS had a 50% chance of dying within 2 years of diagnosis (CDC, 1991). By the time effective treatment was developed in 1996, over 362,000 people had died of AIDS in the United States (CDC, 1996). Given the high costs, it is natural to expect that people at greater risk of infection would adjust their behavior in order to reduce their chances of contracting the disease. I evaluate whether increases in risk of AIDS infection led women to adopt AIDS avoidance behaviors that affected birth rates.

Using data on local AIDS incidence as a proxy for AIDS risk, I analyze the effect of AIDS risk on birth rates with a fixed effects specification. I control for city and year fixed effects

to exploit within-city variation in AIDS incidence over-time. I address threats to causal identification under a fixed effects specification by exploiting characteristics specific to the AIDS epidemic. I add to my primary specification city-specific year trends and time-varying, city-level controls which may plausibly affect both AIDS incidence and birth rates, such as drug use and prostitution. I then conduct analyses that make use of the 10 year incubation period between HIV exposure and AIDS diagnoses, the demographics of AIDS patients, and the relationship between AIDS and other STIs.

I find that the risk of AIDS infection, as measured by local AIDS incidence, has a positive and statistically significant effect on the birth rate among adult women. I find that a 1 standard deviation increase in AIDS incidence increases the birth rate among women 20-44 years old by as much as 1.26 births per 1000 women. At the lower bound, I estimate that a 1 standard deviation increase in AIDS incidence increases the birth rate by 0.88 births. This effect is driven by an increase in the birth rate among white women aged 20-44. I find no effect of the AIDS epidemic on the birth rate among black women. These results are robust to the inclusion of city-specific year trends as well as controls for the crack cocaine epidemic, state Medicaid expansions, poverty rates, prostitution rates, and intravenous (IV) drug use. Results are also robust to the inclusion of birth rates 10 years prior, which proxy for levels of sexual behavior at the time of HIV infection.

I then evaluate how AIDS incidence among people with different sexual identities affects birth rates. I find that there is no effect of AIDS infections among homosexual and bisexual males on birth rates. The positive effect of AIDS on birth rates is due to increases in AIDS incidence among females and heterosexual males. This result further supports a causal interpretation of effects by showing that women are adjusting behavior in response to their true risk of infection. However, we might be concerned that increases in birth rates and AIDS incidence are both driven by increases in unprotected sex among women. I evaluate this alternative hypothesis using data on gonorrhea incidence.

Using a fixed effects specification, I find that AIDS risk led to a decrease in gonorrhea

incidence among women. 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. First, women could switch from effective prescription contraceptives to condoms. Second, women could choose to limit their number of sexual partners. While there are likely heterogeneous responses in the population, I make use of birth record data and survey data to determine which behavioral response is the most common.

I find that the risk of AIDS increased the rate of births to unmarried mothers. However, using information from birth certificates on whether a father's age is recorded, I find that there is no increase in births that lack information on father's age. I also find that births to unmarried mothers with a father present - a proxy for births to cohabiting mothers - increase in response to AIDS risk. 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 that 37.5% of unmarried women adjusted their sexual behavior to avoid AIDS and 16% of unmarried women decided to stop having sex with more than one man (Mosher and Pratt, 1993). Using survey data on pregnancy probabilities, I estimate that 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 1.26 birth increase, but is consistent with additional women in the population combining condoms with existing contraceptive methods or abstaining from sex.

This is the first paper, to my knowledge, to empirically relate AIDS avoidance behaviors and birth rates. Posner (1993) hypothesized an economic model of protection against fertility risk and disease risk. However, he does not extend his theoretical model to an empirical analysis. One reason for the existing gap in the literature is difficulty obtaining sufficient data on sexual behavior and contraceptive use for this time period. I demonstrate that using population-level birth rate and STI data can allow us to draw broad conclusions about AIDS

avoidance behaviors, despite the lack of high-quality individual data.

My results build on the existing, but separate, studies of fertility decisions and STI avoidance. There exists a large literature evaluating fertility decisions and the determinants of birth rates (Levine, 2001; Goldin and Katz, 2002; Bailey, 2006; Goldin, 2006; Kearney and Levine, 2009; Bailey, 2010; Bailey et al., 2012; Bailey, 2013) In comparison, there are few studies which empirically evaluate both STI and pregnancy as a determinant or result of contraceptive use. Delavande (2008) uses a survey to elicit subjective expectations about contraceptive methods and finds that pregnancy prevention and STI avoidance are the two most important characteristics women consider when picking a method of contraception. Both Durrance (2013) and Mulligan (2016) find that expanded access to emergency contraception led to an increase in STI rates. However, there are no studies, to my knowledge which evaluate how STI risk affects births.

Previous studies have evaluated how people adjust their sexual behavior and contraceptive use in response to AIDS. Ahituv et al. (1996) use regional AIDS incidence and estimate that men respond to AIDS risk by adopting condoms as a contraceptive method. They find no effect of regional AIDS incidence on condom use by women. Francis (2008) uses survey data to show that people who know somebody with AIDS switch to sexual behaviors with lower likelihood of HIV transmission. Analyzing the AIDS epidemic in Malawi, Reniers (2008) shows that people respond to AIDS risk by entering into unions with faithful partners. However, his results suggest that this mitigation strategy is more effective for men than women, who have less control over their partner's sexual activity. Furthermore, it is well documented in the literature that individual behavioral changes can affect the rate of AIDS infection in the population (Boozer and Philipson, 2000; Lakdawalla et al., 2006; Greenwood et al., 2019). Despite the wealth of evidence that people adjust their behavior in response to AIDS risk and that these individual changes can affect population level outcomes, there are no studies, to my knowledge, which connect AIDS avoidance behaviors to birth rates. I address this gap in the literature and show that AIDS avoidance behaviors lead to an

increase in birth rates.

Understanding the relationship between AIDS risk and birth rates can inform the development of reproductive health policy and HIV prevention programs. Both HIV infection and unintended pregnancy are very costly. Policies which decrease both STI infections and unintended pregnancies could decrease medical spending, increase labor productivity, and decrease Medicaid expenditures by the federal and state governments. My results 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 joint risks of pregnancy and STI. Screening practices which identify women with one partner as higher risk for pregnancy could lower the unintended pregnancy rate.

This paper proceeds as follows: Section 2 provides background on the AIDS epidemic. Section 3 describes the theoretical effect of AIDS risk on birth rates. Section 4 details the data sources and sample construction. Section 5 describes the empirical approach. Section 6 and 7 present results and robustness checks. Section 8 describes the mechanisms underlying results. Section 9 evaluates the magnitude of estimated effects. Section 10 details the contribution and policy implication of this paper. Section 11 concludes.

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). 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 incidence 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 of 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.

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 population-level data, it is only possible to determine the net effect of AIDS risk on birth rates. The net effect depends on which risk mitigation strategy is most prevalent in the population and thus is an empirical question.

If there is a negative effect of AIDS risk on birth rates, we expect that the majority of affected women are choosing to abstain or are adopting condoms in addition to their current method of contraception. If there is a positive effect of AIDS risk on birth rates, we expect

[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.

that the majority of affected women are adjusting their choices over sexual partners or are switching from more effective contraceptives to condoms. A null finding could be interpreted in multiple ways. One possibility is that the risk of AIDS is not salient to women. Another possibility is that the cost of AIDS infection is very small relative to the cost of unintended pregnancy. A precisely estimated zero effect could also indicate that half of affected women are adopting behaviors that increase their pregnancy risk while the other half are adopting behaviors that decrease their pregnancy risk.

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. To see that AIDS incidence is a reasonable proxy for AIDS risk, consider the following. Let AIDS risk be defined as the probability of contracting AIDS in one year. Holding fixed an individual's sexual behavior and contraceptive practices, an increase in AIDS incidence will increase the likelihood that any given sexual partner has AIDS. If the probability a partner has AIDS increases, then it follows that AIDS risk increases. This relationship holds across the population of sexually-active adults. Thus, AIDS incidence is a positive, monotonic transformation of the average AIDS risk in the population.

Using APIDS data, I create a panel of AIDS incidence across 102 MSAs from 1981 to 2002. 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 1969 to 2003 by mother's race and age. The resulting panel of previous year's AIDS incidence and birth rates contains 2,346 MSA-year observations across the years 1981 to 2003. AIDS incidence is lagged one year to approximate the AIDS incidence at time of conception as opposed to time of birth. Aggregate trends in AIDS incidence and birth rates among adult women are shown in Figure 1. Birth rates decline sharply in the early 1970s with the legalization of the oral contraceptive pill (Bailey, 2010). AIDS incidence increases from 1981 through the early 1990s.

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 incidence in the MSA in the previous year, and γ_m and δ_t are MSA and year fixed effects, respectively. β_1 is the coefficient of interest. In order to interpret β_1 as the causal effect of AIDS risk on birth rates, it must be that AIDS incidence 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 incidence 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 MSA-specific linear trends to address concerns about within-MSA trends which could be affecting both AIDS incidence and birth rates. Third, I add a variety of controls which may

plausibly affect both AIDS incidence and birth rates, including: an index for crack cocaine use, poverty rate, Medicaid coverage, arrests for possession or sale of heroin or crack, and arrests for prostitution.

Additional robustness tests exploit characteristics specific to the setting of the AIDS epidemic. For example, I use the 10 year average latency period between HIV infection and AIDS diagnosis to test for a spurious relationship and reverse causality. I conduct a placebo test by regressing birth rates on next year's AIDS incidence. I analyze the effect of AIDS incidence on gonorrhea incidence to show that estimates are not driven by unobserved sexual behavior. Finally, I show that the relationship between AIDS incidence and birth rates is driven by AIDS diagnoses among females and heterosexual males.

VI. Main Results

A. Effect of AIDS Risk on Birth Rates

Empirical results for the fixed effects specification from equation (1) are presented in Table 1, 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 adult women aged 20-44, I estimate that every additional AIDS diagnosis in an MSA per 100,000 people led to an increase in births by 0.043 per 1000 women.[2] For ease of interpretation, Table 1 also presents standardized coefficients. A 1 standard deviation increase in the previous year's AIDS incidence results in a 0.080 standard deviation increase in birth rates.

Panels B and C of Table 1 show that positive relationship between AIDS incidence and births to all adult women is primarily driven by an increase in births to white women. I find no evidence of a relationship between AIDS incidence and births to black women. I estimate that a 1 standard deviation increase in the previous year's AIDS incidence increases births to white women by 0.120 standard deviations, or 1.26 births.

[2]I also conducted this analysis on births to adolescent women and found no effect. Results for adolescent women can be found in the appendix

Table 1, column (2) presents the results from a weighted version of equation (2). I weight regressions by the number of females aged 20-44 in the MSA each year (or white females and black females, respectively). The estimated coefficients increase when the regressions are weighted. The increase in the estimated effect could be due to heterogeneity across cities (Solon et al., 2015). In other words, the effect of AIDS on birth rates may be greater in cities with a larger population. This is reasonable given that infectious diseases spread more quickly in highly populated areas. Holding AIDS incidence fixed, the risk of contracting AIDS may be higher in more densely populated areas. In an ideal situation, this heterogeneity could be addressed by allowing the effect of AIDS to vary across cities with different population levels. Unfortunately, this is not possible here due to small sample size. Instead, I include a control for female population size in all remaining regressions. Because weighting increases standard errors and inflates the magnitude of estimated results, I choose to use an unweighted specification for the remaining analyses.

B. Inclusion of Controls

To address concerns about within-MSA trends which could be affecting both AIDS incidence and birth rates, I add an MSA-year linear trend to equation (1). Results are presented in Table 1, column (3). For these regressions, I use birth data from 1969 to 2003, resulting in a sample size of 3570. I once again find that there is a positive and statistically significant relationship between births and AIDS incidence which is driven by an increase in births to white women.

Despite the inclusion of fixed effects and linear trends, 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 incidence 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 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.

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 (CPS) 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. To obtain representative averages, I drop MSAs for which I observe less than 20 women of the relevant demographic group. I also use the CPS to calculate the share of women living below the poverty line.

The inclusion of controls substantially decreases my sample size. Not all MSAs in my sample have a corresponding city included in the crack index. Many observations are also dropped due to lack of sufficient observations in the CPS. To ensure results are not driven by the decreased sample size, I report estimates for equation (1) using the control sample both with and without the control variables included. Results using the smaller sample without controls are presented in Table 1, column (4). Results with controls are presented in Table 1, column (5).

Limiting the sample size slightly increases the estimated coefficient on births to white

women. This is not surprising given that the smaller sample excludes many small cities with low AIDS incidence. Adding controls decreases the estimated coefficient, but the magnitude is similar to that estimated in the primary fixed effects specification and the specification with linear trends.

It is puzzling that I find significant results for white women, but no results for black women - particularly given that AIDS incidence was higher among black woman than white women during the 1980s and 1990s. One possibility is that there is greater heterogeneity in response to AIDS risk among black women than white women - such that equal shares of black women are adjusting behaviors in ways that increase (or decrease) their likelihood of pregnancy, thus the net effect is zero. Another possibility is that the AIDS epidemic for black women was of relatively lower concern in light of other risks. For example, I find that the crack cocaine epidemic, as measured by the crack index, led to large increase in births to black women but had no effect on births to white women. However, heroin arrests led to an increase in births to white women but not to black women. These results seem to contradict each other and further research into this area is needed. Given the lack of statistically significant effects among births to black women, I focus the remaining discussion on births to white women. All analyses were also completed for births to black women and can be found in the appendix.

VII. Robustness Tests

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 incidence 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.

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. Results are presented in Table 2, column (1). The effect of AIDS incidence in the previous year on birth rates to white women is positive and statistically significant, even when controlling for birth rates 10 years prior. Furthermore, there is no effect of births 10 years prior on current births. As an additional check, I run a placebo test by regressing birth rates on AIDS incidence one year in the future. The results are presented in Table 2, column (2). There is no effect of next year's AIDS incidence on births to white women. Furthermore, the coefficient on previous year's AIDS incidence remains positive and statistically significant.

One drawback of the fixed effects specification is that it is not robust to concerns of reverse causality. However, given the 10 year incubation period of AIDS, if the birth rate was leading to an increase in AIDS, we would expect that births 10 years prior are predictive of AIDS incidence. In Table 2, column (3), I regress AIDS incidence on birth rates 10 years prior and find no effect. I conclude that the AIDS epidemic led to an increase in the birth rate and not vice versa.

A. Effect of AIDS on other STIs

As an additional argument for causal interpretation of results, I analyze the relationship between AIDS and gonorrhea. 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 2003. This data is publicly available via CDC WONDER. Unfortunately, MSA-level gonorrhea data is only available after 1995.

For this analysis, I drop MSAs that span across multiple states and merge the remaining MSAs with state-level gonorrhea data.

Using a fixed effects specification, I analyze the effect of previous year's AIDS incidence on gonorrhea incidence among women. Results are presented in Table 2, column (4). I find that gonorrhea incidence decreases in response to AIDS incidence. Columns (5), (6), and (7) repeat this analysis with a weighted specification, a smaller sample, and with controls included, respectively. Across all specifications, AIDS incidence 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.

B. Effect of AIDS by Sexuality

As a final robustness check, I analyze the effect of AIDS incidence among among people of different sexual identities on birth rates. 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 females or heterosexual males.[3] In comparison, if the effect is driven by diagnoses among bisexual and homosexual men, it might be that results are driven by unobservable factors such as attitudes towards sexual behavior or access to health care.

Results for this analysis are presented in Table 3. I find that AIDS diagnoses among adult females and heterosexual males both result in an increase in birth rates. Furthermore, the magnitude of estimates is similar to those displayed in Table 1. In comparison, I find there is no effect of AIDS diagnoses among bisexual and homosexual men on births. When

[3]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."

including all groups in one regression, I find no statistically significant results. This is likely because the AIDS incidence among females and heterosexual males is highly correlated, with an unconditional correlation of over 96 percent. Jointly, incidence among females and heterosexual males does have a significant effect on birth rates. Furthermore, the effect of incidence among females and heterosexual males is robust to the inclusion of incidence among bisexual and homosexual males. 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.

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.

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. Condoms have a typical use failure rate of 18% and are much more likely to result in pregnancy than prescription contraceptives. 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 test the robustness of results to multiple additions, including a weighted regression and a regression with MSA-specific linear trends.

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 4. I find that the risk of AIDS increased the birth rate 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.

IX. Magnitude of Estimated Effects

The main results presented in section 6 suggest that the birth rate to adult women increased by anywhere from 0.8 births per 1000 women to 1.2 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 using population-level data 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. It is encouraging that Bailey's estimated effect of the Pill is much larger than the effect I estimate for AIDS.

Kearney and Levine (2009) analyze the effect of expanded Medicaid family planning receipt 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 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. Are these shares 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.[4] 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.

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

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 0.5 to 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 women opting to have only one sexual partner to decrease their chances of AIDS exposure.

X. Contribution 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 my knowledge, 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. My results add to the relatively small literature relating AIDS avoidance behaviors and number of sexual partners (Kremer, 1996; Reniers, 2008; Francis, 2008). More importantly, this paper goes one step further than the existing literature by connecting decisions about AIDS avoidance and number of partners to birth rates. 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.

This research has implications for policy development and future economics research. By establishing a connection between AIDS risk and birth rates, I highlight the importance

of a joint and nuanced approach to addressing STI spread and unintended 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.

The positive relationship between AIDS risk and birth rates also highlights the importance of including STI risk and choices about partners in surveys and in economic models of sexual behavior. There are very few existing models that account for both STI prevention and pregnancy protection (Delavande, 2008; Durrance, 2013; Mulligan, 2016). Furthermore, I am not aware of any joint models that capture nuanced decisions over sexual partners - specifically with regard to how the number and risk level of partners affects both STI likelihood and pregnancy likelihood. Further research is needed to understand how people make decisions regarding of number and choice of sexual partners.

XI. Conclusion

Women consider both risk of STI and risk of unintended pregnancy when making decisions about sexual behavior and contraception. The AIDS epidemic of the 1980s and 1990s created

a large increase in the cost of contracting an STI. I exploit MSA-level variation in the severity of the AIDS epidemic to analyze the effect of AIDS risk on birth rates.

I show that there is a positive and causal relationship between AIDS risk and birth rates. At first glance, this result is puzzling. We might expect that the risk of deadly AIDS infection would cause women to adopt "safer" sex practices that decrease both infection likelihood and pregnancy likelihood. However, the reality is much more nuanced; there are a variety of margins along which women can adjust both their risk of infection and their risk of pregnancy. Specifically, I provide evidence that many women mitigated the risk of AIDS by decreasing their number of sexual partners which then led to an increase in the birth rate. This paper shows that there are unintended consequences of STI avoidance behaviors and highlights the importance of a joint approach to STI and pregnancy prevention.

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Figures

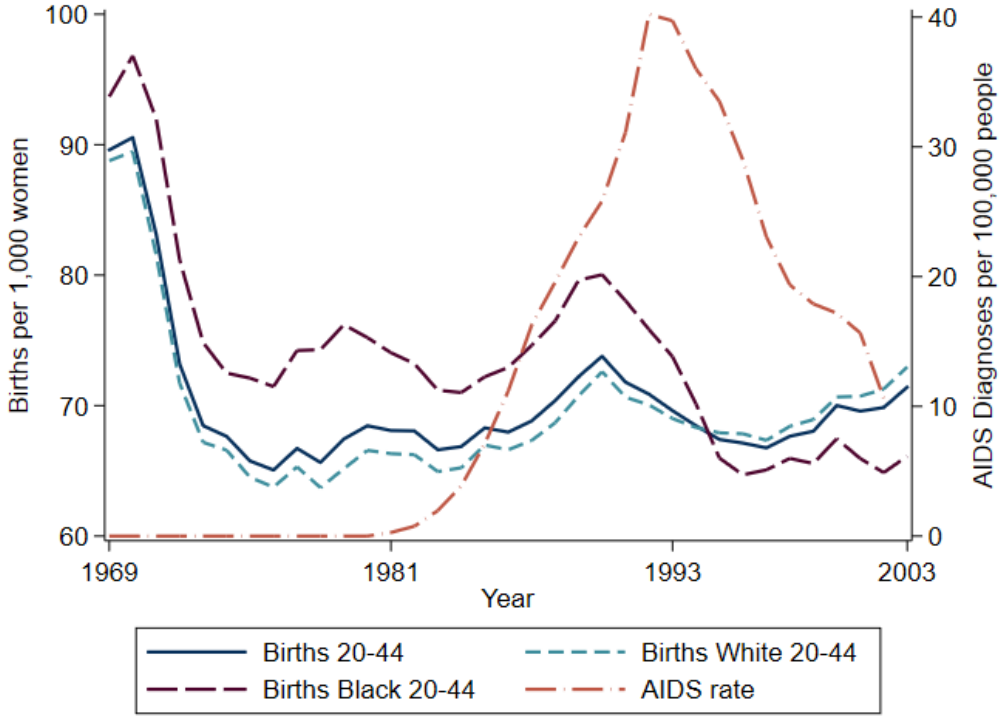

FIGURE 1: TRENDS IN AIDS INCIDENCE AND BIRTH RATES

Notes: This figure plots aggregate trends in AIDS incidence and birth rates among women 20-44 years old from 1969 to 2003. AIDS incidence is defined as number of new AIDS diagnoses per year per 100,000 people. Birth rates are live births per 1000 women of the relevant demographic group. Birth rates declined sharply in the early 1970s. AIDS incidence increased from 1981 to 1993.

Tables

Table 1: Effect of AIDS Risk on Birth Rates

	Fixed Effects (1981-2003)		MSA Trends (1969-2003)	Control Sample (1981-2003)	
	(1)	(2)	(3)	(4)	(5)
Panel A: Birth Rate All Adult Women 20-44					
AIDS incidence in previous year	0.053***	0.067***	0.053***	0.045**	0.032**
	(0.019)	(0.021)	(0.018)	(0.018)	(0.016)
Standardized coefficient	0.080**	0.125***	0.071***	0.117**	0.083**
	(0.032)	(0.039)	(0.024)	(0.047)	(0.041)
N	2346	2346	3570	1307	1307
Panel B: Birth Rate White Women 20-44					
AIDS incidence in previous year	0.070***	0.098***	0.065***	0.089***	0.071***
	(0.022)	(0.028)	(0.022)	(0.027)	(0.025)
Standardized coefficient	0.120***	0.172***	0.084***	0.212***	0.170***
	(0.038)	(0.049)	(0.029)	(0.064)	(0.059)
N	2346	2346	3570	1296	1296
Panel C: Birth Rate Black Women 20-44					
AIDS incidence in previous year	0.031	0.011	0.036	-0.033	-0.060
	(0.032)	(0.021)	(0.024)	(0.061)	(0.046)
Standardized coefficient	0.005	0.016	0.040	-0.083	-0.149
	(0.045)	(0.029)	(0.027)	(0.153)	(0.115)
N	2346	2346	3570	501	501
Weighted		X			
MSA-Specific Linear Trends			X		
Controls Included					X

Notes: Each panel presents the effect of lagged AIDS incidence on birth rates per 1,000 women of the demographic group. 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 the standard deviation change of birth rates. There is a positive effect of AIDS incidence on the birth rate to white adult women. Results are robust to a weighted specification (Column 2), the inclusion of MSA-specific year trends (Column 3), and controls for drug use, poverty rates, Medicaid expansions, and prostitution (Column 5). When including MSA-specific year trends, the sample size increases because I include more pre-AIDS years. Including controls decreases the sample size due to missing observations for control variables. To ensure that results with controls are not driven by sample selection, I present the coefficient from a fixed effects regression on the control sample without the controls included (Column 4). 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 2: Robustness Checks

	Unobserved sexual behavior		Reverse causality	Effect of AIDS on other STI			
Outcome var:	*White Birth Rate*		*AIDS rate*	*Gonorrhea rate*			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
AIDS incidence in previous year	0.072***	0.057***		-0.758**	-0.757*	-0.790**	-1.004**
	(0.026)	(0.020)		(0.311)	(0.448)	(0.370)	(0.415)
Birth rate 10 years prior	-0.089	-0.080	-0.031				
	(0.063)	(0.065)	(0.106)				
AIDS incidence in next year		0.020					
		(0.028)					
N	1296	1296	2244	1759	1759	991	991
Weighted					X		
Controls Included	X	X					X

Notes: This table presents the results of four different robustness checks. I evaluate whether the positive relationship between AIDS and births 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 10 years prior and find that the relationship between AIDS incidence and birth rates remains unchanged (Column 1). I further check whether unobserved sexual behavior is driving results by conducting a placebo test in which I control for AIDS incidence one year in the future (Column 2). 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 3). I show that there is a negative effect of AIDS incidence on gonorrhea incidence (Column 4) and that these results are robust to weighting (Column 5) and the inclusion of controls (Columns 6 and 7). 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 White Birth Rates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Females	0.094***				0.015	0.090***	
	(0.027)				(0.063)	(0.028)	
Heterosexual Males		0.058***			0.047		0.055***
		(0.014)			(0.032)		(0.014)
Bisexual Males			0.129		0.023	0.015	0.026
			(0.080)		(0.084)	(0.086)	(0.083)
Homosexual Males				0.023	0.009	0.011	0.008
				(0.016)	(0.014)	(0.014)	(0.014)
N	2346	2346	2346	2346	2346	2346	2346

Notes: This table presents the effect of AIDS incidence among people of different genders and sexuality on birth rates. When regressed separately, AIDS incidence among females and heterosexual males leads to an increase in birth rates (Columns 1 and 2). When all categories are included in the same regression, nothing is significant (Column 5). However, this is likely because incidence among females and heterosexual males are highly correlated. When these two groups are separated, both are significant (Columns 6 and 7). 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 4: Effect of AIDS on Births to White Women by Marital Status & Age

	Birth Rate			Share of Births		
	(1)	(2)	(3)	(4)	(5)	(6)
Aged 20-29						
Unmarried w/ father	0.015	0.045**	0.043**	0.013	0.022	0.027
	(0.019)	(0.022)	(0.020)	(0.014)	(0.014)	(0.019)
Unmarried no father	0.011	0.029*	0.013	0.011	0.020	0.010
	(0.013)	(0.017)	(0.017)	(0.012)	(0.013)	(0.014)
Married w/ father	0.034*	0.063***	-0.003	-0.027***	-0.046***	-0.040***
	(0.020)	(0.015)	(0.030)	(0.010)	(0.015)	(0.012)
Married no father	0.002	0.004	0.002	0.003**	0.003*	0.002*
	(0.001)	(0.002)	(0.002)	(0.001)	(0.002)	(0.001)
Aged 30-44						
Unmarried w/ father	0.017***	0.028***	0.020***	0.027**	0.040***	0.040**
	(0.005)	(0.007)	(0.007)	(0.011)	(0.011)	(0.017)
Unmarried no father	0.005	0.011**	0.006	0.006	0.014*	0.009
	(0.004)	(0.005)	(0.005)	(0.007)	(0.007)	(0.008)
Married w/ father	0.018	0.002	0.003	-0.035***	-0.055***	-0.051***
	(0.022)	(0.018)	(0.023)	(0.010)	(0.013)	(0.010)
Married -no father	0.001	0.001	0.001	0.001	0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)
N	2346	2346	3570	2346	2346	3570
Weighted		X			X	
MSA-Specific Linear Trends			X			X

Notes: The effect of lagged AIDS incidence on the birth rate to white mothers by marital status is shown in columns (1) to (3). The effect of lagged AIDS incidence on the share of births (out of 100) to each marital group is shown in columns (4) to (6). I use absence of father's age on the birth record as a proxy for father's presence. Results show that AIDS risk increased the number and share of births for which the mother was unmarried but the father was present. 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 A: Additional Tables

Table A1: Summary Statistics for Birth Rates

	Mean	Std. Dev.	Min	Max
Birth rate per 1000 women aged 20-44...				
All	68.64	9.81	48.78	125.85
White	67.66	10.51	46.59	126.59
Black	72.40	13.10	13.99	238.51
Birth rate white women aged 20-29...				
Unmarried w/ father	13.58	10.67	0.00	55.64
Unmarried no father	9.27	5.15	1.23	33.66
Married w/ father	83.24	18.71	38.53	184.94
Married no father	0.60	0.65	0.00	4.47
Birth rate white women aged 30-44...				
Unmarried w/ father	2.38	2.20	0.00	14.36
Unmarried no father	1.54	0.98	0.13	7.00
Married w/ father	37.72	7.54	21.85	70.73
Married no father	0.17	0.19	0.00	1.63
N	2346			

Notes: This table shows descriptive statistics for birth rates used as outcome variables in the main analysis.The sample includes 102 MSAs from 1981 to 2003.

Table A2: Summary Statistics for Disease Incidence

	Obs	Mean	Std. Dev.	Min	Max
AIDS incidence in previous year...					
Total	2346	14.36	18.35	0.00	203.11
Female (20 years or older)	2346	6.43	11.24	0.00	107.84
Heterosexual male (20 years or older)	2346	11.67	19.84	0.00	212.38
Bisexual male	2346	3.80	4.15	0.00	31.96
Homosexual male	2346	12.16	19.00	0.00	323.02
Gonorrhea incidence	1759	195.19	136.74	6.46	1630.13

Notes: This table shows descriptive statistics for disease incidence used in the main analysis. For AIDS incidence, the sample includes 102 MSAs from 1981 to 2003. For AIDS incidence among females and heterosexual males, I limit the data to cases among patients 20 years and older to exclude pediatric cases. The relatively low mean among homosexual and bisexual males is due to using the entire population of males as the denominator to calculate incidence, as it is not possible to obtain information on the number of homosexual and bisexual males in the population. State-level data is used for gonorrhea incidence, resulting in a smaller sample size.

Table A3: Effect of AIDS on Birth Rates - Control Coefficients

	All	White	Black
	(1)	(2)	(3)
AIDS incidence in previous year	0.0318**	0.0713***	-0.0597
	(0.0158)	(0.0248)	(0.0461)
Population	0.0000304***	0.0000423***	0.0000724*
	(0.00000615)	(0.0000122)	(0.0000387)
In previous year...			
Crack Index	0.464	0.305	1.289**
	(0.288)	(0.321)	(0.569)
Share below poverty level	-6.255**	-1.831	-1.776
	(2.691)	(2.533)	(3.136)
Share married	-0.178	1.078	-3.875
	(3.298)	(4.071)	(2.411)
Heroin/crack possession arrests	0.0139**	0.109***	-0.00683
	(0.00594)	(0.0286)	(0.0108)
Prostitution arrests	-0.00871	-0.0245	-0.00736
	(0.00528)	(0.0239)	(0.00891)
Heroin/crack sale arrests	0.0121	0.0608	0.0539
	(0.0114)	(0.0644)	(0.0473)
N	1307	1296	501

Notes: This table expands the results presented in Table 1, column (5) to show the estimated coefficients on control variables. The outcome variable is birth rates among women aged 20-44 of the relevant demographic group. 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 A4: Summary Statistics - Control Variables

	Obs	Mean	Std. Dev.	Min	Max
All Women 20-44...					
Crack index	1307	1.23	1.25	-0.75	8.00
Share below poverty level	1307	0.13	0.06	0.00	0.52
Share on Medicaid	1307	0.09	0.05	0.00	0.38
Arrest rate heroin/crack possession	1307	13.34	20.72	0.00	259.91
Arrest rate prostitution	1307	7.49	17.59	0.00	282.49
Arrest rate heroin/crack sale	1307	6.17	10.64	0.00	213.36
Population	1307	387808	366091	85075	1905439
White Women 20-44...					
Crack index	1296	1.23	1.25	-0.75	8.00
Share below poverty level	1296	0.10	0.06	0.00	0.44
Share on Medicaid	1296	0.06	0.05	0.00	0.33
Arrest rate heroin/crack possession	1296	5.76	7.67	0.00	71.74
Arrest rate prostitution	1296	3.82	6.49	0.00	107.72
Arrest rate heroin/crack sale	1296	2.14	4.23	0.00	108.03
Population	1296	301280	257204	65365	1406409
Black Women 20-44...					
Crack index	501	1.38	1.43	-0.75	8.00
Share below poverty level	501	0.29	0.12	0.05	0.79
Share on Medicaid	501	0.23	0.11	0.00	0.62
Arrest rate heroin/crack possession	501	9.76	18.36	0.00	216.69
Arrest rate prostitution	501	5.74	17.77	0.00	174.10
Arrest rate heroin/crack sale	501	5.41	8.68	0.00	104.63
Population	501	135192	116276	12932	571545

Notes: This table presents descriptive statistics for control variables used in the main analysis. The sample size changes across demographic groups as I drop any observations from the CPS for which there are less than 20 people observed in the MSA of the relevant demographic. Arrest rates are calculated per 100,000 people.