Three Essays on the Effect of Incarceration, Drug Use and Abortion Legalization on STD Risk

by

Anthony Scott Cunningham

(Under the direction of Christopher M. Cornwell)

Abstract

In this dissertation, I argue that the "war on drugs" has increased Black STD risk by creating imbalanced sex ratios which has enabled men with "tastes" for promiscuity to form risky sexual relationships. I test this hypothesis in several ways. First, I use data from the NLSY97 to examine the effect of mating options on promiscuity and condom use and use diverging sex ratios for Blacks in late adolescence to identify the effect of mating options on risky sexual behavior. I find that Black men at the 90th quantile — men I term "promiscuous" – will have between 1.3 and 2.4 more female sex partners a year due to changes in the sex ratio over the sample period. I also find evidence that Black men alter their condom use in response to the sex ratio. Separately, I test for a link between incarceration rates and STD outcomes. I find strong evidence that Black incarceration rates are associated with higher rates of gonorrhea and syphilis among Black females. I also have provided the first quantitative evidence that the crack epidemic increased gonorrhea and syphilis.

Gonorrhea rates began falling in the mid-to-late 1980s as the prison population continued expanding. I argue that abortion legalization, waning crack and the AIDS epidemic are partly responsible for these changes. I exploit the natural experiment offered by early legalization of abortion five states in 1970, compared to universal legalization in 1973, to estimate the

effect of abortion legalization on second generation gonorrhea rates. I find mixed evidence for the abortion legalization hypothesis, combined with consistently strong evidence that the crack epidemic and the AIDS epidemic contributed to the declines. Using a difference-in-difference-in-difference model, I find Black 15–19 year-old gonorrhea rates fell relative to 35–39 year-olds in repeal states compared to Roe legalization states. I also find that for every 100 deaths from AIDS, Black gonorrhea rates fell 7 cases per 100,000. The crack index consistently reveals strong positive correlations with gonorrhea rates for Blacks and Whites, but as the index is based on 3 proxies for crack, interpretation of the coefficients are difficult.

INDEX WORDS: Health Economics, Labor Economics, Applied Microeconomics, Risky Sexual Behavior, Sex Ratio, Crack Cocaine, Incarceration, Abortion, Sexually Transmitted Diseases, Gonorrhea, Syphilis

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Dedication

This dissertation is dedicated to Paige, Miles, Willow and Ren without whom my life would be much, much smaller, and to my fallen brothers, Allen and Madison Mays.

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No one deserves more credit for this project than my wife, Paige Cunningham. She supported our family financially, emotionally, and spiritually and took on far more than her fair share of the household's management. On top of that, she went the extra mile and endured my many monologues about the problem with imbalanced "sex ratios," concurrency, and gonorrhea. Apparently, even sex and gonorrhea can become tedious topics, and she was a sport for listening patiently to my rants on the subject. She is a blessing to her family and friends, and I am fortunate most of all for her companionship, presence and affections.

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I'd also like to acknowledge David Mustard in this work. Every budding "economic imperialist" has a story about a paper that helped them see the potential of economic reasoning for explaining the real world. For me, that paper was Lott and Mustard (1997) – "Crime, Deterrence, and the Right-to-Carry Concealed Handguns." Reading it, I realized that economics was not a body of doctrine, but a way of thinking – an engine of analysis, as Milton Friedman once said, that could be used effectively to understand a broad range of behavior. I applied to the University of Georgia in large part because David Mustard was a professor here, and it has been my pleasure to have studied under him and to have had him on my

committee. Furthermore, I will always be indebted to him for the role he played in helping me get an interview with my future employer, Baylor University.

I also would like to thank Angela Fertig. Though not a dissertation committee member, she might as well have been one given the amount of time she has devoted to helping me with my various projects. She graciously read all my essays, gave me extensive critical comments, and attended all my presentations. Furthermore, she has used her contacts to help me get job interviews and set up meetings. I have learned a great deal from her questions and insightful comments, and I am glad to have gotten to known her.

And then there is Christopher Cornwell. It feels like an cliche to say that I couldn't have written this dissertation without Dr. Cornwell, but in my case, it is completely factual. He has been so involved with this project that at home, my wife has begun calling him jokingly "the other woman" because of how often I reference him or a conversation with him after I come from home work. I spent more time discussing these projects with him in a typical week than many students spend with their adviser in their entire graduate school career. He had an open door policy with me, and encouraged me early on to pursue this project. He gave me endless advice about econometric problems and identification strategies, and when I was discouraged by how slow the project was progressing, he gave me encouragement. I count him not only as my teacher and mentor, but also my friend. I have been blessed to have been in his company these last five years.

And finally, since it's possible I would not be here today if not for the friendship of my brother-in-law, I want to acknowledge Jeffrey Lancaster in closing. When I was lost and confused to the point of suicide, he loved me and in doing so managed to convince me that God loved me too. Learning and experiencing that has changed my life forever, and I will always be in Jeffrey's debt as a result.

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Chapter 1

Introduction

The idea for this project came in the fall of 2004 while teaching a class of undergraduates on supply and demand. Rather than use the market for hot dogs or hamburgers as an illustration, I posed a more interesting thought experiment. Imagine an island with 1000 men and 1000 women, and the king of the island declares war with a distant enemy. After an intense battle, his army is destroyed and not a single man returns home. What would happen to the men and women left behind - specifically in regards to the relationships that they would form? Many answers came from the class. "There would be a shift in supply of men in the marriage market," one student said. "It would reduce marriages." "It would raise the 'price' of marriage that women would pay." That comment started a long conversation that took up most of the class. What exactly is a price in this context? A dowry? A bride-price? Maybe. Or is it more subtle than that? One student offered an interpretation of the "price" in this context — the remaining women would have to start "settling" for men that they previously hadn't seriously considered. Perhaps less attractive men, or men with less income. On the flip side, men would be able to get women they preferred more because of the reduced competition that they faced. I continued to turn the scenario over in my head, long after the lecture had ended. What other effects, besides changing the optimal sorting of monogamous pairings, would a shortage of men have on the mating market?

A few weeks later, a second insight came to me while reading one of Gary Becker's last installments for *Business Week* (Becker 2003). Becker argued that if America wanted to increase high-school graduation rates for Black men, then it should legalize drugs. Since drug trafficking is a potential source of income that raises the opportunity of costs of going to

college for young Black men, drug prohibition had created a black market that was providing students with incentives to drop out. It was not long before I realized that if Black men were not going to college to sell drugs, then they were likely going to jail and dying in relatively larger numbers. I was shocked to learn just how much larger the male incarceration rate was for Blacks than Whites. Over 12 percent of all Black men are in jail or in prison on any given day, and among those with only a high-school degree or less, the number is over 30 percent (Patillo, Weidman and Western 2004). By the time a Black male with only a high-school degree is 35, there is a 60 percent chance that he will have served time in prison or jail. That is to say, the modal experience for the least educated Black male is prison or jail.

I immediately began to make the connection between the thought experiment in class and the real world scenario of Blacks. Here was a unique natural experiment whereby Black men were incarcerated at such a high rate as to effectively shift the supply of men in the marriage and mating market leftward, thus potentially driving up the "price" of matching and altering their bargaining position. Given the weakened bargaining position created by the shortage of men, women would more likely concede to made-dictated relationship terms. These could include toleration of poor treatment from their partners, including promiscuity and infidelity.

The last component of this dissertation fell into place while speaking with a friend in another department. My friend shared with me a book entitled *On the Down Low* by prominent Black novelist, J. L. King and Karen Hunter (King and Hunter 2005). King argues that the increase prevalence of AIDS among Blacks, and especially Black women via heterosexual transmission, was caused by the tendency of homosexual and bisexual Black men to conceal their same-sex preferences by having "straight" relationships with women. King argued that this was possible because of the persistent stigmas against Black male homosexuality in Black culture. Part of this strategy involves having female sexual partnerships, and since the homosexual part of their life is kept hidden, it places Black women at risk for STDs that have higher prevalence in the homosexual network, such as AIDS. I have always found this

hypothesis unbelievable. Though I don't dismiss the idea that such activities occur in Black communities, the high rates of Black HIV/AIDS, gonorrhea and other STDs are simply too high to be caused by the small share of the population homosexuals represent (Laumann, Gagnon, Michael and Michaels 1994). But, the facts still were that AIDS was a growing problem in the Black community, and so largely ignored that it led one observer to call the problem a "silent epidemic" (Cohen 2004). AIDS had become the leading cause of death for Black women aged 25-34, but the media, politicians and those outside the Black community were largely ignorant of the problem, let understood its underlying cause. Since I had already become interested in the effects of sex ratio imbalances on mating market behavior, it seemed to me a good idea to spend a few years studying the relationship between incarceration and STD risk. And thus began this project.

In the next chapter, I explain how male sexual behavior may respond to opportunities in the sexual marketplace. In addition, I show how those opportunities have been affected by a drug policy that has led to mass incarceration of young Black men. Finally, I discuss the direct effects of drug use on risky sex.

The third chapter examines the effect of rising sex ratios and subsequent male risky sexual behavior. Using 1997-2002 data from the National Longitudinal Survey of Youth (1997), I estimate the relationship between sex ratio imbalances and male sexual behavior. Identification is achieved through the divergence of Black and White sex ratios at age 18 when Black males begin leaving the general population for prison and jail *en masse*. By "sex ratio," I mean the ratio of men to women in the population. To facilitate interpretation of results, though, I usually rely on the inverse of the sex ratio, or $sr-1$. I find that the state-level sex ratio is associated with increased promiscuity among Black males, but not White males, and the effects are largest for men in the far right tail of the sexual partnership distribution. I find that the most promiscuous Black males (i.e., men in the 90th percentile of the sexual partnership distribution) have between 1.3 and 2.4 more female sex partners a year because of the sex ratio change. Interestingly, I also find evidence that both Black men

and promiscuous men increase their condom use as the sex ratio rises, possibly suggesting that individuals are cognizant of the increased risks of STD infection and are therefore compensating by more vigilant condom use.

The fourth chapter studies the impact of incarceration and aggregate crack use on gonorrhea and syphilis rates for Blacks and Whites. I show that the Black male incarceration rate for 15-29 year olds increased the Black female gonorrhea rate for the same corresponding age bracket. I do not find any effect of incarceration on White disease rates for that group. Furthermore, I present first empirical evidence using nationally representative data that gonorrhea and syphilis rose among Blacks and Whites because of the introduction of crack cocaine. However, I do find that the incarceration rate is associated with White gonorrhea rates among 30-44 year olds. It is likely the case that a separate mechanism from a sex ratio imbalance connects White incarceration to White STD risk because while White males are incarcerated in larger aggregate numbers, the percentage incarcerated is very small and thus cannot be considered a shock to the sex ratio itself.

Gonorrhea and syphilis rates peaked in 1985/1986, and began to fall precipitously thereafter. Chapter five investigates why gonorrhea rates fell when they did, focusing on the abortion-legalization hypothesis. Gruber, Levine and Staiger (1999) showed that legal abortion improved the quality of the birth cohort treated while *in utero*. For instance, fertility rates fell approximately 6 percent following abortion legalization in the US, followed by a reduction in the children living in poverty, on welfare, and with low birth weight. A series of new papers, prompted by Donahue and Levitt (2001), has sought to track these policy changes with future outcomes, such as decreased crime. In their original study, Donahue and Levitt (2001) claimed abortion legalization could account for as much as 50 percent of the decline in crime in the 1990s. I argue that such large reductions in crime could translate into reductions in STD cases, because crime and risky sex share many of the same family determinants. Using a Difference-in-difference framework (DD), I compare the gonorrhea rates of "early repeal" states to the rest of the country, and find that indeed gonorrhea rates

fell from 1985-1989 for Blacks in the repeal states – consistent with the hypothesis. But, closer examination of the difference by age cohort casts doubt on the abortion legalization hypothesis, since 1985-1989 saw secular declines in gonorrhea across *all* cohorts — even cohorts who had not been exposed to abortion legalization *in utero*. Therefore, I estimate a difference-in-difference-in-difference (DDD) model using various older cohorts as controls for period effects. I find that except for when I use the 35–39 year-olds to control for the period effects, the earlier DD results vanish. This suggests either that 15–19 year-olds with gonorrhea were in sexual relationships with men and women from 25–34 years of age, and thus the declines among youth triggered by 15–19 year-olds moved throughout the network at the same time, or that the only proper control group is 35–39 year-olds. I conclude that the abortion legalization hypothesis is inconclusive based on these results.

Chapter 2

STD Risk, Incarceration and Drugs

Sexually transmitted disease (STD) and drug abuse patterns are closely related. Epidemiologists have cited the abuse of certain drugs like crack cocaine and meth-amphetamine, as well as intravenous drugs, as risk factors in disease transmission. To decrease drug consumption, the US has historically relied on imprisonment and interdiction. But, even if imprisonment is successful in reducing drug use, it may also exacerbate the spread of STDs by creating imbalances in the sex ratio. In this chapter, I will explain how drug use and drug policy, specifically incarceration, affects STD risk.

2.1 Relating Drug Abuse to STD Risk: Evidence from Crack Cocaine

Drug abuse may directly affect the spread of STDs by encouraging risky sexual behavior and inducing sex-for-drugs exchanges and prostitution. This is particularly true for crack cocaine. Crack's entry into the illegal drug market was a technical innovation that delivered an intense high for a considerably lower price than powder cocaine.

Crack is made by preparing a water-based solution of cocaine-hydrochloride with ammonia to alkalinize the solution. This material is then heated until a crystallized form breaks off from the solution. This solid crystal is what is then marketed and sold as crack. It is ingested through smoking as opposed to snorting or injection. This method delivers more cocaine into the lungs, creating a stronger euphoric effect than is obtained by snorting powder cocaine (Lancet 1987).

Because of its low price and intense high, crack greatly expanded the population of users. It was also associated with an increase in the behavioral complements of drug abuse —

namely crime (specifically prostitution) and risky sex. Edlin, Irwin, Faruque, McCoy, Word, Serrano, Inciardi, Bowser, Schilling and Holmberg (1994) reported that approximately 16 percent of a sample of crack smokers from a small number of cities tested positive for HIV, as compared to 5.2 percent of the non-smokers in their sample. Furthermore, HIV prevalence was highest among the crack-smoking females from New York (30 percent) and Miami (23 percent). In these two cities, over 30 percent of the women who had exchanged sex for drugs or money was infected with HIV as compared to 9 percent of the non-smoking women. Their regression analysis shows that commercial sex work and anal sex between men accounted for the higher prevalence of HIV infection among the crack smokers as compared with those who did not smoke crack. Crack use may also increase risky sex independent of its effect on commercial sex work. Flom, Friedman, Kottiri, Neaigus, Curtis, Jarlais, Sandoval and Zenilman (2001) document more associations between sex risks and drug use. Specifically, they find that crack users were more likely to report more sex partners than non-smokers, and that they were more likely to report a history of concurrent sexual partnerships. Thus, given the addictive nature of crack, users may resort to income-generating crimes to support their consumption, including commercial sex work. Addicts may also simply take fewer precautions against infection due to crack's pharmacological effects.

Crack may have triggered increases in violence as well. Grogger and Willis (2000) find that crack's appearance in various metropolitan areas was followed by spikes in violent crimes in those areas. They report large increases in aggravated assault and sharp rises in property crime that they attribute solely to the appearance of crack cocaine. Grogger and Willis (2000) argue that the effect of crack on crime was so large that if it had not appeared, the crime rate of 1991 would have been as low as the earlier peak from the early 1980s.

Triggers in crime may have led to increased prosecution of drug-related arrests. Drug trafficking and drug possession were 13.0 percent and 8.3 percent (respectively) of all felony cases brought in state court in 1990, and 22.8 percent and 15.0 percent in 2000 (Charles and Luoh 2006). Thus, increases in drug-related crimes in the 1980s and 1990s may have expanded

the prison population. I will discuss the effect of increased prosecution of drug-related crimes in detail below.

2.2 Showing Causality and Devising Policy

The data linking drug abuse and STDs is largely suggestive. To date, no study has shown that crack cocaine causes the spread of STDs. Many of the studies that shown crack's association with HIV and other STDs either are based on non-representative cross-section surveys, or fail to control for important unobserved variables that could determine both crack use and risky sex. Such research is needed to devise appropriate policy responses. The exception, to my knowledge, is Johnson and Raphael (2006). Using data on AIDS incidence from the AIDS Public Information Data (PID) project and incarceration rates constructed from the Census 1980-2000 IPUMS 5 percent sample, the authors estimate the effect of crack on AIDS incidence. Their measure of crack, which I also employ, is the Fryer, Heaton, Levitt and Murphy (2006) index. Fryer et al. (2006) use a variety of proxies for crack cocaine available at the state and MSA level, which they load into a factor regression to create this index. The proxies are cocaine related deaths, cocaine busts and cocaine related arrests. Johnson and Raphael (2006) find inconclusive evidence that crack was associated with the spread of AIDS among minorities. Johnson and Raphael (2006) find positive and significant effects of the crack index on contemporaneous through 6 lags, but negative and significant effects for lags 10-13. They argue that this pattern "casts some doubt on the hypothesis that crack cocaine explains the rise of AIDS infections among minorities in the US." I claim that this is a problematic association because HIV incubates for an average of eight years, before transforming into full-blown AIDS. Thus to find a positive *contemporaneous* association should be considered spurious. In addition, the quality of AIDS data is poor because of numerous refinements in HIV and AIDS definitions since the start of the epidemic. A more suitable test of the link between crack cocaine and risky sex would be to use a bacterial

infection which has high prevalence, is not transmitted through intravenous drug use, and is more common in heterosexual networks.

Alcohol studies may provide some guidance for studying drug use and STD risk. For example, a large literature on the effects of alcohol tax policy on reducing social "bads" has grown up over the last three decades. On the whole, it has found that increased taxing and regulation of alcohol has reduced underage drinking (Coate and Grossman 1988), traffic fatalities (Chaloupka, Saffer and Grossman 1993; Saffer 1997; Dee 1999; Dee and Evans 2001), and violence (Markowitz, Kaestner and Grossman 2005). Chesson, Harrison and Kessler (2000) added STDs to the list of bads that may be affected by alcohol policy. Using a variety of ingenious methods and robustness checks, they find that beer taxes lower gonorrhea rates. Some scholars have criticized this paper on methodological grounds, as well as because the magnitudes of their estimates do not fit the critics' prior assumptions. For instance, Sen and Lee (2002) criticize the paper for using lagged dependent variables within a fixed effects framework, noting that the estimates are inconsistent. Using an Arrelano-Bond dynamic panel estimator, they find no relationship between beer taxes and gonorrhea rates. Chesson, Harrison and Kessler (2000) find that a 1 dollar increase in the tax on beer reduced gonorrhea rates by 45 percent, which seems too large given our knowledge of the individual price elasticities of demand for alcohol. Other studies have estimated both the effect of alcohol taxes on STD incidence (Sen and Lee 2002; Grossman, Kaestner and Markowitz 2004) and risky sexual behavior more generally (Sen and Lee 2002; Grossman, Kaestner and Markowitz 2002; Rashad and Kaestner 2004; Markowitz, Kaestner and Grossman 2005) and childbearing specifically (Dee 2001; Sen 2003), and the results are generally inconclusive. While the evidence on the connection between alcohol policy and STDs may be inconclusive, the alcohol policy literature highlights the importance of establishing the causal effects of crack.

What is lacking for illicit marks is an equivalent tax mechanism. Policy-makers cannot tax illegal drugs to raise its price, but must rely on legal methods, such as harassing sellers and buyers or restricting inputs. DeSimone (2001) is an exceptional study that links changes

in cocaine prices — triggered by the invention of crack which presumably shifted the supply of cocaine, lowered prices and expanded the overall market for cocaine — with crime. Using data from the System for the Retrieval of Drug Evidence (STRIDE), which is a composition of drug seizures and undercover drug purchases by DEA agents going back to the early 1980s, and FBI Uniform Crime Report data on the seven index crimes, DeSimone (2001) estimates two-stage least squares equations to estimate the impact of crack on crime price, and finds that crack's appearance was associated with falling cocaine price in the first stage. Instrumenting for cocaine price with crack, he finds cocaine price is negatively associated with six of the seven FBI index crimes. This paper supports the policy of using legal enforcement to raise the price of an illegal good as to reduce crime.

2.3 The Role of Incarceration

It is often reported that the so-called "war on drugs" has resulted in a massive expansion of the prison population in the US, which has disproportionately removed low-educated, young, minority men from the general population (Patillo, Weidman and Western 2004; Western 2006; Raphael 2004; Charles and Luoh 2006). I discuss the evidence for this below. I argue widespread incarceration may itself be a new risk factor in the spread of STDs.

2.3.1 Trends in Incarceration

The Bureau of Justice Statistics estimated that by end 2005, 2.2 million men and women were incarcerated in state or federal prisons or local jails. This is the highest level of incarceration and amounts to the highest incarceration rate of any OECD country. The US has always had relatively high incarceration levels and rates, but in the mid-1970s they began to rise sharply. Between 1974 and 2001, the number of persons incarcerated in prisons and jails grew from approximately 100 inmates er 100,000 to almost 700 per 100,000 (Patillo, Weidman and Western 2004).

This increase in incarceration has been unevenly distributed across race, age and class. Raphael (2004) reports that the share of all Black males who were institutionalized grew from 0.03 in 1970 to 0.08 in 2000, whereas the share of all White males remained at 0.01 over the same period. Racial disparities exist even controlling for education. For Black (White) males with less than a high-school education, 19 (4) percent were institutionalized. Most of this is concentrated among Black men in their 20s and 30s. The percentage of Black male high-school dropouts aged 18-25, 26-30 and 31-40 who were institutionalized in 2000 is 23 percent, 34 percent, and 28 percent respectively. Raphael (2004) reports that for Black male high-school dropouts aged 26-30, more of these men (34 percent) are institutionalized than are employed (30 percent). Roughly a third of all young Black males in the US with only a high-school degree or less are in jail or in prison on any given day (Western 2006).

The lifetime risk of ever being incarcerated reveals even more disturbing inequalities. Approximately 14 percent of all 30-34 year-old White male high-school dropouts were either dead or had been imprisoned at least once in their life, compared to 5.5 percent of White males with a high-school degree or GED equivalent. For Black males, the numbers were much higher: approximately 62 percent of all Black male high-school dropouts will be dead or imprisoned at least once by their mid-30s, compared to 21.9 of those with a high-school degree or GED equivalent. In other words, for young Black males with little schooling, incarceration is the modal experience in ones transitioning to adulthood. Most young black men who do not graduate from high-school look forward to living at least some share of their young adulthood behind bars.

2.3.2 Examining the Rise in Incarceration

Why did the prison population grow so dramatically from the 1970s to 2005? There are two possibilities: changes in crime and changes in law enforcement. Thirty years ago (Levitt 2004), criminologists and other social scientists were largely in agreement that US crime rates would continue to rise. However, crime rates that had been on the rise throughout the mid-to-late

1980s suddenly and unexpectedly began to steadily drift down (Donahue and Levitt 2001; Levitt 2004). When the labor market prospects of young, unskilled men began to improve in the 1990s, crime rates among the underclass fell – even while imprisonment rates continued to climb. This has led some observers to suggest other causes besides underlying economic and crime trends to explain the continual rise in imprisonment.

One of the most important law-enforcement changes over the past 30 years is the so-called "war on drugs", which was initiated during the Nixon administration and continued during the Reagan years. For instance, drug-related arrests grew from 580,900 in 1980 to 1.58 million in 2001 (Patillo, Weidman and Western 2004). Patillo, Weidman and Western (2004) calculates that the number of people admitted to prison per drug-related arrest was .02 in 1980 and grew to .12 in 2001. The number of parolees per admission also grew from .11 in 1980 to .22 in 2001, which Western argues reflects the increased strictness parolee drug tests during their parole. Failing a drug test, he writes, is a common trigger of re-arrest among parolees, and became more consistently enforced during the period in question. The drug-crime incarceration rate also grew dramatically over this period, from 8 drug-crimes per 100,000 in 1980 to 86 per 100,000 in 2001. All of this is viewed as supporting evidence that the "war on drugs" played a prominent role in the expansion of prison populations in the latter quarter of the 20th century.

The "Just Say No" campaign in 1986, as well as the creation of the Office of the Drug Czar in 1988 also represented key points in the expanding war on drugs. Coincident with these were the passage of a series of laws meant to increase the severity of sentencing for drug offenders and other felons, such as three strikes, minimum sentencing legislation, truth-in-sentencing legislation, and sentencing guidelines. Altogether, the combined effects of expanding the scope of enforcement rules to include the strict enforcement of drug prohibition, as well as the increased severity of penalties in the form of longer sentences, is probably the driving force in the expansion of the prison population. As has been said, this expansion was felt keenly by the underclass namely, uneducated, young, Black males, who now face a lifetime

risk of imprisonment of over 60 percent by the age of 35 (Patillo, Weidman and Western 2004).

2.3.3 Consequences for Mating Markets

Removing (Black) men from the general population in such larger numbers creates a significant imbalance in the sex ratio as well as disrupts monogamous sexual relationships in the general population. Either of these can have implications for the spread of STD, but differ in magnitude. Incarcerating men will disrupt a monogamous relationship and increases the probability of the (now) unattached female forming a new sexual relationship. Insofar as the new relationship is itself monogamous, lifetime partnerships increase because of incarceration and non-infected women will face new risks if they match with an infected partner.

A separate effect is incarceration's impact on the kinds of relationships non-incarcerated men form when facing a surplus of unattached women. The shortage of men will have three main effects on the mating market. First, it will reduce the probability of a woman finding a mate. Second, the matches in equilibrium will generally favor the men; men will likely match with women whom they prefer more and women with men they prefer less, all else equal (Roth and Sotomayor 1990). Evidence for this has been found in Angrist (2002) and Charles and Luoh (2006), just to name two of the many studies done along these lines. Becker's model of marriage (Becker 1973, 1981) point to a third, somewhat less obvious effect. A reduction in the supply of men should shift the gains from relationships away from women towards men. Women will therefore make transfers to men so long as the net gain from the relationship compensates them for their next best alternative on the market.

The theoretical models of marriage are largely silent about the nature of the intra-household transfers, usually choosing to model them in terms of cash or utility (Manser and Brown 1980; McElroy and Horney 1981; Lundberg and Pollak 1993). Yet the nature of the transfers matters since some may possess network externalities important to the study of STDs. For instance, some men may respond to their position by forming semi-polygamous

sexual relationships, which they implicitly expect the women to tolerate. For such relationships, which are technically called "concurrent sexual partnerships," can greatly amplify the spread of an STD throughout a network. Would some men prefer semi-polygamous matches to monogamous ones? Biological differences in male and female reproductive capacity suggest that the answer is yes (Posner 1992; Willis 1999).

Concurrency facilitates the speed with which an STD moves throughout a population by decreasing the average distance between infected and non-infected individuals, holding constant the average number of sexual partnerships in a network (Morris and Kretzschmar 1995; Kretzschmar and Morris 1996; Morris and Kretzschman 1997). Thus, small increases in the share of concurrent relationships can have significant multiplier effects throughout the network, because the STD can immediately spread to another partner without waiting for the previous relationship to dissolve. The growth of the epidemic is contained by the extent of the network, but the speed with which it grows and the infectious agents' ability to move throughout the network are critically affected by the number of nodes simultaneously linked. Morris and Kretzschman (1997) show that when one-half of all partnerships in a population are concurrent, the size of the epidemic after five years is ten times larger than if all partnerships were serially monogamous and holding constant the number of partners.

Adimora and Schoenbach (2005) have suggested that concurrency (among other factors), brought upon by high rates of Black male incarceration, could be an important factor in explaining racial disparities in STD incidence. Given the information asymmetries inherent to sexual partnerships (Over 1999), imbalanced sex ratios could amplify those asymmetries by providing men with more opportunities to form sexual partnerships with women other than their primary partners.

2.3.4 Incapacitation, HIV and Prisons

STD risk may also be related to incarceration simply through incapacitation. Prison is a high-risk environment where STDs are more likely to spread. In 2004, 1.8 percent of all US

prison inmates were HIV-positive (Okie 2007) — more than four times the estimated rate in the general population — and condoms are not legally distributed in prisons. In addition, the instruments used for tattooing prisoners are extremely unsanitary, creating a separate risk for infection. Reliable data on sexual violence does not yet exist, but with the Prison Rape Elimination Act of 2003, the Justice Department has begun collecting comprehensive statistics on prison rape, which will lead to better understanding of the health risks of that current environment.

Johnson and Raphael (2006) examine the relationship between Black male incarceration and AIDS incidence among Black females. As I discussed earlier, the long incubation period and the quality of AIDS data make identifying causal effects of risk factors difficult. Johnson and Raphael (2006) use prisoner-overcrowding litigation to identify exogenous variation in prison releases, following Levitt (1996). Any legislation that increases prison release shortens a prisoner's sentence, and therefore places men back into their communities. If it is the case that HIV infection occurs during a prison sentence, then this strategy may be able to isolate the effect of imprisonment on AIDS incidence in the general population. To test this, the authors use five states identified by Levitt (1996) where overcrowding lawsuits caused correctional systems to increase their release rate. The authors match each of these five states to three controls states using matching estimators to find the nearest neighbors with comparable incarceration, prisoner-admission, and prisoner-release rates. First, Johnson and Raphael (2006) examined the effect of the litigation-induced shocks to the prisoner release rates on all AIDS infections. The AIDS infection rates among the treatment group resembles those for the control group for the first three years after litigation occurs. After three years, though, AIDS incidence diverges between the treatment and control states. Specifically, at years four through ten, AIDS infections grow at a faster rate in the treatment groups, and for a period continue to grow even while AIDS incidence falls in the control states. The differences, per year, are between 3 and 5 additional AIDS cases per 100,000 in the treatment

states. This can be interpreted as the effect of the early release of infected offenders on AIDS cases in the treatment states.

Second, Johnson and Raphael (2006) analyze the effects of early release by race. Because of the high rates of incarceration among Black males, early release should be disproportionately skewed towards BLacks. AIDS infection rates for Blacks in treatment states again follow a similar trajectory to control states for years 1-3 following overcrowding litigation, and then diverge from the control states for years 4 and on. In years 4-6, the annualized AIDS differential for Blacks following overcrowding litigation was in the range of 20 additional cases of AIDS per 100,000, and by year 10, it had fallen to approximately 10 additional cases per year. Johnson and Raphael (2006) also report that prison release was causing a spike in AIDS incidence among women suggesting prison transmission is a real disease vector. The divergence in female AIDS incidence between treatment and comparison states happens immediately following the early release. These differentials increase continually until the 8th year after the court decision, at which point they decline. At their peak, there is around a 30-point difference in AIDS incidence among females in treatment states. Johnson and Raphael (2006) show that for Blacks and for women, post-decision years are typified with spikes in AIDS incidence that are statistically significant and meaningful in magnitude. However, they do not tell us whether it is concentrated among Black women or not.

2.4 Conclusion

I have reviewed the broad literature on the twin epidemics of drug abuse and sexually transmitted diseases, showing that there are in fact two separate, yet related, disease vectors that connect drug markets to STDs. The relative numbers of men and women in the population can affect not only the number of people in a sexual relationship, but also the kinds of sexual relationships. Men facing a surplus of unattached women have both opportunities and bargaining advantages to form concurrent partnerships, which will amplify an STD epidemic. High rates of male incarceration is one source of such sex ratio imbalances. In the US, as a

result of the so-called "war on drugs," 12 percent of all Black men are incarcerated, which has effectively altered the mating environment such that Black women face a shortage of eligible Black men, and in turn, may be forced to accept arrangements that are less advantageous to them, such as tolerating promiscuity in their mates. In the next chapter, I test this hypothesis using individual-level data from the NLSY97 and state-level data from the 2000 Census 5 percent sample from IPUMS. I estimate several different models to examine the relationship between sex ratio changes from the time Black and White males are teens until they are in their late 20s so as to determine the degree to which this can account for racial differences in promiscuity. In another chapter, I use state-level data on gonorrhea and syphilis from the CDC and incarceration rates from the Census so as to examine the contemporaneous association between incarceration and STDs. I also test the separate disease vector relating drugs to STD outcomes. Drug abuse, especially crack cocaine, can cause an increase in risky sexual behavior, including commercial sex work, multiple partners and inconsistent condom use, as well as delay treatment for STDs. I test the impact of crack on gonorrhea and syphilis, independent of incarceration's effect, using a crack index created by Fryer et al. (2006) and STD data from the CDC.

Chapter 3

Sex Ratios and Risky Sexual Behavior

3.1 Introduction

> "It's hard because men have it easy. They have two to three women per man, so it's very easy for him to not stay committed. A woman like me is looking for commitment and will try almost anything just to keep that commitment going ... I'm gonna accept this BS he's giving me because ... without him ... it's gonna be hard for me to find someone else to [be with] ... seeing it as, 'if I let him go, this [other] woman's gonna have him.' ... I don't want to be alone."
>
> — Black woman, Syracuse, New York, 2003 from Lane, Keefe, Rubinstein, Levandowski, Freedman, Rosenthal, Cibula and Czerwinski (2004)

As of 2003, nearly one million people had been infected by HIV/AIDS in the US alone, and over 500,000 had died. Behind these aggregate totals is a picture of remarkable racial disparity in HIV/AIDS, and sexually transmitted diseases (STD) in general. According to 2005 Vital Statistics, HIV was the seventh leading cause of death among Blacks but only the twenty-second among Whites. For Black women aged 25-34 years, it is the leading cause.

Black Americans constitute 12.3 percent of the US population, yet account for over 50 percent of estimated new HIV/AIDS diagnoses. Table 5.1 presents AIDS case-rate data from the CDC HIV Surveillance Report for 2003. Black infections outnumbered White infections 21,174 to 12,175, with a Black case rate (per 100,000) of 75.2 and a White case rate of 7.2. The Black male case rate is 10 times that of Whites; the Black female case rate is 25 times greater.

Blacks not only account for more new cases, but overwhelmingly more of those arising through heterosexual contact. Table 5.2 reports the numbers of adults and adolescents living with HIV/AIDS at the end of 2003, by race, sex and exposure category. Only 5 percent of infected White males contracted the infection through heterosexual contact, while 22 percent of Black males report heterosexual transmission. Similar exposure disparities exist for Black and White women. Heterosexual contact accounts for 64 percent of White females, compared with 75 percent for Black females. In 2003, there were 3.5 times more Black women living with HIV/AIDS who had contracted the disease heterosexually.

Epidemiologists do not fully understand the causes of these racial disparities (Adimora and Schoenbach 2005), but several explanations have been proposed. Recent research has emphasized compositional differences in sexual networks (Laumann and Youm 1999). For instance, Whites with many sexual partners who comprise a "core group" in the sexual network mix less often with the rest of the population than their Black counterparts. To the extent core-group members mix with others, disease will spread more broadly through the population. Adimora, Schoenbach, Martinson, Donaldson, Fullilove and Aral (2001); Adimora, Schoenbach, Martinson, Donaldson, Stancil and Fullilove (2004) have focused on the number of concurrent relationships in a sexual network as a primary factor. Concurrency, the temporal overlap of sexual partnerships, facilitates STD growth and transmission by decreasing the average distance between infected and non-infected individuals, even holding constant the average number of sexual partnerships (Morris and Kretzschmar 1995; Morris and Kretzschman 1997). Thus, small increases in the share of concurrent relationships can have significant multiplier effects on the infection's growth rate. They show that when one-half of all partnerships in a population are concurrent, the size of the epidemic after 5 years is 10 times larger than if all partnerships were sequentially monogamous.

Concurrency is hard to measure, but I am aware of at least two studies that document racial differences in concurrency patterns. Adimora et al. (2004) find higher rates of concurrent sexual partnerships among heterosexual Blacks, aged 18-59, in rural North Carolina

(a region with unusually high STD rates). Using the 1995 wave of the National Survey of Family Growth, Adimora et al. (2002) report that Black females are more likely to have had a concurrent sexual partner over the last year compared to White and Hispanic females.

Citing concurrency as a proximate cause invites another question: why do Blacks have more concurrent sexual encounters? In this paper, I emphasize the relative shortage of men in Black communities. The sex ratio, as quantified by the relative number of non-institutionalized females to males for a race-, age-, and geographic-specific "relationship" market, tends to be higher for Blacks because of high levels of Black male incarceration (Raphael 2004; Charles and Luoh 2006). Research has linked imbalanced Black sex ratios to out-of-wedlock childbirth (Wilson 1987; Willis 1999; Neal 2004), female-headed households (Fossett and Kiecolt 1993; South and Lloyd 1992), marital delays (Lichter, McLaughlin, Kephart and Landry 1992; Brien 1997), wages and schooling(Angrist 2002; Charles and Luoh 2006) and welfare dependency (Darity and Myers 1984). I am not the first to propose a connection between the sex ratio and concurrency. For example, Adimora, Schoenbach, Bonas, Martinson, Donaldson and Stancil (2002), Adimora and Schoenbach (2005) and Lane et al. (2004) all argue that a shortage of men may cause women to feel desperate about the prospects of finding a stable partner, encouraging short-term relationships with less-committed mates. But their empirical evidence is largely qualitative and ethnographic in nature. Posner (1992) hypothesizes that differences in the "effective sex ratio" lead to more male-centered couplings in Black networks, but does not attempt to test this hypothesis. However, I am the first to test for such a relationship econometrically using a nationally representative dataset.

Closely related to my work is a study by Johnson and Raphael (2006), who attempt to directly link incarceration and AIDS outcomes among Blacks. Using aggregate data from the 1970-2000 Census and AIDS case data from the CDC, the authors present evidence that incarceration dynamics account for the racial difference in AIDS outcomes among females. Their findings suggest two separate mechanisms for disease transmission. First, by removing

large numbers of males from the population, incarceration hampers the bargaining position of females in the relationship market. Second and separately, incarceration exposes males to STD risk via incapacitation. Prisons and jails have both higher STD rates and increased risky sexual behavior between male inmates. Upon release into the community, former inmates place females at risk as they re-enter the relationship market. However, because HIV incubation spells are so long (the median spell is 9 years), Johnson and Raphael (2006) are unable to distinguish between these two mechanisms.

My focus on risky sexual behavior allows me to explore the first mechanism. Utilizing data from the 1997 National Longitudinal Survey of Youth (NLSY97), I first examine the effect of the sex ratio on recent sex partners — my measure of concurrency. Next, I investigate the sex ratio's effect on defensive STD choices, as represented by condom use. The direction of this relationship is theoretically ambiguous. On the one hand, a shortage of males improves a man's ability to negotiate sex without condoms. On the other hand, sexual behavior can have negative externalities throughout the network if it increases the probability of others matching with an infected agent (Jackson 2005; Ballester, Calvo-Armengol and Zenou 2006). A rational male will consider disease prevalence in deciding whether to use a condom. Therefore I estimate the effect of sex ratios on condom use separately for those with lower and higher risks of infection. Broadly, I find strong evidence that the sex ratio increases recent partners, and by extension, concurrency. The evidence is somewhat weaker that sex ratios affect condom use. Taken together, my findings provide additional support for the proposition that incarceration, operating through the sex ratio, likely increases the spread of STDs in general and HIV/AIDS in particular.

The paper is organized as follows. Section 2 reviews the racial differences in sex ratios and section 3 explores the relationship between STD prevalence and optimal STD risk. Section 4 describes my data and measures for the sex ratio, concurrency, condom usage, and the control variables, and the results from regressions. Section 5 concludes the paper.

3.2 Racial Differences in Sex Ratios

Using data from the 2000 Census longform, I calculated White and Black sex ratios for six age intervals: 0-1 year-olds, 1-5 year-olds, 6-10 year-olds, 11-15 year-olds, 16-20 year-olds, 21-25 year-olds, and 26-30 year-olds[1]. Traditionally, the sex ratio (sr) is defined as the ratio of men to women. Figure 5.1 depicts the *inverse*, which I utilize in estimation to facilitate interpretation of the sex ratio's effect. Figure 5.1 compares the (inverse) national sex ratio (i.e., sr^{-1}) for Blacks and Whites across age cohorts. The data show little racial disparity from birth or early adolescence, but by early adulthood the Black and White ratios begin to diverge sharply. This pattern is consistent with the racial differences in male incarceration reported in other studies (Raphael 2004; Charles and Luoh 2006). By the time Blacks reach their late twenties, there are about 128 Black women for every 100 Black men. I therefore use a change of 28 (required to establish parity) to interpret my estimate of the partial effect of the sex ratio on male sexual partnerships.

The 2000 Census longform was also used to compute the ratio of non-incarcerated 18-24 year-old males to 18-24 year-old females for Blacks and Whites in all 50 states. Figures 5.5 and 5.5 show the geographic variation in Black and White sex ratios. Darker shading indicates greater shortages of males. States with the largest Black populations generally have sr^{-1} values greater than 110. By contrast, there is little variation in White sex ratios across states.

Over 90 percent of the US Black population lives in 24 states. Figure 5.4 shows sr^{-1} by age interval in each of these states. The pattern exhibited by the national sex ratio is repeated and generally more pronounced. By their mid-20s, Black women significantly outnumber Black men in every state.

[1]The details of this calculation are discussed in section 4 below.

3.3 Relationship Markets, Concurrency and Male STD Risk

Standard matching models predict that the removal of men from the marriage market reduces female probability of marriage, and empirical studies have borne this out (Brien 1997). But there are other, more subtle, predictions that flow from such models. First, as shown by Roth and Sotomayor (1990), removing men from the market will affect the optimal sorting of both males and females into matches. Assuming that males and females have complete, ordered preferences, removing males creates a new, stable pairing where men move up their preference ordering, and women move down. As a result, females will have a higher chance of matching with a male they preferred less, if they match at all. Second, females who do match are hurt because the division of output will be renegotiated in response to the removal of men (Becker 1973). This effect induces women to make transfers to their partners because their bargaining position is weakened. The types of transfers that might take place range from the trivial, such as who picks which restaurant when going on a date, to the much more serious, such as toleration of sexual infidelity (or worse). I estimate the sex ratio's effect on concurrency as a test of the latter.

Sex ratio induced concurrency raises a question about male STD risk. What effect might the removal of men from relationship markets have on contraceptive decisions? On the one hand, if some men prefer vaginal intercourse without a condom—because the sensual pleasure is greater presumably—men in a strong bargaining position might successfully negotiate less frequent condom usage, ceteris paribus. Babcock and Laschever (2003) discuss research that found women are reluctant to negotiate condom use with their husbands, since doing so was tantamount to accusing them of sexual infidelity. On the other hand, if the sex ratio increases the degree of concurrency across the sexual network, then the risk of contracting an STD has increased for all people attached to that network. The more partners a person has, the more likely they will eventually come into contact with an infected female, and thus contract an STD themselves (Holmes, Sparling, Mardh, Lemon, Stamm, Pio and Wasserheit

1999; Finer, Darroch and Singh 1999). Thus, a lower sex ratio might induce some men to wear condoms more frequently to compensate for the increased risk.

I cannot identify which men in our sample prefer unprotected sex and which men would likely respond to the increased risk of their sexual network. Therefore, I use quantile regressions to separately estimate the effect at the .10 and .50 quantiles. These two quantiles are important because they represent two extremes: men who never or rarely wear condoms, and men who nearly always wear condoms.

3.4 Data

My data come from two sources: the 2000 Census longform survey, also known as the 5-percent sample, and the NLSY97 Geocode. The Census data are used to construct the sex ratios and the NLSY97 provides information on the sexual behavior of adolescents and young adults.

3.4.1 Sex Ratio Construction

Although the sex ratio is easy to understand conceptually, there is no consensus on the correct way to measure it empirically. One issue concerns the relevant age cohort. Angrist (2002) staggers the male-to-female ages in such a way that men are assumed to search among women roughly two years younger than themselves. Numerous demographers and sociologists have followed this strategy (Gutenberg and Secord 1983). But it is unclear whether even this added level of realism is correct since there is substantial dispersion around the mean age difference between partners. The relevant geographic area or market is another issue. Fossett and Kiecolt (1991) recommend constructing sex ratios defined by race, age and either MSA or county-level. However, Brien (1997) presents evidence that county- and MSA-level sex ratios constructed from Census longform data contain serious measurement error. Using the 5-percent sample at the county/MSA level creates measurement error because some cells have so few observations. The problem is exacerbated in counties/MSAs with small Black

population shares. The correlation of the measurement error with race makes an analysis based on counties or MSAs unappealing.

Measurement error is not the only reason to be skeptical about a county- or MSA-level analysis. One obvious adjustment to a sex ratio imbalance in your relationship market is to move your search to another neighboring market. Females who face a deficit of men in their immediate vicinity may look outside for a mate. This is much more likely to be a problem across counties and MSAs than across states. As an example, Adimora et al. (2004) report that women in North Carolina traveled to the military base, Fort Bragg, because men were lacking in their immediate communities.

So, I base my empirical work on sex ratios calculated at the state level. For any geographic area, distinguishing the non-institutional population from the population at large is key. The Public Use Microdata Samples (PUMS) for each census includes an indicator for the institutionalized and non-institutionalized population by age, race, sex and state. I construct a race-, age- and state-specific sex ratio based on the number of non-institutionalized men and women reported in the 2000 Census longform survey. As Charles and Luoh (2006) note, institutionalized mainly consists of incarcerated individuals, particularly for a cohort as young as the one I am examining.

Following Charles and Luoh (2006), I exploit the fact that the overwhelming majority of sexual relationships occur between women and men of similar age, race and geographic location (Laumann et al. 1994). I measure the contemporary non-institutionalized sex ratio for each demographic group defined by state of residence, age group, and race/ethnic group according to the following formula:

$$sr_{a,r,j} = \frac{\sum_{a-b}^{a+b} M_{a,r,j}}{\sum_{a-b}^{a+b} F_{a,r,j}}, \tag{3.1}$$

where $M_{a,r,j}$ ($F_{a,r,j}$) denotes the number of non-institutionalized men (women) of age a, race r, living in state j. Using a strategy similar to Helmchen (2005), I estimate sex ratios that vary by age according to the age ranges depicted in Table 5.3. I will be exploiting variation in the state-age-race-cohort cell to identify the effects of the sex ratio.

3.4.2 Concurrency, Condom Use and Control Variables

My measures of concurrency and condom use come from the NLSY97, a household survey representative of people living in the US in 1997 who were born during the years 1980 through 1984. I obtained the NLSY97 Geocode through special request, which includes geographic indicators for each respondent that allow me to match each respondent to a specific state.

Concurrency reflects temporal overlap in sexual couplings. Mathematically, it is expressed as two links to two separate nodes emanating from one node. This has been modeled using graph theory, and as such, approximates what might be concurrent pairings in the "real world." For instance, if a male had sex with Female A on June 2nd, Female B on June 5th, and Female A again on June 10th, then one might describe this as a concurrent match. Thus, the ideal data source would contain information on both the number of recent encounters and the dates of each encounter. The National Survey of Family Growth (NSFG) provides such information but it is a repeated cross-section (not a panel) and males were not surveyed until 2002. The NLSY97 is currently comprised of eight waves, following the same individuals in a panel that extends through 2004. Although it does not provide information on encounter dates, the NLSY97 does report the number of vaginal intercourse partners defined in terms of the last twelve months for 1997 and since the date of the last interview for all subsequent waves. I use the recent partner data to compute rp — the number of "recent female sexual partners" for each respondent in the sample.

Male NLSY97 respondents are not only asked their number of sexual encounters during the past year, they are also queried about the number of times they wore a condom and the number of times they had vaginal intercourse since the date of their last interview. As before, I construct measures of sexual precaution using the months since the date of the last interview and respondents' answers to how much sex and how many condoms they have had since the date of the last interview, then multiplied that number by 12. I combine the measures based on these questions to calculate a condom use rate, c/s (where c is the frequency of condom

use in the last twelve months, and s is the number of vaginal sex encounters), which I use as my safe-sex measure.[2]

The NLSY97 also provides information on a number of important covariates. In particular, I control for a person's age on the date of the interview (measured in months), education level (measured as the respondent's highest school grade completed on the date of the interview), marital status and family structure (proxied by an indicator of whether respondent's biological or adoptive parents are still married).

My sample starts with a total of 8,984 NLSY97 male respondents in 1997. From those individuals I select Black and White non-Hispanics who have complete data for all six available years of the survey.[3] There are 2702 Whites and 1198 Blacks who are observed from 1997 to 2002. Individuals in my sample were not asked questions about their sexual histories until they reached the 14, but in 1997, 12–13 year-olds were in the sample. I imputed rp for 12- and 13-year-olds by examining the sexual histories of these individuals when they answered the sex history questions in 1998 and 1999. Most male children 12 to 14 years-old have not yet made their sexual debut, and so respondents who reportedly were virgins at 14 were necessarily virgins when they were 12–13. I assigned values of 0 to all 12–13-year-olds who were reportedly still a virgin when they were 14. Individuals who were sexually active by age 14, and who had made their debut when they were 12 or 13, could still have partnerships imputed to their earlier histories. First, if they had only one lifetime partner, and had made their debut when they were 12 or 13, I assigned a 1 to the appropriate year. Second, if they had more than one lifetime partner, and made their debut when they were 13, I could subtract their number of recent partners from their lifetime partners, and assign the appropriate value to both age 12–13. If individuals had lost their virginity when they were

[2]In a few instances, an individual would report wearing condoms more times than he reported having sex. These observations were dropped from the sample.

[3]I ignored Hispanic and Latinos for the simple fact that Census data on Hispanic/Latino characterization is currently very poor. Further, Hispanic/Latino relationship markets are diverse, making it even difficult to match individuals in the NLSY97 sample with an appropriate Census race category.

12, I distributed an equal number of partners to 12–13 depending on their lifetime partners in 14.

I then experiment with various cuts of the data which include balancing the panel on individuals who were in the survey for each wave from 1997-2002, for 1999-2002, restricting the sample to only those individuals who live in the 24 states with the highest black populations, and restricting the sample to individuals who do not show unusually large deviations in partnerships year to year. The results are roughly the same - I find large associations between changes in Black sex ratios and reported sexual partnerships at the 90th quantile for each of the cuts, suggesting that Black males are responsive to changes in their own sex ratio.

3.5 Estimation and Results

My general empirical strategy is to estimate models of the form

$$y_{ijt} = \beta_1 r_i + \beta_2 (sr)^{-1}_{ijt} + \beta_3 (sr)^{-1}_{ijt} \times r_i + \mathbf{x}_{ijt}\gamma_{ijt} + \epsilon_{ijt}, \tag{3.2}$$

where y is either rp or c/s, r is a race indicator set to one for Blacks, sr is the sex ratio, $\mathbf{x}$ contains the covariates, and ϵ_{ijt} is an error term that, in principle, includes individual (i), state (j), and time (t) effects. As mentioned earlier, sr enters the regression in its inverse (i.e., as the ratio of females to males), multiplied by 100. For example, for early adulthood, sr^{-1} is 128 on average for Blacks, ages 18–24. Since I predict that the disappearance of males is positively associated with increased concurrency among Black males, this formulation allows us to easily interpret an increase in sex partners in terms of an increase in the surplus of females.

The extreme right tail of the rp distribution should reflect individuals with a taste for promiscuity. Because our main interest lies in the effect of changes in the sex ratio on promiscuity, I focus most of our attention on the 90th quantile. However, the same argument applies to the left tail of the condom use distribution. I estimate pooled OLS (POLS) and linear

fixed effects (FE) versions of (2). Comparing these results will allow me to determine whether unobserved heterogeneity is driving my main results. The POLS and FE standard errors are corrected for within-individual clustering. Then, I turn to quantile regression to estimate the partial effect of sr^{-1} at different points of the recent-partner and condom-use distributions. The standard errors for the quantile regressions were obtained by bootstrapping.

3.5.1 Effects of Sex Ratios on Recent Partners

In principle, the NLSY97 respondents were interviewed once a year, but in practice, there was some variation in how often the surveys were conducted. For instance, in 1998, the mean number of months since the date of the last interview was 19.7 with a standard deviation of 2.9. Subsequent waves had means closer to 12 months, but numerous individuals were asked questions after only 7 months had passed, and others were not asked questions until 16 months from the date of the last interview. Therefore, to minimize measurement error, I constructed an annualized rate of recent partners (*rprate*) equal to the number of recent partners reported since the date of the last interview, divided by the numbers of months since the date of the last interview multiplied by 100.

Table 5.5 reports the average and quantile mean *rprate*, by age and race. The White male *rprate* grew steadily from age 13 to 21, and from age 13 to 19 for Black males. The 12–13-year-old data has a smaller standard deviation than all other years, which is not surprising given that a majority of children do not make their sexual debut until well into their teenage years. Blacks have a much higher mean number of partners than Whites throughout most of the sample, and especially during the late teenage years. Unlike other studies which top-code at 7 partners in the last 12 months, the NLSY97 top-codes recent sexual partnerships at 99. When top-coding is dealt with, these figures are consistent with other data sources, such as the National Survey of Family Growth 2002 (NSFG). Thus, it becomes clear that the higher average number of Black female partners is due to the relatively larger numbers of reported partnerships at the right tail. While the median number of annual sexual partners

is larger for Blacks at all age brackets, the difference is much smaller in magnitude than when comparisons are made at the mean.

Table 5.5 reports descriptive statistics for respondent age (measured in months), highest grade completed, a dummy variable equalling 1 if their biological/adoptive parents have remained married, a dummy variable equalling 1 if they are married, and sr^{-1}. These summary statistics combine Blacks and Whites, which push down the sex ratio since White sex ratios are relatively balanced across all years of the sample. Nevertheless, sex ratios are at around 98 for the mean of the sample, meaning there are approximately 98 women for every 100 men. At the right tail, that statistic grows to around 109. The education variable is centered around the high school years, though for the last few years of the survey sample, numerous individuals have begun taking college courses, which can be seen in the 90th quantile. Approximately 50 percent of the sample have parents that are still married, and only 1 percent of the sample is married at any point in they survey years.

I first estimate (2) omitting the sr^{-1} terms so as to both highlight the racial disparities in sexual partners and derive baseline regressions for all that follows. The results of this exercise are reported in Table 5.7. The first column presents POLS estimates of the mean difference, while the remaining columns show the racial disparities at the .5, .75, and .9 quantiles. Panel A gives the results for the entire US and panel B those pertaining to the 24 states that account for over 90 percent of the US Black population. All estimates are conditional on state and year fixed effects.

Reading across the first row of Table 5.7, the racial disparity in *rprate* among adolescents and young adults is clear. On average, Blacks have .82 more partners than whites; that difference is small at the median but increases to 1.8 partners at the .90 quantile. This pattern is essentially the same in the states where Blacks are concentrated.

The estimated mean effects of the covariates are as expected. Recent partners increase with age (to a point); decrease with schooling; and are lower among young people whose parents are still married. However, there is some heterogeneity in the estimated coefficients

across quantiles, with magnitudes generally increasing as you move into the right tail of the *rprate* distribution. Again, the results are similar, whether the analysis is conducted for all 50 states or the subset of 24 with substantial Black populations. For most estimates, dropping individuals from the lowest Black population states has a negligible effect. The largest effect is on the effect of the household variable. When using all 50 states, the mean difference on the household variable is –.45, but falls to –.35 when I drop individuals from the states which have few Blacks in the NLSY97.

Table 5.8 introduces the sex ratio terms and presents the results for the entire sample. The first four columns repeat the specifications in 5.7. The last column reports the results from the specification that adds individual fixed effects. The focus is on the Black$\times sr^{-1}$ interaction and in almost every case it enters positively, though not always significantly. I employed both the POLS and FE estimators to assess the role of unobserved heterogeneity. The estimates are marginally different, although neither result is statistically significant. For instance, POLS estimates are positive, but small and insignificant, whereas the FE estimates are zero at three decimal points with very large standard errors. This is important because estimating FE quantile regressions is problematic (Koenker 2004).

When I examine the effect of the sex ratio at different points in the *rprate* distribution, the story is different. The estimated effect of the interaction term is 0.007 at the median; it is four times larger at the 75th percentile and almost ten times larger at the 90th percentile. The estimated effects are significant at approximately the 1 percent level in each case.[4] As I noted earlier, the Black sex ratio was approximately 100 for Black teenagers and reaches as high as 128 for Blacks in their late 20s. Thus using a 28-point change in the Black sr^{-1}, these estimates translate into 0.20 more partners a year at the median and approximately 1.9 more partners a year at the 90th percentile.[5] In contrast, except for the weak evidence at the median, the sex ratio does not appear to matter at all Whites, which makes sense given the lack of variability in the White sex ratio both across states and over the life-cycle.

[4]The p-value for the estimated median coefficient is 0.02.

[5]Multiplying the estimated coefficient by 28 gives 1.876

Given the quantile regression results, the failure to find a significant effect in the conditional mean is a little surprising. Measurement error could explain the small interaction coefficient estimate. Geographic regions with small Black populations yield demographic cells that are especially sensitive to changes in the denominator and numerators. Therefore, I repeated the analysis in Table 5.8 using only respondents living in the 24 highest Black population states. Table 5.9 presents these results. First the POLS and FE estimates are both positive and larger in magnitude after having dropped the states with small Black populations, though only the POLS estimate is significant at the 10 percent level. Again, the similarity of the POLS and FE estimates alleviates some of the concern about neglected heterogeneity.

Second, dropping the states with low Black populations increased both the magnitude and precision of the quantile estimate of the interaction coefficient (see Table 5.9). The estimated effect of the interaction term is 0.011 at the median, is almost five times larger at the 75th percentile and nearly eight times larger at the 90th percentile. The precision is at the 1 percent level for all quantile estimates. Black males at the far right tail of the *rprate* distribution are estimated to have approximately 2.4 more female sex partners a year due to a change in the sex ratio.

There are two sources of error that could affect the sex-ratio results: measurement error in *rprate* and under-representation of the highly sexually active males for ages 12 and 13. First, in regards to the measurement error in *rprate*, I drop individuals who show implausible volatility in the reported number of female sex partners over time. When the NLSY97 collected data on sexual history, the respondent was provided a self-administered questionnaire which was filled out without anyone else present. This approach has been found to increase response rates and is also believed to yield more accurate results for sensitive areas such as sexuality, fertility and reproductive health. But it also means that other kinds of measurement error can creep in because of the lack of professional oversight. I define implausible volatility as a year-to-year difference in the number of partners that exceeded 40 in absolute

value, or "DRP> |40|." Table 5.10 presents the estimates from these regressions. Dropping these observations has little effect on the results from Table 5.9. The POLS estimate of the interaction term is slightly more precise, but the others are basically the same in magnitude and precision.

To address the potential under-representation of sexually active males among the 12–13 year-olds, I drop the first two years of the panel and balance on the 1999-2002 waves. While this creates a shorter panel, I am able to test the importance of including 12 and 13-year-old values for *rprate*. Because the average male has his sexual debut at 15 year old, the shorter panel begins after most men have become sexually active. Therefore debut is much less important to identification in this case. Nevertheless, I should still find an effect at the upper quantiles, since individuals with tastes for promiscuity should still be responsive to changes in mating options. Table 5.11 shows the effect of the sex ratio on individuals in the highest Black population states with the most volatile sexual histories dropped, and for the 1999-2002 balanced panel. Unsurprisingly, the coefficient on the sex ratio at the mean and median is no longer statistically significant. The coefficient on the interaction term at the 90th quantile, on the other hand, remains positive and is significant at nearly the 5 percent level, and shows that Black males at the 90th quantile have 1.3 additional partners a year because of a change in the sex ratio.

3.5.2 Effects of Sex Ratios on Condom Use

In section 3, I discussed two possible behavioral responses to changes in the sex ratio. On the one hand, men with tastes for unprotected sex could negotiate less frequent condom use by using their improved bargaining position. In this case, one should find a negative relationship between the sex ratio and condom use. On the other hand, if the sex ratio causes the degree of concurrency to increase, then the probability of contracting an STD increases and lowers the expected utility from random matches within the network. If so, one should expect the sex ratio to have the opposite effect as predicted by the bargaining hypothesis, since higher risks

of infection from higher concurrency should cause men to increase condom use. Theoretically, therefore, the effect of the sex ratio on condom use is ambiguous.

Table 5.6 reports summary statistics for the condom use rate. Because I measure condom use as the ratio of condoms worn (c) to all sexual encounters (s) from the last twelve months (multiplied by 100), it is necessarily a subset of sexually active individuals.[6] Furthermore, I make additional restrictions on this data. I drop all observations in which a person reported wearing more condoms than he had sexual encounters, thus restricting the $(c/s) \times 100$ variable to be bounded between 0 and 100. Second, I dropped all individuals who reported having a non-zero number of recent partners, but zero sexual encounters. Over the entire period, of those who were sexually active, respondents wore condoms during vaginal intercourse approximately 70 percent of the time. The 10th percentile and the median correspond to "never" and "always" wearing condoms. Other variables differ slightly from the *rprate* sample, and usually in a way that is indicative of which individuals are included in this sample. These are primarily sexually active individuals — they are individuals who reported values for the number of condoms worn in the last twelve months and/or the number of times they had sexual intercourse. As such, the covariates in Table 5.6 reflect sexual activity in the sample. They are older, for instance – mean age is 19.25 vs. 17.83. The sex ratios are higher at the mean at median than in the previous *rprate* sample. The sample has a higher proportion of individuals who come from divorced families and a higher proportion of the sample are married. These individuals also have a higher educational attainment, on average, as well. For all subsequent regressions, I focus on the mean, the 10th and the median percentiles.

I include all of the covariates from the *rprate* sample and add an indicator equalling 1 if the respondent had 5 or more partners in the previous year (rp_{t-1}^{5+}). The purpose of rp_{t-1}^{5+} is to control for revealed promiscuity, which poses great risk to the sexual network. These individuals have the highest probability of matching with an infected partner and are at

[6]For this reason, the number of observations does not divide by the number of years. Some individuals were sexually active in only some of the years of the survey, and thus had missing values for the years in which they did not have sexual partner.

the highest risk for both contracting and spreading STDs (Laumann et al. 1994). I assume last year's promiscuity is exogenous to this year's condom-use decision. I assume last year's promiscuity is exogenous to this year's condom use decision

Table 5.12 presents estimates of the effects of race and the covariates on condom use using the balanced 1999-2002 waves, after dropping the low Black population states and removing individuals who displayed extreme sexual volatility. The results are the same regardless of which cut of the data is used, though. I find that Black males have considerably higher condom use rates than White males, and the difference varies across the distribution. Black males have a condom use rate that is 14.6 points higher at the mean, 5 points higher at the 10th percentile and 7 points higher at the median than White males. In all cases, the racial difference is precisely measured. This may reflect the greater risks of infect in Black networks, corresponding to higher STD prevalence among Blacks. Alternatively, the difference might reflect lower monogamy rates among Black males. Monogamy is itself a protection against STD risk, and if Whites are more monogamous, then this would translate into higher Black condom use rates. I also see that condom use declines with age, increases with years of schooling completed, increases depending on whether respondent's parents are still married, and decreases dramatically with marriage. I also find that condom use rates are approximately 5 points higher for men who had more than 5 partners in the previous period.

Next, I add sr^{-1} which is interacted with race and separately with rp_{t-1}^{5+}. These estimates are included in Table 5.5. First, I consider the results from the race–sex-ratio interaction regressions, which are listed in column '(a)' for each model in Table 5.5. The POLS and FE estimates are identical in magnitude, but neither are statistically significant at the 10 percent level. The only time the sex ratio enters negative and significant is at the 10th percentile. What this indicates is that the divergence in the Black/White sex ratio corresponds to some Black men (at the 10th percentile) wearing condoms rarely or never. This could be evidence of some Black men negotiating less condom use as a function of their improved bargaining position in the market.

I also find that White males *reduced* condom use at the median by roughly 0.41 percentage points for every 1-point change in the sex ratio. While there is very little variation in the White sex ratio over time or across space, the removal of White men from the mating market is associated with reduced condom use. This is plausible if the imprisonment of White men reduces the risks of contracting an STD.

Now turn to the results reported in the '(b)' columns of Table 5.5, which include the terms involving rp_{t-1}^{5+}. First, I find the increased sex ratio is associated with an increase in Black male condom use at the median. The net effect of a 28-point change in the sex ratio was associated with Black men at the median wearing condoms 5.4 percentage points more of the time.[7] Second, I consistently find that promiscuous men will increase their condom use by roughly 0.78 points for every 1-point change in the sex ratio, or 22 points on average.[8] The result holds up in the FE model, as well, though it is of slightly smaller magnitude. It also appears at the median, but is not reportedly unique for promiscuous men.

I interpret the positive association between the sex ratio and condom use to be largely supportive of the sex ratio hypothesis, for it suggests that indeed the sex ratio does correspond to increased risks of contracting and STD and/or decreased monogamy such that condom use increases as sex ratios diverge. I see this for Black men (who experience substantial changes in the sex ratio), and I see this broadly for promiscuous men (who either may face higher risks of contracting an STD, or who prefer sex with women without complications from having children, and thus wear condoms more regularly) - both of which effects are predicted by the theory.

3.6 Conclusion

The goal of this paper has been to identify the effect of the sex ratio on risky sexual behavior among Black men who are promiscuous (i.e., have multiple sexual partners in a year). Using

[7]This finding is based on column '(a)' of the median regression. (0.601-0.407)×28=5.432.
[8]This is based on column '(b)' of the POLS regression. (0.543+0.232)×28=21.7.

data from the Census and the NLSY97, I present strong evidence that Blacks take on more sexual partners when there is a surplus of women; this is particularly true for promiscuous men. Moving from parity (100) in sr^{-1} to 128 translates into between 1.3 and 2.4 more female sex partners at the 90th percentile a year. Furthermore, we find evidence that condom use among Blacks and promiscuous males changes in response to changes in the rising sex ratio. First, I find evidence that Black men at the 10th percentile of the condom-use distribution reduce condom use as the surplus of Black women rises, suggesting male opportunism in the face of a favorable bargaining position – the condom use rates for Black men at the left tail falls 14 percentage points due to the changing sex ratio. Second, I find that Black and promiscuous men appear, on average and at the median, to increase their condom use in light of changing opportunities. Black men, for instance, appear to increase their condom use at the median as the sex ratio changes by roughly 5 percentage points, whereas promiscuous men increase their condom use by closer to 20 points, in response to the changing sex ratio.

In light of this evidence, the net effect of changes in the sex ratio on STD risk in the population is possibly ambiguous. Though I find strong evidence for incarceration-induced concurrency, and even some significant reductions in condom use, there is likewise a ramping up of condom for Black and promiscuous males, on average. Therefore it is conceivable that the increased risk of STD infection due to higher degrees of concurrency are muted somewhat by the reductions in risk accomplished by more vigilant condom usage. But it is important to note that I am unable to measure whether Black and promiscuous males have fully compensated their partners' risks of infection due to their own promiscuity — thus, even a rise in condom use may not be enough given the failure rates of condoms and the higher risks associated with male-to-female transmission than female-to-male transmission (Laumann et al. 1994). A better test would be to move beyond individual-level survey data towards counts of STD incidence in the population, so as to quantify the relationship between rising incarceration and STD epidemics. I follow this strategy in the next chapter.

Chapter 4

Male Incarceration, Crack and Sexually Transmitted Diseases

4.1 Introduction

Chapter 2 documented the dramatic rise in incarceration levels and rates since 1975 due to the increased strictness of prison sentencing and the so-called "war on drugs." Today, the prisoner rate for Black men is almost 7 times larger than the prisoner rate for white men, despite accounting for a mere 12 percent of the US population. By mid-year, 4,682 (per 100,000) Black men were sentenced in state or federal prisons and local jails compared to 709 (per 100,000) White men. Similar disparities exist for Black and White females rates, as well. We can see how significant of a change has occurred in the last two decades by noting the change in the incarceration rate at the median state (see Figure 5.5). The incarceration rate of Black men grew from 3.17 in 1980 to 10.04 percent in 2000, compared to a White male incarceration rate that grew from 0.43 in 1980 to 1.53 percent in 2000.

High rates of male imprisonment creates significant imbalances in community sex ratios. As discussed in chapter 3, the resulting surplus of women shifts the bargaining power in sexual relationships to men, leading to risky behavior like concurrent partnership that facilitate the spread of STDs. As shown in the third chapter, I found the surplus of women in Black communities induced Black men to take an additional sex partner. The response is particularly strong for the most promiscuous men. The ratio of Black women to black men rises from parity during the teenage years to about 128 by their 20s. For Black men at the 90th quantile, this change is associated with between 1.3 and 2.4 additional female sex partners a year. At the same time, I find that the surplus of women led to higher condom-use rates. Both Black men and promiscuous men were found to increase their condom use rates as the

sex ratios in their state rose, which suggests that the increased risks of concurrency may have been partially offset. I also found evidence that some Black men at the 10th quantile reduced their condom use in response to the rising sex ratios, which taken with the higher rates of concurrency suggests incarceration may lead to increasing risky sexual behavior among men. It is therefore vital to move beyond self-reported sexual behavior and focus on the effect that rising male incarceration has had on actual STD outcomes.

As discussed in chapter 2, Johnson and Raphael (2006) find that lagged rates of race, age, and state-specific male incarceration account for all of the racial disparities in AIDS incidence among Black and White women. However, as they acknowledge, the unique epidemiological features of advanced stage HIV makes it difficult to identify the precise mechanism by which incarceration influences AIDS incidence. Incarceration may increase AIDS incidence by inducing risky behavior, like concurrency, or exposing prisoners to HIV risk during their imprisonment. Prison have much higher rates of AIDS than the general population, after all, and male-to-male intercourse is believed to occur with some regularity and without contraceptive protection. Needle-sharing, due to the underground tattoo culture of prisons, could also facilitate HIV transmission behind bars. The distinction between transmission vectors is important for policy. If incarceration-induced concurrency is fueling the spread of STDs, then prisoner interventions will have modest effects. If, though, transmission between prisoners is the primarily factor, then prisoner interventions such as the quarantine of HIV-positive prisoners or the distribution of condoms to prisoners may play a significant role in slowing down disease spread.

Like HIV/AIDS, gonorrhea and syphilis have higher prevalence rates among Blacks than Whites. Using CDC data on gonorrhea and syphilis incidence for 1981-2004, I constructed national gonorrhea and syphilis rates for the White and Black population. In 1981, the Black gonorrhea rate was 1,601 per 100,000; in 2004, it had fallen to 484. The White rate, on the other hand, was 155 in 1981 and only 25 in 2004. Syphilis patterns are qualitatively similar, though much smaller in magnitude because of the much lower prevalence of syphilis in the

population. In 1981, Black syphilis rates were 42 (per 100,000) and 8 in 2004. White syphilis rates were 4 in 1981 and only 1.5 in 2004. Both gonorrhea and syphilis rates rose sharply during the 1980s and peeked in the early 1990s, before reversing trend and falling.

The timing of this explosion in Black and White STD rates occurs coincident to a nationwide expansion of the prison population (Patillo, Weidman and Western 2004), as well as the appearance of crack cocaine and the subsequent expansion of the cocaine market (Fryer et al. 2006). In this chapter, I examine the relationship between Black male incarceration on gonorrhea and syphilis, controlling for the effects of crack. I focus my attention on gonorrhea and syphilis because both allow me to assess the influence of incarceration-induced concurrency and serial monogamy on disease transmission. First, gonorrhea and syphilis have very short duration spells before symptoms appear (less than a month). Second, they are also bacterial infections, which means treatment with clinical dosages of antibiotics will cure the victim entirely. When syphilis and gonorrhea outbreaks appear in prisons, they are quickly eliminated. FInally, unlike HIV/AIDS, gonorrhea and syphilis are only transmitted through sexual contact.

I match the gonorrhea and syphilis data, constructed from the CDC data, with male incarceration data. STD data is measured as cases of gonorrhea (syphilis) per 100,000 disaggregated by race, age, gender and state for 1981-2004. Incarceration data comes from the 1980, 1990 and 2000 Census 5 percent samples. To address possible sources of endogeneity, I include a variety of demographic and economic control variables, as well as state-specific linear and quadratic time trends. All models are estimated using a fixed effects strategy, with the unit of observation being a state, age and gender case rate for Blacks and Whites. To control for substance abuse, I use two proxies for alcohol abuse and crack cocaine abuse. While including a control for alcohol consumption is not new, crack cocaine is, and one of the more significant contributions of this study is to quantify, for the first time, the role of crack cocaine in the gonorrhea and syphilis epidemics of the late 1980s. I also control for

state unemployment rates, state per capita income, the percent of the state that is Black, and the percent of the state that is 15-19 years old.

First, I find evidence that male incarceration is a strongly related to female gonorrhea incidence, even controlling for numerous variables that are likely correlated with the underlying incarceration rate. Net of all these other factors, I consistently find that Black male incarceration is associated with Black STD incidence, and the effects are large and significant. Specifically, I find that for every 1 point increase in the Black male incarceration rate, gonorrhea cases among Black females increased by 64 cases (per 100,000). The effect among Whites is comparable, though smaller and less precisely estimated — 17 new cases of White female gonorrhea for every 1-point increase in the White male incarceration rate. Second, in all specifications, I find that a 1-unit change in the crack cocaine index was independently associated with 66 new Black cases of gonorrhea, and 11 cases among Whites. In all specifications of the data, the relationship between crack and gonorrhea – while differing somewhat in magnitude – is large and precisely estimated. Third, I find some evidence that White male incarceration increases White female gonorrhea incidence. Because White male incarceration is not high enough to cause imbalanced sex ratios, I consider this evidence for incarceration-induced serial monogamy among White females. Finally, in contrast to the gonorrhea results, incarceration and syphilis appear to be negatively related. Syphilis prevalence is much lower in the population and may be concentrated among those at-risk for imprisonment. As such, removing these individuals from the population tends to reduce individuals infected with syphilis and the overall efficiency of transmission within that network. As with gonorrhea, though, I find that crack drove growth in the syphilis rate considerably.

4.2 Gonorrhea and Syphilis Epidemiology and U.S. Trends

4.2.1 Gonorrhea

Gonorrhea is a bacterial infection caused by *Neisseria gonorrhoeae*, which can grow and multiply in the reproductive tract, including the cervix, uterus, and fallopian tubes in women, in

the urethra in women and men, as well as grow in the mouth, throat, eyes, and anus of women and men (CDC 2006) (Holmes et al. 1999). Unlike HIV, gonorrhea has both a relatively short incubation period, and is acquired almost exclusively through sexual intercourse.

In a study involving 81 men who acquired urethral infection at a defined time, the mean time to development of symptoms was 3.4 days, and only 2 men (2.5 percent) remained asymptomatic for 14 days (Harrison, Hooper, Wiesner, Campbell, Karney, Reynolds, Jones and Holmes 1979). Other studies place the incidence of asymptomatic urethral gonococcal infection in the general population at approximately 1 to 3 percent. These symptoms for men include urethral discharge, dysuria and penile edema in men. For the most part, men develop these symptoms immediately after inoculation.

The incubation period for urogenital gonorrhea in women is less certain and likely more variable than for men. Most women who develop local symptoms do so within 10 days of infection. Women experience symptoms that are relatively mild, and even when a woman does have symptoms, they are so non-specific as to be easily mistaken for a bladder or vaginal infection (CDC 2006). The initial symptoms include a painful or burning sensation when urinating, increased vaginal discharge, or inter-menstrual uterine bleeding (Holmes et al. 1999).

Also unlike HIV, gonorrhea is curable with clinical dosages of antibiotics. First reports of 100 percent utility of penicillin for gonorrhea therapy were published in 1943 (Mahoney, Ferguson, Bucholtz and Slyke 1943), following which gonorrhea rates declined. Increased virulence by the *N. gonorrhoeae* strain has led to regular revisions of the recommended gonorrhea therapy. Between 1943 and 2006, numerous antibiotic families have been introduced into gonorrhea treatment. In 1976, new strains of gonorrhea were discovered that were nearly impervious to clinically appropriate doses of penicillin, and within 10 years, gonococci with high-level resistance to tetracycline were discovered, reducing the efficacy of tetracycline family of drugs as well. The most common antibiotic used today is ciprofloxacin and members of related antibiotic families (Holmes et al. 1999). The CDC continues to regularly

document the diversity of anti-microbial-resistant *N. gonorhoeae* strains in the US, but it is expected that this process of increased virulence will eventually limit the utility of the current ciprofloxacin treatments.

While gonorrhea is easily cured, left untreated it can lead to serious health problems. Among women, it is a major cause of pelvic inflammation disease, which can itself lead to ectopic pregnancy and infertility. In men, untreated gonorrhea can cause epididymitis, which is a painful condition of the testicles that can also result in infertility. In addition, studies have suggested that the presence of gonorrhea infection causes a person to be more likely to acquire HIV, if exposed (Oster 2005).

Beginning in the early 1980s, gonorrhea incidence in the US began a dramatic decline across all race, age and gender cohorts. It is difficult to compare the US experience to other nations, since few countries have reporting systems that permit accurate estimation of the true incidence of gonorrhea, but those that do show a general decline in incidence from the 1950s on. The CDC has collected information from state health agencies on gonorrhea incidence in the US disaggregated by race, age, gender and state since 1981. Figure 5.6 plots these data for Blacks and Whites by age cohort.

The number of Black Americans aged 15-44 with gonorrhea in 1981 was 3,128 cases (per 100,000) and 326 cases for Whites. Black gonorrhea rates climbed from a low of 2,183 cases in 1983 to a high of 3,109 in 1989, before reversing and starting a steady decline throughout the 1990s. Figure 5.6 shows that 15-34 year olds account for most of this movement. The patterns are similar for Whites, although the case rates are considerably smaller.

4.2.2 Syphilis

Like gonorrhea, syphilis is a bacterial infection that is acquired through sexual contact, although in rare cases it can be acquired *in utero*. Also like gonorrhea, syphilis has a short incubation period and is easily cured with clinical dosages of antibiotics.

Syphilis progresses through various stages of maturity. In the earliest stage, the illness typically results in a lesion on the genitals 14-21 days from the time of inoculation. Usually, the lesion is not painful, though there are exceptions. The lesion will grow to a size of 0.5-1.5 cm in diameter. Left untreated, it will heal within a few weeks of appearing. Because the lesion is visible on the penis, syphilis is commonly identified in the primary stage for heterosexual men. But because it appears on the labia, fourchette or cervix of women - areas which are not visible - syphilis is more commonly identified among women after it progresses to the secondary stage. The same applies to homosexual men with lesions on or near the rectum or anus.

The secondary stage of syphilis, occurring within a few weeks or months from the time of exposure, is a variable illness. Symptoms include headaches, low-grade fever, malaise, sore throat, adenopathy and cutaneous rashes. Latent syphilis occurs in some cases and is difficult to detect on physical examination since it may be asymptomatic or subtle. Morbidity and mortality are principally caused by late manifestations of the illness in adults' skin, bones, central nervous systems, heart and great vessels (Holmes et al. 1999). Although highly infectious, syphilis is curable in its primary and secondary stage using antibiotics.

If untreated, it can lead to serious long-term complications, including nerve, cardiovascular, and organ damage, and even death. Syphilis is also a co-factor in the spread of HIV, and like gonorrhea, it increases the transmission of the virus by two-five-fold. This is due to the lesions and discharge that is associated with syphilis, and the role that bodily fluids and blood play in the increasing the transmission rate of HIV to an uninfected person. But, unlike gonorrhea, syphilis has considerably high co-infection rates with HIV. This is due to the low prevalence of syphilis in the heterosexual population and the high prevalence in the homosexual population (Laumann et al. 1994).

Syphilis rates were constructed both for all 15-44 year olds at the national level and disaggregated by age using the series provided by the CDC. Because syphilis has very low prevalence in the population, I limited the sample to states which had syphilis rates higher

than the median state in 1985, because the 1981-1984 sample has measurement error.[1] Data after 2000 was excluded from analysis due to the fact that incarceration rates are based on decennial data, making the 2001-2004 years impossible to interpolate. Nonetheless, Figure 5.7 plots the syphilis series for Blacks and Whites for 1981-2004.

In 1981, the syphilis rate for Blacks aged 15-44 was 80 (per 100,000). After falling 10 points in 1982, the Black case rate suddenly reversed trend and climbed for six consecutive years. In 1990, the Black syphilis rate had grown to 257 cases (per 100,000). This represented the highest point for Blacks in the sample. From 1991 to 2003, the Black syphilis rate fell from 219 to 13. In 1981, the syphilis rate for Whites aged 15-44 was 10 (per 100,000). That was the high point for syphilis among Whites – since then, it has steadily fallen to an all-time low of 1.10 cases (per 100,000) in 1998. Yet, since 1998, syphilis rates have steadily risen, and as of 2004, stand at 3.42 cases (per 100,000) – a 210 percent increase from its low in 1998. Most of the change in syphilis rates has been among the 20 and older cohort, as syphilis is relatively rare among 15-19 year olds. Figure 5.7 shows the complete breakdown of syphilis trends by race for various age brackets.

4.3 Incarceration Trends

Starting in 2006, 2.2 million American men and women were imprisoned in state of federal prisons local jails. Our prison population is high today even for American standards, which have always been high from a global perspective. In the mid-1970s, US incarceration rates began to rise steadily, after being relatively stable throughout the 20th century. Between 1974 and 2001, the number of persons in jail or in prison grew from 100 inmates (per 100,000) to 700 (Patillo, Weidman and Western 2004).

[1]These states are Alabama, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Georgia, Louisiana, Maryland, Massachusetts, Mississippi, Nevada, New Mexico, New York, North Carolina, North Dakota, Oklahoma, Oregon, Pennsylvania, South Carolina, Tennessee, Texas, and Utah.

Behind this aggregate statistic are sharp racial disparities. Raphael (2004) notes that the share of all Black men who were "institutionalized" in 1970 was 3 percent and in 2000 was 8 percent, whereas the share of all White males remained at approximately 1 percent over the same period. Not surprisingly, most of this imprisonment is concentrated among the youth and uneducated. In 2000, 23 percent of all Black male high-school dropouts were in jail or in prison, compared to 34 percent of all 26–30-year-old Black male high-school dropouts Patillo, Weidman and Western (2004). Roughly a third of all young Black male high-school dropouts are in jail or in prison on a given day in the US.

The prison population grew in part because of the crack epidemic, rising crime, and a stricter approach to crime. The "war on drugs" has been a significant factor in the expansion. Crack was accompanied by significant outbreaks in crime (Grogger and Willis 2000), and it became a national goal to reduce drug consumption because of the external costs that consumption was believed to impose on the rest of society. To do this, the US government has largely utilized enforcement in the form of harassing drug sellers through imprisonment, tougher sentences, and larger fines for possession. The net effect of this has been to increase the prison population with drug offenders (Charles and Luoh 2006).

I constructed a time series of male incarceration rates disaggregated by race, age, state, and year using the Census 5 percent samples for 1980, 1990 and 2000. Following Johnson and Raphael (2006), I linearly interpolated the inter-census years for each demographic cell. Figure 5.5 shows the changes in the male incarceration rate for Whites and Blacks at the median state. As one can see, the Black male incarceration rate grew dramatically from 1980 to 2000. By 2005, approximately 12 percent of all Black men are in jail or prison. While the median state shows a slight increase in White male incarceration rates, the overall level is still far below that of Whites.

4.4 Empirical Framework

4.4.1 Data

Relationship matches tend to follow positive assortative matching by race, age and geography. For instance, over 95 percent of all Black men married between 1970 and 2000 were married to a Black woman. Likewise, men and women tend to closely match by age and geography as well (Charles and Luoh 2006). Therefore, the identification strategy I use in this paper exploits the variation within a racially defined, age-specific, and geographic-specific male incarceration rate over time.

Data on gonorrhea and syphilis were acquired from the CDC Division for STD Prevention. This data was collected by the CDC from state health departments. Starting in 1981, the data was available by race, age, state, gender and year – although there is some measurement error in the tabulations for 1981-1984 due to incomplete records and poorly recorded race and age characteristics[2]. I show results both with and without the 1981-1984 results.

Data on US incarceration delineated by race, age, sex, state and year is difficult to acquire. The FBI Uniform Crime Reports contain information on total arrests, but not incarceration. Secondly, the Uniform Crime Reports do not provide arrest information on age-race-sex-state-year specific demographic cells. One can find total arrests by age, or total arrests by race, for every state in the series, but not for a single age-race (i.e., Black males aged 15-19 from Alabama in 2000) demographic cell. Another possibility is the Bureau of Justice Statistics (BJS). The BJS collects data on prison populations annually but not for any age band narrower than 18-59 years of age. Thus, to use that data, I must forego our empirical strategy of using the within variation of racially-defined, age- and geographic-specific demographic cells to identify the effect of male incarceration on STD incidence. Other datasets I considered suffered from similar problems.

[2]Personal correspondence with CDC economist, Harrell Chesson, confirms this problem.

An alternative might be to find a dataset that contains information at the appropriate demographic cells, but is available only periodically, and use interpolation to complete the missing years. Johnson and Raphael (2006) use this approach and I follow their approach[3]. Data on incarceration rates was collected from the University of Minnesota's IPUMS Census project. Longform (5 percent sample) Census years 1980, 1990 and 2000 were used. The data for census years 1980 are "flat", meaning the samples are nationally representative needing no additional weighting. This was not the case for 1990 and 2000, which require weights to make the data representative. Adjustments were made according to directions contained in the IPUMS User Guide.

Incarceration is calculated by referencing the group quarter variable. The Census defines a person as living in a group quarter if the person lives in a dwelling unit containing 5 or more non-related individuals. Group quarters are broken down into different categories, and those categories are not consistent from 1980 to 1990. In 1980, individuals were flagged as living in correctional facilities as opposed to college dormitories, nursing homes, or other group quarters. In 1990, the Census no longer flagged a person as living in a correctional facility. Individuals were said to live in either an"institutionalized" group quarter or a "non-institutionalized" group quarter. I treat men characterized as "institutionalized" as incarcerated. Charles and Luoh (2006) argue that for 1990-2000 most young men, "institutionalized" effectively means incarcerated since most of the other categories contained in the Census definition would not apply to younger cohorts[4]. The exception is mental institutions. But,

[3]Johnson and Raphael (2006) address the robustness of linear interpolation in their paper. They used a non-parametric method to interpolate the inter-census years, and found that it was highly correlated with the linear interpolation (high at 0.93). They also abandoned interpolation altogether and used other data to replace the inter-census years. Their results were robust to such extensions. Future work on this project will require similar robustness checks, but at present, I report only our linear interpolations.

[4]In 1990 and 2000, institutionalized persons were living in jails and prisons, mental institutions, institutions for the elderly handicapped and poor, nursing and convalescent homes, homes for neglected/depend children, other institutions for children, deaf/blind schools, schools for "feeble-minded," sanitaria, poor houses and almshouses, poor farm/workhouses, homes for unmarried mothers, widows and single women, and detention homes. See Charles and Luoh (2006), footnote 8.

Grob (2000) has shown that the number of persons living in mental institutions has fallen in the past few decades, which would mean that in later years, mental institutions constitute a diminishing portion of the "institutionalized" group quarter. Finally, the pattern of incarceration found from using the "institutionalized" data is consistent with the aggregate information on incarceration from the Bureau of Justice Statistics (Charles and Luoh 2006).

Sex, age and state-specific rates were constructed using the 1980, 1990 and 2000 IPUMS Census Longform surveys for Blacks and Whites. Incarceration rates are the ratio of incarcerated individuals to the general population for each age, race, and state cell. Linear interpolations of the inter-census years were then performed for each demographic cell. Six age cohorts were constructed: 15-19, 20-24, 25-29, 30-34, 35-39, and 40-44. Incarceration rates were constructed for each of the 50 American States and District of Columbia.

To consistently estimate the effect of incarceration on gonorrhea and syphilis, it is important to control for crack cocaine use. It is believed that the crack epidemic increased the number of concurrent sexual partnerships during the 1980s and 1990s via the drug's unique pharmacological effects. Ethnographic studies of the crack epidemic showed the promiscuity and unprotected sex were centrally involved with crack exchanges. Addicted female users were known to exchange sex for crack or for money to buy crack cocaine (Jones, Irwin, Inciardi, Bowser, Schilling, Word, Evans, Faruque, McCoy and Edlin 1998; Edlin et al. 1994; Flom et al. 2001). The crack trade was a factor in the prison population expansion, because of its connection violence and theft (Grogger and Willis 2000).

I use the crack index created by Fryer et al. (2006) as a measure of crack cocaine consumption.[5]. While my focus is on incarceration, this is the first study to examine the relationship between crack and STD rates. I also include a measure of alcohol consumption so as to capture other substance abuse factors that could be correlated with risky sex and high rates

[5]Using factor analysis on a variety of separate variables correlated with underlying crack cocaine usage at the state level, Fryer et al. (2006) created an index that is believed to correspond to movement in crack consumption and trade at the state level.

of incarceration in an area (Chesson, Harrison and Kessler 2000). These data were collected from the National Institute on Alcohol Abuse and Alcoholism.

Finally, because incarceration is correlated with socio-economic status, I control for other various economic and demographic variables, such as the percentage of the state living below the poverty threshold, the percent of the state between the ages of 15 and 19 and the percent of the state that is Black. I also include state-level income and unemployment rates collected from the Bureau of Economic Analysis to address economic factors that might be related to the propensity to commit crimes and engage in risky sexual behavior. Table 5.14 shows the summary statistics for 1981, 1990 and 2000 for each of the covariates. Crack grows throughout the entire sample, but it jumps considerably from 1981 to 1990.[6] The relatively large standard deviation in 1990 reflects the fact that crack's influence was heterogeneous across the US. Alcohol consumption, on the other hand, falls throughout the entire sample. In 1981, Americans consumed approximately 2.9 gallons per capita, and 2.3 by 2000. Economic prosperity can also be seen in the per capita income, unemployment and poverty rates. Income per person rose considerably from 1981-2000, while state unemployment rates bottomed. Poverty rates, also, fell roughly 3.5 percentage points over the two decades observed. Demographic changes are also observed during this period. The percentage of states with youth fell from 9.1 on average in 1981 to 7.3 in 2000. And the Black share of the US rose from 11.6 to 12.6.

4.4.2 Basic Setup

The basic setup for my analysis is a regression equation of the form

$$STD_{s,a,g,t} = \beta_1 IR_{s,a,g,t} + \beta_2 Female_{s,a,g,t} + \beta_3 Female_{s,a,g,t} \times IR_{s,a,g,t} + \alpha \mathbf{x}_{s,t} + \epsilon_{s,a,g,t} \quad (4.1)$$

where $STD_{s,a,g,t}$ is Black (White) gonorrhea and syphilis cases diagnosed per 100,000 individuals for the state (s), for a given age cohort (a), for a given gender (g) in a given year

[6] Fryer et al. (2006) note that the negative value on the index in 1981 is entirely an artifact of the data and the process that generated the index.

of infection (t), $IR_{s,a,r,t}$ is the Black (White) male incarceration rate, $Female_{s,a,g,t}$ is an indicator variable equalling 1 if the demographic cell is describing female STD rates and 0 otherwise, $\mathbf{x_{s,t}}$ are the covariates previously discussed which vary by state and year, including the crack index, and the error term ($\epsilon_{s,a,g,t}$) contains unobserved demographic cell effects which I control for in estimation. Each model is estimated using fixed effects so as to net out any unobserved (time-invariant) variables that are correlated with the included covariates at level of the demographic cell, year effects, and state-specific linear and quadratic time trends. I adjust the standard errors to allow for clustering at the level of the demographic cell.

4.5 Results

Table 5.15 presents the results from baseline regressions for gonorrhea and syphilis, by race, covering the 1981-2000 period. In each case, I estimate models first without the female dummy variable interacted with the male incarceration rate. Black male incarceration is positively related to gonorrhea incidence both with and without the gender interaction. When gender is controlled, it becomes apparent that the results are strongest for Black females – 69 additional cases of gonorrhea for a single-point increase in the Black male incarceration rate over the period. This effect is precisely estimated at the 1 percent level. The patterns is similar for Whites. The White male incarceration rate increases White female gonorrhea rates by 77 cases for every 1-point change. Crack cocaine is large and statistically significant for Blacks and Whites – a 1-point change in the index moves Black gonorrhea up 37 cases, and White gonorrhea up 14 cases.

As with previous studies (Chesson, Harrison and Kessler 2000; Carpenter 2005), I find that alcohol is positively related to gonorrhea incidence for both Blacks and Whites, though the effect on Blacks is imprecise. An additional tenth of a gallon of alcohol consumed per capita in a year is associated with 36 additional cases of gonorrhea among Whites. Black and White gonorrhea rates move in opposite directions with the unemployment rate; down for

the former, but up for Whites. Both are significant below 1 percent. The only other result that is significant is the "percent Black" variable for Whites – the larger the Black share of the population, the higher the gonorrhea rates among Whites.

The results for syphilis paint a different picture than that of gonorrhea. As noted in the section 4.2, the syphilis sample includes only those states that were at or above the median rates of incidence in 1985. First, the effect on Blacks and Whites is negative and significant (though the effect is positive for the interaction). Male syphilis rates appear to be falling when male incarceration rates are rising — for both Blacks and Whites. Secondly, the association between crack and syphilis is quite strong — a 1-point change in the crack index is associated with 21 new cases of Black syphilis, and 1 new case for Whites. The difference in magnitude between these two estimates is not surprising given the relatively low syphilis rates for Whites in the first place (see Figure 5.7). Syphilis responds to alcohol and unemployment rates similarly to what was observed in the gonorrhea regressions.

In Table 5.16, I drop the 1982-1989 and 1991-1999 interpolated years so as to determine the degree to which the results are sensitive to interpolation. Though some variables change sign and significance (for instance, alcohol is negative and insignificant for Blacks), the overall story remains the same for estimated effects of incarceration on gonorrhea and syphilis.

In Table 5.18, I focus only on the 1985-2000 panel, since the 1981-1984 suffer from measurement error correlated with race, age and gender. Dropping these data only strengthens the relationship between Black male incarceration and Black female gonorrhea. Black incarceration increases Black female gonorrhea by 64 cases a year, compared to 69 in the previous estimate, and is small and imprecise for Whites unlike before. The estimate of crack on Blacks, on the other hand, is roughly double – a 1-point increase in the crack index is associated with 66 additional cases of gonorrhea among Blacks, and 11 cases among Whites. Alcohol consumption differs slightly when focusing only on 1985-2000 – its effects are estimated to be larger for Blacks (116 additional cases with an additional tenth of a gallon of alcohol consumed per year), but smaller for Whites (14 additional cases for an addi-

tional tenth of a gallon consumed). Both estimates are significant at the 1 percent level. State unemployment rates are no longer negative and statistically significant for Blacks, but remain positive and significant for Whites - a 1 percentage point increase in the state unemployment rate increase White gonorrhea by 11 cases a year. The percentage of the state that is young is positive and significant for Blacks, unlike before – a tenth of a percentage point increase in the share of the state that is young is associated with 23 additional cases of Black gonorrhea. Percent Black, on the other hand, is no longer significant for Whites, though it remains positive.

The effect of Black male incarceration on syphilis rates are similar to the previous estimates – negative and precisely estimated for all Black males and positive for Black females, with very little change in the point estimates themselves. The estimates on White syphilis are somewhat lower, but nearly identical to before. Crack is also positive and significant for Blacks and Whites, and virtually identical to before. The rest of the covariates retain their approximate value and significance.

In Table 5.5, I re-estimate the models using the 1985-2000 panel but dropping states with small Black populations. My prior is that dropping these states should only strengthen the relationship between crack and STDs, as well as incarceration and STDs, since only relatively high Black population states should exhibit the kinds of mating market shocks I'm examining. For the most part, that is what I find. Dropping states which have small Black populations strengthens the association between male incarceration and gonorrhea. In the first column, Black gonorrhea rates increase by 64 cases for ever 1-point percentage change in the Black male incarceration rate. The second column reveals that this is primarily due to changes in Black female gonorrhea incidence, as the impact on Black male gonorrhea incidence is negative and insignificant. Focusing only on states in which at least 5% of the population is Black, a 1-point change in the Black male incarceration rate increases Black female gonorrhea by almost 200 additional cases every year. Moving to columns 3 and 4, I find that the results only grow in magnitude if I focus on states with at least 10% of the

population Black — a 1-point change in the Black male incarceration rate is associated with around 260 additional gonorrhea cases among Black females in these high Black population states.

The association between crack and gonorrhea strengths the more I limit the analysis just on states with significant Black populations. In the 5% sample, crack increases Black gonorrhea by 188 cases a year, and 252 cases a year in the 10% sample. Other covariate estimates are similar. For instance, alcohol is positive and large for both the 5% and the 10% sample (though it is only significant in the 5% sample). As alcohol consumption increased by a tenth of a gallon (per person), Black gonorrhea rates increased by 96 cases a year. Unemployment rates enter negative for both samples, but are only significant for the 10% sample – a 1-point increase in the state unemployment rate reduced Black gonorrhea rates by 81 cases a year in the 10% sample.

The syphilis results do not differ considerably much from the previous estimates, either. For instance, I again find that male incarceration rates are negatively associated with Black syphilis rates, and though this effect is significantly larger from the complete sample, it does not differ much between the 5% and 10% samples. Incarceration rates were associated with a fall in Black syphilis rates on the range of 14 to 15 cases a year for every 1 point increase in Black male incarceration, but this effect is strongest among the males. Incarceration increases Black female syphilis by roughly 16-19 additional cases a year. Crack, on the other hand, is sensitive to the sample used. Though marginally significant with the 5% sample, crack is positive and very precise when focusing just on the 10% sample. Black syphilis rates increased by 24 cases a year for every 1-point change in the crack index. Alcohol estimates is also positive, large and very precisely estimated. As alcohol consumption increases by a tenth of a gallon per person, Black syphilis rates increased by 100–175 cases a year, depending on the sample used. Also, I continue to find a strong positive association between per capita incomes and syphilis rates, and in a range that is comparable to the previous estimates — a $100 increase in real incomes (per person) is associated with 2 new cases of Black

syphilis a year. Other measures of economic prosperity show the opposite effect, though. As with gonorrhea, unemployment rates are negatively associated with Black syphilis. For the 5% sample, a 1 percentage point increase in unemployment rates lowered Black syphilis by approximately 17 cases, and 27 cases for the 10% sample. And finally, I also find that the higher the Black share of the state's population, the higher the syphilis rate among Blacks – a 1 percentage point increase in the Black share is associated with 12 additional cases of syphilis.

Finally, I divide the sample into groups: 15-29 year olds and 30-44 year olds. Table 5.19, reports the results for the Blacks; Table 5.20 for Whites. Black male incarceration is strongly associated with Black female gonorrhea for both young (i.e., 15–29 year-olds) and old (i.e., 30–44 year-olds). A 1-point change in the Black male incarceration rate is associated with 65 (61) additional 15–29 (30–44) year-old Black female gonorrhea cases a year. Black male incarceration is negatively associated with Black male gonorrhea for both sets of ages, but is statistically significant only for the 30–44 year-old sample — a 1-point increase in the incarceration rate caused Black male gonorrhea rates to fall 32 cases a year.

Crack enters positively for both 15–29 year-olds and 30–44 year-olds, but is only marginally significant for 15–29 year-olds, and is precisely estimated for the 30–44 year-olds. A 1-point increase in the crack index is associated with between 60 and 78 additional cases of gonorrhea per year, depending on the sample. Alcohol consumption, furthermore, has a positive effect on Black gonorrhea rates for both young and old, though the effect is larger among the young sample. A tenth of an additional gallon of alcohol consumed a year is associated with 152 additional cases of 15–29 year-old gonorrhea cases a year, and 917 additional cases of 30–44 year-old gonorrhea cases a year. The only other statistically significant gonorrhea estimate in 5.19 is the percent of the state that is 15–19 years-old. A tenth of a percentage point increase in the share of youth in the state is associated with 41 additional cases of gonorrhea among the young. There is no comparable effect on the older sample.

Black syphilis estimates are similar to what has previously reported, though differs in possibly important ways. For instance, most of the negative effect of incarceration on Black syphilis appears to be concentrated among the 15–29 year-old sample. A 1-point increase in the Black male incarceration rate is associated with a 9 fewer cases of Black syphilis cases a year, 14 fewer cases of Black male syphilis cases a year, and 9 additional cases of Black female syphilis cases a year. The negative effects are much milder when focusing on the 30–44 year-olds. A 1-point increase in the incarceration rate is associated with 6 fewer Black male cases a year, and 10 additional Black female cases a year.

Crack, on the other hand, is extremely precise and predictably large among both demographics (though its effect on the youth is roughly twice as large as its effect on the 30–44 year-old sample). A 1-point increase in the crack index is associated with 29 additional cases of Black 15–29 year-old syphilis a year, and 16 additional cases of Black 30–44 year-old syphilis a year. Other proxies of substance abuse also show positive signs – a tenth of a gallon increase in the per capita alcohol consumption rate is associated with 15 more cases of Black youth syphilis a year, and 8 additional cases of 30–44 year-old Black syphilis. Real incomes are associated with higher syphilis rates among the younger sample than the older sample. An increase in $100 per person (real terms) increases 15–29 year-old syphilis rates by 1.5 cases a year, and 0.7 cases a year among 30–44 year-olds. Unemployment rates, on the other hand, are negatively correlated with each, and are comparable in size — a tenth of a percentage point increase in the state unemployment rate is associated with roughly 2 fewer cases of Black syphilis a year for both young and old. None of the other syphilis estimates are statistically significant.

Table 5.20 shows the White results. Unlike the Black results, the impact of White male incarceration rate is strongest among the 30–44 year-old sample. None of the estimates on the young sample are statistically significant, and point estimates are a fraction of the 30–44 year-old sample. Among the older sample, White male gonorrhea rates fall by 25 cases, and increase among White female gonorrhea rates by 30 cases, for every 1-point change in the

incarceration rate. That the effect is concentrated among the older group is inconsistent with the incarceration-concurrency hypothesis. Not only are the incarceration rates so low among White males as to have a negligible effect on the White sex ratio, but these results find incarceration only effective among the older group — individuals who are presumably less likely to be on the mating market, delaying marriage. It's possible that these results indicate a separate pathway by which incarceration is affecting STD rates among Whites than the incarceration-concurrency pathway previously hypothesized.

Crack enters the equation positive and statistically significant for all age groups, but is three times larger in magnitude among White 15–29 year-olds. A 1-point change in the crack index increased White youth gonorrhea 16 additional cases a year, and only 5 additional cases a year among the older sample. Alcohol also has large and significant effects on both groups, but as with crack, has a larger effect on the 15–29 year-old sample. A tenth of a gallon increase in the per capita alcohol variable is associated with 19 additional cases of 15–29 year-old gonorrhea, and roughly 9 additional cases among 30–44 year-olds. A $100 increase in real incomes is associated with 1 additional cases of gonorrhea among the youth, but has no effect on the older sample. Unemployment is positively associated with gonorrhea for young and old, but the magnitudes are six times as large among the youth — a tenth of a percentage point increase in the unemployment rate increased youth gonorrhea by 2 cases a year, but only 0.3 cases a year among the older sample. And finally, the higher the share of the population that is Black, the higher the White 30–44 year-old gonorrhea rate. Every tenth of a point increase in the share of the population that is Black is associated with 1 additional case of gonorrhea among the older White sample.

The syphilis results for Whites holds few surprises, and is qualitatively similar to what has been discussed before. Briefly, I find incarceration to have a large and negative effect on White male youth syphilis (9 fewer cases a year), a large and positive effect on White female youth syphilis (9 additional cases), a negative effect on White male 30–44 year-old syphilis (5 fewer cases), and a positive effect on White female 30–44 year olds (8 additional

case). Crack, on the other hand, is extremely precise for White syphilis, and point estimates are similar for both young and old – 1 additional case for young and old White individuals for every 1-point change in the crack index. Alcohol estimates differ somewhat for the two demographics. Among 15–29 year-olds, a tenth of an additional gallon of alcohol consumed in a year is associated with 0.7 more cases of syphilis among the 15–29 year-olds, and 1.1 additional cases among the older White sample. Poverty enters the equation very small, but significant, effect for the 30–44 year-olds. A 1 percentage point increase in the state poverty rate lowers White 30–44 year-old syphilis rates by 0.2 cases a year.

These regression results show that effects differ by age, as well as race. That we see such strong positive effects of incarceration on Black youth gonorrhea rates is consistent with the concurrency hypothesis. These are the years where men would be considering there many options on the mating market and delaying marriage. However, because we see these effects persisting among the older cohorts as well suggests that incarceration affects STD transmission through other pathways as well. Furthermore, finding a positive effect of incarceration on White 30–44 year-olds, but not among the old, also suggests that incarceration affects STD transmission in other ways besides merely creating concurrent sexual partnerships.

4.6 Conclusion

In this chapter, I have examined the relationship between incarceration and gonorrhea and syphilis, controlling for crack cocaine, alcohol consumption, and other economic and demographic control variables. High rates of Black male incarceration removes young Black men from the mating market and creates an effective shortage of men for the remaining Black women. Faced with the surplus of young, unattached Black women, non-imprisoned Black men have incentives and bargaining position to form multiple partnerships, assuming they are able to find partners who prefer this arrangement to their next best alternative. I estimated fixed effects models controlling for substance abuse, economic and demographic variables which may be correlated with the incarceration, as well as state and year fixed effects and

state-specific linear and quadratic time trends. I consistently find that Black male incarceration is correlated with Black female gonorrhea. This effect is strong among all age groups, and increases in strength as the sample is limited to states with sizeable Black populations. Incarceration is positively associated with Black female syphilis rates, as well, but is negatively associated with Black male syphilis. We also find that crack has a very strong and large positive correlation with Black and White gonorrhea and syphilis rates. It enters positive and statistically significant in all of the models estimated. Other variables included are largely consistent with previous estimates in the literature. The association between White incarceration and gonorrhea/syphilis, on the other hand, is much less suggestive of the concurrency hypothesis. For instance, White incarceration rates have small and statistically insignificant effects on youth White gonorrhea rates, but positive effects among the 30–44 year-olds. These patterns suggest that incarceration may amplify the spread of STDs through pathways separate from the concurrency hypothesis, as the 30–44 year-olds, since the hypothesis largely suggests that men in their early years will use their advantageous position to have numerous partners, but Whites neither face a significant sex ratio imbalance, nor do the positive associations show up for the youth. Overall, we suggest that both both the growth in the prison population and the abuse of crack cocaine during the 1980s contributed to an increase in Black female gonorrhea incidence.

Chapter 5

STDs and Abortion Legalization

5.1 Introduction

The legalization of abortion, along with the development of oral contraception is one of the most profound technological changes related to fertility in all of human history (Goldin and Katz 2002). It provided American women with the ability to time their childbearing, allowing them to pursue other activities (such as invest in their labor market skills) at lower cost in the absence or failure of contraception. Social scientists have extensively documented the impact of abortion access and abortion-related costs on a range of fertility outcomes, such as birth rates, pregnancies, abortion utilization and contraception use (see Levine (2004) for an review).

More recently, though, attention has shifted to non-fertility outcomes of abortion legalization. A major reason for this shift is the now-famous paper, Donahue and Levitt (2001) (DL01). Their argument is that the legalization of abortion changed the composition of subsequent birth cohorts, and robbed the emerging "treated" cohort of future criminals. DL01 regress crime rates from 1985 to 1996 on abortion ratios lagged 15 to 25 years with controls for year and state fixed effects and find that 50 percent of the increase in the mean abortion ratio is associated with an 11 percent decrease in violence crime, 8 percent reduction in property crime and 12 percent decrease in murder. Furthermore, these effects generally are more precise and larger in magnitude than other explanations offered by the crime literature, such as the size of the police force and the effects of incarceration.

This result has been disputed, most notably by Joyce (2004, 2006), Foote and Goetz (2005, 2006) and Dills and Miron (2006). Joyce (2004) uses within-state comparison groups

to difference out changes in crime that are unrelated to unmeasured so-called "period effects" like crack cocaine, and is unable to find evidence that abortions reduced crime in the 1990s. Donahue and Levitt (2004) (DL04) respond by arguing that the estimates of abortion on crime in Joyce (2004) suffer from omitted variable bias because Joyce uses a panel during which the crack epidemic was at its peak. Extending the sample period, they show, causes the DL01 results to return.

Foote and Goetz (2006) claim that DL01 is fundamentally flawed for two different reasons. First, they point out that DL01 failed to control for state-year fixed effects. Secondly, Foote and Goetz (2006) criticize DL01 for using crime measures that had not been adjusted for population levels, despite using per capita adjustments in the right-hand side variables. When both of these problems are corrected, the evidence for a relationship between abortions and crime disappears. Donahue and Levitt (2006) (DL06) acknowledge the omission of state-year fixed effects, but argue that the results disappear because of measurement error in the abortion ratio[1] caused the estimated coefficients to be biased towards zero when state-year fixed effects are included. To correct for this, DL06 instrument for the CDC abortion ratio using a separate measure of abortions from the Alan Guttmacher Institute (AGI). Though both variables have measurement error, the error is uncorrelated, and thus makes the AGI abortion ratio variable a suitable instrumental variable. The IV results produce an abortion effect, although smaller in magnitude than the DL01 finding.

The lack of robustness in the evidence surrounding the abortion-crime hypothesis encourages additional scrutiny. One strategy is to examine abortion's effect on the correlates of crime. For instance, Charles and Stephens (2006) finds illicit drug use falls for individuals born in the early abortion repeal states. In Donahue, Grogger and Levitt (2002) and Ozbeklik (2006), both papers find that teen pregnancy rates fell for those in the early repeal states. A potentially broader connection between legalized abortion and "bad" cohort outcomes is argued by Joyce (2006).

[1]DL01 used the ratio of abortions to live births as their proxy for unwanted children. These data were drawn from historic CDC surveys on abortions performed.

> "If abortion lowers homicide rates by 20 to 30 percent, then it is likely to have affected an entire spectrum of outcomes associated with well-being: infant health, child development, schooling, earnings and marital status. Similarly, the policy implications are broader than abortion. Other interventions that affect fertility control and that lead to fewer unwanted births — contraception or sexual abstinence — have huge potential payoffs. In short, a causal relationship between legalized abortion and crime has such significant ramifications for social policy and at the same time is so controversial, that further assessment of the identifying assumptions and their robustness to alternative strategies is warranted."

In this vein, I examine the relationship between abortion legalization and STDs. The same mechanism that connections abortion to crime could explain risky sexual behavior and STD incidence — moreso in light of the evidence produced by Charles and Luoh (2006), Donahue, Grogger and Levitt (2002) and Ozbeklik (2006). In this project, I utilize a quasi-experiment to identify the effect of legal abortion on state-level, gonorrhea incidence. While some studies have used this strategy to identify the immediate effect of abortion legalization on gonorrhea (Klick and Stratmann 2003), I am the first to study the connection between cohorts exposed to legal abortion and subsequent gonorrhea rates 15–19 years later.

First, I estimate a difference-in-difference (DD) model using repeal states as the control group. Similar to Charles and Luoh (2006), I find that 15-19 year old gonorrhea rates fell in repeal states approximately 16-19 years after legalization, followed by similar declines 16–19 years later in the other 45 states that legalized abortion under *Roe v. Wade* (hereafter: "Roe states"). Because DD cannot disentangle cohort effects from the potentially important period effects, I use difference-in-difference-in-difference (DDD) estimation with older age groups in both the repeal and Roe states. The DDD results show that gonorrhea fell for all age cohorts approximately 16–19 years after early abortion repeal, followed by 16–19 years after Roe. I take this as evidence that rejects the abortion-legalization hypothesis, since as falsification, gonorrhea should not have fallen among the older individuals who were not *in utero* during

abortion repeal. An alternate explanation is that the AIDS epidemic reduced gonorrhea via the deterrence of risky sexual behavior. Since the AIDS epidemic hit the repeal states first (California, particularly), followed by the Roe states a few years later, this could explain why the DD tests had shown evidence in support of the hypothesis.

5.2 History and Implication of Abortion Legalization

Abortion legalization is believed to influence child and adult outcomes through two related, though distinct, mechanisms. First, legalization reduces birth cohort sizes, and thus may decrease outcomes through this mechanical reduction. Second, depending on which individuals were aborted, the composition of the birth cohort will change as well. Gruber, Levine and Staiger (1999) found that the "marginal child" who would have been born had abortion not been legal was "60 percent more likely to live in a single-parent household, 50 percent more likely to live in poverty, 45 percent more likely to be in a household collecting welfare and 40 percent more likely to die in the first year of life." This evidence provides a strong motivation for studies that attempt to track abortion legalization's effect as the birth cohort aged.

Being born into difficult circumstances as an unwanted child may lead to a variety of poor adult outcomes. Beyond crime, only three have been studied. Angrist and Evans (1999) use state abortion reforms to estimate the effect of teen and out-of-wedlock childbearing on schooling and labor market outcomes of mothers. They find that black women exposed to abortion reform experienced large reductions in teen fertility and teen childbearing that, in turn, caused schooling and employment rates to increase. Donahue, Grogger and Levitt (2002) and Ozbeklik (2006) find that abortion legalization reduced teenage pregnancy among the individuals who were *in utero* during abortion reform. And finally, Charles and Stephens (2006) present evidence that abortion legalization lowered alcohol and illicit drug use among the treated birth cohort.

Taken together, these findings suggest a role for abortion legalization in STD rates. First, according to Grossman's theory of health capital (Grossman 1972*a*,*b*; Lleras-Muney 2005), education is causally associated with improved health outcomes. Individuals more likely to graduate from high-school and attend college face higher wages and thus have an incentive to protect their human capital by reducing their STD risk. Secondly, Gruber, Levine and Staiger (1999) found the marginal child was more likely to be born to single-parent households, and since I found that children whose biological or adoptive parents are no longer married have higher rates of promiscuity (see 5.7) and less consistent condom use patterns (see 5.12), this suggests that abortion legalization could reduce gonorrhea rates through the family structure mechanism. And third, STD and drug-use rates are highly correlated. For instance, it is suspected that the crack epidemic was associated with the dramatic rise in gonorrhea and syphilis incidence during the 1980s, partly fueled by increased female prostitution and the practice of exchanging sex for drugs or money to purchase drugs (Edlin et al. 1994; Jones et al. 1998; Flom et al. 2001). In addition, others (Chesson, Harrison and Kessler 2000; Carpenter 2005) have found gonorrhea rates to be responsive to limits on alcohol consumption, thus suggesting that anything which reduces substance abuse may inadvertently reduce risky sex and gonorrhea as well.

Abortion was legal in the US until the late 1800s, at which time it was made universally illegal because of public health concerns[2], though not all of the bans were enforced. As late as 1961, the Supreme Court in *Poe v. Ullman* upheld a lower court ruling that banned contraception even for married individuals. In response to *Poe*, Connecticut activists immediately opened a birth control clinic and were arrested. These events ultimately led to the 1965 *Griswold v. Connecticut* Supreme Court ruling, which reversed the earlier ban on contraception, arguing that the Connecticut law was unconstitutional because of an individual's right to privacy. In 1973, the Supreme Court ruled that a Texas statute prohibiting abortion was unconstitutional. In *Doe v. Bolton* — a second ruling in 1973 — it was ruled unconstitutional

[2]The following summary is taken from chapter 2, "Abortion Law and Practice," from Levine (2004).

for the state to regulate where an abortion is performed (e.g., certain hospitals only) as well as to intervene in the decision at all. With these two rulings, liberalized abortion access was ensconced in US law.

Although the 1973 ruling had the most widespread changes in abortion access and availability, there were nonetheless several state-level changes in abortion access during the 1960s and early 1970s which altered the abortion landscape prior to *Roe*. First, Alaska, California, Hawaii, New York and Washington either repealed or had their ban revoked through judicial decision in 1970. A 1969 State Supreme Court decision ruled that the pre-1967 California abortion law was unconstitutional and evidence shows that abortions were commonly practiced in California as early as 1970. The other four states all repealed their abortion statutes through formal repeal legislation. Second, Arkansas, Colorado, Delaware, Florida, Georgia, Kansas, Maryland, New Mexico, North Carolina, Oregon, South Carolina, and Virginia implemented more modest reforms in the late 1960s and 1970 which made abortions legal for women under special circumstances. Finally, in Connecticut, District of Columbia, Illinois, Michigan, Missouri, New Jersey, South Dakota and Vermont, a lower level court decision left the legal status of an abortion somewhat ambiguous, although there is no evidence that this ambiguity had any meaningful effect on abortions in those states.

Using the early repeal of Alaska, California, Hawaii, New York and Washington as a comparison group, Levine, Staiger, Kane and Zimmerman (1999) estimate that abortion legalization lowered birth rates approximately 4.13%. The effects were largest among 15-19 year olds (–12.08%), 35-44 year olds (–7.86%), non-White women (–11.63%) and non-married women (–5.49%). I focus, therefore, on these same states. Cohorts born from 1970-1972 in early "repeal" states were "treated" with abortion legalization, and as such, would be expected to translate into discontinuous reductions in gonorrhea incidence among 15-19 year olds 15-19 years later. Since *Roe v. Wade* effectively legalized abortion in the remaining 45 states in 1973, I expect gonorrhea rates to fall 15-19 years later as that "treated" cohort

matures and enters the sample. Thus, "treatment" refers to the legalization of abortion 15-19 years after legalization occurred in that state.

5.3 Gonorrhea Epidemiology and State Trends

Gonorrhea is a sexually transmitted bacterial infection caused by *Neisseria gonorrhoeae* which grows and multiplies in the reproductive tract as well as the mouth, throat, eyes and anus of men and women (Holmes et al. 1999). Gonorrhea also has a very short incubation period and is therefore highly correlated with contemporaneous sexual behavior (Harrison et al. 1979), making it ideal for a study seeking to determine the effects following a change in the composition of an aging cohort. Symptoms for women include painful urination, vaginal discharge and inter-menstrual bleeding for women. Men experience painful urination, discolored discharge and penile edema (Holmes et al. 1999). While gonorrhea is easily cured with clinical dosages of antibiotics, left untreated it leads to serious health problems, such as PID and infertility in women, and infertility and epididymitis in men. It also will accelerate the transmission of HIV from an infected to non-infected person due to genital sores and discharge created by the infection (Holmes et al. 1999; Oster 2005). Gonorrhea is also ideal for a study of this kind because of its high prevalence in the heterosexual and homosexual network, the strong link between infection and sexual behavior, and the high correlation between bacterial infections and multiple heterosexual and homosexual partnerships.

As was discussed in chapter 4, data on gonorrhea were acquired from the CDC Division for STD Prevention. This data was collected by the CDC from state health departments starting in 1981 and extending to 2004. The data is disaggregated at the state-level by age, race and gender. For 1981-1982, the CDC reports that while every state reported, information on race and sex was highly problematic, and so I drop those 2 years from our sample. Furthermore, years 1983 and 1984 have many missing values, notably for New York and several southern states, and so based on recommendations from CDC officials, I have also opted to drop these

years from our sample[3]. For figures which report national gonorrhea rates, I include the entire 1981-2004 series, but for regression analysis, I include only 1985-2000 because of the measurement error at the state level and the need to match our time series with the covariate series (some of which end in 2000). The outcome variable - gonorrhea - is expressed as the number of cases per 100,000.

Figure 5.9 shows US gonorrhea rates for Black and White 15–19 year-olds. The first panel shows each trend using the same left-hand-side scale, while the second graph uses two scales so as to help the reader see the relatively small fluctuations in White gonorrhea rates against the large movements in Black rates. First, as all the figures show, Black rates vastly outnumber White rates by a magnitude of 5–30, depending on the year. Though rates have fallen among Blacks from their heights in the late 1980s, by 2004 Black cases still outnumbered White cases by a factor of 17 (see Figure 10). As the second panel of Figure 5.9 shows, most of the movement in gonorrhea as measured by aggregate state-level statistics reflects the Black series pattern. First, both Blacks and Whites saw dramatic decreases in gonorrhea incidence throughout the 1990s. Secondly, both groups experienced a temporary increase in gonorrhea around 1985, although this spike persisted longer for Blacks. Nevertheless, the broad trends of the two groups are indeed similar.

The abortion legalization hypothesis predicts that 15–19 year-old gonorrhea rates should fall 15–19 years after 1970 in the repeal states (relative to the Roe states), followed by a subsequent decline around 1990 in the Roe states. Figure 5.11 show the change in gonorrhea rates for repeal and Roe states for both Black and White 15–19 year-olds. The pattern for Blacks is consistent with the prediction. Figure 5.11 indicates that state gonorrhea rates began their decline in 1985, and as the 15–19 year-old demographic cell is covered by the treatment initiated in 1970, the gonorrhea rates began falling. Gonorrhea rates do not begin to drop in the Roe states until 1990. Figure 5.11 also shows that while it is the case that

[3]Including 1981-1984 does not qualitatively change the ensuing results.

White gonorrhea rates fell in the repeal states starting in 1985, they also began falling in the Roe states as well, suggesting the hypothesis does not fit the White data.

5.4 Empirical Methodology

5.4.1 Description of Data

A test of the abortion legalization hypothesis must include other factors that may have affected the spread of gonorrhea who timing correspond to changes in the abortion law. The crack and AIDS epidemics fit these criteria. Several studies have shown that men and women who regularly smoked crack were far more likely to be infected with HIV and AIDS than non-users (Edlin et al. 1994). Some evidence suggests that because of its highly addictive nature, regular users committed income-generating crimes, including commercial sex work, to support their habits (Jones et al. 1998; Grogger and Willis 2000; DeSimone 2001). Some commercial sex workers reported to also exchange sex directly for crack, and that those who did used condoms inconsistently and increased the number of partners they had over a given time frame. Hence, crack should be positively correlated with risky sex, and therefore STDs like gonorrhea. To address this, I use a crack cocaine index constructed by Fryer et al. (2006). The crack index varies by state and year for 1980-2000 and is the product of factor analysis using cocaine-related arrests, cocaine-related and crack-related drug seizures, and cocaine-related deaths – all at the state level. I plot the mean crack index for repeal and non-repeal states in Figure 5.12. The plots show that crack spiked in both the repeal and non-repreal states during the coverage years of 1985-1989, and increased more sharply in the repeal states – most notably California and New York. This is surprising given the *decline* in gonorrhea that begins in 1985 in the repeal states at the time.

AIDS was initially concentrated among gay and bisexual men, and two of the repeal states had very high homosexual and bisexual male populations at the time (CA and NY). If AIDS awareness reduced risky sex, as some studies suggest (Ahituv, Hotz and Philipson 1996; Chesson, Dee and Aral 2003), then the declines in gonorrhea in repeal states could merely be

picking up this relationship, and not earlier abortion legalization. As Figure 5.12 shows, the per capita AIDS mortality rates spiked earlier in the repeal states, reaching approximately 50 deaths per 100,000 by 1989, which is roughly equal to the highest ever AIDS mortality rates in the non-repeal states. Furthermore, at its peak, the repeal states experienced death rates from AIDS that were roughly 2.5 times larger than that of non-repeal states.

Following Chesson, Harrison and Kessler (2000); Chesson, Dee and Aral (2003); Carpenter (2005), I control for a variety of economic and demographic variables. The economic variables include a measure of the percentage of the state living in poverty, the annual state unemployment rate, and the real state per capita income. As in chapter 4, I also include per capita alcohol consumption and the male incarceration rate. Finally, I control for the percentage of the state that is Black and the percentage of the state that is between 15–19 years of age.

5.4.2 Identification Strategy

The abortion legalization hypothesis has been tested in two ways — the 2SLS approach of Gruber, Levine and Staiger (1999) and the "effective abortion ratio" strategy of DL01. In Gruber, Levine and Staiger (1999), state-level birthrates for the early 1970s are regressed onto state and year fixed effects and a dummy variable indicating whether abortion had been repealed in the state. These predicted birthrates are then included in the second stage regression. For instance, Gruber, Levine and Staiger (1999) regress infant birth weight onto the predicted birthrate of the cohort, while Charles and Luoh (2006) regress drug and alcohol use onto the predicted birthrates of each individual in his sample. DL01's identification strategy relates crime rates and arrests to lagged abortion ratios adjusted for state and year fixed effects, making abortion ratios a proxy for unwanted childbearing. Arrests of 15–24 year-olds are regressed by single year of age onto the abortion ratio in the year before

a cohort was born (e.g., arrests of 20-year-olds in 1990 in state j are correlated with the abortion ratio in state j in 1969, which is t-20-1.[4]

My data is disaggregated by state, gender, race and age intervals. I know the gonorrhea rate for 15–19 year-olds, but not the gonorrhea rate for 15-year-olds. As such, I cannot match the sample of 15-19 year olds to a precise birth cohort. Future work will incorporate DL01's strategy into the analysis.

Alternatively, I exploit repeal status as a quasi-experiment and estimate the difference-in-difference (DD) in gonorrhea rates between repeal and non-repeal states. Figure 5.13 shows graphically the basic DD identification strategy. Repeal and non-repeal states have different STD rates because of difference in the underlying population, the magnitude of which is measured by k in Figure 5.13. In 1970, 5 states legalized abortion[5], and three years later in 1973, abortion became nationally legal because of the Supreme Court ruling, *Roe v. Wade*. Therefore, the value of k should fall in or around 1985 and remain low for 3 years before rising back to its original point. Figure 5.13 leads to a regression model of the form:

$$GON_{s,g,t} = \beta_1 Repeal_s + \beta_2 D_t + \beta_3 Repeal_s \times D_t + \alpha X_{s,a,t} + \epsilon_{s,g,t} \tag{5.1}$$

where $X_{s,a,t}$ is a matrix of state-varying and time-varying covariates, including the crack index, the AIDS mortality rate, male incarceration rates, alcohol consumption, percent of the state Black, percent of the state 15-19 years of age, percent of the state living in poverty, real income per capita, and state unemployment rates. D_t is a year dummy and $\epsilon_{s,g,t}$ is an error term. The parameter of interest in this regression is β_3 which estimates the average difference in gonorrhea rates for repeal states (compared to Roe states) for each year.

If the abortion legalization hypothesis is correct, then repeal states should experience declines in 15-19 year old gonorrhea rates for approximately 3 years (roughly 1986-1989),

[4]In a recent NBER working paper, Ananat, Gruber, Levine and Staiger (2006) proposes a more general empirical framework that encompasses both approaches as a special case, and which separately estimates impacts of the marginal birth and the marginal pregnancy.

[5]Alaska, Hawaii, New York and Washington all legalized abortion in 1970, whereas California's supreme court ruled in 1969 that their statutes prohibiting abortion were illegal, making the practice legal by *de facto* in 1970.

which corresponds to the period of time where the gonorrhea variable consists of 20 percent to 80 percent treated individuals from repeal states, and virtually no treatment in the Roe states. Furthermore, the effect should go to k, or at least be statistically insignificant from 1990 on, since Roe states will presumably begin to experience declines which cancel out the observed differences.

While finding statistically significant and negative values for β_3 is consistent with the abortion legalization, other tests are available which can better establish this relationship. Because abortion legalization applies exclusively to the 15-19-year-old cohort, though, I should not find a policy effect on older cohorts. To test this, I employ a DDD strategy in which the 15-19-year-olds are compared to older cohorts. Then, the estimating equation becomes:

$$\begin{aligned} GON_{s,g,t} &= \beta_1 Repeal_s + \beta_2 D_t + \beta_3 Repeal_s \times D_t + \beta_4 Age_{15-19} + \beta_5 Age_{15-19} \times Repeal_s \\ &\quad + \beta_6 Age_{15-19} \times D_t + \beta_7 Age_{15-19} \times Repeal_s \times D_t + \alpha X_{s,g,t} + \epsilon_{s,g,t} \end{aligned} \tag{5.2}$$

where now the parameter of interest if β_7. The Age_{15-19} is an indicator variable equalling 1 if the cell corresponds to 15-19-year-olds, and 0 if older. If the abortion legalization hypothesis is correct, β_7 should be negative and statistically significant for 1986-1989, and small and/or insignificant for 1990-2000.

5.4.3 Results

Table 5.5 shows the results from the DD model for both Blacks and Whites with and without the control variables. The first column reports the DD regressions for the Black demographic cells. I find that for 1986–1991, the Black (base model) estimates are negative and statistically significant. In 1986, Black, youth gonorrhea rates fell 643 cases a year compared to the rest of the country. That statistic grew to –728 in 1987, –1136 in 1988, –1543 in 1989, –1398 in 1990, and –1305 in 1991. The subsequent years are statistically insignificant for the most part, and continue to fall in absolute value. This pattern largely resembles that predicted

by the abortion legalization hypothesis, as these years represent the proportion of the 15–19 year-old sample that consists of individuals who were exposed to abortion legalization while *in utero*. The effect grows as the year's progress, then fall as the Roe states begin experiencing their own declines.

Including the extensive demographic and economic controls causes some of these estimates to shrink and lose their significance, but the overall pattern remains roughly the same. For instance, cases begin falling in the repeal states in 1986, and more or less grow until 1989 (–1211 cases) before then beginning to fall. Including the controls causes the effect to show up only in the 1986–1990. Crack is positive, but not statistically significant for Black gonorrhea. AIDS mortality, furthermore, is strongly negative and statistically significant — for every 100 men who died from the AIDS virus, Black gonorrhea fell 7 cases a year. Incarceration is also positive and highly significant for this demographic – an additional point increase in the Black male incarceration rate is associated with a 59 additional cases of Black gonorrhea. The alcohol variable is also significant: a tenth of an additional gallon of alcohol consumed every year is associated with 179 more cases of gonorrhea.

For Whites, the pattern is less convincing. Point estimates remain largely in the 150–220 range after 1987, even though estimates are only statistically significant for the bulk of the 1987-19991 years. When controls are included, point estimates do not change much at all. Crack increases White youth gonorrhea by around 17 additional cases a year. AIDS mortality is positive, but is both small in magnitude and statistically insignificant. Alcohol increases White gonorrhea by a fraction of the amount it did for Blacks — a tenth of an additional gallon of alcohol consumed every year is associated with 12 additional cases of White youth gonorrhea. And finally, state unemployment is positively related with White gonorrhea — for every tenth of a percentage point that the unemployment rose, White gonorrhea cases increase by approximately 1.6 more cases a years.

Figure 14 plots the DD coefficient estimates, which correspond to $\hat{\beta}_3$. The shaded regions for 1986-1989 represent the years in which the treated cohort enters the sample, and therefore

the years in which we should expect to see declines in gonorrhea if the hypothesis is true. The vertical bars are confidence intervals around the black circle, which is the point-estimate. As can be seen, Black estimates are strongly negative and statistically significant for 1986-1989, and then either begin rising and/or become imprecise. The effects on Whites, on the other hand, is less convincing. Estimates never change much over the entire sample, suggesting repeal and Roe states do not exhibit significantly different patterns from 1981-2000.

The DD framework cannot separate period effects from age effects, so I estimate a DDD model using older cohorts as control groups. There is a tradeoff with using older cohorts. On the one hand, they were not *in utero* during abortion legalization, and thus provide a counterfactual to the 15–19 year-olds. On the other hand, if 15–19 year-olds have sex with older individuals, then declining gonorrhea rates among 15–19 year-olds would reduce gonorrhea rates in the older cohort, and therefore wash away the possibility of identification. Table **??** shows the results from the DDD estimates. Since models were estimated separately for Blacks and Whites, the columns are labeled for the race of the sample (B or W). Different control groups were used for each race, as well. In the first column of the estimation results (B2529), the 15–19 year olds were compared to the 25–29 year-olds. The third column (B3034) uses 30–34 year-olds as the control group, and so on. When I include controls for period effects, I lose most of the results on Blacks and Whites.

First, none of the $\hat{\beta}_7$ are statistically significant for column B2529. Crack enters positive (97 additional cases of Black gonorrhea for every 1 point increase in the crack index) and so does AIDS mortality (7 fewer cases of gonorrhea for every 100 men who died of AIDS). Alcohol consumption is positive and significant as before. But, none of the abortion legalization results enter significantly. For Whites, the story is slightly different — years 1987 and 1989 are negative (–161 and –212 respectively) and significant at the 10% level. But, White estimates had never been very convincing in the raw DD estimation since repeal and Roe states began falling at approximately the same time among youth. Crack is positive (15 additional cases), but AIDS mortality is not.

It's possible that the 25–29 year-olds are too close in age to the 15–19 year-olds to serve as adequate control groups, so I also estimated DDD models with 30–34 year-olds and 35–39 year-olds as implicit controls for period effects. AIDS mortality enters negative and significant for Blacks always, though slightly smaller in magnitude than previously reported, suggesting that the largest effect of AIDS mortality on reducing gonorrhea was among youth. Crack enters positive and significant for nearly all the Black and White results. The only place where we find evidence for the abortion legalization hypothesis is in the 35–39 year-old results, and there the effects are very strong. Focusing on column B3539, I find that 15–19 year-olds in repeal states fell relative to the Roe states and the 35–39 year-olds in their own state by 924 cases in 1986, 760 cases in 1987, 977 cases in 1988, 1,356 in 1989. The effects are negative but insignificant in 1990, and –1,103 in 1991 and significant at the 10% level. Following 1991, the coefficient estimates fall dramatically, where they remain for the rest of the sample period. AIDS mortality also enters negative and statistically significant for Blacks in this sample (100 deaths lowered Black gonorrhea rates by 4 cases a year).

5.5 Conclusion

I test a variation of the DL01 hypothesis that abortion legalization reduced criminal behavior by focusing on a correlate of crime – gonorrhea incidence. Gonorrhea trends show abrupt declines in abortion repeal states starting in 1985, followed by dramatic declines in 1990 in the Roe states. Previous studies have suggested that the average living circumstances of children born after abortion improved because it reduced the number of unwanted children and allowed women greater control over their life-cycle fertility such that they could wait until they possessed the resources needed to raise a child.

First, I find strong evidence in the DD model that Black 15–19 year-old gonorrhea rates are being affected by abortion legalization because this group's gonorrhea rates fall in the abortion repeal states 15–19 years after abortion legalization, followed by similar declines in the Roe states 15–19 years 1973. This effect is robust to the inclusion of state and year fixed

effects, linear and quadratic time trends, economic and demographic controls, a measure of crack cocaine, and the cumulative number of men who died from AIDS in the 3 years prior to the year. The estimates on Whites are far less persuasive. Because DD cannot control for period effects, I use DDD with older cohorts who were unexposed to abortion legalization while *in utero* as implicit controls for period effects. Using 25–29 and 30–34 year-olds as controls for the period effects causes the estimated effect of abortion legalization on Black youth gonorrhea to disappear. The estimated effects return, though, when I include the 35–39 year-olds as controls. Since network externalities play a role in the transmission of gonorrhea, it's possible that 15–19 year old-declines in gonorrhea led to 25–34 year-old declines, as well, if there is considerable matching among this broad age interval. In all specifications of the data, AIDS mortality and crack cocaine have predictably large effects on reducing Black gonorrhea rates. I ultimately conclude that AIDS, crack and the legalization of abortion may have played a role in the timing of the decline in Black gonorrhea in the United States.

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Table 5.1: Estimated US AIDS cases and rates by race and gender, 2003

Ethnicity	Infections	Rate
White (non-Hispanic) Males	10,450	12.8
White (non-Hispanic) Females	1,725	2.0
White (non-Hispanic) Total	**12,175**	**7.2**
Black (non-Hispanic) Males	13,624	103.8
Black (non-Hispanic) Females	7,551	50.2
Black (non-Hispanic) Total	**21,174**	**75.2**

Source: CDC HIV Surveillance Report 2003

Table 5.2: Estimated numbers of adults and adolescents living with HIV/AIDS at the end of 2003 by race, sex, and exposure category

Exposure Category	Infected	Percent	Infected	Percent
Male	**White**		**Black**	
Male-to-male sexual contact	86,674	76	50,675	47
Injection drug use	10,550	9	23,658	22
Male-to-male sexual contact and inject drug use	10,431	9	7,817	7
Heterosexual contact	5,178	5	23,513	22
Other	1,524	1	1,198	1
Subtotal	**114,358**	**100**	**106,861**	**100**
Female	**White**		**Black**	
Injection drug use	6,625	34	13,244	23
Heterosexual contact	12,494	64	43,957	75
Other	447	2	1,118	2
Subtotal	**19,566**	**100**	**58,319**	**100**

Source: 2003 CDC HIV Surveillance Report

Table 5.3: Definition of Age-specific, Race-specific sex ratios

Male Ages	**Searching Among Women Ages**	**Competing with Men Ages**
a$\in [13, 19]$	[a-1, a+1]	[a-1, a+1]
a$\in [20, 24]$	[a-2, a+2]	[a-2, a+2]

Table 5.4: Statistics for *rprate* Distribution, by Age

White Males, Unbalanced Panel, All States, 1997-2002												
rprate	12 yo	13 yo	14 yo	15 yo	16 yo	17 yo	18 yo	19 yo	20 yo	21 yo	22 yo	23 yo
Mean	0.036	0.023	0.171	0.449	0.981	1.392	1.776	2.055	2.130	2.540	2.120	1.518
SD	0.234	0.281	1.433	1.712	4.643	6.042	5.997	6.895	6.012	7.476	5.672	1.717
.50 percentile	0	0	0	0	0	0	0.800	1.000	1.000	1.091	1.091	0.923
.75 percentile	0	0	0	0	0.923	1.091	1.714	2.000	2.182	2.400	2.000	2.118
.90 percentile	0	0	0	1.000	2.000	3.000	3.652	4.000	4.000	4.615	3.690	4.615
N	305	472	959	1588	2004	2129	2220	1707	1243	795	373	22
Black Males, Unbalanced Panel, All States, 1997-2002												
	12 yo	13 yo	14 yo	15 yo	16 yo	17 yo	18 yo	19 yo	20 yo	21 yo	22 yo	23 yo
Mean	0.128	0.066	0.633	1.533	2.163	2.904	2.921	3.684	3.622	3.252	3.399	1.360
SD	0.707	0.394	2.766	4.220	6.496	8.299	6.385	8.394	7.627	6.253	6.527	1.580
.50 percentile	0	0	0	0	0.600	1.000	1.333	1.412	1.500	1.657	1.200	1.091
.75 percentile	0	0	0	1.500	2.000	2.769	3.273	3.692	3.646	3.646	3.273	1.714
.90 percentile	0	0	1.714	3.692	4.800	5.333	6.000	8.000	7.714	6.667	7.385	1.846
N	94	148	342	677	853	926	981	773	568	352	155	15

Table 5.5: Summary Statistics for *rprate* sample – Balanced (1997-2002), All States

	Mean	SD	.50	.75	.90	$N \times T$
sr^{-1}	97.974	8.779	95.974	101.440	108.550	13,488
Age	214.345	28.280	215	235	252	13,488
Highest Grade Completed	10.311	2.144	11	12	13	13,488
Parents Married	0.478	0.500	0	1	1	13,488
Marital Status	0.014	0.118	0	0	0	13,488

Table 5.6: Summary Statistics for $(c/s) \times 100$ sample – Balanced (1999-2002), 24 States, $DRP > |40|$

	Mean	SD	.10	.50	$N \times T$
$(c/s)\times 100$	69.622	39.834	0	100	4039
sr^{-1}	100.860	9.679	91.183	98.285	4039
rp_{t-1}^{5+}	0.165	0.372	0	0	4039
Age	231.423	20.238	205	231	4039
Highest Grade Completed	11.360	1.644	9	12	4039
Parents Married	0.333	0.471	0	0	4039
Marital Status	0.032	0.176	0	0	4039

Table 5.7: Estimated Effect of Race on Recent Sex Partners (Clustered SE)

	POLS	.50	.75	.90	Linear FE
A. Balanced (1997-2002), All States					
Black	0.820	0.129	0.773	1.790	-
	(0.172)	(0.023)	(0.072)	(0.202)	-
Age	0.046	0.016	0.041	0.079	-0.058
	(0.006)	(0.002)	(0.002)	(0.007)	(0.029)
Education	-0.291	-0.074	-0.217	-0.521	-0.023
	(0.065)	(0.013)	(0.021)	(0.081)	(0.091)
Parents Married	-0.449	-0.140	-0.247	-0.426	0.060
	(0.111)	(0.016)	(0.034)	(0.101)	(0.317)
Married	-0.785	-0.072	-0.877	-2.025	-0.777
	(0.470)	(0.052)	(0.091)	(0.214)	(0.478)
Year Effects	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	Yes
$N \times T$	13488	13488	13488	13488	13488
R^2	0.054	0.068	0.103	0.117	0.033
B. Balanced (1997-2002), 24 States					
Black	0.799	0.114	0.765	1.806	-
	(0.184)	(0.024)	(0.083)	(0.192)	-
Age	0.051	0.015	0.042	0.080	-0.046
	(0.007)	(0.002)	(0.002)	(0.007)	(0.032)
Education	-0.324	-0.070	-0.233	-0.527	-0.077
	(0.075)	(0.012)	(0.028)	(0.077)	(0.115)
Parents Married	-0.349	-0.124	-0.209	-0.367	-0.051
	(0.134)	(0.024)	(0.043)	(0.090)	(0.427)
Married	-1.388	-0.080	-0.931	-1.967	-1.323
	(0.406)	(0.050)	(0.114)	(0.352)	(0.502)
Year Effects	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	Yes
$N \times T$	9990	9990	9990	9990	9990
R^2	0.048	0.062	0.100	0.114	0.029

Table 5.8: Estimated Effect of Sex Ratio on Recent Sex Partners (Clustered SE)

Balanced (1997-2002), All States					
	POLS	.50	.75	.90	Linear FE
Black	-0.175	-0.629	-2.229	-5.038	-
	(1.373)	(0.243)	(0.732)	(1.158)	-
sr^{-1}	0.005	0.003	0.002	-0.005	0.011
	(0.011)	(0.002)	(0.003)	(0.008)	(0.011)
Black$\times sr^{-1}$	0.009	0.007	0.029	0.067	-0.000
	(0.014)	(0.003)	(0.007)	(0.012)	(0.016)
Age	0.045	0.015	0.040	0.077	-0.059
	(0.006)	(0.002)	(0.002)	(0.007)	(0.029)
Highest Grade Completed	-0.287	-0.071	-0.211	-0.496	-0.015
	(0.065)	(0.014)	(0.022)	(0.089)	(0.090)
Parents Married	-0.452	-0.132	-0.263	-0.425	0.054
	(0.111)	(0.016)	(0.032)	(0.101)	(0.317)
Married	-0.770	-0.063	-0.866	-1.906	-0.782
	(0.465)	(0.051)	(0.070)	(0.232)	(0.477)
Year Effects	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	Yes
N	13488	13488	13488	13488	13488
R^2	0.054	0.069	0.104	0.120	0.034

Table 5.9: Estimated Effect of Sex Ratio on Recent Sex Partners (Clustered SE)

Balanced (1997-2002), 24 States					
	POLS	.50	.75	.90	Linear FE
Black	-2.637	-1.035	-4.113	-7.020	-
	(1.791)	(0.379)	(0.829)	(2.599)	-
sr^{-1}	-0.016	0.002	-0.007	-0.019	0.001
	(0.016)	(0.003)	(0.004)	(0.013)	(0.014)
Black$\times sr^{-1}$	0.034	0.011	0.048	0.087	0.017
	(0.019)	(0.004)	(0.009)	(0.027)	(0.018)
Age	0.049	0.014	0.038	0.079	-0.047
	(0.007)	(0.002)	(0.003)	(0.007)	(0.032)
Highest Grade Completed	-0.317	-0.069	-0.206	-0.529	-0.063
	(0.076)	(0.010)	(0.025)	(0.072)	(0.115)
Parents Married	-0.356	-0.116	-0.224	-0.387	-0.067
	(0.135)	(0.020)	(0.037)	(0.114)	(0.426)
Married	-1.345	-0.055	-0.864	-1.952	-1.305
	(0.402)	(0.067)	(0.118)	(0.378)	(0.499)
Year Effects	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	Yes
$N \times T$	9990	9990	9990	9990	9990
R^2	0.048	0.063	0.102	0.117	0.029

Table 5.10: Estimated Effect of Sex Ratio on Recent Sex Partners (Clustered SE)

Balanced (1997-2002), 24 States, Dropping $\lvert DRP \rvert > 40$					
	POLS	.50	.75	.90	Linear FE
Black	-1.589	-1.011	-4.031	-6.701	-
	(1.199)	(0.305)	(1.072)	(1.835)	-
sr^{-1}	-0.013	0.003	-0.008	-0.014	-0.005
	(0.009)	(0.002)	(0.005)	(0.011)	(0.008)
Black$\times sr^{-1}$	0.023	0.011	0.047	0.083	0.014
	(0.012)	(0.003)	(0.011)	(0.020)	(0.011)
Age	0.038	0.013	0.037	0.072	-0.067
	(0.005)	(0.002)	(0.002)	(0.007)	(0.020)
Highest Grade Completed	-0.212	-0.064	-0.190	-0.479	0.002
	(0.052)	(0.013)	(0.026)	(0.059)	(0.076)
Parents Married	-0.280	-0.111	-0.222	-0.340	0.009
	(0.103)	(0.024)	(0.033)	(0.116)	(0.185)
Married	-0.828	-0.050	-0.814	-1.673	-0.824
	(0.359)	(0.040)	(0.123)	(0.331)	(0.389)
Year Effects	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	Yes
$N \times T$	9834	9834	9834	9834	9834
R^2	0.098	0.072	0.123	0.148	0.060

Table 5.11: Estimated Effect of Sex Ratio on Recent Sex Partners (Clustered SE)

Balanced (1999-2002), 24 States, Dropping $\|DRP\| > 40$					
	POLS	.50	.75	.90	Linear FE
Black	0.549	0.508	1.396	-2.287	-
	(1.484)	(0.937)	(0.969)	(2.279)	-
sr^{-1}	-0.014	-0.003	-0.011	-0.013	-0.004
	(0.012)	(0.006)	(0.009)	(0.018)	(0.011)
Black$\times sr^{-1}$	0.005	-0.000	-0.000	0.045	-0.005
	(0.015)	(0.009)	(0.010)	(0.024)	(0.015)
Age	0.028	0.016	0.034	0.060	-0.129
	(0.005)	(0.001)	(0.003)	(0.008)	(0.036)
Highest Grade Completed	-0.210	-0.034	-0.197	-0.551	0.203
	(0.057)	(0.016)	(0.035)	(0.116)	(0.110)
Parents Married	-0.479	-0.391	-0.571	-0.894	-0.040
	(0.139)	(0.046)	(0.083)	(0.212)	(0.250)
Married	-0.854	-0.213	-1.050	-2.410	-0.871
	(0.340)	(0.053)	(0.106)	(0.379)	(0.381)
Year Effects	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	Yes
$N \times T$	7760	7760	7760	7760	7760
R^2	0.050	0.046	0.059	0.067	0.019

Table 5.12: Effect of Race and Other Covariates on Condom Use (Clustered SE)

Balanced (1999-2002), 24 States, Dropping $\|DRP\| > 40$				
	POLS	.10	.50	Linear FE
Black	14.595	5.115	7.111	-
	(1.762)	(2.582)	(1.455)	-
rp_{t-1}^{5+}	5.095	4.624	0.550	5.715
	(1.610)	(2.237)	(0.604)	(2.393)
Age	-0.432	-0.083	-0.201	1.064
	(0.059)	(0.039)	(0.072)	(0.623)
Highest Grade Completed	2.595	0.617	1.053	-0.697
	(0.643)	(0.295)	(0.605)	(1.402)
Parents Married	5.331	0.712	2.714	2.528
	(1.662)	(0.695)	(1.028)	(4.132)
Married	-34.894	-0.817	-81.331	-19.265
	(4.087)	(1.572)	(3.662)	(7.516)
Year Effects	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	Yes
$N \times T$	4039	4039	4039	4039
R^2	0.123	0.010	0.071	0.068

Table 5.13: Estimated Effect of Sex Ratio on Condom Use (clustered SE)

Balanced (1999-2002), 24 States, Dropping $\|DRP\| > 40$								
	POLS		.10		.50		FE	
	a.	b.	a.	b.	a.	b.	a.	b.
Black	-17.290		62.313		-51.674		-	
	(22.408)		(23.235)		(23.891)		-	
sr^{-1}	-0.157	0.543	-0.006	0.065	-0.407	0.298	-0.190	0.009
	(0.208)	(0.091)	(0.058)	(0.063)	(0.255)	(0.060)	(0.271)	(0.126)
Black$\times sr^{-1}$	0.318		-0.491		0.601		0.319	
	(0.228)		(0.191)		(0.256)		(0.298)	
rp_{t-1}^{5+}		-21.085		-20.304		-4.758		-37.683
		(16.764)		(21.870)		(13.514)		(20.311)
$rp_{t-1}^{5+} \times sr^{-1}$		0.232		0.215		0.046		0.369
		(0.163)		(0.229)		(0.122)		(0.196)
Age	-0.446	-0.523	-0.010	-0.056	-0.233	-0.278	0.969	0.984
	(0.061)	(0.061)	(0.027)	(0.038)	(0.057)	(0.060)	(0.620)	(0.618)
Education	2.506	2.486	0.060	0.240	0.923	1.042	-0.597	-0.529
	(0.644)	(0.653)	(0.216)	(0.230)	(0.724)	(0.474)	(1.401)	(1.403)
Parents Married	5.322	3.744	0.105	0.348	2.551	1.740	2.583	2.444
	(1.667)	(1.646)	(0.318)	(0.408)	(0.568)	(0.998)	(4.147)	(4.145)
Married	-35.044	-36.534	-0.172	-1.109	-79.243	-82.121	-19.314	-19.190
	(4.120)	(4.030)	(1.867)	(0.627)	(3.966)	(3.436)	(7.505)	(7.489)
Year Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Individual Fixed Effects	No	No	No	No	No	No	Yes	Yes
$N \times T$	4039	4039	4039	4039	4039	4039	4039	4039
R^2	0.122	0.110	0.011	0.006	0.074	0.070	0.065	0.066

Table 5.14: Summary Statistics for Covariates (Mean and Standard Devation)

	1981		1990		2000	
	Mean	SD	Mean	SD	Mean	SD
Crack	–0.19	0.28	1.34	1.08	1.40	0.87
Alcohol Consumption	2.86	0.81	2.50	0.61	2.28	0.48
Per capita Income	$10,896.04	1639.99	$18,644.29	3088.10	$28,572.45	4696.82
State Unemployment Rate	7.29	1.85	5.40	1.16	3.87	0.93
Poverty	14.18	4.38	13.28	4.27	10.87	2.95
Percent 15-19	9.05	0.41	7.20	0.55	7.33	0.66
Percent Black	11.63	12.85	11.38	12.57	12.55	12.73

Table 5.15: Effect of Incarceration on Gonorrhea and Syphilis (Clustered SE)

	1981-2000							
	Gonorrhea (All States)				Syphilis (States≥0.50)			
	Blacks		Whites		Blacks		Whites	
Male Incarceration Rate (IR)	16.320	-18.143	-24.230	-63.374	-3.268	-7.971	-2.782	-6.790
	(7.447)	(11.362)	(15.276)	(19.566)	(2.023)	(2.419)	(0.929)	(1.077)
Female × IR		69.057		77.949		9.394		8.080
		(14.452)		(20.078)		(2.249)		(0.951)
Crack	37.757	37.750	14.638	14.654	21.567	21.568	1.072	1.069
	(27.480)	(27.247)	(4.216)	(4.202)	(4.602)	(4.609)	(0.198)	(0.182)
Alcohol Consumption	329.662	329.007	360.553	360.398	116.879	116.667	9.190	9.203
	(236.662)	(232.802)	(66.282)	(65.814)	(24.262)	(23.553)	(2.180)	(2.037)
Per capita Income	0.016	0.016	0.005	0.005	0.011	0.011	-0.000	-0.000
	(0.031)	(0.031)	(0.005)	(0.005	(0.002)	(0.003)	(0.000)	(0.000)
State Unemployment Rate	-52.245	-52.275	15.809	15.800	-15.830	-15.854	-0.020	-0.020
	(19.705)	(19.661)	(4.666)	(4.631)	(2.817)	(2.793)	(0.225)	(0.206)
Poverty	-12.070	-12.070	0.646	0.641	-0.040	-0.042	-0.116	-0.118
	(10.209)	(10.167)	(1.091)	(1.085)	(1.187)	(1.178)	(0.054)	(0.051)
Percent 15-19	69.736	69.437	3.435	3.371	6.085	6.040	0.395	0.399
	(99.054)	(98.996)	(11.434)	(11.468)	(9.331)	(9.207)	(0.604)	(0.582)
Percent Black	-118.856	-118.861	30.131	30.164	-8.954	-8.977	0.435	0.436
	(116.139)	(115.359)	(14.445)	(14.473)	(6.151)	(6.079)	(0.451)	(0.463)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
$N \times T$	12180	12180	12180	12180	5376	5376	5376	5376
R^2	0.209	0.215	0.358	0.365	0.361	0.367	0.255	0.316

Table 5.16: Effect of Incarceration on Gonorrhea and Syphilis (Clustered SE)

	1981, 1990, 2000							
	Gonorrhea (All States)				Syphilis (States≥0.50)			
	Blacks		Whites		Blacks		Whites	
Male Incarceration Rate (IR)	10.710	-23.391	-43.838	-99.127	-2.849	-8.367	-0.507	-1.260
	(10.500)	(14.827)	(22.861)	(28.555)	(4.101)	(5.433)	(1.032)	(1.237)
Female × IR		68.739		104.582		11.614		1.408
		(18.830)		(27.892)		(7.633)		(1.312)
Crack	134.217	136.521	27.661	27.835	45.211	44.253	0.998	1.007
	(73.170)	(72.625)	(18.738)	(18.682)	(20.028)	(19.791)	(0.556)	(0.549)
Alcohol Consumption	-218.151	-227.194	689.674	691.312	100.153	97.732	2.221	2.130
	(359.129)	(358.320)	(149.017)	(148.434)	(73.375)	(71.036)	(2.006)	(1.991)
Per capita Income	-0.037	-0.039	0.001	0.001	-0.003	-0.003	-0.000	-0.000
	(0.041)	(0.040)	(0.010)	(0.010)	(0.007)	(0.007)	(0.000)	(0.000)
State Unemployment Rate	24.958	23.156	33.945	33.969	-1.932	-3.006	-0.038	-0.027
	(60.025)	(58.926)	(13.979)	(13.887)	(21.430)	(21.470)	(0.679)	(0.672)
Poverty	-17.224	-14.884	12.343	12.459	5.356	5.622	0.138	0.141
	(33.598)	(33.603)	(6.005)	(5.983)	(7.360)	(7.346)	(0.195)	(0.194)
Percent 15-19	-156.435	-157.806	0.528	1.369	121.557	122.490	0.647	0.624
	(164.728)	(164.050)	(18.235)	(18.170)	(43.064)	(43.052)	(0.945)	(0.938)
Percent Black	36.148	35.237	57.811	58.090	-8.061	-8.274	-0.319	-0.309
	(121.314)	(120.572)	(32.605)	(32.576)	(18.274)	(18.372)	(0.475)	(0.473)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
$N \times T$	1836	1836	1836	1836	600	600	600	600
R^2	0.338	0.348	0.453	0.462	0.697	0.703	0.530	0.536

Table 5.17: Effect of Incarceration on Gonorrhea and Syphilis for Blacks Only (Clustered SE)

	Low Black Population States Dropped, 1985-2000							
	Gonorrhea				Syphilis			
	<5% Dropped		<10% Dropped		<5% Dropped		<10% Dropped	
Male Incarceration Rate (IR)	63.761	-35.324	138.712	10.964	-13.742	-21.995	-14.821	-24.169
	(34.598)	(42.730)	(55.213)	(64.765)	(4.371)	(4.640)	(6.351)	(6.417)
Female × IR		198.669		257.070		16.460		18.652
		(38.159)		(52.785)		(3.591)		(4.283)
Crack	188.549	188.413	251.950	252.248	9.145	9.158	23.502	23.591
	(53.779)	(53.213)	(80.355)	(78.412)	(6.211)	(6.117)	(7.484)	(7.353)
Alcohol Consumption	963.990	965.047	444.327	444.846	174.536	174.575	99.650	99.514
	(407.379)	(394.611)	(470.796)	(447.437)	(38.239)	(36.928)	(31.977)	(30.783)
Per capita Income	0.004	0.005	0.055	0.057	0.018	0.018	0.020	0.020
	(0.041)	(0.040)	(0.072)	(0.070)	(0.003)	(0.003)	(0.005)	(0.005)
State Unemployment Rates	-16.829	-16.912	-81.193	-81.608	-16.979	-17.008	-26.926	-26.992
	(36.952)	(36.751)	(37.597)	(37.081)	(3.792)	(3.740)	(3.657)	(3.691)
Poverty	-10.276	-10.638	-11.897	-12.442	-1.851	-1.845	1.455	1.481
	(13.598)	(13.418)	(13.723)	(13.774)	(1.553)	(1.537)	(1.519)	(1.502)
Percent 15-19	332.707	332.587	72.600	71.136	-4.392	-4.405	6.614	6.688
	(208.885)	(206.791)	(307.490)	(298.436)	(18.866)	(18.464)	(22.168)	(21.552)
Percent Black	68.043	68.653	214.874	216.318	8.489	8.476	12.017	11.981
	(124.771)	(123.869)	(120.232)	(118.398)	(6.897)	(6.829)	(6.833)	(6.740)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
$N \times T$	6180	6180	4140	4140	3672	3672	2796	2796
R^2	0.324	0.339	0.356	0.375	0.444	0.454	0.509	0.522

Table 5.18: Effect of Incarceration on Gonorrhea and Syphilis (Clustered SE)

	1985-2000							
	Gonorrhea (All States)				Syphilis (States≥.50)			
	Blacks		Whites		Blacks		Whites	
Male Incarceration Rate (IR)	25.721	-6.405	10.521	1.729	-5.191	-10.257	-2.053	-4.816
	(7.302)	(10.626)	(8.527)	(11.176)	(2.357)	(2.908)	(0.694)	(0.812)
Female × IR		64.263		17.572		10.167		5.561
		(13.923)		(12.669)		(2.905)		(0.811)
Crack	65.933	65.618	10.685	10.683	26.629	26.638	0.881	0.882
	(27.763)	(27.647)	(1.932)	(1.933)	(5.471)	(5.491)	(0.148)	(0.142)
Alcohol Consumption	1163.104	1163.882	136.971	136.957	149.181	149.414	7.593	7.590
	(322.266)	(319.679)	(28.273)	(28.232)	(36.511)	(35.973)	(1.863)	(1.795)
Per capita Income	-0.038	-0.038	0.004	0.004	0.012	0.012	-0.000	-0.000
	(0.038)	(0.038)	(0.003)	(0.003)	(0.003)	(0.003)	(0.000)	(0.000)
State Unemployment Rate	2.656	2.920	10.522	10.529	-15.789	-15.786	0.009	0.010
	(23.804)	(23.786)	(2.586)	(2.585)	(3.941)	(3.923)	(0.207)	(0.199)
Poverty	-5.352	-5.318	-0.871	-0.874	0.708	0.707	-0.103	-0.103
	(10.353)	(10.291)	(0.743)	(0.743)	(1.326)	(1.318)	(0.056)	(0.054)
Percent 15-19	229.369	230.070	3.990	3.998	8.038	7.996	0.940	0.949
	(112.681)	(112.744)	(9.404)	(9.410)	(11.301)	(11.217)	(0.682)	(0.671)
Percent Black	-125.211	-125.269	7.777	7.777	-7.527	-7.576	0.378	0.380
	(113.966)	(113.577)	(7.205)	(7.224)	(6.648)	(6.587)	(0.405)	(0.413)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
$N \times T$	9792	9792	9792	9792	4800	4800	4800	4800
R^2	0.286	0.292	0.344	0.344	0.374	0.379	0.245	0.275

Table 5.19: Effect of Incarceration on Gonorrhea and Syphilis by Age for Blacks Only (Clustered SE)

	1985-2000							
	Gonorrhea (All States)				Syphilis (States≥.50)			
	15-29 year-olds		30-44 year-olds		15-29 year-olds		30-44 year-olds	
Male Incarceration Rate (IR)	26.088	-6.247	-2.058	-32.069	-9.184	-13.451	-0.719	-5.794
	(12.222)	(20.243)	(4.773)	(12.001)	(2.849)	(3.641)	(2.663)	(3.129)
Female × IR		64.649		60.556		8.537		10.202
		(22.883)		(13.167)		(3.610)		(2.149)
Crack	77.848	77.872	59.854	59.950	29.092	29.123	16.037	16.053
	(45.727)	(45.708)	(18.938)	(18.415)	(7.332)	(7.337)	(5.146)	(5.153)
Alcohol Consumption	1521.008	1519.112	917.178	918.518	152.625	152.643	77.847	78.415
	(485.776)	(484.740)	(258.673)	(248.998)	(33.689)	(33.548)	(28.035)	(25.577)
Per capita Income	-0.064	-0.064	-0.015	-0.015	0.015	0.015	0.007	0.007
	(0.065)	(0.064)	(0.025)	(0.025)	(0.004)	(0.004)	(0.003)	(0.003)
State Unemployment Rates	2.295	2.216	-2.759	-2.565	-17.591	-17.626	-15.541	-15.483
	(39.837)	(40.046)	(16.478)	(15.927)	(4.436)	(4.369)	(3.144)	(3.130)
Poverty	-4.434	-4.761	-3.477	-3.520	0.395	0.421	-0.578	-0.572
	(17.304)	(17.247)	(6.423)	(6.216)	(1.884)	(1.866)	(1.360)	(1.347)
Percent 15-19	409.715	410.161	68.685	68.757	14.874	14.806	-3.788	-3.726
	(186.056)	(186.463)	(65.995)	(65.815)	(14.187)	(14.111)	(12.119)	(11.583)
Percent Black	-158.197	-157.751	-76.164	-76.691	-11.899	-11.941	-6.599	-6.549
	(193.551)	(192.956)	(81.828)	(81.212)	(8.374)	(8.305)	(8.494)	(8.387)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
$N \times T$	4896	4896	4896	4896	2688	2688	2688	2688
R^2	0.405	0.408	0.241	0.263	0.414	0.418	0.337	0.349

Table 5.20: Effect of Incarceration on Gonorrhea and Syphilis by Age for Whites Only (Clustered SE)

	1985-2000							
	Gonorrhea (All States)				Syphilis (States≥.50)			
	15-29 year-olds		30-44 year-olds		15-29 year-olds		30-44 year-olds	
Male Incarceration Rate (IR)	-6.117	-7.808	-9.990	-24.708	-5.009	-9.395	-1.362	-5.098
	(12.141)	(16.793)	(3.630)	(5.972)	(1.265)	(1.622)	(1.349)	(1.502)
Female × IR		3.382		29.678		8.786		7.538
		(19.775)		(5.831)		(1.595)		(1.085)
Crack	16.003	16.001	5.274	5.254	1.222	1.223	0.947	0.947
	(3.196)	(3.196)	(1.567)	(1.561)	(0.264)	(0.236)	(0.289)	(0.267)
Alcohol Consumption	192.601	192.596	85.259	85.237	7.414	7.465	11.005	11.010
	(40.019)	(40.028)	(21.125)	(20.682)	(2.518)	(2.302)	(3.673)	(3.487)
Per capita Income	0.010	0.010	-0.002	-0.002	-0.000	-0.000	-0.000	-0.000
	(0.005)	(0.005)	(0.003)	(0.003)	(0.000)	(0.000)	(0.000)	(0.000)
State Unemployment Rates	17.157	17.156	3.122	3.116	-0.225	-0.218	0.175	0.173
	(4.034)	(4.036)	(1.338)	(1.310)	(0.296)	(0.261)	(0.360)	(0.339)
Poverty	-1.497	-1.497	-0.059	-0.062	-0.061	-0.061	-0.152	-0.150
	(1.142)	(1.142)	(0.347)	(0.339)	(0.073)	(0.070)	(0.073)	(0.069)
Percent 15-19	0.669	0.666	10.286	10.263	-0.303	-0.288	1.064	1.086
	(14.848)	(14.855)	(8.535)	(8.516)	(0.660)	(0.620)	(0.968)	(0.936)
Percent Black	7.564	7.564	10.648	10.636	-0.107	-0.101	1.034	1.031
	(11.511)	(11.521)	(6.615)	(6.634)	(0.414)	(0.433)	(0.757)	(0.768)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
$N \times T$	4896	4896	4896	4896	2688	2688	2688	2688
R^2	0.521	0.521	0.254	0.266	0.309	0.376	0.252	0.309

Table 5.21: Difference in Difference Estimates of Abortion Legalization on Second Generation Gonorrhea Incidence (Clustered SE)

A. Baseline Model and Full Model (15-19 year olds)				
	Black (base model)	Black (w/ controls)	White (base model)	White (w/ controls)
$Repeal \times D_{1986}$	-643.273	-602.828	-59.818	-74.673
	(312.515)	(324.507)	(49.429)	(52.899)
$Repeal \times D_{1987}$	-728.248	-564.749	-171.411	-181.886
	(363.465)	(360.061)	(79.229)	(76.764)
$Repeal \times D_{1988}$	-1135.687	-947.995	-184.563	-201.979
	(381.554)	(355.489)	(107.149)	(99.846)
$Repeal \times D_{1989}$	-1532.122	-1210.769	-224.562	-244.257
	(431.300)	(400.354)	(124.718)	(120.527)
$Repeal \times D_{1990}$	-1397.931	-853.424	-181.684	-190.720
	(673.557)	(621.486)	(105.997)	(105.090)
$Repeal \times D_{1991}$	-1305.128	-406.171	-183.677	-191.955
	(604.465)	(543.888)	(108.464)	(105.998)
$Repeal \times D_{1992}$	-992.888	29.526	-170.428	-188.135
	(694.604)	(675.240)	(115.054)	(110.951)
$Repeal \times D_{1993}$	-262.725	1113.244	-133.928	-142.451
	(872.223)	(821.950)	(106.693)	(112.713)
$Repeal \times D_{1994}$	-574.661	765.708	-151.402	-171.436
	(827.917)	(783.622)	(101.168)	(99.851)
$Repeal \times D_{1995}$	-559.456	790.065	-204.894	-233.044
	(789.462)	(755.986)	(132.682)	(129.697)
$Repeal \times D_{1996}$	-264.652	1298.800	-211.821	-213.434
	(732.018)	(836.046)	(129.849)	(125.894)
$Repeal \times D_{1997}$	-274.759	1207.915	-206.211	-193.411
	(730.394)	(799.543)	(129.518)	(127.022)
$Repeal \times D_{1998}$	-552.104	852.717	-215.196	-179.677
	(779.994)	(869.794)	(126.581)	(131.582)
$Repeal \times D_{1999}$	-432.013	830.785	-198.754	-171.939
	(807.501)	(930.033)	(133.103)	(133.118)
$Repeal \times D_{2000}$	-632.574	675.327	-208.423	-165.506
	(737.248)	(888.688)	(143.004)	(144.873)
Crack		75.614		16.865
		(71.507)		(5.550)
AIDS Mortality Rate		-0.071		0.004
		(0.036)		(0.004)
Male Incarceration Rate		59.276		25.858

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Table 5.21 – Continued

	Black (base model)	**Black (w/ controls)**	**White (base model)**	**White (w/ controls)**
		(20.130)		(25.289)
Alcohol Consumption		1789.349		132.721
		(795.149)		(66.757)
Percent Black		169.038		16.047
		(322.043)		(16.061)
Percent 15–19		-284.866		5.065
		(303.812)		(25.515)
Poverty		25.571		-2.919
		(28.069)		(2.303)
Real Income Per Capita		-0.045		0.010
		(0.091)		(0.007)
State Unemployment Rate		-54.071		15.841
		(64.477)		(7.529)
Year Effects	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes
N	1632	1632	1632	1632
R^2	0.738	0.753	0.663	0.678

Table 5.22: Difference-in-Difference-in-Difference Estimates Using Older Cohorts (Clustered SE)

	B. DDD on Each Older Cohort					
	B2529	**W2529**	**B3034**	**W3034**	**B3539**	**W3539**
$Age_{15-19} \times Repeal \times D_{1986}$	-571.645	-70.003	-604.113	-19.884	-923.959	-74.630
	(443.044)	(72.815)	(382.642)	(70.500)	(350.342)	(64.216)
$Age_{15-19} \times Repeal \times D_{1987}$	-163.731	-161.377	-292.247	-126.770	-759.717	-140.047
	(508.685)	(91.612)	(409.143)	(86.854)	(360.749)	(82.148)
$Age_{15-19} \times Repeal \times D_{1988}$	-573.039	-161.874	-452.915	-138.969	-977.388	-174.535
	(518.519)	(120.983)	(469.731)	(115.091)	(401.358)	(113.846)
$Age_{15-19} \times Repeal \times D_{1989}$	-786.742	-211.900	-783.581	-167.954	-1356.297	-205.477
	(606.525)	(122.515)	(523.992)	(117.902)	(458.315)	(114.720)
$Age_{15-19} \times Repeal \times D_{1990}$	-312.765	-172.544	-378.701	-126.688	-1058.360	-150.563
	(812.184)	(117.792)	(734.379)	(110.118)	(677.059)	(105.137)
$Age_{15-19} \times Repeal \times D_{1991}$	-578.751	-139.029	-465.714	-106.714	-1103.034	-149.529
	(788.753)	(124.060)	(627.816)	(112.778)	(581.968)	(109.176)
$Age_{15-19} \times Repeal \times D_{1992}$	-475.258	-123.721	77.528	-113.204	-425.677	-110.301
	(794.279)	(111.156)	(734.601)	(106.836)	(661.865)	(94.601)
$Age_{15-19} \times Repeal \times D_{1993}$	-233.309	-105.829	491.623	-61.772	-85.812	-109.850
	(930.008)	(113.222)	(888.033)	(107.788)	(854.389)	(104.303)
$Age_{15-19} \times Repeal \times D_{1994}$	-371.358	-133.167	72.505	-76.232	-308.044	-116.143
	(929.772)	(114.201)	(863.153)	(105.117)	(831.981)	(97.051)
$Age_{15-19} \times Repeal \times D_{1995}$	-243.909	-161.882	200.626	-142.892	-332.751	-151.084
	(889.009)	(132.360)	(808.142)	(126.934)	(744.374)	(119.297)
$Age_{15-19} \times Repeal \times D_{1996}$	-176.281	-137.832	165.148	-132.946	-169.475	-147.820
	(957.382)	(140.348)	(839.392)	(132.531)	(771.343)	(127.799)
$Age_{15-19} \times Repeal \times D_{1997}$	94.113	-126.906	325.400	-130.886	141.923	-136.898
	(913.352)	(131.555)	(820.800)	(126.067)	(764.293)	(119.681)
$Age_{15-19} \times Repeal \times D_{1998}$	-143.336	-158.383	18.738	-135.191	-176.796	-154.850
	(1012.294)	(149.611)	(899.945)	(139.156)	(830.792)	(136.862)
$Age_{15-19} \times Repeal \times D_{1999}$	-86.483	-175.755	134.964	-146.797	-175.579	-160.635
	(951.063)	(146.083)	(858.959)	(138.307)	(796.994)	(131.779)
$Age_{15-19} \times Repeal \times D_{2000}$	-204.926	-148.813	-36.613	-119.504	-221.137	-147.044
	(958.273)	(146.668)	(839.687)	(140.409)	(779.642)	(136.072)
Crack	97.393	14.629	106.551	12.786	54.126	9.915
	(46.281)	(3.429)	(42.509)	(3.179)	(36.413)	(2.934)
AIDS Deaths (cum. over prev. 3 years)	-0.069	0.001	-0.059	0.002	-0.042	0.002
	(0.024)	(0.003)	(0.021)	(0.002)	(0.019)	(0.002)
Male Incarceration Rate	-19.459	-27.322	-14.599	-33.793	4.924	-44.437
	(15.803)	(17.505)	(19.004)	(17.505)	(12.690)	(16.621)
Alcohol	1778.371	132.503	1626.297	115.457	1341.009	89.142

Table 5.22 – Continued

	B2529	**W2529**	**B3034**	**W3034**	**B3539**	**W3539**
	(536.166)	(40.816)	(509.872)	(37.755)	(488.446)	(34.696)
Percent Black	111.607	24.517	39.522	19.875	86.671	19.188
	(227.578)	(15.482)	(212.606)	(12.386)	(193.803)	(10.450)
Percent 15-19	133.856	11.266	58.014	14.018	-58.685	14.470
	(200.319)	(14.761)	(184.114)	(14.159)	(155.047)	(13.136)
Poverty	7.865	-0.605	10.391	-0.693	14.511	-0.565
	(18.345)	(1.290)	(15.284)	(0.895)	(14.755)	(0.920)
Income	-0.039	0.005	-0.034	0.002	-0.025	0.001
	(0.059)	(0.005)	(0.051)	(0.005)	(0.046)	(0.004)
State Unemployment Rate	-16.153	10.730	-10.014	9.407	-20.913	8.465
	(40.615)	(4.464)	(36.214)	(4.163)	(34.475)	(4.061)
Year Effects	Yes	Yes	Yes	Yes	Yes	Yes
State Effects	Yes	Yes	Yes	Yes	Yes	Yes
State Trends	Yes	Yes	Yes	Yes	Yes	Yes
N	3264	3264	3264	3264	3264	3264
R^2	0.656	0.493	0.671	0.497	0.699	0.513

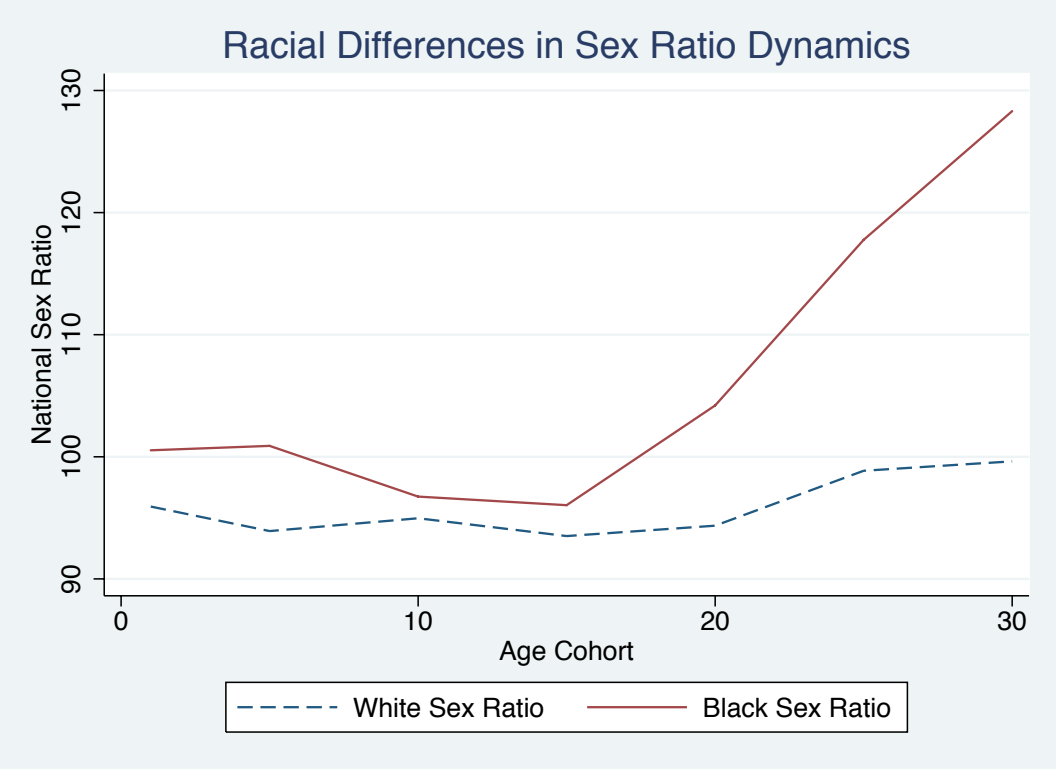

Figure 5.1: U.S. Sex Ratio^{-1} by Race and 5-Year Age Cohorts

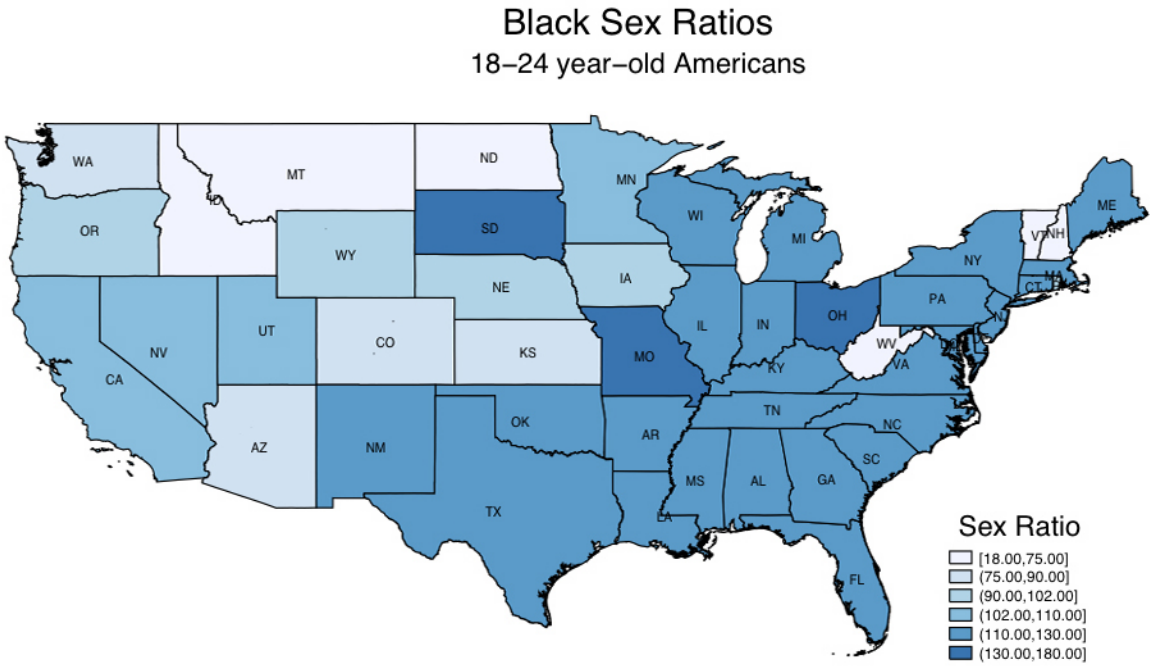

Figure 5.2: Black 18-24 Year olds State Sex Ratios^{-1} by Race

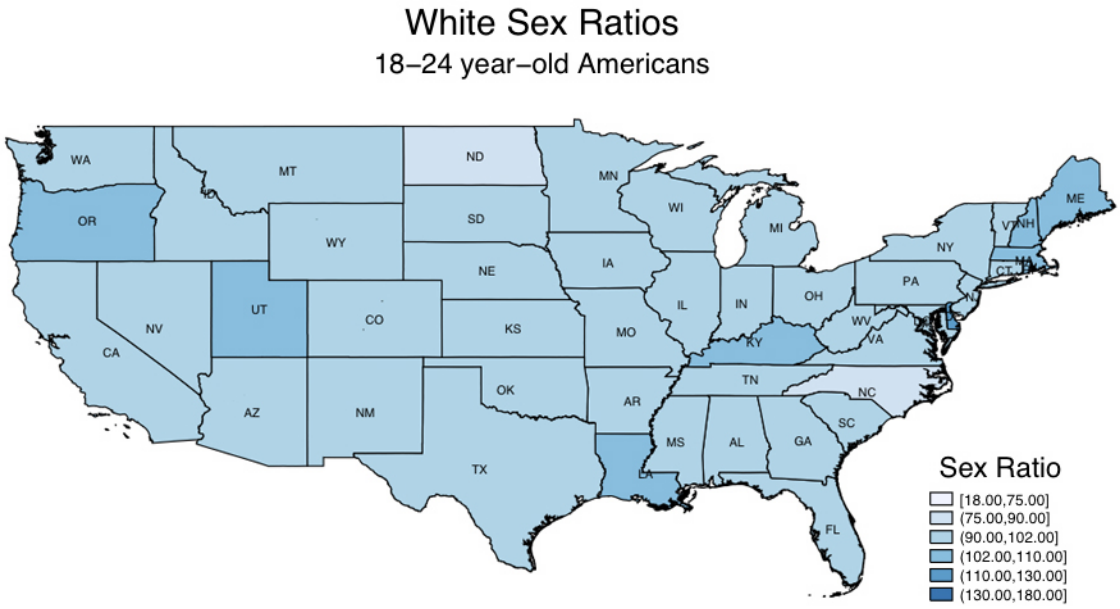

Figure 5.3: 18-24 White Year olds State Sex Ratios^{-1} by Race

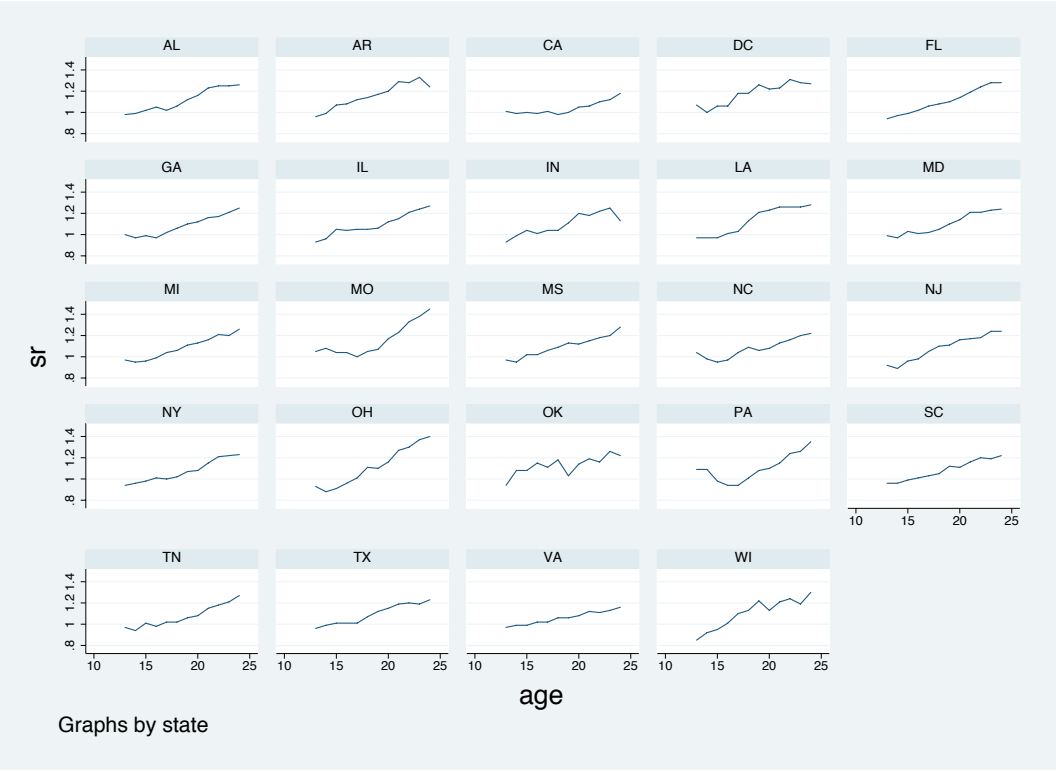

Figure 5.4: Black Sex Ratios for 24 States with Most Black Population (Source: Census 2000 Longform Survey)

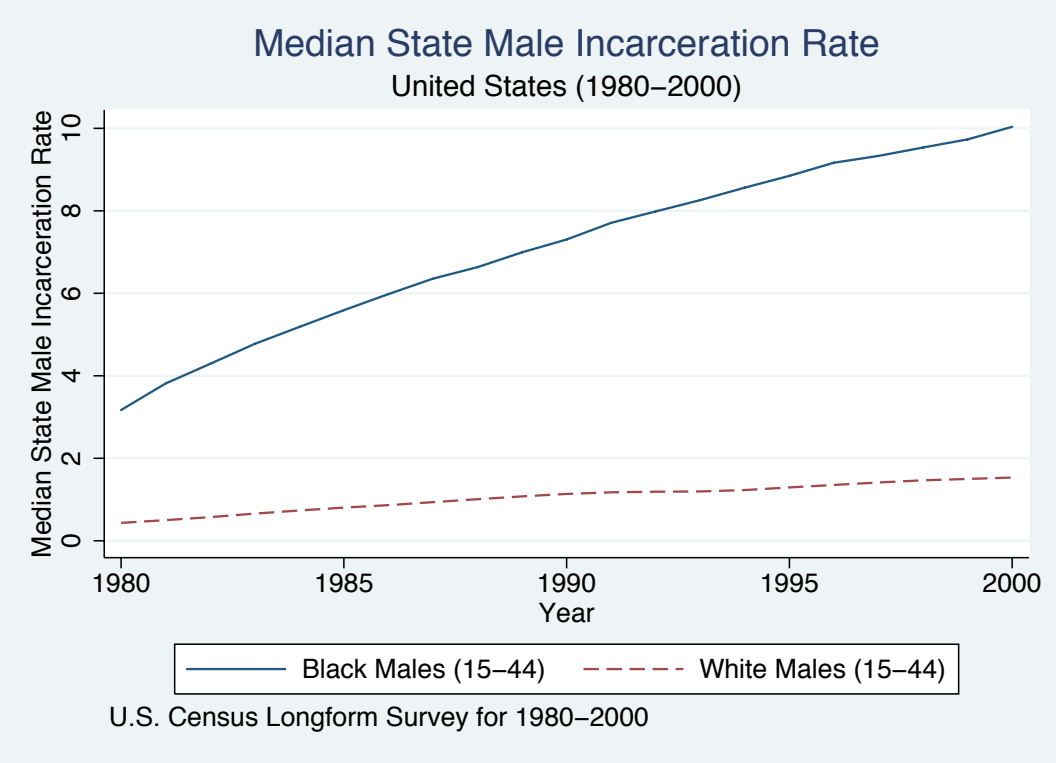

Figure 5.5: Male Incarceration Dynamics by Race using Linear Interpolation of 1980, 1990 and 2000 Census

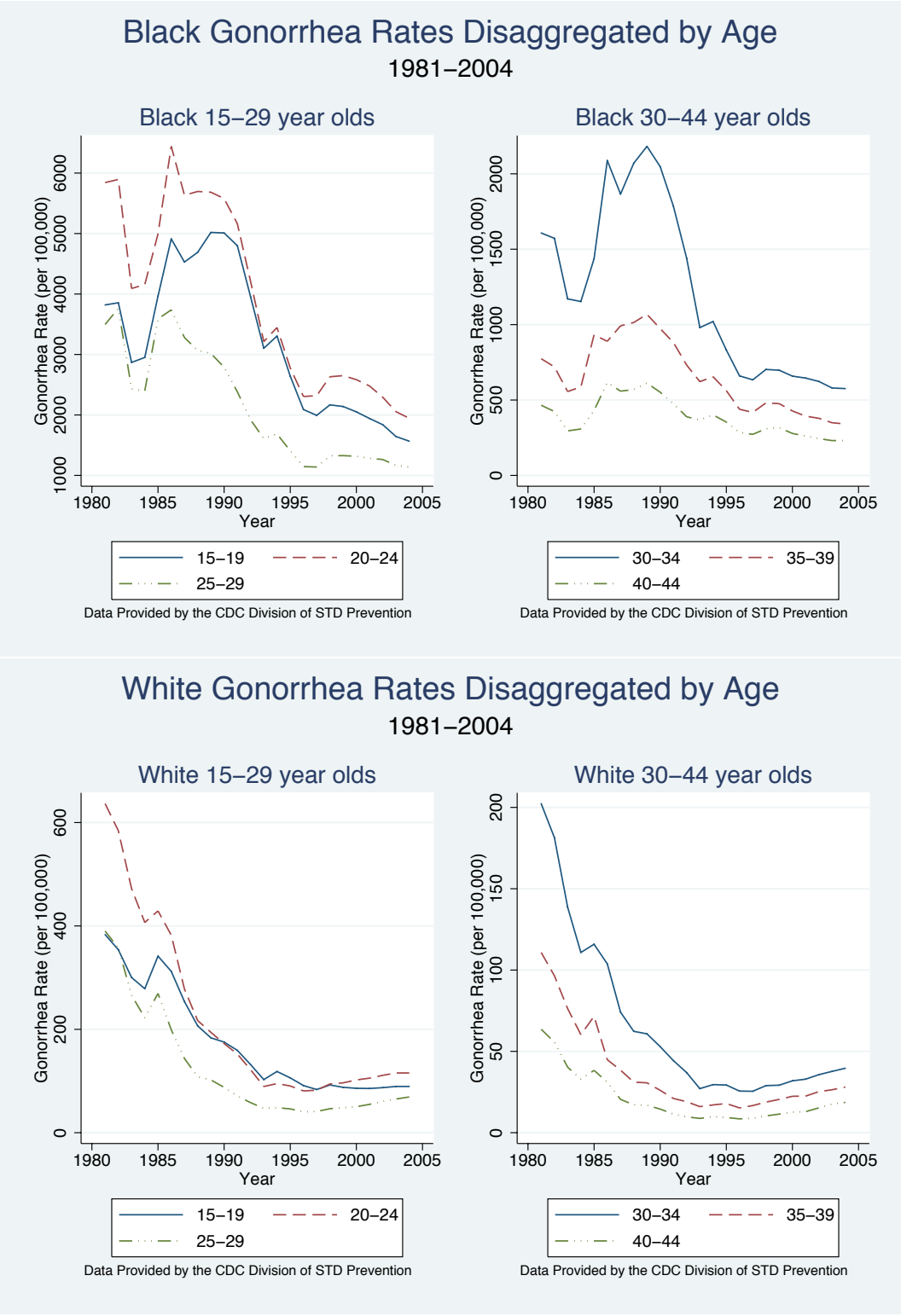

Figure 5.6: Gonorrhea Rates for Blacks and Whites for 1981-2004

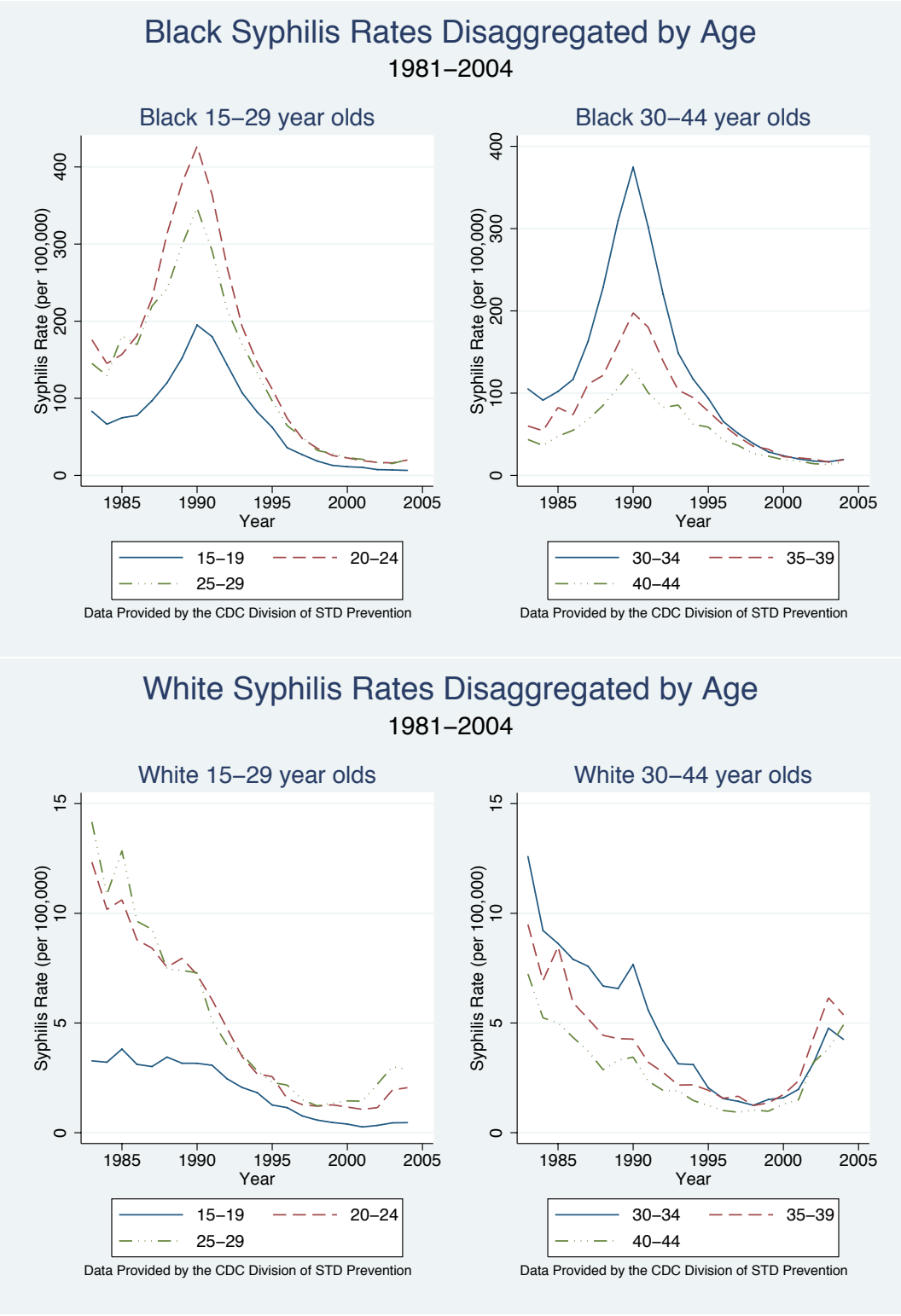

Figure 5.7: Black and White syphilis rates

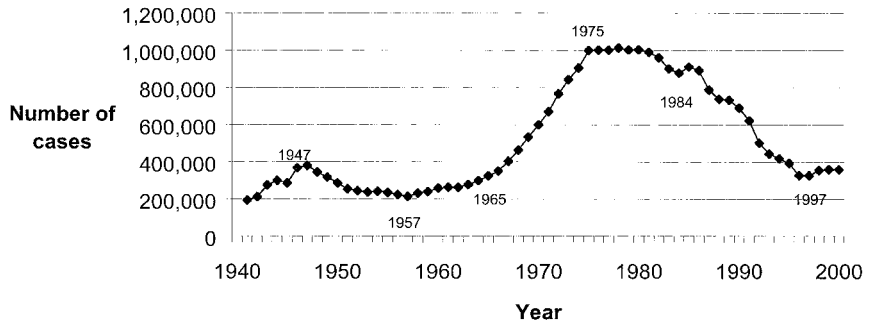

Figure 5.8: Total Cases of Gonorrhea in the US, 1941-2000, from Rothenberg and Potterat (2002)

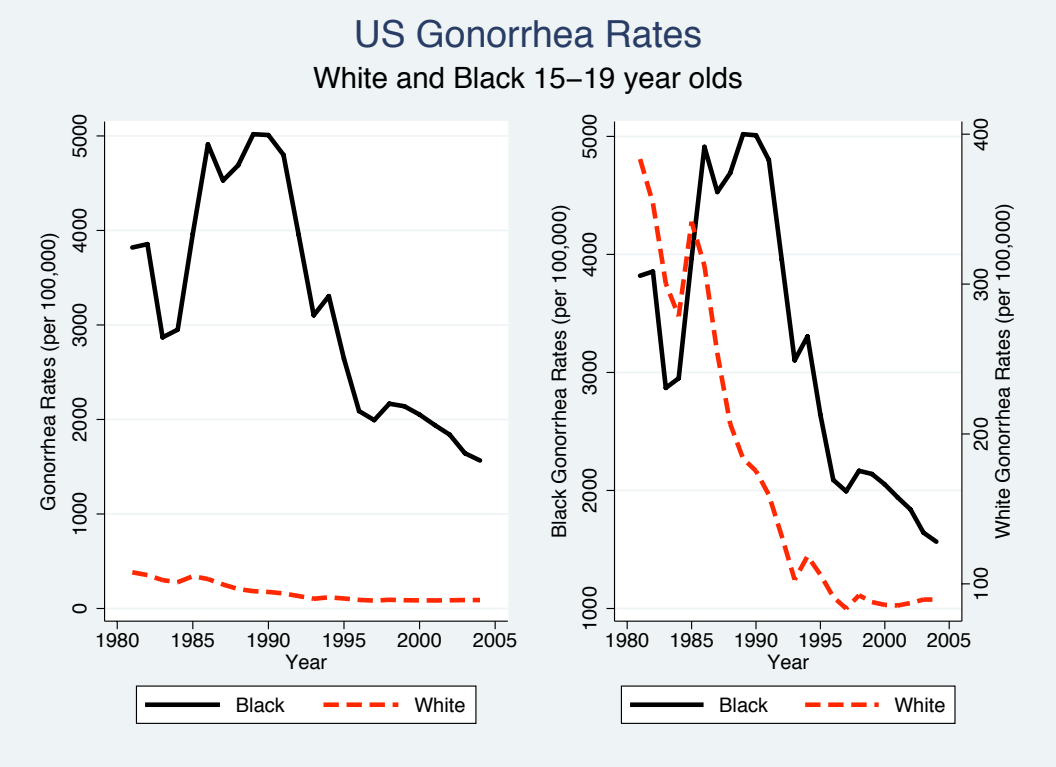

Figure 5.9: Black and White Gonorrhea Rates, 1981-2004

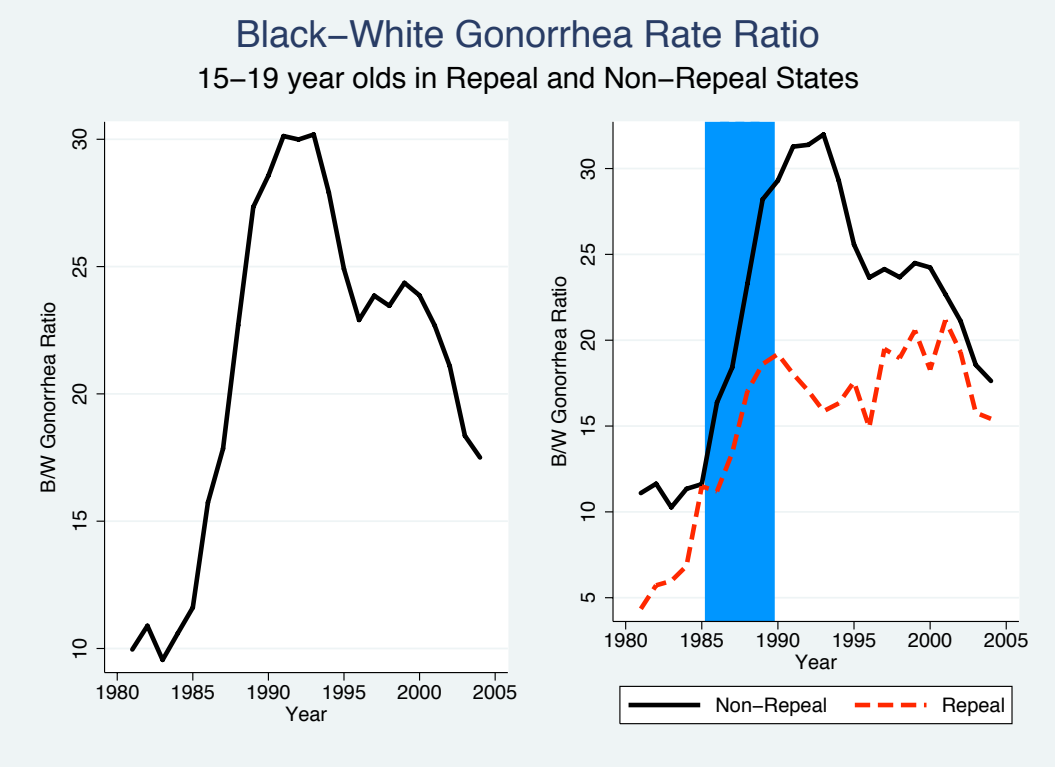

Figure 5.10: Black-White Gonorrhea Gap, 1981-2004. The shaded region represents the "treated" cohort entering the sample because of 1970 abortion repeal in the AK, CA, HI, NY, and WA.

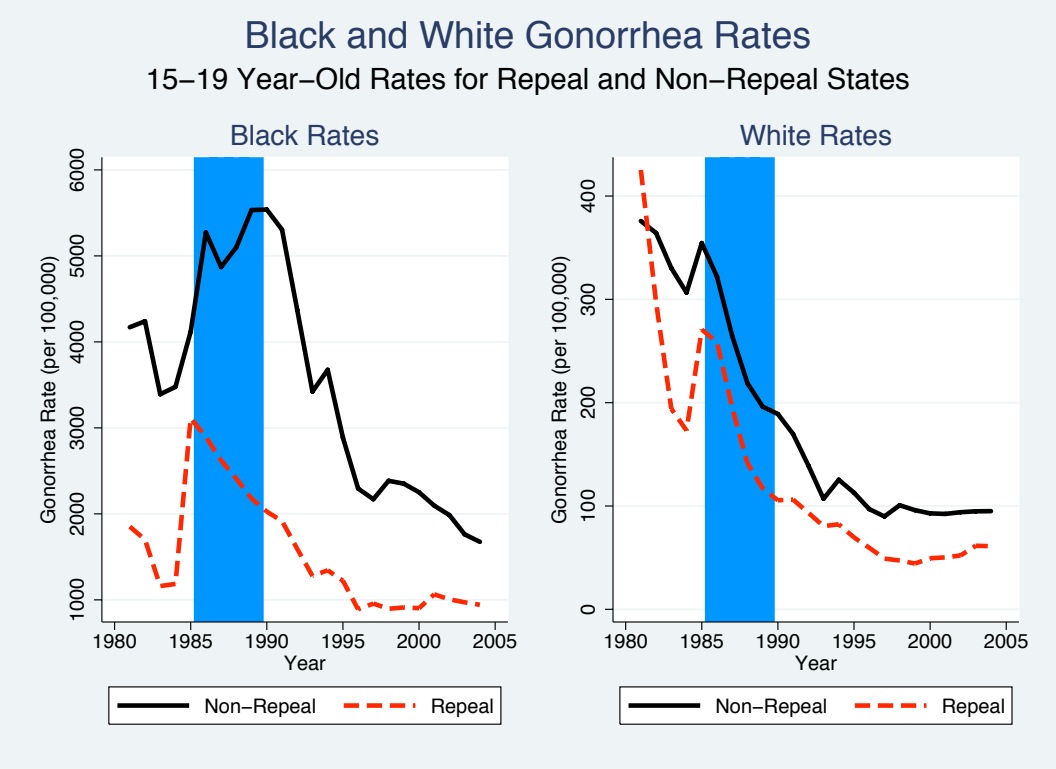

Figure 5.11: Black and White Gonorrhea Rates for Repeal and Non-Repeal States

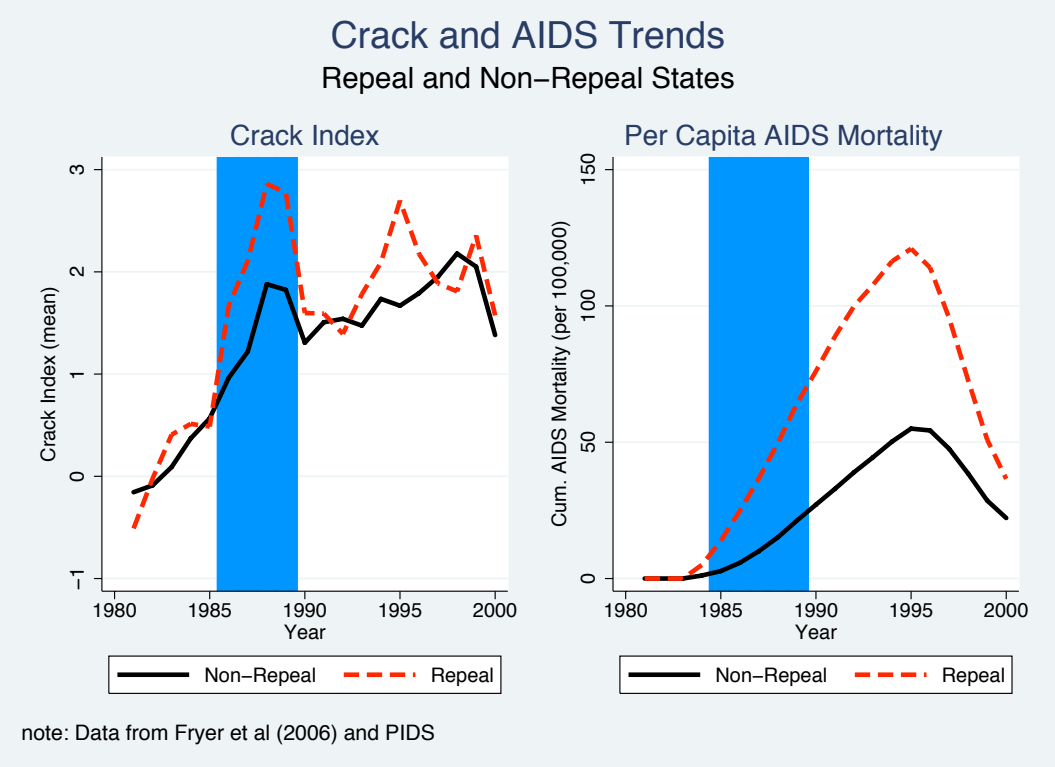

Figure 5.12: Mean Crack Index and Cumulative Per Capita AIDS Mortality Rates for Repeal and Non-Repeal States

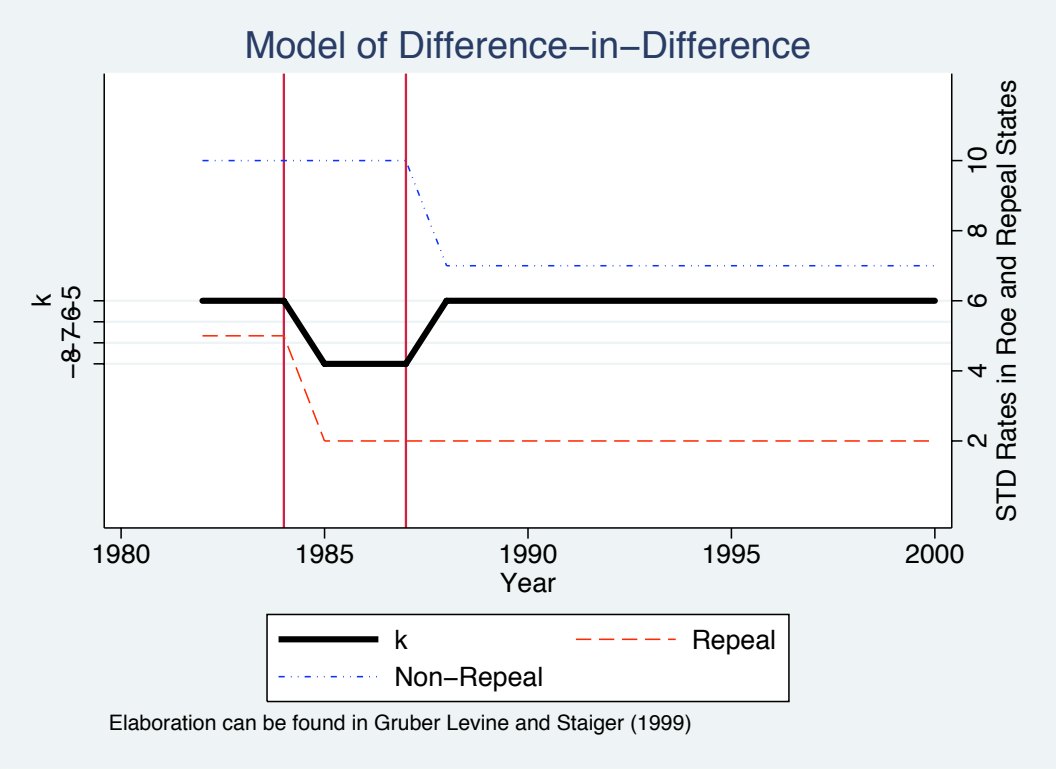

Figure 5.13: Diagram of the Difference-in-Difference Method

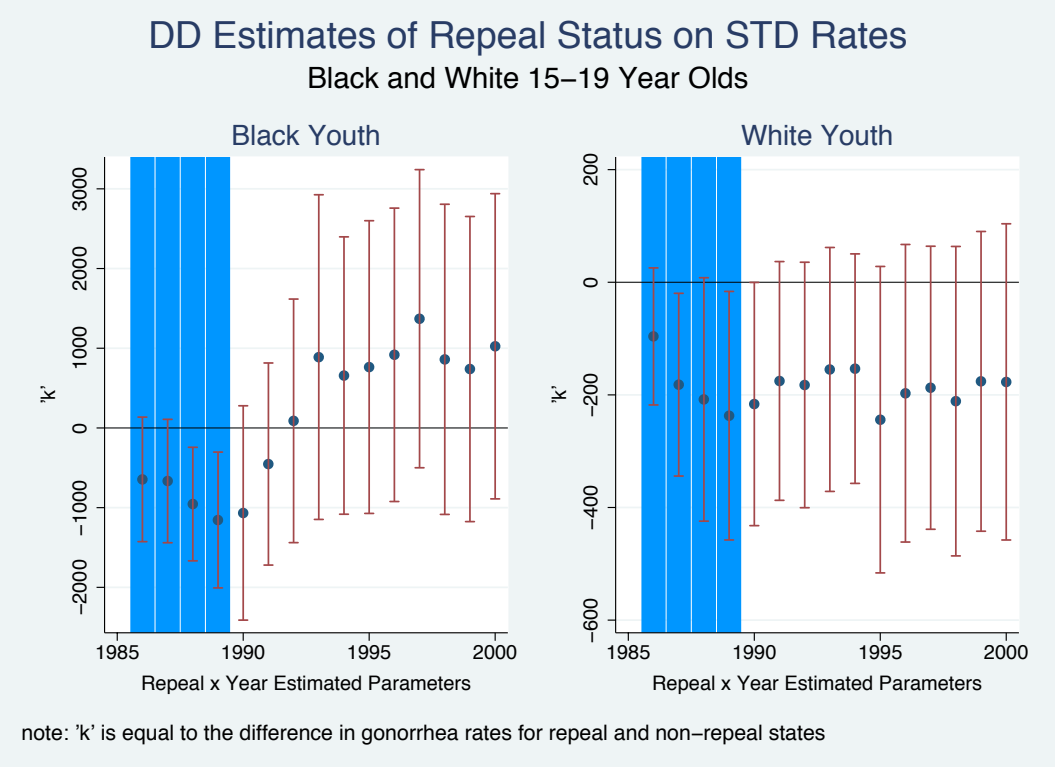

Figure 5.14: *Repeal* $\times$ D_t estimated coefficients from DD estimation (ie, columns 2 and 4 in Table 2)

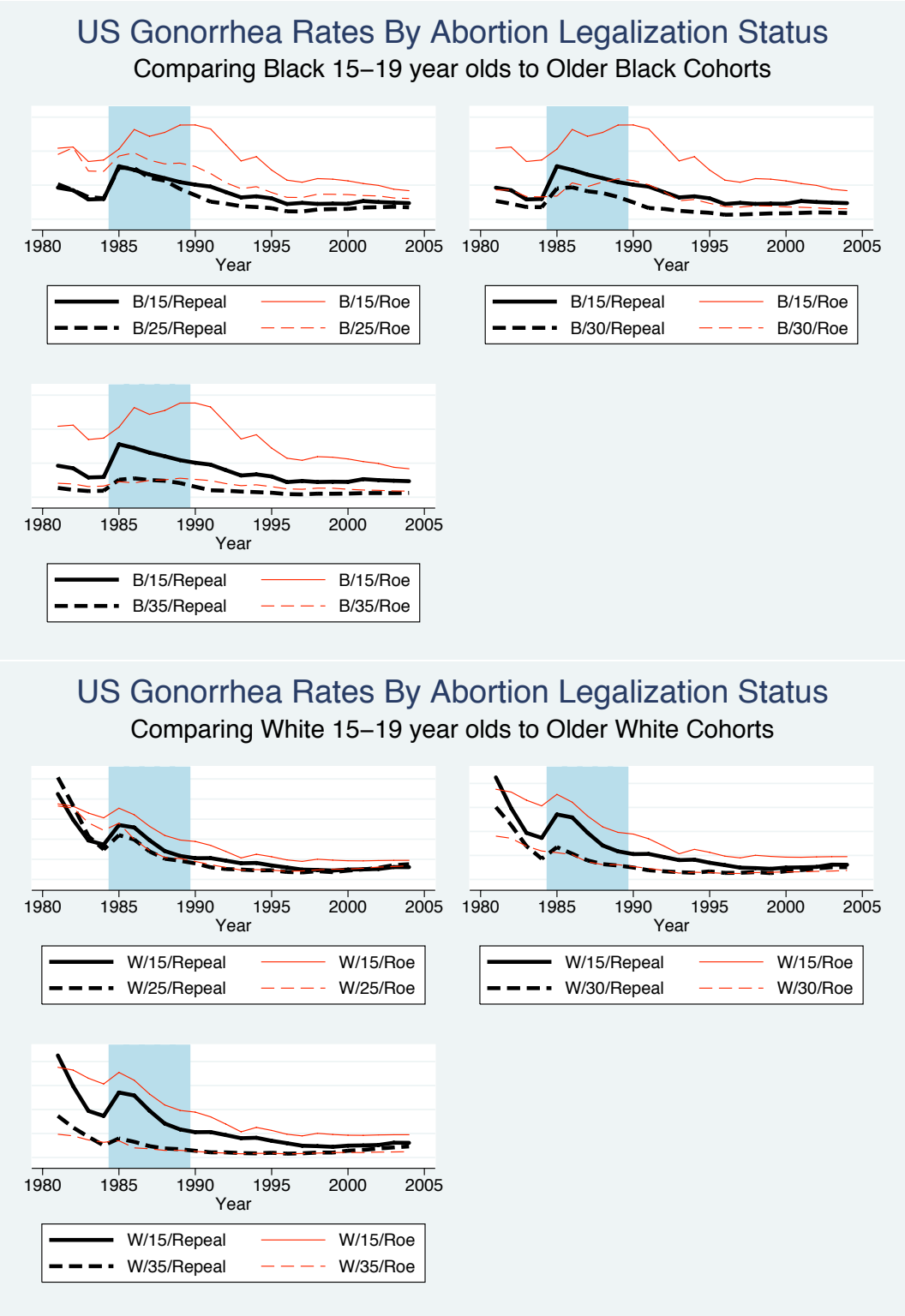

Figure 5.15: Bivariate Comparisons of Older Cohorts