The Effects of Male Incarceration Dynamics on AIDS Infection Rates among African-American Women and Men[*]

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Abstract

In this paper, we investigate the potential connection between incarceration dynamics and AIDS infection rates, with a particular emphasis on the black-white AIDS rate disparity. Using case-level data from the U.S. Centers for Disease Control and Prevention, we construct a panel data set of AIDS infection rates covering the period 1982 to 1996 that vary by year of onset, mode of transmission, state of residence, age, gender, and race/ethnicity. Using data from the U.S. Census, we construct a conforming panel of male and female incarceration rates. We use this panel data to model the dynamic relationship between the male and female AIDS infection rates and the proportion of men in the age/state/race-matched cohort that are incarcerated. We find very strong effects of male incarceration rates on both male and female AIDS infection rates. The dynamic structure of this relationship parallels the distribution of the incubation time between HIV infection and the onset of full-blown AIDS documented in the medical and epidemiological literature. These results are robust to explicit controls for (race-specific) year fixed effects and a fully interacted set of age/race/state fixed effects. Our results reveal that the higher incarceration rates among black males over this period explain the lion's share of the racial disparity in AIDS infection between black women and women of other racial and ethnic groups. The magnitude and significance of these effects persist after controlling for measures of crack cocaine prevalence and flow rates in and out of prison. In a separate analysis, we exploit the occurrence of system-wide state prison overcrowding litigation as an instrumental variable for the flow rate of prison releases. We find short-run increases in prison release rates that were induced by final court decisions on relief of prisoner overcrowding resulted in significant increases in subsequent AIDS infection rates among women and blacks, manifesting 5-10 years following the increase of prison releases.

I. Introduction

Coincident with the large increase in black male incarceration rates is a pronounced increase in AIDS infections among African-American women and men. Between 1970 and 2000, the proportion of black men incarcerated increased from 0.03 to 0.08, with a much larger increase in the proportion that has ever been to prison. There is no comparable increase among non-Hispanic white men. Concurrently, the HIV/AIDS infection rate among African-American women went from zero during the pre-epidemic period to an annual rate of 55 per 100,000 between 2000 and 2003, a figure nineteen times higher than that for non-Hispanic white women. For African-American men, this rate exceeds 100 per 100,000, in contrast to less than 15 per 100,000 among non-Hispanic white men. Moreover, African-Americans (12 percent of the overall population) accounted for half of the AIDS cases reported in 2002.

The sources of racial differences in HIV/AIDS infection rates are not well understood. Individual-level risk factors alone have proven inadequate to explain the substantial geographic heterogeneity in the diffusion patterns of the AIDS epidemic in the U.S. both between and within racial/ethnic groups. Researchers have yet to identify the mechanisms by which the AIDS epidemic transformed from one almost exclusively impacting young gay men to a disease that is increasingly transmitted through heterosexual sex and that disproportionately afflicts minority women.

In this paper, we investigate the potential connection between incarceration dynamics and AIDS infection rates. We estimate the effects of changes in male incarceration rates on male and female AIDS infection rates and assess the extent to which high levels of black male incarceration explain the black-white AIDS rate disparity. To our knowledge, this research represents the first systematic analysis of the relationship between incarceration and AIDS

infection rates using nationally representative population data. We hypothesize that changes in male incarceration rates alter HIV transmission risks within defined sexual relationship markets through a number of channels. In particular, male incarceration lowers the sex ratio (male-to-female), disrupts the continuity of heterosexual relationships, and increases the exposure of incarcerated men to high-risk sex amid a high HIV-prevalence population. All of these factors elevate an individual's or group's AIDS infection risk and should disproportionately affect the AIDS infection rates of black women and men.

Following Charles and Luoh (2005), we exploit the fact that the overwhelming majority of sexual relationships, as well as marriages, occur between women and men of similar age, race/ethnicity, and geographic location (Laumann et. al. 1994). To identify the effect of incarceration on AIDS infection rates, we exploit this social stratification and the tremendous variation in the incarceration trends over the past two decades within these groups. Accordingly, we use sexual relationship markets defined by age, state, race, and year.

We construct a panel data set of AIDS infection rates covering the period 1982 to 1996 that varies by year of onset, mode of transmission, state of residence, age, gender, and race/ethnicity. Using data from the U.S. Census, we construct a conforming panel of current and cohort-specific lagged male and female incarceration rates. We use this panel data to model the dynamic relationship between the male and female AIDS infection rates and contemporaneous and lagged changes in male incarceration rates within one's sexual relationship market. The impact of incarceration is identified from variation within sexual relationship markets over time.

To preview the results, we find very strong effects of male incarceration rates on both male and female AIDS infection rates. The dynamic structure of this relationship — i.e., the lagged effects of the proportion of males incarcerated — parallels the distribution of the

incubation time between HIV infection and the onset of full-blown AIDS, with small effects for early lags and relatively large effects for later lags. These results are robust to explicit controls for (race-specific) year fixed effects and a fully interacted set of age/race/state fixed effects. We find similar results in models estimated separately by racial group. The magnitudes of the results suggest that higher incarceration rates among black males explain the lion's share of the black-white disparity in AIDS infection rates.

To probe the robustness of these results to alternative hypotheses such as differential drug use, we explore several extensions of our base model. First, using state-year level data on AIDS infections and incarceration rates, we expand our base specification to include contemporaneous and lagged controls for the flow rates of inmates in and out of prison (i.e., correlates of prison turnover), as well as contemporaneous and lagged controls for crack cocaine prevalence. We find that crack cocaine prevalence has independent, significant effects on subsequent AIDS infection rates, but that the effects of incarceration rates remain significant and of similar magnitude with and without controls for cocaine prevalence. Regarding the flow variables, we find patterns consistent with an incapacitation effect of prison admissions on AIDS infections in the short-run (i.e., reduced community AIDS incidence in the year of an increase of prison admissions), followed by elevated community risks in the longer-run (i.e., accelerated community AIDS incidence in years after increased prison releases). However, adjusting for these flow measures does not impact our basic finding relating the scope of incarceration to infection rates.

Finally, in a separate analysis we explore the effects of statewide court-ordered mandates to relieve prison overcrowding on contemporaneous and future AIDS infection rates. Levitt (1996) demonstrates that relative prison population growth declines in years when state prison

systems are under order to relieve overcrowding. We find that this occurs primarily through a temporary increase in prison releases. Using a simple nearest-neighbor match for the five states experiencing such a court order during the AIDS epidemic, we find a spike in AIDS infection rates 5 to 10 years following such court orders relative to a group of matched comparison states. This spike is larger for women than men, large for African-Americans, and non-existent for whites.

These results taken together indicate that high levels of black male incarceration rates are a principal explanation for the relatively high rate of infection among black women and men.

II. Incarceration and HIV/AIDS Transmission Among Inmates and the Community

Our paper builds upon and extends two strands of literature. The first analyzes the intended and unintended consequences of incarceration. The second concerns the sources of racial/ethnic differences in HIV/AIDS infection rates and the divergent patterns that have emerged over the past two decades. At the nexus of these two topics, we explore the connection between AIDS, the number of men relative to women in sexual-relationship markets, and the incarceration rates of specific groups of men.

How, then, do changes in male incarceration rates affect the rate at which HIV/AIDS propagates through a given population? Perhaps the most mechanical effect may occur through the incapacitation of a group of high-risk individuals. To the extent that prisons remove from society those whose behavior accelerates the spread of infectious diseases, an increase in incarceration may reduce the overall incidence of HIV/AIDS. Indeed, the prevalence of HIV/AIDS among inmates is quite high[2], and many HIV positive inmates were infected prior to

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[2] Two to three percent of U.S. prisoners are HIV+, roughly five times the infection rate for the general population (Hammett et. al. 2002). Comparably high infection rates occur among prisoners in Western and Eastern Europe

the commencement of the current prison term. In addition, the profile of the average inmate clearly indicates that the incarcerated population is drawn from a sub-population at high risk of having HIV/AIDS.[3] Whether the incarceration of these high-risk individuals epidemiologically incapacitates them and reduces HIV/AIDS transmission is an unanswered question. However, there is ample evidence of an incapacitation effect of incarceration on crime (Levitt 1996, Raphael and Stoll 2005), and thus the proposition that similar effects exist for the transmission of infectious diseases is not implausible.

Nonetheless, several factors associated with serving time are likely to elevate the risk of infection while incarcerated. To the extent that prison independently elevates transmission risk, increases in incarceration rates may increase HIV infections among inmates as well as members of the communities from which they come and to which they return. We discuss these potential factors below.

Avenues by which prison may accelerate the transmission of HIV

The determinants of sexually-contracted HIV incidence (i.e., the number of newly-infected individuals in year *t*) is a function of: (a) the initial prevalence of the disease in the population, (b) the riskiness of the sexual activity involved, (c) the rate at which sexual relationships form and dissolve, and (d) the degree of concurrent sexual relationships in the population. An increase in male incarceration rates may affect HIV/AIDS infection rates among inmates and members of the community at large through each of these channels.

(WHO 2001). U.S. prisoners also have high rates of hepatitis B (Macalino et. al. 2004) and C (Hammett et. al. 2002, Macalino et. al. 2004).

[3] The typical inmate in the United States is relatively young, poor, minority, with very low levels of educational attainment (Raphael and Stoll 2005), and is likely to engage in risky sexual activity and drug abuse prior to becoming incarcerated (Swartz et. al. 2004).

First, the prevalence of HIV infections in U.S. prisons is particularly high.[4] Holding constant sexual practices, the relatively high HIV prevalence should directly translate into a higher rate of new infections, as sex partners encountered behind bars are more likely to be infected than non-institutionalized sex partners.

Second, transmission risk differs considerably by type of sexual activity, with male unprotected homosexual activity (the predominant sexual activity in U.S. prisons) being by far the most risky. Figure 1 displays the per-contact probability of HIV transmission by sexual activity, defined as the likelihood of acquiring HIV from having unprotected sex with an HIV infected individual.[5] As shown, anal receptive sex has an estimated per-contact probability of HIV transmission of 0.82%, while vaginal intercourse carries a female-to-male transmission probability between 0.01-0.03% and a male-to-female transmission probability of 0.05-0.09%.[6] Given the composition of sexual activity in prison, these statistics suggest that, holding frequency constant, HIV transmission rates should be higher in prison.[7]

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[4]The overall HIV prevalence is 2 to 3 percent among inmates but has been reported as high as 17.4 percent in some states (New York in 1993). Roughly one-fourth of those living with HIV in 1997 passed through a correctional facility in that year (Hammett et al. 2002). According to a U.S. Department of Justice report (1993, p.15), between November 1992 and March 1993, 11,500 AIDS cases were reported in Federal prisons, and the number of new cases grew at approximately 50 percent a year between 1994-1997. Thomas and Moerings (1994) give reports from a number of other countries that highlight HIV/AIDS in prison systems as a worldwide concern.

[5] These statistics are taken from the best estimates of the existing medical literature (Royce et al. 1997, Vittinghoff et al 1999, Downs and de Vincenzi 1996).

[6] This is average risk. Actual risk depends on the infectivity of the carrier, the condition of the immune system and genital tissues of the exposed person, presence of pre-existing other sexually transmitted disease, and possibly other factors.

[7] There are a handful of studies that attempt to measure the rate at which HIV is transmitted within prisons. One strand of this literature estimates the rate at which new inmates seroconvert (test negative upon entering prison and positive at a follow-up date) while incarcerated (Brewer et. al. 1988, Horsburgh et. al. 1990, Castro et. al. 1994, Macalino et. al. 2004). A second strand assesses the degree to which long-term prisoners that had been incarcerated since before the start of the AIDS epidemic become infected with the HIV virus (Mutter et. al. 1994, Krebs and Simmons 2002). The subset of these studies that tabulate annual transmission rates suggest transmission rates on the order of 0.1 to 0.5 percent per year served, a figure roughly 7 to 35 times the rate of transmission for the nation overall. A fundamental weakness of this evidence as a test of the extent of HIV transmission in prison is that only three state prison systems have mandatory HIV testing on entry and exit (none of which extant studies are based), so the estimates from other prison systems are vulnerable to the non-random selection process of who voluntary gets tested.

To put these relative transmission risks into context, we use these per-contact transmission probabilities to answer the following question: for an HIV negative male, how many unprotected sexual encounters with an HIV positive woman yields a transmission risk equal to that of one unprotected sexual encounter with an HIV positive man?[8] A simple computation[9] suggests that the HIV transmission risk associated with one unprotected male homosexual experience (via anal receptive sex) is equivalent to the risk of 85 unprotected heterosexual encounters with HIV+ women.[10] Thus, even if the frequency of sexual contact diminishes while incarcerated, the higher per-contact risk can potentially offset the effect of a reduction in frequency on the likelihood of becoming infected over a given time period. [11,12]

Third, the configuration of sexual relationship networks – who has sex with whom and over what time frame -- impacts transmission risk. Moreover, the common configuration of such networks in prison is likely to enhance the efficiency of HIV transmission. Sexual networks characterized by serially monogamous relationships provide a less efficient environment for a communicable disease to spread. On the other hand, network configurations where a small number of individuals have repeated sexual encounters with a large number of partners – either through rape, prostitution, or otherwise – more speedily connects members within a network

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[8] We assume sex is unprotected since the overwhelming majority of prisons and jails (95 percent) in the United States do not allow the distribution of condoms to inmates (Hammett 2004).

[9] We calculate this figure by solving the equation P(Infection|sex with HIV+ man)=1-[1-P(Infection|sex with an HIV+ female)]^N for N where N is the number of sexual encounters with and HIV+ woman.

[10] There is some evidence that the relationship between within-partnership HIV transmission and the number of sexual contacts with the partner may be highly nonlinear, increasing at a decreasing rate (Kaplan 1990).

[11] Research pertaining to the prevalence of sex in prison is quite imprecise, with estimates suggesting between 20 (Tewksbury 1989) and 65 percent (Wooden and Parker 1982) have sex while incarcerated. Evidence from the National Health and Social Life Survey (NHSLS), reveals that men who have ever served time in jail were 62 percent more likely to report "ever having sex with a man" (men with jail history=12.8 percent; men without a jail history=7.9 percent) (Francis 2005); and ethnographic evidence and prisoner surveys indicate the overwhelming majority of the first experiences occurred while incarcerated.

[12] The Bureau of Justice Statistics recently completed a national survey of administrative records on sexual violence in correctional facilities (as mandated by the Prison Rape Elimination Act of 2003). The estimates are likely to be conservative, however, since victims are often reluctant to file reports. In 2004, there were 8,210 sexual violence allegations in correctional facilities — 3.2 allegations per 1,000 inmates. See http://www.ojp.usdoj.gov/bjs/abstract/svrca04.htm.

through the high-risk central network member. Since few prison systems segregate HIV+ inmates or have mandatory testing on entry, HIV+ inmates may occupy central positions within their sexual risk networks (in part due to predatory patterns of victimization), which may help explain the relatively high, observed levels of infection among the incarcerated population.[13]

Thus, while reducing the aggregate exposure time of the non-incarcerated to the incarcerated, imprisonment may raise the AIDS incidence among those who are currently serving or have served time. Furthermore, as most inmates are returned to society after a relatively short prison spell, an accelerated transmission rate among inmates may spillover to the non-institutionalized population post-release.

A fourth avenue by which higher male incarceration rates may augment AIDS incidence is through the effect of incarceration on the number of lifetime sex partners and the likelihood of concurrency[14] (partnerships that overlap in time). To start, the dynamics of incarceration spells destabilize existing relationships. The typical U.S. prisoners serve relatively short spells (the median length is 2 years) followed by even shorter spells usually triggered by a parole violation (Raphael and Stoll 2005). Moreover, spells outside of prison are often punctuated by jail time while awaiting trial. These periodic absences from non-incarcerated partners are likely to result in the formation of new relationships by the partners left behind, as well as new sexual relationships among inmates, thus increasing the total lifetime number of partners. To the extent

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[13] Prior research indicates that the behavior of a small group of highly sexually active people is central to the spread of HIV as they contribute a disproportionate share of HIV incidence, and their behavior can fuel sustained transmission (Laumann 1994). Under the standard epidemiological susceptible-infected (SI) model, the growth rate of a new epidemic is proportional to $\left(\mu + \dfrac{\sigma^2}{\mu} \right)$, where μ and σ^2 denote the mean and variance of the number of partners per period (Anderson and May 1991).

[14] Concurrency involves having more than one sexual partner and going back and forth between them, a pattern that increases the probability of transmission as earlier partners can be infected by later partners. These factors are known to augment the risk of contracting and spreading sexually transmitted diseases as evidenced in Morris (1997), who showed concurrency alone can spur an epidemic even if the average number of partners is relatively low.

that these ancillary relationships continue after an inmate is released and returns to previous partners, the churning in and out of prison may augment the extent of concurrency.

A more subtle transmission mechanism may occur through the effect of incarceration on the relationship market conditions within which sexual partners match. Given that inmates are overwhelmingly male and minority, incarceration disproportionately reduces the ratio of minority men to minority women. This relative scarcity of minority men improves the bargaining position of non-institutionalized minority men in negotiating personal relationships.[15] This change in the terms of trade will decrease women's ability to be discriminating in partner choice and to negotiate safer sex practices, and may result in exposure to relatively more high-risk men. The improved terms of trade for men may also translate into men having the bargaining power to display less commitment or loyalty in seeking sexual relations, resulting in less stable relationships.

While there is little direct evidence of an effect of incarceration on relationship formations and concurrency operating through this channel, Charles and Luoh (2005) show that higher male imprisonment has lowered the likelihood that women marry, reduced the quality of their spouses when they do, and caused a shift in the gains from marriage away from women and towards men. In addition, low sex ratios have been shown to be associated with higher rates of single motherhood (Myers and Darity 1995), teen pregnancy (Sampson 1995), syphilis (Kilmarx et. al. 1997) and gonorrhea (Thomas et. al. 2003).

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[15] While market forces tend to stabilize the rate of exchange within a community, the social stratification of sexual relationships along race and class lines means that terms of trade will not be equal across relationship markets.

Can private behavioral responses undo the elevated risk associated with changes in male incarceration rates?

The pathways discussed above suggest a number of avenues by which increases in male incarceration rates may elevate the transmission rate (and thus incidence) of HIV among inmates as well as the non-institutionalized partners of inmates. To the extent that these factors disproportionately impact certain sub-populations of the United States (for example, minority women), prison provides a potential explanation for recent trends in AIDS infection rates.

However, one might argue that behavioral responses among those facing an elevation in risk may partially undo the effect of incarceration on the overall spread of the disease. The general epidemiological assumption that behavior is exogenous to environment can lead to erroneous conclusions about the cause of disease patterns, a point emphasized as a cautionary note in Kremer's (1996) work on AIDS. For example, an economic model of the spread of infectious diseases hypothesizes that individuals will alter their demand for risky activities as risk increases, holding all else constant (Philipson and Posner 1993, Francis 2006). In fact, there is some evidence that individuals do indeed take greater precaution in the face of greater risk. Ahituv, Hotz, and Philipson's (1996) find an increase in the demand for safer sex, such as the use of condoms, in response to the increase in the prevalence of AIDS.[16]

However, information asymmetries, driven by delays in onset and awareness of HIV prevalence, are likely to significantly limit the efficacy of this salutary behavioral response. Since the cost of sexual activity, in terms of the probability of infection, depends on the activity levels of the other individuals in one's relationship market (network), risky behavior by any

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[16] They find that, while there was no difference in condom demand among U.S. census regions in 1984, the incidence of condom usage became geographically heterogeneous as the AIDS epidemic progressed, with higher rates of condom utilization in states with higher AIDS prevalence rates. The authors also find more rapid growth in condom use among black men, single men, those in urban areas, and those who were more sexually active.

person within the market imposes a negative externality on all others. With asymmetric information regarding the sexual history of potential partners, it is impossible for individuals to accurately incorporate this higher cost into their decisions.

Moreover, if the behavioral response is heterogeneous and systematically related to sexual behavior, more conservative behavior by some may actually elevate the transmission risk faced by others. For example, Kremer (1996) shows that increases in the probability of infection from an additional partner will create incentives for people to reduce risky sexual activity, but greater proportional reductions in risky sexual activity by low activity people will worsen the composition of the pool of available partners.

Incarceration trends and racial differences in AIDS infection rates

The net effect of an increase in incarceration rates on AIDS transmission is thus theoretically ambiguous. In this paper, we do not attempt to separately identify structural estimates of the magnitudes of these individual mechanisms, but instead focus on uncovering the reduced-form net effect. The mechanisms noted above – incapacitation effects, elevated transmission rates while incarcerated, and effects of incarceration dynamics on the formation of new sexual relationships and concurrency – should disproportionately impact the African-American community in the United States. Roughly one-fifth of black adult males in the U.S. have served time (Raphael 2005), and many of these men have cycled in and out of correctional institutions for fairly long periods of their early adult lives. The ratio of men to women among the non-institutionalized is markedly lower for non-Hispanic blacks than for non-Hispanic whites (Adimora and Schoenbach 2005). Moreover, black women are nearly twice as likely to have recently had concurrent partnerships relative to white women (Adimora and Schoenbach 2005, Laumann and Youm 1999); and, on average, they have higher lifetime numbers of partners,

holding age constant (all factors that may result theoretically from high black male incarceration rates).[17] Whether these factors translate into greater AIDS infection rates among African-Americans is the question to which we now turn.

III. Empirical Framework and Data Description

With ideal data, we would model the effects of current and prior incarceration spells on the likelihood of becoming HIV positive among current and former inmates, as well as among men and women within the same sexual relationship markets as current and former inmates. Thus, one might model group-level HIV incidence in year t as a function of the proportion of men who have ever served time in jail or prison. In the parlance of the experimental literature, the "treatment" for incarcerated men can be viewed as exposure to a high-risk prison environment. For women, the treatment amounts to having men in the sexual relationship market exposed to higher transmission risks, and thus in turn, exposing their non-institutional female partners. Beyond these two effects, there still may be additional spillover effects on men and women who have never had sex with a current or former inmate, or who have never been to prison, via secondary infections from those who have. The possibility of these secondary infections coupled with a time-served delay between changes in male incarceration and changes in female HIV infections suggests a dynamic relationship between incarceration and HIV incidence with lagged effects of the former on the latter.

Unfortunately, representative data on HIV infections do not exist for the United States. However, nearly complete national data on the universe of advanced-stage HIV infections in the

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[17] Our tabulations of the 2001-2002 National Health and Nutrition Examination Survey Sexual Behavior Component revealed that black women on average have greater lifetime numbers of sex partners relative to white women. These differences are on the order of 20 percent and disappear for women over 50. We observed these differences for only younger cohorts.

United States is publicly available. Thus, in this paper we model the determinants of AIDS (or advanced-stage HIV) infections. One benefit of analyzing the determinants of AIDS cases rather than early-stage HIV (which is often asymptomatic) is that it minimizes the differences in reported rates across groups that are simply an artifact of differential interaction with the health care system, resulting in differences in early detection.

A further complication, however, is that the average lag for a model of AIDS cases is likely to be larger than the comparable lag for HIV infections due to reasons beyond the factors already noted. For both genders, variance in the AIDS incubation distribution – where incubation is defined as the time between HIV infection and the development of a measurably suppressed immune system – will induce a lag between any incarceration-induced infections and newly diagnosed AIDS cases. Estimates of the cumulative distribution function (CDF) of incubation for the pre-1996 period[18] reveal sharp increases in the proportion developing full-blown AIDS starting three years post seroconversion and a flattening of the CDF at around ten years post infection (Bacchetti 1990, Brookmeyer 1991, U.K. Register of HIV Seroconverters Steering Committee 1998).[19] Thus, our model specification must account for the likely long lag function relating changes in incarceration to changes in AIDS infection rates.

Following Charles and Luoh (2005), our empirical strategy builds on the fact that the overwhelming majority of marriages occur between men and women of similar age, race/ethnicity, and geographic location. Moreover, these endogamous patterns mirror the stratification of sexual relationships along these lines, thus creating sharp and distinct sexual

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[18] The AIDS incubation period was altered considerably by the introduction of antiretroviral drugs in 1996, with the variance increasing considerably along with the median and mean time to the development of symptoms. For this reason, the empirical tests below will focus on the pre-1996 period.

[19] These estimates suggest that roughly one-quarter of HIV-positive individuals develop AIDS within six years, one-half within nine years, and three-quarters within twelve years. For women who are at risk of infection via heterosexual relationships with former inmates, time served will also induce a lag between men becoming infected while incarcerated and the ultimate infection of female partners. There is evidence that people are most infectious in the first few months after becoming infected and again when the disease develops into AIDS (Jacquez et al. 1994).

relationship markets (Laumann et. al. 1994). We exploit this empirical regularity and the substantial variation in the incarceration trends over this period occurring within these demographic groups. Accordingly, we define sexual relationship markets by the interaction of race, age, and state of residence. We use the proportion of men incarcerated to capture the proportion of the relationship market's population at risk in a given year. We then model AIDS incidence as a function of contemporaneous as well as lagged changes in the fraction of men incarcerated at the relationship market level.

Specifically, our principal estimates come from estimation of the regression equation:

$$AIDSRate_{rsat} = \sum_{\tau=0}^{13} \omega_{m\tau} IM_{rsa,t-\tau} + \sum_{\tau=0}^{13} \omega_{f\tau} IF_{rsa,t-\tau} + \delta_{rsa} + \lambda_{rt} + \phi_{st} + \pi_{at} + \varepsilon_{Rsat}, \quad (1)$$

where r indexes racial/ethnic groups, s indexes state of residence, a indexes age groups, and t indexes year of infection. The variable $AIDSRate_{rsat}$ measures the number of new AIDS cases diagnosed per 100,000 individuals from race group r, age group a, in state s, during year t; $IM_{rsa,t-c}$ provides the male incarceration rate (defined as the proportion incarcerated at a point in time for the given year) for the demographic group rsa for the contemporaneous year of infection and for thirteen lagged years; $IF_{rsa,t-c}$ provides the comparable incarceration rates for women; δ_{rsa} denotes a complete set of sexual-relationship market fixed effects defined by the interaction of race, age, and state of residence; λ_{rt} denotes a complete set of race-specific year effects; ϕ_{st} denotes a complete set of state-specific year effects; π_{at} provides a complete set of age-specific year effects; and ε_{rsat} is the random error term. Finally, the parameters $\omega_{m\tau}$ and $\omega_{f\tau}$ provide the coefficients on the contemporaneous and lagged incarceration rates and provide the principal parameters of interest.

An important aspect of the specification of Equation (1) is the fact that we match the contemporaneous and lagged male and female incarceration rates to be cohort consistent. This means that we model AIDS infection rates for a specific gender and sex market as a function of the contemporaneous incarceration rates and the lagged incarceration rates for the specific group. Thus, the 1990 AIDS infection rate for 30 to 34 year old black women in Georgia is modeled as a function of the current incarceration rate for 30 to 34 year old black men in Georgia, the 1989 incarceration rate for 29 to 33 year old black men in Georgia, the 1988 incarceration rate for 28 to 32 year old black men, and so on.

The inclusion of sexual market fixed effects adjusts for time-invariant market-specific characteristics, such as drug use prevalence or behavioral norms—factors that are otherwise difficult to quantify. Allowing for race-specific, age-specific, and state-specific individual year effects controls for race- and age-specific trends that might exist in AIDS incidence at the national level, and overall trends that may vary by state. Collectively, the inclusion of the sexual market effects and the various time effects means that we are identifying the effect of incarceration on AIDS infection rates using variation in both series occurring within sexual relationship networks after accounting for race, age, and state-level time trends in both variables. We estimate equation (1) using weighted least squares, where we weight by the population size of each group defined by race, state, age, and year. Finally, to ensure that our statistical inferences are robust to serial correlation in the error term, we estimate the standard errors of the model by bootstrapping.

We estimate separate models by gender. Since sexually-transmitted AIDS infection is the hypothesized chief mechanism linking incarceration and AIDS, we also estimate the equation separately for new AIDS infections contracted through heterosexual (for women), homosexual

sex (for men), in addition to estimating models for overall AIDS infection rates. Given the high degree of correlation between current and lagged incarceration rates, we use a third-order polynomial distributed (Almon) lag for both male and female incarceration rates to reduce multicollinearity problems and yet allow a fairly flexible structure on the shape of the lag distribution. Our modeling of the lag structure is guided by the medical and epidemiological evidence regarding the pre-1996 incubation period (which suggests no more than two inflections in the incubation probability distribution function). We tested alternative lag lengths and higher-order polynomials, but none significantly improved the fit of the model.

We further constrain the lag coefficients to zero for those whose transmission effects correspond with time periods that predate the AIDS epidemic (i.e., before 1980). For example, for AIDS rates in 1985 we constrain all coefficients on lagged incarceration rates in excess of five years to zero; for AIDS rates in 1986 we constrain all coefficients on lagged incarceration rates in excess of six years to zero, and so on. These constraints essentially mean that later lags are identified using fewer years of data and AIDS infection rates occurring later in the panel. This specification of the distributed-lag model parallels that of Pakes and Griliches (1984) and Andrews and Fair (1992) in other applications.

The model in Equation (1) measures the effects of incarceration on AIDS transmission using variation within sexual relationship markets after netting out race-, age-, and state-specific time trends in both variables. Thus, any unobserved determinants of the incidence of AIDS that vary across but not within sexual relationship markets, or that drive year-to-year changes for specific racial groups, age groups, or states, are accounted for in this model specification. Nonetheless, there may be omitted variables that vary within the remaining slice of variation that we are using to identify the incarceration lag coefficients, such as changes in high-risk behavior.

Perhaps the strongest contender for a contaminating omitted variable is crack cocaine usage. There is ample speculation that the use of crack cocaine during the late 1980s and early 1990s increased the degree of concurrent sexual relationships, both due to pharmacological effects of the drug as well as users prostituting themselves for money to support their habits (Levenson 2004).

In addition, the model specified in Equation (1) does not allow for independent effects of turnover holding the scope of incarceration constant. For example, with a front-loaded infection hazard function in prison, higher turnover may yield higher infection rates, holding the overall incarceration rate constant. Moreover, our model in Equation (1) constrains the effects of incarceration to be constant over time. Following the presentation of the initial results for Equation (1), we will present some additional results using data varying at the state-year level that incorporates correlates of prison turnover as well as measures of crack cocaine usage. In these extensions, we will allow effects of incarceration to vary over the course of the AIDS epidemic. In addition, we will explore the effects of a plausibly exogenous shock to prison releases on subsequent AIDS infections.

A detailed discussion of our data is provided in the data appendix. Here, we provide a brief overview of our data sources and the manner in which we construct our panel. We use data from the 2001 CDC AIDS Public Information Data Set (PIDS) as well as the 1980, 1990, and 2000 five percent Public Use Microdata Samples (PUMS) from the U.S. Census of Population and Housing. The AIDS PIDS database provides case level information on all known AIDS cases measured by the national AIDS surveillance system. To construct AIDS infection rates, we first tabulate the total number of newly diagnosed AIDS cases by the state of residence, race, age, gender, and year of diagnosis. We then use data from the 1980, 1990, and 2000 Census

PUMS to estimate the national population corresponding to each state/race/age/gender/year cell for each census year and linearly interpolate population estimates for inter-census years. These two variables are then used to tabulate an AIDS infection rate per 100,000 individuals.

We employ four race/ethnicity categories: non-Hispanic white, non-Hispanic black, non-Hispanic Asian, and Hispanic. We use nine of the ten age groupings from the AIDS PIDS data.[20] The introduction and widespread use of medical therapies, particularly medical advances introduced since 1996, have significantly elongated the lagged structure of the relationship between incarceration and AIDS incidence. Thus, we focus on the period from 1982 to 1996.

One problem with the annual AIDS PIDS data disaggregated at the race-age-state level concerns the ability to identify the state of residence at the time of diagnosis. To protect confidentiality, roughly 15 percent of AIDS cases observed over this period lack state identifiers. Thus, the infection rates in our panel data set are estimated using only 85 percent of the total number of AIDS cases recorded in the U.S., encompassing data from 38 states plus Washington, D.C.[21] To make use of all cases, we also estimated the models below using the four-category region of residence to define geographic location. The results are qualitatively and numerically similar to what we present below. With fifteen years, 39 states, four race/ethnicity groups, and 9 age groups, the dimensions of the panel define 21,060 individual cells for each gender.[22]

Figures 2 and 3 present our estimates of the annual newly diagnosed AIDS cases (expressed per 100,000) for men and women for 1982 through 2001. The figure for men reveals

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[20] The age ranges describing each infected individual refer to age at infection and are 20 to 24, 25 to 29, 30 to 34, 35 to 39, 40 to 44, 45 to 49, 50 to 54, 55 to 59, 60 to 64, and 65 plus. We drop the 65 plus category since many of those 65 plus in the census defined as institutionalized are in nursing homes.

[21] The twelve states with missing disaggregated AIDS case-level information are Alaska, Iowa, Idaho, Maine, Mississippi, Montana, North Dakota, New Hampshire, South Dakota, Vermont, West Virginia, Wyoming. There are also missing state identifiers for some AIDS cases in small rural areas disproportionately in the South.

[22] For cells with a positive population's estimate and zero new AIDS cases, we set the AIDS infection rate to zero. After omitting those cells where the population estimates from the census are zero, there are 21,018 observations for men and women.

that black men are newly diagnosed with AIDS at a rate that is between three and nine times the comparable rate for white men (with the larger figures pertaining to the latter periods). The rate of new AIDS cases for black women is between 12 and 24 times the annual rate of new diagnoses for white women.

To estimate male and female incarceration rates, we use the group-quarters identifier included in the PUMS data. This variable identifies individuals residing in non-military institutions (inmates of federal and state prisons, local jail inmates, residents of inpatient mental hospitals, and residents of other non-aged institutions). We use this variable as our principal indicator of incarceration. The patterns of incarceration using the PUMS data on the proportion "institutionalized" is consistent with the aggregate information on incarceration from the Bureau of Justice Statistics (BJS) (Raphael 2005).[23]

For the census years 1980, 1990, and 2000, we measure the contemporaneous incarceration rate for each demographic group defined by state of residence, age group, race/ethnic group, and gender as the proportion of the members of the demographic cell that is institutionalized. For non-census years, we linearly interpolate the incarceration rate using the estimated rates for the two years bracketing the year in question.[24] Our model also requires that we estimate lagged incarceration rates for each demographic group defined by our panel data set. Using a procedure similar to that for estimating contemporaneous incarceration rates, we construct thirteen cohort-specific lagged incarceration rates, where the age bracket and year are adjusted for the lag length. Again, the details of this imputation are discussed at length in the data appendix.

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[23] Annual incarceration data disaggregated at the race-age-state level is not available in the BJS data.
[24] We have also estimated our models using the time path of the overall state incarceration rates between census years to non-linearly interpolate group-specific incarceration rates. The results using this alternative are similar to the results presented below.

IV. Descriptive Statistics and Preliminary Analysis

Our empirical strategy relies heavily on the variation occurring within relationship markets and how this within-group variation differs across groups. In addition, the model as laid out in Equation (1) imposes, a priori, several functional form restrictions. In this section, we provide a descriptive analysis of the data with an eye on demonstrating the great heterogeneity in the time path of incarceration and AIDS infection rates for our defined relationship markets. We also provide some simple descriptive analyses showing the within-group relationship between these variables, descriptive evidence pertaining to the dynamics of the relationship between incarceration and AIDS, as well as some simple falsification tests that probe the reasonableness of our specification choices.

Variation in incarceration and AIDS infections

Figure 4 displays key percentiles of the distribution of incarceration rates across states for men between the ages of 20-34. Table 1 presents key percentiles of the distribution of the total *change* in incarceration rates between 1982-1996 across states for men between the ages of 20-64 and 25-34, respectively. The levels in Figure 4 as well as the changes in Table 1 highlight the considerable geographic variation across states in incarceration levels and growth rates both between and within race among young men that occurred over this 15-year period. While incarceration among young men increased for all groups, young black men exhibited markedly higher increases, followed by young Hispanic men, and small increases for young white men.

Even within these groups, however, there is substantial variation in the levels and changes. For example, Figure 4 reveals that in 1996 the state-level incarceration rate for young black men varied from roughly 0.08 at the 10th percentile to 0.13 at the 90th. Moreover, the distribution of the changes in incarceration rates for young black men over the entire study

period reveals a change at the 90th percentile (7.2 percentage points) nearly 2.5 times the change at the 10th percentile. As it pertain to AIDS infections, the tails of these distributions are particularly important since a small minority of individuals who engage in high-risk behaviors (or are exposed to high-risk environments) contribute a disproportionate share of HIV infections.

Similar disparities as well as large cross-state variation are also evident in AIDS infection rates. Figure 5 displays mean AIDS incidence per 100,000 as well as key percentiles of the cross-state distributions of this variable by race for men between 30 and 44 years of age (the age groups with a particularly high incidence level at the height of the epidemic). Figure 6 presents comparable figures for women. By 1996, the average AIDS infection rate for black men in this age group was 280 compared to 50 and 160 for non-Hispanic whites and Hispanics, respectively. Similar, yet proportionally larger, racial disparities emerge for women, with overall averages of 125, 10, and 35 cases per 100,000 among black, white and Hispanic women, respectively. The tremendous cross-state variation in the extent of the AIDS epidemic (as evidenced by the difference between the 90th and 10th percentiles) within racial groups is equally striking. For example, in 1996 the infection rate for black men between 30 and 44 in the state at the 90th percentile of the cross-state distribution is nearly four times the comparable rate of the state at the 10th percentile. The comparable 90/10 ratio for black women 30 to 44 year of age in 1996 is nearly ten.

Figures 7 and 8 focus on sexually transmitted infection rates (as opposed to transmission via intravenous drug use). Comparing these figures to Figures 5 and 6 reveals that the largest component of the growth in the racial disparity in female AIDS infection rates resulted from infection occurring through heterosexual sex, while homosexually-contracted AIDS was a growing component of the black-white AIDS gap among men.

Correlations between AIDS infections and contemporaneous and lagged incarceration rates

We proceed with a simple graphical analysis of the relationship between changes in incarceration and changes in AIDS infection rates. We present simple contemporaneous and lagged correlations and provide a first-pass analysis of the timing of the relationship between change in incarceration and change in AIDS infections.

To do so, we divide the 15-year period spanning 1982-1996 into three 5-year periods of the epidemic: 1982-1986—the early years; 1987-1991—the middle years; and 1992-1996—the peak years. For each period and each sex market (defined by race, age and state) we calculate the total number of new AIDS cases (per 100,000) over the five-year period as well as the corresponding five year change, the one-period lagged five year change, and the two periods lagged five year change in male incarceration rates. The one- and two-period lagged changes are calculated for the same cohort – i.e., when the individuals were five and ten years younger, respectively.

Figure 9 presents scatter plots of cumulative new male infections per 100,000 over the five year peak period (1992 through 1996) against the contemporaneous, once-lagged, and twice-lagged change in incarceration rates. Figure 10 presents the comparable scatter plots for women. For both male and female AIDS infection rates, there is no visible relationship between contemporaneous changes in incarceration and AIDS infections. However, we find that the lagged five-year changes in incarceration rates are more strongly associated with changes in the number of new AIDS cases than are the contemporaneous five-year changes.[25] We find the strongest effect of incarceration rates in the two-period lagged change.

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[25] For both men and women, the slope coefficients on the contemporaneous and lagged incarceration changes are statistically distinguishable from one another at the one percent level of confidence.

The larger effects of changes in lagged incarceration rates are most clear in the regression results presented in Table 2. Here the dependent variable is the cumulative number of new AIDS infections over five year periods for the latter two periods (1987 through 1996), while the explanatory variables include the contemporaneous and the once-lagged five-year changes in male incarceration simultaneously. For the male AIDS infection rate model, we find no significant effect of the contemporaneous five-year change in male incarceration, yet strong significant effects of the one-period lag change. The same holds true when the dependent variable is changed to female AIDS infection rates. These patterns are quite suggestive of incarceration-induced infections, in that we should not expect to see a contemporaneous effect since few HIV-positive transmissions develop into full-blown AIDS in the first several years after acquiring HIV. We should, however, see effects emanating from changes in male incarceration that occurred in previous time periods; and indeed, that is the pattern of effects we find.

This simple exercise suggests two falsification tests we can perform that should yield no evidence of incarceration effects, assuming these relationships are not spurious. The first involves testing whether lagged increases in *female* incarceration rates are independently associated with female AIDS infection rates. The rationale for this test builds on the fact that the transmission risk faced by imprisoned women should be negligible and thus the incapacitation effect should clearly dominate. Therefore, if one were to find significant positive effects of female incarceration rates on AIDS infections, one might be concerned that changes in incarceration may serve as a proxy for changes in drug use or some other behavior.

This first specification test is displayed in columns (2) and (4) in Table 2. While the principal test involves the model where the female infection rate is the dependent variable, we

present results for male AIDS infections as well. The contemporaneous change in female incarceration rates exhibits a negative and significant effect on the female AIDS infection rate while the once-lagged change is statistically insignificant. Note, the effects of the male incarceration variables on female infections are robust to inclusion of these additional variables. In the male infection rate model, the female incarceration variables are both statistically insignificant, while the results for the male incarceration variables are not altered by their inclusion.

The second falsification test considers whether lagged changes in incarceration rates that correspond to periods that predate the AIDS epidemic (for our purposes, pre-1982) are associated with subsequent increases in AIDS infection rates. For the early and middle periods of the epidemic, we can define lagged five-year changes in incarceration rates that pre-date the AIDS epidemic. We should see no significant effects of changes in incarceration rates during these periods (occurring before 1982) and significant effects of changes in incarceration rates occurring during the epidemic (1982 and later). Note, since we constrain the effects of incarceration rates pre-1980 to zero in our specification of Equation (1), this also serves as a specification check for our more detailed model results presented below.

Since we cannot estimate incarceration rates for the 1970s due to data constraints,[26] here we use data from the Bureau of Justice Statistics to measure the contemporaneous and lagged changes in incarceration rates. Again, we use five-year cumulative AIDS cases, but measured at the state level rather than the state-age-race level. We use the same three five-year time periods (early, middle, and peak) and define three time periods over which changes in incarceration rates are measured: contemporaneous (t-5 to t), once lagged (t-10 to t-5), and twice lagged (t-15 to t-

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[26] The 1970 PUMS is a one-percent sample, less than one fifth the size of the 1980 sample. Incarceration rates at the sex-race-age-state level are quite imprecise for many groups. For this reason, we focus on state-level data here.

10). We define the time dimension as $t=$(*early, middle, peak*), and the geographic location

dimension as *s* corresponding to state. We use these data to estimate the model

$$
\begin{aligned}
ChangeAIDS_{st} = {} & \alpha_{st} + \chi * early_{st} + \phi * middle_{st} + \beta * \Delta Incarceration_{st}^{contemporaneous} + \\
& \delta * \Delta Incarceration_{st}^{oncelagged} + \gamma * \Delta Incarceration_{st}^{oncelagged} * early_{st} + \\
& \kappa * \Delta Incarceration_{st}^{twicelagged} + \pi * \Delta Incarceration_{st}^{twicelagged} * early_{st} + \\
& \lambda * \Delta Incarceration_{st}^{twicelagged} * middle_{st} + \varepsilon_{st}
\end{aligned}
\tag{2}
$$

where *early*$_{st}$ and *middle*$_{st}$ are dummy variables corresponding to the early and middle periods of

the epidemic. The regression specification allows the effects of lagged incarceration rates to

differ when the change occurs during the pre-AIDS period. For example, the effect of the once-

lagged change for the early period of the epidemic (a change in incarceration rates corresponding

to 1977 to 1981) is given by the sum of the coefficients δ and γ, while the once-lagged effects for

the middle and peak periods is given by δ alone. The effects of the twice-lagged changes in

incarceration rate are given by $\kappa+\pi$ for the early period, $\kappa+\lambda$ for the middle period, and κ for the

peak period. Our specification/falsification test involves testing whether the once-lagged for the

early period as well as the twice-lagged effects for the early and middle periods are zero. To

isolate the tests of the significance of changes in incarceration rates during pre-AIDS period, we

impose two constraints on the model in Equation (2) to simplify the specification. First, the

contemporaneous effects are constrained to be equal across the three periods. Second, the once-

lagged effects are constrained to be equal for the middle and peak period.

Table 3 presents estimates of the contemporaneous, once-lagged, and twice-lagged

incarceration effects for the three five year periods. Coefficients corresponding to pre-AIDS

epidemic periods are bolded. The results show an insignificant once-lagged effect of a change in

incarceration for the early period while the once-lagged effect is positive and significant for the

middle and peak periods. The results also show insignificant twice-lagged effects for the early

and middle periods, with a significant and large twice-lagged effect for the peak period. Thus, we find no evidence of significant effects of changes in male incarceration rates occurring during the pre-epidemic period on later AIDS infections. However, we find large and significant effects of changes occurring during the AIDS epidemic. These dynamic patterns and specification checks lend support to our specification of the dynamic model in Equation (1).

V. Empirical Results from the Dynamic Regression Models

In this section we present various estimates of the dynamic model of AIDS transmission presented in Equation (1). Our goals are two-fold: first, we aim to estimate the overall dynamic relationship between incarceration rates and AIDS infection rates among men and women; second, we wish to use these results to provide a statistical accounting of the fraction of the racial differences in AIDS infection rates attributable to differences in incarceration rates.

Controlling for incarceration and the overall race/ethnic differences in infection

Tables 4 and 5 present preliminary estimates of the lagged effects of incarceration on AIDS incidence using a restrictive version of the model in Equation (1). Table 4 models the AIDS infection rate for men. For each dependent variable, the table presents two specifications: (1) a model including race, year, state, and age effects, and (2) a model with all of these fixed effects plus the contemporaneous and thirteen years of lags of the incarceration rates for men and the comparable incarceration rates for women. For each model, we display the results for the race coefficients and the male incarceration variables only to conserve space.

Regression (1) indicates an average black-white AIDS disparity of 87 cases per 100,000 over the course of the panel. The comparable Hispanic-white and "other race"-white differentials are 21 and –35, respectively. Controlling for incarceration rates substantially

reduces the black-white difference, eliminates the Hispanic-white differences, and slightly widens the "other race"-white difference in infection rates. There are no measurable effects of contemporaneous incarceration rates, while the magnitude of the lagged effects increase with time (becoming significant at lag four, reaching a maximum at lag year 10, and remaining significant through lag 13).

Regressions (3) and (4) reproduce these models for homosexually-contracted AIDS incidence. The race effects presented in regression (3) are considerably smaller than the effects presented in regression (1). This is consistent with the fact that transmission through homosexual contact is a proportionally less important avenue of transmission for black men relative to white men. Nonetheless, the average annual infection rates for black men are considerably higher than those for white men (by roughly 29 per 100,000) while the transmission rates for Hispanic men are slightly lower. For the black-white difference, controlling for incarceration reduces the coefficient on the black dummy from roughly 29 to -14, while for the Hispanic-white difference adding incarceration rates widens the negative differential.

The lag coefficients on the male incarceration rates parallel those in regression (2) with two important differences. First, the magnitudes of the lag coefficients are considerably smaller. Second, the contemporaneous incarceration effect is positive. Given that only a small fraction of those who contract HIV develop AIDS within the same year of being infected, any contemporaneous effects are likely to be driven by something other than transmission while incarcerated. For example, a contemporaneous effect may be indicative of an effect of wide-scale testing of the incarcerated on the number of new diagnoses.

Table 5 presents comparable regression results for women. For AIDS cases transmitted by any source, there are large average racial/ethnic differentials in the annual average infection

rate. The black-white difference in incidence for women is on the order of 31, the Hispanic-white differential is approximately 10, while the other-white differential is approximately -3. These absolute differentials are smaller than those observed for men, reflecting the relatively lower infection rates among women. The inclusion of the male incarceration rate variables completely eliminates the positive black-white and Hispanic-white differentials in infection rates, while not affecting the other-white differential. Concerning the lag coefficients on male incarceration rates, there is no measurable effect of the contemporaneous incarceration rate and lagged effects that increase monotonically with the lag length.

The results in regressions (3) and (4) modeling heterosexually transmitted infections are similar. Adding the incarceration variables to the specification again eliminates the black-white differential and the Hispanic-white differentials in these variables. In fact, the black-white differential becomes negative and significant, suggesting that holding incarceration rates constant, black women are infected at a lower rate than white women. The shape of the lag function is similar to that observed for the model using the overall AIDS infection rate, although the coefficients are smaller.

Allowing for sexual relationship market fixed effects

Table 6 presents estimates of the lagged effects of male incarceration rate on AIDS infection rates for men (first column) and women (second and third columns) using the full specification from Equation (1). Each model in Table 6 includes the male and female contemporaneous and lagged incarceration rates, a complete set of fixed effects for race/state/age groups (our sex market fixed effects), and race-specific, age-specific, and state-specific year effects. For the male infection rate model, we only report the coefficients on the contemporaneous and lagged male incarceration rates. For the female infection rate model, we

report the coefficients for both the set of male incarceration variables and the set of female incarceration variables.

For the male infections model, the parameter estimates of the lag coefficients are quite similar to the parameter estimates from the lag coefficients using the somewhat restrictive model in Table 4. There is little evidence of a positive contemporaneous effect of incarceration on male AIDS infections, or of effects of the first three lags. The lag coefficients become positive and significant at the 4th lag, increase through the 10th year (the 11th in the third specification), and decline thereafter. Similarly, the results for women are not appreciably altered by the inclusion of the more liberal set of fixed effects. The lag structure using this more complete specification is nearly identical to that from the restrictive model presented in Table 5. Interestingly, we find little evidence of positive effects of contemporaneous and lagged female incarceration rates on female AIDS infections. We do find significant negative effects of the latter lags (lags 11 through 13), suggesting that holding male incarceration rates constant, increases in female incarceration rates actually reduces subsequent AIDS infections among women. These results are consistent with the more informal falsification test presented in the previous section.

The lag structures revealed in Tables 4 through 6 (as well as in the less formal graphical analysis of the previous section) suggest that the effects of male incarceration on AIDS incidence do not surface for several years and increase considerably over a ten-year period for men and over at least a thirteen-year period for women. As we have already discussed, factors that may drive these delayed responses include delays between prison entry and infection, the known incubation delay between seroconversion and becoming severely immuno-compromised, as well as time delays between male prison admission, female infections, and any other secondary infections that may occur. Thus, for both men and women, the expected patterns of the lagged

effects of incarceration would parallel the incubation distribution of the disease, but with additional delays. In other words, the lag structure should peak later than the peak in the incubation distribution due to factors that cause delay between an increase in incarceration rates and a new HIV infection.

To assess whether this is the case, Figure 11 plots the lagged coefficients on male incarceration rates from the models in Table 6 along with two alternative estimates of the probability distribution functions of the incubation period between seroconversion and the onset of AIDS. The first incubation distribution is calculated using the United Kingdom AIDS registry and pertains to HIV infections in the U.K. occurring prior to 1996 (U.K. Register of Seroconverters Steering Committee 1998).[27] The second incubation distribution estimate comes from an analysis of the incubation period among homosexual men in San Francisco during the pre-1996 period (Bachetti 1990). Based on both incubation period distribution estimates, the probability of becoming advanced-stage HIV (following seroconversion) increases in each of years one through seven, reaching a peak likelihood in the seventh year, and declining thereafter. By comparison, the lagged effects of male incarceration on overall AIDS infections for men follow a similar shape, though delayed an additional four years (with a peak at the 11th lag). For women, the delay appears to be greater, as the lag coefficients increase through the thirteen-year period suggesting a maximum effect beyond the lag length allowed in our panel regressions.

Simulating the effect of racial differences in incarceration on AIDS infection rates

The results in Tables 4 and 5 indicate that racial differences in incarceration rates largely explain the sizable overall black-white differential in annual AIDS infection rates in models where the racial differential is constrained to being constant through time. However, the more

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[27] The figure in the graph smooths the raw estimate of the pdf reported by the U.K. Register of Seroconverters Steering Committee using a third-order polynomial regression of the infection probability on the time since seroconversion.

complete model specification results in Table 6 allow for a more detailed decomposition of the time path of this differential. Using these latter models, we simulate the counterfactual black-white difference in AIDS infection rates that would have occurred had black male incarceration rates equaled white male incarceration rates. We do so by subtracting the predicted AIDS differential caused by male differences in incarceration rates from the overall black-white difference in AIDS infection rates.[28]

Figure 12 displays the actual black-white differential in overall AIDS incidence among men along with the predicted black-white differentials after accounting for black-white differences in male incarceration rates. Figure 13 presents the comparable series for women. Figure 12 reveals that racial differentials in incarceration rates explain little of the racial differentials early in the course of the epidemic, but account for a proportionally increasing share as we progress through the time period of the panel. In the latter years of the panel, racial differences in incarceration rates account for between 70 and 100 percent of the black-white differences in AIDS infection rates. For women, Figure 13 reveals that accounting for the effect

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[28]To illustrate this decomposition, here we present a simplified version of Equation (1). Suppose that the AIDS infection rates depends on a set of sexual relationship market fixed effects, and race-, age-, and state-specific year effects, and the contemporaneous incarceration for males only (the decomposition can be easily extended to the dynamic model we estimate in Tables 4 through 6). In other words, we would estimate the equation

$AIDS_{rast} = \alpha_{ras} + \gamma_{rt} + \delta_{at} + \theta_{st} + \beta MI_{rast} + \varepsilon_{rast}$. Taking expectations of this equation conditional on

race=B and t=t_0 and allowing the subscript, Bt_0, to denote this conditional expectation gives the expression

$AIDS_{Bt_0} = \alpha_{Bt_0} + \gamma_{Bt_0} + \delta_{Bt_0} + \theta_{Bt_0} + \beta MI_{Bt_0}$, where the first fixed effect is the average network effect for

blacks, the second effect is the black time effect for the given year, the following fixed effect is the average age-time effect for blacks, and the remaining provides the average state effect for blacks. If we take a similar expectation for whites and subtract this expectation from that for blacks, we get the final expression

$AIDS_{Bt_0} - AIDS_{Wt_0} = (\alpha_{Bt_0} - \alpha_{Wt_0}) + (\gamma_{Bt_0} - \gamma_{Wt_0}) + (\delta_{Bt_0} - \delta_{Wt_0}) + (\theta_{Bt_0} - \theta_{Wt_0})$

$+ \beta(MI_{Bt_0} - MI_{Wt_0})$. The first term in the decomposition provides the average black-white differential for the

whole panel, the second difference provides the additional period specific difference, the third term provides the differential attributable to difference in the age distribution, while the next term provides the portion of the differential attributable to difference in the geographic distribution. The final term provides the portion of the difference in AIDS infection rates attributable to racial difference in incarceration rates. The tabulations in figures 12 and 13 show the overall unadjusted differential (the left hand side of this equation) as well as the overall differential less the final component of the decomposition due to incarceration.

of racial differences in male incarceration rates yields negative black-white differentials in overall AIDS infection rates. In other words, the model predicts that, if black male incarceration rates had been at the lower level experienced by whites, black women would have been infected with AIDS at a rate that fell short of that for white women between 1982 and 1996.

VI. Probing the Robustness of the Results

Thus far, we have documented strong partial correlations between the rate at which men and women become infected with full-blown AIDS and the lagged values of the incarceration rate for males in one's demographic group. These correlations persist when we focus only on variation occurring within sexual relationship markets over time and after removing race-, age-, and state-specific year-to-year changes in both AIDS infection and incarceration rates. These partial correlations are highly significant and the implied lagged effects of incarceration parallel estimates of the pre-1996 AIDS incubation period distribution. Moreover, the effect sizes suggest that much of the racial differential in AIDS infection rates are attributable to historical differences in the rates at which black men are incarcerated.

In this section, we probe the robustness of our results to a number of our specification choices. While we discuss many specification tests, we present results for only a select set in order to conserve space.

- **Robustness to the linear interpolation of incarceration rates:** As noted above, in constructing our panel, we linearly interpolate contemporaneous and lagged incarceration rates for non-census years. We re-estimated all of these results using a panel where incarceration rates in non-census years are estimated using the time path of state-level incarceration rates (taken from Bureau of Justice Statistics (BJS) data) to non-parametrically apportion the decade-to-decade change across inter-census years.[29] The correlation between the linearly interpolated incarceration rates and this

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[29] For example, if the aggregate incarceration pattern in Minnesota increased linearly between 1980 and 1990, we would linearly interpolate for non-census years for the detailed demographic sub-groups in Minnesota; but if New Jersey's incarceration time pattern instead exhibited nonlinearities, increasing at a decreasing rate during the decade

more flexible alternative interpolation of non-census years was high at 0.93. The results from these models were nearly identical to the results presented above. We also estimated our models using current and lagged incarceration data varying at the state-year level, which are not linearly interpolated. These results were also very similar and will be discussed in greater detail below.

- **Race/ethnicity-specific estimates of the lag functions:** Equation (1) constrains the effects of incarceration on AIDS to be similar across race groups. In Table 7, we relax this constraint. The table presents results from models where the dependent variables are now race/ethnicity-specific. The only notable departures from the constrained results presented in Table 6 are the contemporaneous effects and the somewhat weaker lagged effects for white women. The lagged effects are particularly strong for white, black, and Hispanic males, and all minority female groupings presented. The lagged effects are quite close to the constrained results used to simulate racial differences above.

- **Allowing the effects to vary by age group:** We estimated separate models where the effects were permitted to differ between younger (less than 45) and older groups, but were constrained constant across racial groups. We find somewhat larger male incarceration effects on AIDS incidence for younger age groups. This result is to be expected since the rate of partner change, which affects the transmission speed of HIV, is greater at younger ages.

- **Allowing for alternative cross-gender age matching:** We re-estimated the incarceration rate lag functions using alternative panel data sets where female race/state/age groups were matched to men from similar race/state groups but who were either older (by one or two five-year age groups) or younger (by one or two age groupings). The rationale of this test is that since sexual transmission is the primary pathway linking male incarceration dynamics to female AIDS infection rates, we would not expect increases in young men's incarceration rates to have large consequences for much older women's or much younger women's AIDS incidence. Indeed, this is the case, as we found much smaller and, in most cases, insignificant effects of male incarceration rates on female AIDS infection rates for incompatible relationship age-matched groups (results available upon request). The largest male incarceration effects were found on female AIDS incidence in the same five-year age range matched group.

- **Testing the stationarity of AIDS and prison population data:** We use annual state-level data in incarceration rates from the BJS and state-level AIDS incidence to test for unit roots in both the AIDS incidence and incarceration rate time series. OLS regressions performed on non-stationary data series can yield spurious results unless the trend is removed by direct subtraction or by differencing. The unit root tests, which include state-specific time trends, show that these series appear to be stationary or I(0) processes. We also used the state-level BJS data to re-estimate the model in

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of the 1980s, then we would use that structure to connect the end points of the demographic sub-groups that reside in New Jersey.

first-difference form. The first-difference form of the model estimates the annual *change* in the state's AIDS incidence rate on 13-year distributed lags of annual *changes* in the state's incarceration rate—in other words, the model estimates the effects of an increase in incarceration on the acceleration in the growth of AIDS cases distributed over the subsequent 13-year period. The first-difference results again show significant lagged effects: increases in incarceration rates accelerate the growth rate of AIDS infections, with peak acceleration in years 7 and 8 following the incarceration rate increase (these results are available from the authors upon request).

- **Altering sample periods, lag lengths, and order of the Almon lag:** We examined the sensitivity of the results to modest changes in the choice of the analysis period and lag length, and allow higher-order polynomials of the lag structure for the Almon lag. The results from these checks were not fundamentally altered from the qualitative patterns of results reported in the paper.

- **Allowing for time-varying effects of incarceration:** Another potential threat to uncovering unbiased estimates of the effects of incarceration dynamics on AIDS infection rates stems from the fact that the strength of any underlying relationship between the probability of acquiring HIV and the proportion who has ever served time in prison may change as the AIDS epidemic progresses. In particular, we expect any relationship between HIV incidence and prison population size to grow stronger over time, as the prevalence of HIV increases in the population. This resulting lack of stability in the strength of the relationship could lead to biased estimates of the dynamic structure linking incarceration rates and AIDS, since the later lagged incarceration coefficients are identified disproportionately from the most recent observation years on AIDS incidence. We examine this issue directly by extending our primary models to allow incarceration effects to vary linearly over the course of the epidemic. We found some evidence of positive interaction effects, suggesting that these effect sizes have increased over the course of the epidemic.

- **Exploring whether male incarceration has a measurable effect on female AIDS transmission via intravenous drug use:** As a final robustness check, we explored whether male incarceration rates have any measurable effect on HIV infections among women occurring via intravenous drug use. The causal factors that we have discussed that link male incarceration to female AIDS infections are primarily sexual. While one can argue that prison-induced AIDS infections among men may lead to higher prevalence among non-institutionalized IV drug users, this path of infection is less direct. Thus, one might expect to see smaller effects of changes in male incarceration on changes in female AIDS infection through IV drug use relative to infections occurring through heterosexual sex. We explored this possibility by estimating separate models for female AIDS infections contracted through heterosexual sex and through IV drug use. We present a sub-set of these results (for black women) in Table 8. In this table, we allow the incarceration effects to vary by time and specify the model so that the base effects pertain to the year 1996. Thus, the results reported in Table 8 are the estimated lagged effects in the final year of our panel (the year when the effects should be the largest). The results reveal once again

a lag structure of male incarceration effects that mirrors the incubation period distribution with an additional 2-3 year lag for heterosexually-contracted AIDS infection rates; but interestingly, we do not observe comparable statistically significant lagged effects on black women's AIDS infection rates contracted via intravenous drug use. This lends some support that changes in intravenous drug use prevalence within relationship markets do not appear to be driving these results.

VII. Extending the Model to Incorporate Prison Turnover and Crack Cocaine Usage

Our estimation results thus far have modeled AIDS incidence among specific demographic groups as a function of the fraction of the males in the groups' cohort that are currently serving time or who have served time in the past. While we have demonstrated a robust relationship between these sets of variables, one can easily think of some potentially important extensions of this basic empirical model.

First, our specification does not allow for an independent effect of prison turnover on AIDS infections, a potentially important complication. Prison populations can expand in two ways: (a) a larger proportion of convicted offenders may be sent to prison, or (b) the sentences given out may be increased in length. The effects of each of these sources of increases in prison population size on a community's HIV incidence/risk need not be of the same magnitude or even have the same sign/direction. For example, increasing the time served of the current stock of inmates will, all else equal, increase the size of the prison population. Such a change should reduce the AIDS infections among women as current high-risk inmates are kept out of society for a marginally longer time. Conversely, reducing incarceration rates via early releases may increase AIDS infections among the non-institutionalized via the reverse effect.[30]

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[30] The effect on male infections is theoretically ambiguous and depends on the shape of the infection-time-served hazard function. If inmates are most at risk when first entering prison, extending sentences may have little affect on infection. On the other hand, if this hazard function increases with the length of time served, longer sentences may result in more male-to-male transmission of the disease.

On the other hand, expanding incarceration rates along the extensive margin may have a short-term incapacitation effect on the disease but may elevate transmission rates in the long term. The long-term effect would occur through the exposure of a greater proportion of men to a high risk prison environment, a factor that should elevate transmission rates among men and possibly that from men to women post release.

For community HIV infection risk, we expect the consequences of releasing one inmate early to differ from that of contracting the scope of incarceration to exclude one additional inmate. In particular, the early release may be of an inmate who has already experienced an elevated risk of acquiring HIV due to imprisonment, whereas reductions in the scope of incarceration may expose fewer men to transmission risk behind bars. On the other hand, the "new" inmate is likely to have faced a higher probability (relative to the general population) of acquiring and transmitting HIV due to risky behaviors prior to imprisonment. Whether the stock of prisoners matters more than the flow rates in and out of prison for HIV risk in the short and longer-run is an empirical question.

An additional factor that we have yet to control for (that some have argued helped propagate the AIDS epidemic throughout the black community) is the introduction of crack cocaine. Emergency room admission statistics suggests that the use of crack cocaine in American cities began in earnest between 1984 and 1987 (Grogger and Willis 2000); the precise time when HIV infections were on the rise in African-American communities. In his ethnography of the AIDS epidemic in the black community, Levenson (2004) intimates that promiscuity and unprotected sex are integrally related to the crack cocaine trade, as users trade sex for crack or for money to buy crack and may be affected through a psycho-pharmacological effect of the drug itself. Moreover, the introduction of crack cocaine has been linked to a

number of negative outcomes, including homicide rates, and infant mortality (see Blumstein 1995, Fryer et. al. 2005).

Here, we make use of incarceration rates and AIDS infection rates measured at the state-year level to extend the model specification in these directions. Analysis of state-level data permits inclusion of a measure of crack cocaine prevalence tabulated by Fryer et al. (2005), as well as inclusion of annual prison admissions and prison release flow rates in addition to overall incarceration rates (all three variables from the BJS). In addition to expanding the model specification to incorporate these additional control variables, we also allow the incarceration effects to vary over the course of the epidemic. Finally, the state-level data do not require linear interpolation of incarceration rates for non-census years and permit inclusion of all AIDS cases in the dependent variable (i.e., we do not lose the roughly 15 percent of cases due to confidentiality restrictions).[31]

Table 9 presents the results for this model. For each of the key explanatory variables, the table includes contemporaneous measures and 13 lags. Given the high degree of correlation between current and lagged incarceration rates, we again use a third-order polynomial distributed (Almon) lag for both incarceration rates and prison flow rates to reduce multicollinearity problems and yet allow a fairly flexible structure on the shape of the lag distributions. The three sets of prison variables are all interacted with a linear time trend. The coefficients for these variables reported in the table pertain to the lag function as of the year 1996. Beginning with the results for the prison incarceration rates, we find a pattern very similar to that of our results using the sex-market based panel. There is no significant contemporaneous effect of incarceration, while there are significant incarceration effects after lag five, reaching a peak in lag year eight,

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[31] The AIDS PIDS provides total counts by state for overall AIDS cases by gender and by race. The data do not include counts by race interacted with gender.

and remaining significant through lag eleven. The results for prison admissions and releases suggest that holding the scope of incarceration constant, releasing an additional offender results in more AIDS infections six to thirteen years later, while admitting an additional offender reduces AIDS infections seven to thirteen years later. In other words, while expanding the scope of incarceration increases AIDS infections, we also find evidence that incarceration has an incapacitation effect on AIDS infection transmission risk, holding the level of incarceration constant.

Regarding crack cocaine, we do find several significant coefficients for crack cocaine usage. However, the pattern of the lag function is not consistent with the incubation distribution of HIV. There is a positive and significant contemporaneous effect of the crack index and significant positive effects for the first through sixth lags. However, we also find negative and significant effects of the tenth through thirteenth lag. This pattern casts some doubt on the hypothesis that crack cocaine explains the rise of AIDS infections among minorities in the U.S. Nonetheless, our principal results withstand controlling for this factor.

VIII. Using Prisoner Overcrowding Litigation to Identify Exogenous Variation in Releases

Our results thus far have relied on an identification strategy that makes use of within-group variation in incarceration and AIDS infection rates. An alternative strategy for estimating the causal relationship between prison and AIDS would be to identify sources of exogenous variation in incarceration, and use this variation to estimate the effects of changes in incarceration on current and subsequent changes in AIDS infection rates.

Ideally, we would like to have exogenous variation in new admissions (or shifters of the scope of incarceration) as well as exogenous variation in releases. As our previous discussion

indicated, incarceration rates can be increased (or decreased) by changes along the extensive or intensive margins of the prison population, and such changes are likely to have different impacts on overall AIDS infection rates. Unfortunately, we have only been able to find a plausible source of exogenous variation in prison releases. This precludes us from examining the effect of changes in the scope of incarceration on infection rates and limits our analysis to the potential incapacitation effects of extending or shortening the sentences of those already incarcerated. Nonetheless, this analysis permits a direct documentation of the connections between incarceration dynamics and AIDS infection. For this reason, this exercise is meant to complement the model results presented above.

We present a simple non-parametric analysis of the effect of an exogenous increase in prison release rates on subsequent AIDS infection rates. Any factor that increases prison release rates (effectively shortening the time served of some) will reduce the incarceration rate. The effect of this change on AIDS infections will depend on the time path of infection transmission while incarcerated. It is plausible that the HIV transmission hazard is highest early in prison spells due to, for example, a declining likelihood of being victimized with time served (due to learning or forming protective social networks in prison, and/or negative duration dependence). If this is the case, then for incarcerated men, shortening sentences should not significantly reduce infections among male inmates. However, shorter sentences will expose non-institutionalized women and, via secondary infections, non-institutionalized men to high-risk inmates for a longer time period. Thus, under these conditions, one would expect an increase in releases to increase AIDS infections among women and have theoretically ambiguous effects for men.

On the other hand, if the transmission risk increases with time served, shorter sentences may reduce infections among inmates. For non-institutionalized women, the effect would be

theoretically ambiguous, as the lower infection rate among released men is in part offset by the greater exposure of these women to relatively high-risk men.

We use the five states identified by Levitt (1996) where (1) the state department of corrections was placed under court supervision to relieve prisoner overcrowding, and (2) the decision placing the corrections systems under court supervision occurred during the period within which the AIDS epidemic was well on its way.[32] We match each of these five states to three comparison states using the average incarceration for the five-year period preceding the year of the court decision, the average prisoner admission and release rates over this period, and the changes over this period in each of these three variables.[33] We then define a time scale where $t = 0$ corresponds to the year that the state corrections departments among the treatment states are placed under court supervision.[34] We provide a simple comparison of AIDS infections for the four years preceding the year of the final court decision, the year of the court decision, and the following post-decision decade. Table 10 displays the five treatment states, the years when each state is placed under court order, as well as the three comparison states identified through the matching procedure.

Levitt (1996) demonstrates that the growth in incarceration rates declines (in the short-run) in states with correction systems placed under court order, relative to all other states. Table 11 shows that for the five treatment states, this occurs primarily through increases in the annual flow rate of prison releases. The table displays average annual incarceration rates, admission rates, and release rates for year –4 through –1 (the pre-period), 0 through 3 (the final decision

[32] Levitt (1996) identifies twelve states that were sued for prison overcrowding and that were subsequently placed under court supervision. The final court decisions for six of these states predate the AIDS epidemic. For one state, the decision occurs at the start of the epidemic, and is thus excluded.
[33] We match the five treatment states to the three nearest neighbors where the between-observation distance is a function of the weighted sum of the difference in the six matching variables. The imbalance in the covariates is weighted inversely by the variance in the explanatory variable observed for all 50 states.
[34] Note, treatment states are matched exactly to comparison states by year.

period), 4 through 7 (the first post-period), and 8 through 10 (the second post-period). While the pre-decision incarceration rates were higher on average by 62 per 100,000 among treated states relative to the comparison states, this differential narrows to roughly 43 for years 0 through 7 and then widens thereafter. However, the treatment-comparison differences in admission rates are approximately constant through the final decision period. We do observe a relative spike in average annual releases corresponding to the time the state was placed under court order. Thus, the table suggests that the lull in the growth in incarceration is driven primarily by the early release of offenders in order to satisfy the court order to relieve overcrowding.

We first explore the effects of this exogenous shock to the flow rate of prison releases on all AIDS infections. Figure 14 presents the average annual AIDS incidence for the treatment states and the comparison states. The AIDS infection rates among the treatment states charts closely with the AIDS infection rates for the comparison states up through the third year following the final court decision. In years four through ten, however, AIDS infections increase in the treatment states above those observed for the comparison states. These annual differentials are on the order of 3 to 5 additional AIDS cases per 100,000 residents per year. Thus, the figure suggests that the early releases of offenders created a subsequent spike in AIDS cases among treatment states.[35]

Figures 15 and 16 present similar comparisons for AIDS infections rates among whites and blacks. Given the relatively high rates of incarceration among black men, one would expect a disproportionate impact of prison releases on AIDS infections among African-Americans. Indeed, this is what the data suggest. The AIDS infection rates for whites in the treatment states

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[35] The overall shape of the AIDS infection-time profile reflects the shape of the course of the AIDS epidemic more generally. Since three of the states have decision years in the early 1990s, the increase in the first 6 or 7 post years and the subsequent decrease reflect the peak of the AIDS epidemic around 1996 and the subsequent decline in new infections.

are consistently below those for the comparison states and charts their movements quite closely. The AIDS infection rate among blacks in treatment states follows a similar path to the comparison states through the three years following the final court decision. Black infection rates in treatment states then spike relative to comparison states in the fourth year and remains above the comparison states thereafter.

Figures 17 and 18 present similar results for men and women. For men, we observe only a slight up-tick in relative infection rates in treatment states four years after the final court decision. Among women, relative infection rates in states placed under court supervision increase in the first year following the decision. The treatment-comparison differential increases through year 8 and declines thereafter.

To assess whether the post-decision disparities in AIDS infection rates are statistically significant, Table 12 presents a comparison of annual AIDS infection rates for the treatment and comparison for the four time periods analyzed in Table 11. For each time period, we calculate the average difference in annual infection rates and test the significance of this difference. We also present a regression-adjusted difference estimate, where comparison-specific fixed effects are included in the specification.[36] For overall AIDS infections, the treatment-comparison difference in AIDS infections increases from –1.31 in the pre-decision period, to roughly 3.4 in the last two periods analyzed. These latter differentials, however, are not statistically significant. Among whites, the treatment-comparison differentials are consistently negative and generally insignificant.

Annual infections per 100,000 are lower in treatment states for blacks during the pre-decision period and the period encompassing the court decision. However, the relative infection

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[36] Since the five states are treated in four different years (corresponding with the year of the final court decision), the comparison fixed effects capture differences in time periods as well as the composition of comparison states.

rate differential increases to 16.73 in the first post decision period and 11 in the second. For the first post-decision period, the unadjusted differential is significant at the ten percent level, while the adjusted differential is statistically significant at the five percent level of confidence. For the second period, the adjusted differential is statistically significant. Finally, while there are no statistically significant treatment-comparison differences among men, among women the treatment-comparison difference in AIDS infection rates goes from small and insignificant in the pre-period and the decision period, to larger and statistically significant in the first post-decision period (at one percent) and the second post-decision period (at ten percent).

To summarize, these results suggest that the early releases caused by the corrections department being placed under court supervision increase subsequent AIDS infections. However, these increases in AIDS infections were concentrated among African-Americans and among women. The timing of these changes suggests a delayed response, similar to the timing of the lagged incarceration effects that we estimated above using an alternative identification strategy.

IX. Conclusion

The findings of this study are several. We demonstrate a strong positive correlation between increases in incarceration rates occurring among men within narrowly defined demographic groups and corresponding increases in the incidence of new AIDS infection among both men and women. This relationship survives detailed controls for sexual relationship market fixed effects, overall national time trends, and time trends that are specific to age, racial, and state groups. The estimated dynamic relationship between male incarceration and AIDS infections resembles estimates of the probability distribution of the incubation period between

seroconversion and the onset of symptomatic, advanced-stage HIV. Moreover, given the sizable racial differentials in incarceration rates at the beginning of the AIDS epidemic, and the increases in these differentials thereafter, our model estimates suggest that the lion's share of the racial differentials in AIDS infections rates for both men and women are attributable to racial differences in incarceration trends.

We also find evidence that an exogenous increase in prison releases created spikes in AIDS infection rates that manifest 5 to 10 years later, primarily for African-Americans and women. While we are unable to identify suitable instruments for new prison admissions (reflecting an expansion in the scope of incarceration), the results for releases establish a direct connection between incarceration dynamics and subsequent AIDS infections among the non-institutionalized.

While we have focused explicitly on the transmission of HIV/AIDS, our theoretical story and empirical analysis can easily be extended to other communicable diseases that have high prevalence among prisoners. For example, we have cited existing evidence of relatively high inter-personal transmission of the Hepatitis-B and Hepatitis-C viruses as well as tuberculosis among inmates. Given the large numbers of individuals cycling through corrections systems in the United States, the more general issue of how prison impacts the transmission of communicable diseases broadly defined is clearly an issue in need of further research as well as attention from policy makers.

What do these results imply for national and state-level policy debates regarding the optimal level of incarceration? Existing research clearly documents the benefits of prison in terms of crime reduction that extend beyond society's desire to punish those who transgress the law. However, imprisonment is costly, and some of the costs come in the form of unintended

consequences. To assess whether we are at, below, or beyond the optimal level of incarceration, one would need to assign a monetary value to the benefits to society, in terms of the crime reduction of incarcerating the last offender, and compare these benefits to the costs. Donohue (2005) estimates that we are currently incarcerating people at a rate beyond the point where the benefits exceed the costs. Based on an annual per-inmate cost of $46,000 per year, Donohue argues that the optimal incarceration level is roughly 300,000 persons less than the current level.

The findings of our study suggest that imprisonment places on society additional costs that extend beyond the per-inmate per-year costs of incarceration. These additional costs include increased medical expenditures for post-release treatment of offenders and others who are infected as a result of incarceration, as well as the loss of health and happiness among those affected. While it is difficult to place a monetary value on these factors, they certainly add to overall costs of incarceration, and their incorporation into cost-benefit accounting would certainly lower the optimal incarceration point even further than that estimated by Donohue.

Our results suggest that there are large and important unintended health consequences for former offenders and for non-incarcerated members of the communities that disproportionately send young men into the state and federal prison systems. A comprehensive assessment of criminal justice policy in the United States should clearly take these considerations into account.

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DATA APPENDIX

To estimate the model discussed in the previous section, we construct a panel data set covering the period 1982 to 1996 that measures the rate of advanced-stage HIV infection[37] for sub-populations of the United States as well as a host of own- and opposite-gender incarceration rates. The dimensions of the panel are defined by the interactions between the year of diagnosis, the state of residence at the time of diagnosis, age group, racial/ethnic group, and gender. We calculate AIDS infection rates using data from the 2001 CDC AIDS Public Information Data Set (PIDS) as well as the 1980, 1990, and 2000 five percent Public Use Microdata Samples (PUMS) from the U.S. Census of Population and Housing. We calculate incarceration rates using the census data. In this section, we discuss the construction of these variables and the details of our panel data set.

Calculating the AIDS infection rate

The AIDS Public Information Data Set provides case level information on all known AIDS cases measured by the national AIDS surveillance system. Since 1985, all states require that health service providers report diagnosed AIDS cases to state and local health departments. In turn, these departments voluntarily report the details of such cases to the CDC.[38]

Since the onset of the AIDS epidemic, the definition of a case has changed several times. Prior to the ability to identify the HIV antibody, AIDS cases were defined by the presence of a disease indicative of a suppressed immune system, such as pneumocystis carinii pneumonia, Kaposi's sarcoma, and other opportunistic infections. The definition was changed in 1985 reflecting the discovery of HIV as a causative agent of AIDS. The 1985 change added additional medical conditions as well as the restriction to those with HIV infections. The number of admissible conditions for an AIDS diagnosis was expanded again in 1987. Finally, the definition of AIDS was expanded once again in 1993 to reflect more generally those with HIV infections and measurably suppressed immune systems. The redefinition also expanded the number of medical conditions that would lead to an AIDS diagnosis for an HIV positive individual.

The three redefinitions of an AIDS case increased the likelihood of an AIDS diagnosis independent of actual prevalence. The CDC reports that the 1985 redefinition added 3 to 4 percent to total annual new diagnoses, while the 1987 change augmented cases by nearly 25 percent. Similarly, the expanded definition based on a gauge of a suppressed immune system caused a discrete change in reported cases. Moreover, there is evidence that the redefinitions had larger effects on reporting for racial and ethnic minorities and on AIDS cases that were not contracted through men having sex with men. To control for the effects of these case reporting redefinitions and any other common temporal changes, we include complete controls for year of diagnosis as well as complete sets of race-specific, age-specific, and region-specific time effects.

Using the AIDS PIDS database, we first tabulate the total number of newly diagnosed AIDS cases by the state of residence, race, age, gender and year of diagnosis for individuals with advanced-stage HIV. We then use data from the 1980, 1990, and 2000 Census PUMS to estimate the national population corresponding to each state/race/age/gender/year cell. For census years, we directly calculate the population with the sample data by summing the provided sample weights within cells. For inter-census years, we linearly interpolate the population using

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[37] Advanced-stage HIV is commonly referred to as a full-blown AIDS case.

[38] Evaluation studies of the completeness of the reporting of AIDS cases has been estimated to be more than 85 percent complete, with the level of reporting completeness varying by geographic area. For a complete discussion, see Rosenblum et. al. (1992).

the population estimates for the respective cell for the two census years bracketing the year in question. With these population estimates, we then tabulate an AIDS diagnosis rate expressed per 100,000 individuals. This variable is the principal dependent variable of our analysis.

An individual's race/ethnicity is defined by the four mutually-exclusive categories non-Hispanic white, non-Hispanic black, non-Hispanic Asian, and Hispanic. We use nine of the ten age groupings used to characterize new diagnoses in the AIDS PIDS data, effectively limiting the analysis to AIDS cases among individuals between 20 and 65 years of age.[39] The introduction and widespread use of medical therapies, particularly medical advances introduced since 1996, have slowed the progression of HIV to AIDS and therefore may have altered/elongated the lagged structure of the relationship between incarceration and AIDS incidence. In light of this fact, our analysis focuses on the period from 1982 to 1996.

One problem with the annual AIDS PIDS data disaggregated at the race-age-state level concerns the ability to identify the state of residence at the time of diagnosis. Because of confidentiality restrictions due to small cell size within some dimension of our panel, roughly 15 percent of AIDS cases observed over this period lack state identifiers. For the levels of disaggregation of AIDS cases required by our analysis, the PIDS identified metropolitan area of residence for those individuals residing in large metropolitan areas. This accounts for 85 percent of documented AIDS cases and includes AIDS cases for 38 state plus Washington, D.C.[40] For the remaining 15 percent of documented AIDS cases, the only geographic identifier is the region of residence (defined as west, south, Midwest, and northeast). Thus the infections rates in our panel data set are estimated using only 85 percent of the total number of AIDS cases recorded in the U.S. To make use of all cases, we also estimated the models below using the four-category region of residence to define geographic locations rather than state of residence. The results are qualitatively and numerically similar to what we present below and are available from the authors upon request.

Given that the panel spans fifteen years (1982 to 1996) and cover 38 state plus Washington D.C., the dimensions of the panel define 21,060 individual demographic groups for each gender.[41]

Calculating Incarceration Rates from the PUMS

Estimating Equation (1) requires data on current and lagged incarceration rates for both men and women. Here we first describe how we estimate incarceration rates with data from the U.S. Census. We then describe the lagged structure of our panel data set and the manner in which we calculated the lagged incarceration rates.

To estimate the proportion incarcerated for each sub-group of our panel, we make use of the group-quarters identifier included in the PUMS data. The decennial Census enumerates both the institutionalized as well as the non-institutionalized population. The PUMS data for each census includes a flag for the institutionalized as well as micro-level information on age, education, race and all other information available for other non-institutionalized long-form

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[39] The age ranges describing each infected individual refer to age at infection and are 20 to 24, 25 to 29, 30 to 34, 35 to 39, 40 to 44, 45 to 49, 50 to 54, 55 to 59, 60 to 64, and 65 plus. We drop the 65 plus category since many of those 65 plus in the census defined as institutionalized are in nursing homes.

[40] The twelve states with missing disaggregated AIDS case-level information are Alaska, Iowa, Idaho. Maine, Mississippi, Montana, North Dakota, New Hampshire, South Dakota, Vermont, West Virginia, Wyoming. There are also missing state identifiers for some AIDS cases in small rural areas disproportionately in the South.

[41] For cells with a positive populations estimate and zero new AIDS cases, we set the AIDS infection rate to zero. After omitting those cells where the population estimates from the census are zero, there are 21,018 observations for men and women.

respondents. The group-quarters variable allows one to identify those individuals residing in non-military institutions, a category that includes inmates of federal and state prisons, local jail inmates, residents of inpatient mental hospitals, and residents of other non-aged institutions. We use this variable as our principal indicator of incarceration.[42] Raphael (2005) presents a comparison of incarceration estimates from the census to those tabulated by the Bureau of Justice Statistics using alternative data sources and shows that the institutionalized in the decennial census provide a good proxy for the incarcerated population.

For the census years 1980, 1990 and 2000, we measure the contemporary incarceration rate for each demographic group defined by state of residence, age group, race/ethnic group, and gender as the proportion of the members of the demographic cell that is institutionalized. For non-census years, we linearly interpolate the incarceration rate using the estimated rates for the two years bracketing the year in question.

Our model requires that we estimate lagged incarceration rates for each demographic group defined by our panel data set. We assume that the AIDS epidemic begins in 1980 and allow for up to 13 lags of the incarceration rate.[43] We calculate the lagged incarceration rates in the following manner. First, we redefine the age groupings of our panel to reflect the effect of a time lag. For example, for 30 to 34 year old black women in New Jersey who are infected in 1990, the one-year lagged incarceration rate should correspond to New Jersey black women who are 29 to 33 years of age in 1989, the two-year lagged incarceration rate should correspond to New Jersey black women that are 28 to 32 years of age in 1988, and so on. Given that the maximum number of lags in our panel is 13 years, we must adjust the age structure 13 times.

Next, for each of these 13 additional age structures interacted with the other dimensions of our panel, we estimate the contemporary incarceration rate for each year from 1980 to 2000 using the PUMS. This essentially creates 13 ancillary panel data sets using 13 alternative age groupings.

Finally, we match observations from our original panel to the corresponding observations from each of the 13 ancillary panels that gauge the appropriate time lags. For example, using the ancillary panel where the age structure is lagged one year, the 1995 incarceration rates provide the one-year lag for 1996, the 1994 incarceration rate provides the one-year lag for 1995 and so on. Using the ancillary panel where the age structure is lagged two years, the 1994 observations provide the two-year lag for 1996, the 1993 observation provides the two-year lag for 1995, and so on.

Each observation in our final data set is matched to 13 lags of the own-gender incarceration rate, where observations with infection years between 1982 and 1992 will have missing values for lags that date prior to 1980. In addition, each observation is also matched by year of infection, state of residence, race/ethnicity, and age to the contemporary and lagged incarceration rates for individuals of the opposite gender.

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[42] See Butcher and Piehl (1998) for an analysis of incarceration among immigrant men that also uses the group quarter variable to identify the incarcerated.
[43] Recall from our methodological discussion above, for any year where lags one through thirteen occur prior to 1980, we constrain the coefficient on that lag for that year to zero.

Figure 1: Per Contact Probability of HIV Transmission by Sexual Activity
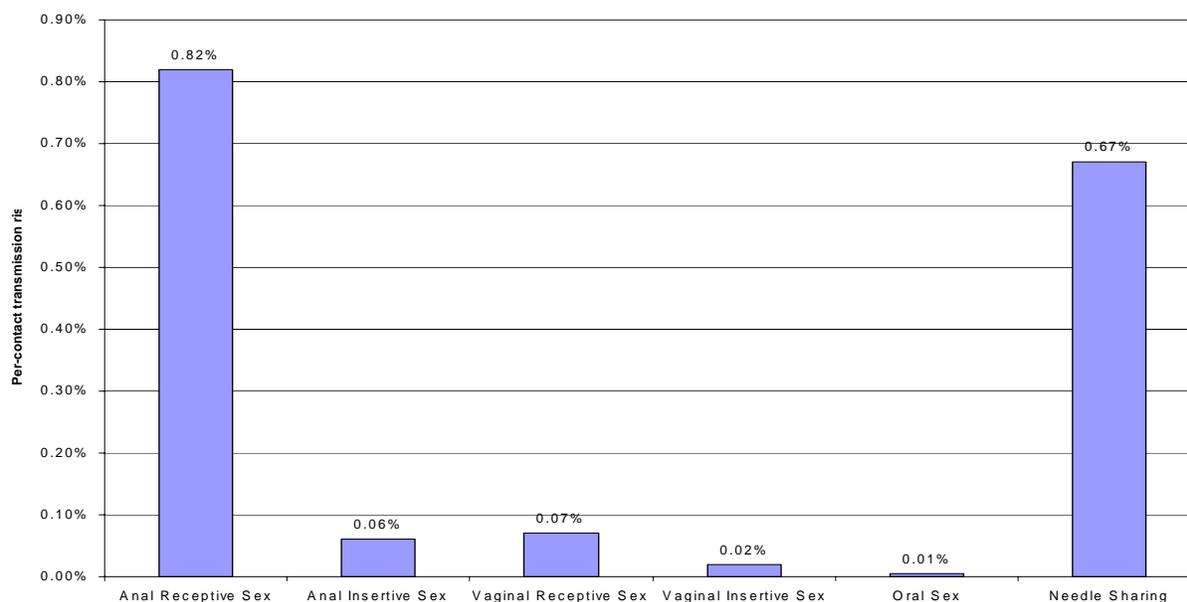
Figure 2

Annual Newly-Diagnosed AIDS Cases Per 100,000 Men Age 20t to 64 by Race/Ethnicity, 1982 to 2001

Figure 3

Annual Newly-Diagnosed AIDS Case Per 100,000 Women Age 20 to 64 by Race/Ethnicity, 1982
to 2001

Figure 4

Proportion of Men Incarcerated, Age 20-34:
Median, 90th, 10th Percentile of Distribution Across States

Data: 1980, 1990, 2000 Census PUMS
(Linear interpolation used for non-census yrs)

Figure 5

AIDS Infection Rate, Men Age 30-44: Mean, 90th, 10th Percentile of Distribution Across States

Data: CDC AIDS cases (PIDS), 1982-1996

Figure 6

AIDS Infection Rate, Women Age 30-44: Mean, 90th, 10th Percentile of Distribution Across States

Data: CDC AIDS cases (PIDS), 1982-1996

Figure 7

Homosexually-contracted AIDS Infection Rate, Men Age 30-44:
Mean, 90th, 10th Percentile of Distribution Across States

Data: CDC AIDS cases (PIDS), 1982-1996

Figure 8

Heterosexually-contracted AIDS Infection Rate, Women Age 30-44:
Mean, 90th, 10th Percentile of Distribution Across States

Data: CDC AIDS cases (PIDS), 1982-1996

Figure 9

Effects of Changes in Male Incarceration
on New AIDS Infections('92-'96), Men

Figure 10

Effects of Changes in Male Incarceration
on New AIDS Infections('92-'96), Women

Figure 11a

Figure 11b

Figure 12

Actual Black-White Differences in Overall AIDS Infection Rates for Men and the Black-White Difference After Accounting for Male Incarceration Rates

Figure 13

Actual Black-White Difference in Overall AIDS Infection Rates for Women and the Black-White Difference After Accounting for Male Incarceration Rates
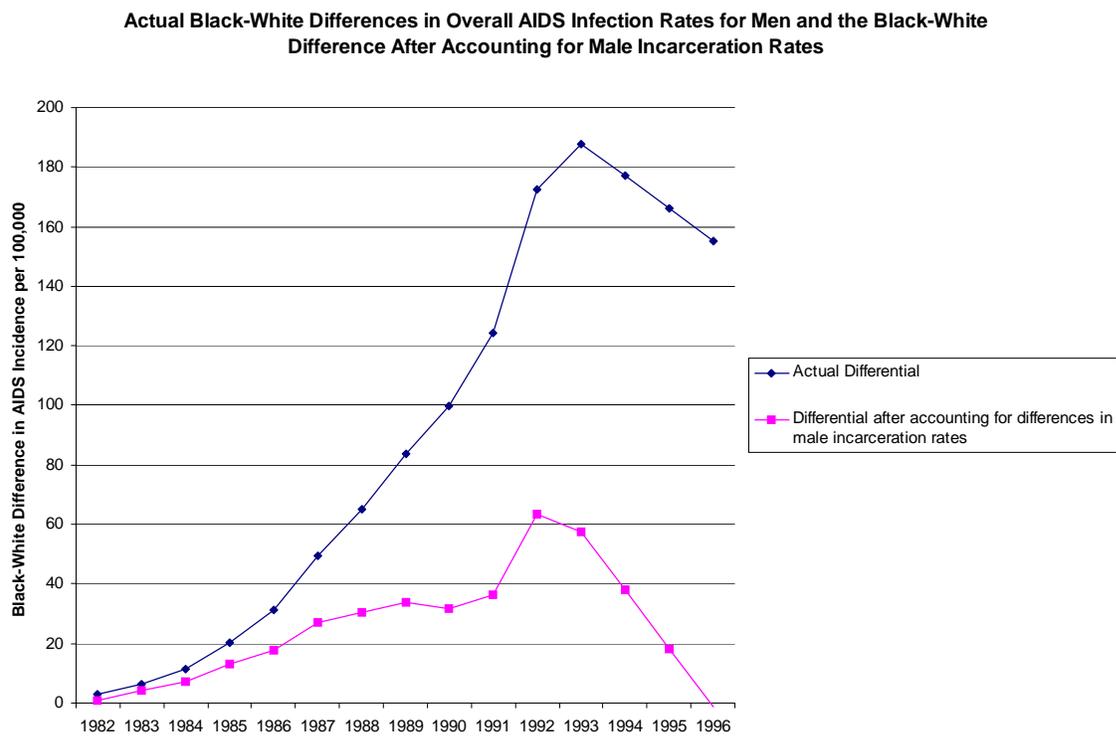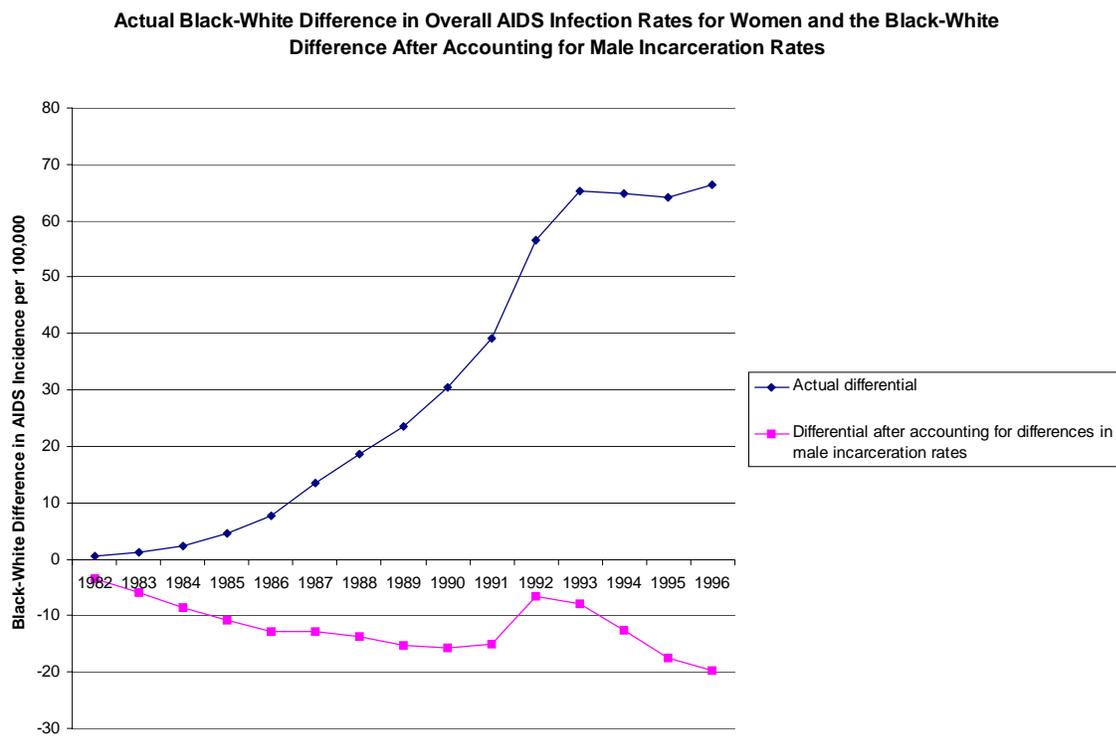
Figure 14

Comparison of New AIDS Cases per 100,000 in States Under Court Order to Relieve Prison Overcrowding and Comparison States
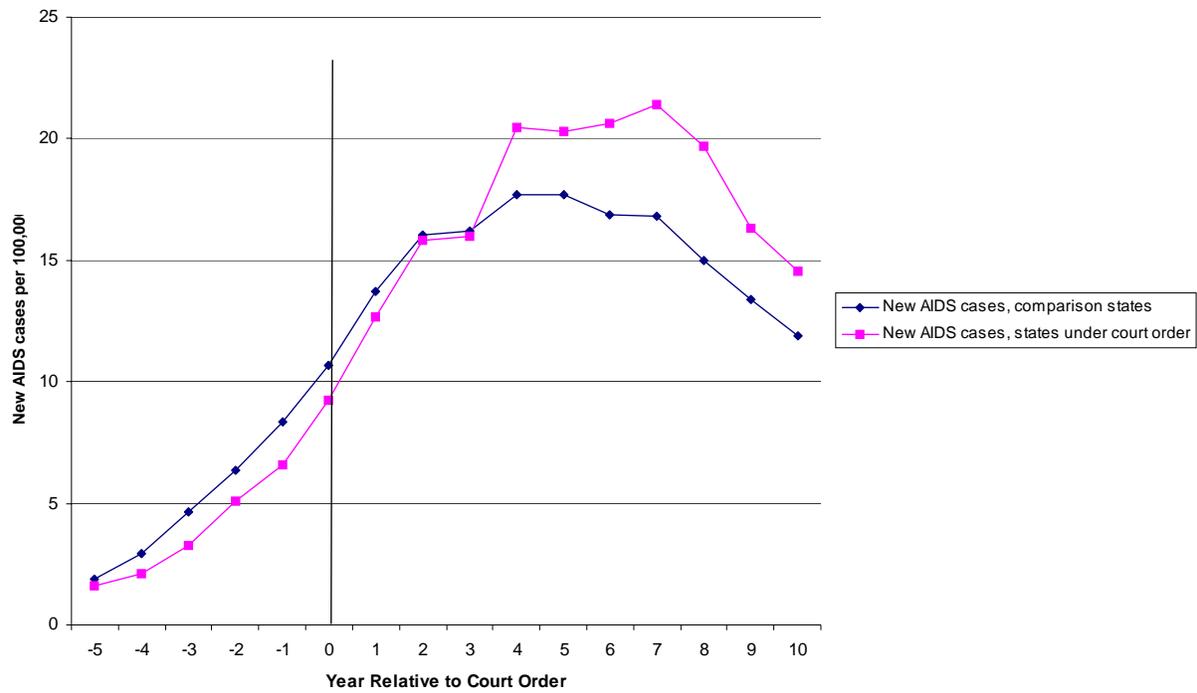
Figure 15

Comparison of New AIDS Cases per 100,000 Among Non-Hispanic Whites in States Under Court Order to Relieve Prison Overcrowding and Comparison States
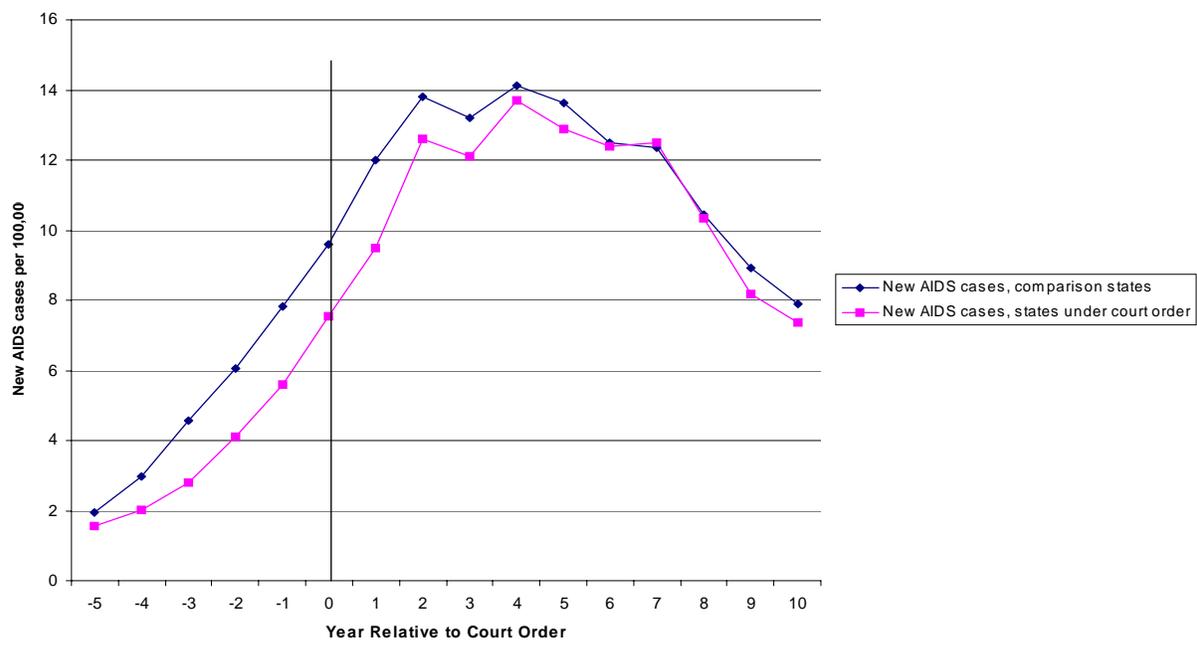
Figure 16

Comparison of New AIDS Cases per 100,000 Among African-Americans in Stated Under Court
Order to Relieve Prison Overcrowding and Comparison States
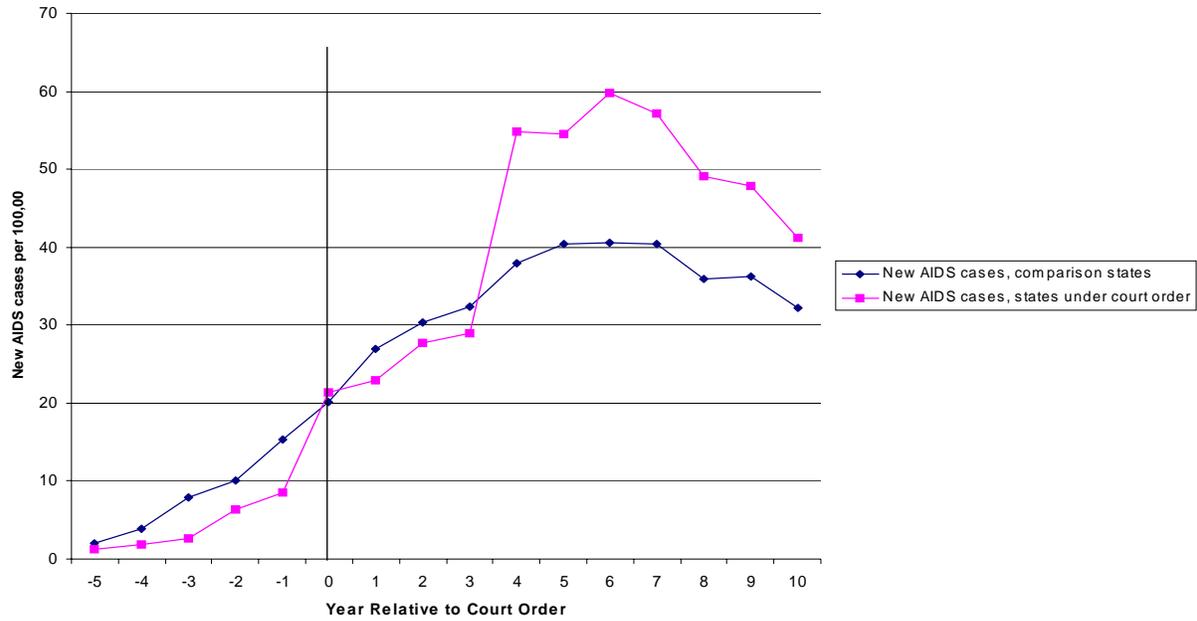
Figure 17

Comparison of New AIDS Cases per 100,000 Among Men in States Under Court Order to
Relieve Prison Overcrowding and Comparison States
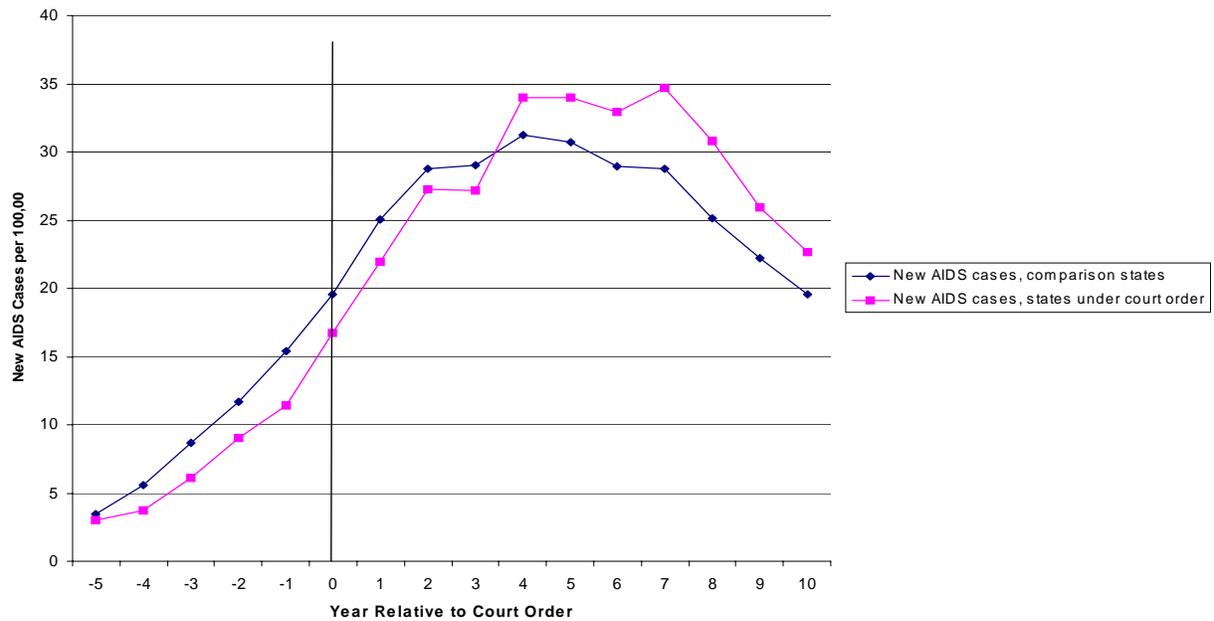
Figure 18

Comparison of New AIDS Cases per 100,000 Among Women in States Under Court Order to Relieve Prison Overcrowding and Comparison States
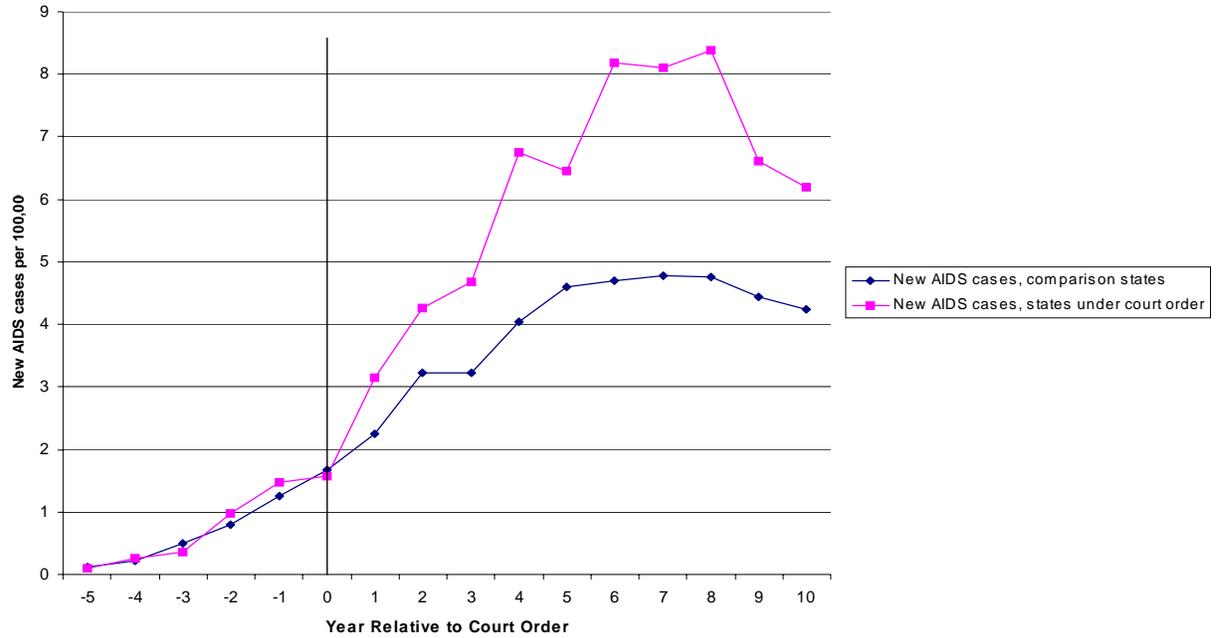
Table 1
Key Percentiles of the Distribution of the Change in the Proportion of Men Incarcerated Between 1982 and 1996

Panel A: Using All Age Categories for Men 20 to 64 Years of Age

	10th	25th	50th	75th	90th
White	-0.003	-0.000	0.002	0.006	0.009
Black	0.005	0.015	0.035	0.050	0.064
Hispanic	0.001	0.007	0.012	0.016	0.020
Other	-0.009	-0.001	0.002	0.004	0.010

Panel B: Using Two Age Categories for Men 25 to 34 Years of Age

	10th	25th	50th	75th	90th
White	0.001	0.004	0.006	0.008	0.012
Black	0.030	0.038	0.049	0.064	0.072
Hispanic	0.005	0.012	0.013	0.017	0.021
Other	-0.001	0.001	0.002	0.006	0.011

The figures present percentiles of the distribution of the change in male incarceration rate between 1982 and 1996 across all age-state cells used in the main analysis of the paper within racial groups. The distributions are weighted by the average of the 1982 and 1996 male population of each cell.

Table 2
Regression of New AIDS Cases Reported Over the Five Year Periods 1987 through 1991 and 1992 through 1996 Against Contemporaneous Change in Male and Female Incarceration Rates and Cohort-Consistent 5-Year Lagged Changes in Male and Female Incarceration Rates

	Dependent Variable=New AIDS Cases per 100,000 Among Men		Dependent Variable=New AIDS Cases per 100,000 Among Women	
	(1)	(2)	(3)	(4)
ΔContemporaneous Male Incarceration Rates	1,906 (3,416)	1,997 (3,498)	-1,026 (1,292)	-363 (1,268)
ΔCohort-consistent 5-Year Lagged Male Incarceration Rates	55,228[a] (4,763)	55,473[a] (4,756)	17,456[a] (2,104)	17,488[a] (2,089)
ΔContemporaneous Female Incarceration Rates	-	-385 (4,976)	-	-5,739[a] (1,795)
ΔCohort-consistent 5-Year Lagged Female Incarceration Rates	-	-2,408 (7,584)	-	1,501 (1,919)
R^2	0.266	0.266	0.243	0.247
N	3,120	3,117	3,119	3,117

Standard errors are in parentheses. All regressions include a constant term.

<u>Note</u>: incarceration rate changes here expressed in decimal form: .01 for one percentage-point change.

a. Statistically significant at the one percent level of confidence.

Table 3
Estimated Effects of Contemporaneous and Lagged Five-Year Changes in Incarceration Rates on Cumulative AIDS Infections (per 100,000) in the Early, Middle, and Peak Periods of the AIDS Epidemic

	Early Period	Middle Period	Peak Period
Contemporaneous	143.44	143.44	143.44
ΔIncarceration	(87.77)	(87.77)	(87.77)
Once-Lagged	**172.71**	463.16[b]	463.16[b]
ΔIncarceration	**(451.60)**	(188.5)	(188.5)
Twice-Lagged	**20.21**	**625.13**	852.11[a]
ΔIncarceration	**(389.14)**	**(458.49)**	(250.97)

Standard errors are in parentheses. Contemporaneous and lagged effects are estimates from the coefficients of the model specified in equation (2). Coefficients in bold correspond to time periods pre-dating the AIDS epidemic.

Note: in this table and remainder of paper, incarceration rates are expressed as a percent: e.g., 1.5 represents a one point five percentage-point increase in incarceration rates.

    a.   Statistically significant at the one percent level of significance.
    b.   Statistically significant at the five percent level of significance.
    c.   Statistically significant at the ten percent level of significance.

Table 4. Regression Models Examining the Role of Male Incarceration Rates and Overall Racial/Ethnic Differences in AIDS Infection Rates among Men

Variables	Men: 1982-1996			
	AIDS^{rsat} (any source)		Homosexually-contracted AIDS^{rsat}	
	(1)	(2)	(3)	(4)
Black (ref cat. White)	87.3739***	10.9101***	28.8359***	-14.7595***
	(2.8102)	(3.2807)	(1.1620)	(1.3481)
Hispanic	20.9899***	0.4095	-3.4704**	-13.9691***
	(2.8613)	(2.6441)	(1.6813)	(1.5089)
Asian	-35.0814***	-35.4066***	-31.3973***	-30.3543***
	(1.5153)	(1.3630)	(1.2082)	(1.1359)
Male Incarceration Rate^{rsat}		-0.8050		2.1212***
		(0.7792)		(0.2124)
Male Incarceration Rate^{rsat-1}		-0.8211***		0.7073***
		(0.2546)		(0.0937)
Male Incarceration Rate^{rsat-2}		-0.3940		-0.0088
		(0.3885)		(0.1070)
Male Incarceration Rate^{rsat-3}		0.3732		-0.1660
		(0.4850)		(0.1229)
Male Incarceration Rate^{rsat-4}		1.3770***		0.0969
		(0.4303)		(0.1102)
Male Incarceration Rate^{rsat-5}		2.5143***		0.6407***
		(0.3045)		(0.0906)
Male Incarceration Rate^{rsat-6}		3.6818***		1.3266***
		(0.2855)		(0.1016)
Male Incarceration Rate^{rsat-7}		4.7762***		2.0156***
		(0.4393)		(0.1410)
Male Incarceration Rate^{rsat-8}		5.6942***		2.5686***
		(0.5940)		(0.1762)
Male Incarceration Rate^{rsat-9}		6.3326***		2.8468***
		(0.6453)		(0.1842)
Male Incarceration Rate$^{rsat-10}$		6.5881***		2.7112***
		(0.5419)		(0.1526)
Male Incarceration Rate$^{rsat-11}$		6.3573***		2.0228***
		(0.3933)		(0.1091)
Male Incarceration Rate$^{rsat-12}$		5.5372***		0.6426***
		(0.8447)		(0.2222)
Male Incarceration Rate$^{rsat-13}$		4.0243**		-1.5683***
		(1.8953)		(0.4960)
Year controls?	yes	yes	yes	yes
State controls?	yes	yes	yes	yes
Age group controls?	yes	yes	yes	yes
Observations	21,060	21,018	21,060	21,018

Bootstrapped standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%

Columns 2 and 4 estimate constrained 13-yr distributed lag models, using a third-order polynomial to represent the lag weights. These models include the same series of lagged female incarceration rates as shown for men--coefficient estimates on these variables are suppressed in the Table. All regressions are weighted by cell frequency.

Table 5. Regression Models Examining the Role of Male Incarceration Rates and Overall Racial/Ethnic Differences in AIDS Infection Rates among Women

Variables	Women: 1982-1996			
	AIDS^{rsat} (any source)		Heterosexually-contracted AIDS^{rsat}	
	(1)	(2)	(3)	(4)
Black (ref cat. White)	31.0285***	-5.1316***	15.6272***	-4.2077***
	(1.5282)	(1.1888)	(0.7105)	(0.7644)
Hispanic	10.1768***	0.4197	5.6110***	0.5184
	(1.0101)	(0.7569)	(0.4214)	(0.4434)
Asian	-2.5483***	-2.4078***	-1.0534***	-0.7773***
	(0.3482)	(0.4415)	(0.1564)	(0.1473)
Male Incarceration Rate^{rsat}		-0.0907		0.1304
		(0.3371)		(0.1660)
Male Incarceration Rate^{rsat-1}		0.2442***		0.3085***
		(0.0883)		(0.0480)
Male Incarceration Rate^{rsat-2}		0.5367***		0.4245***
		(0.1334)		(0.0684)
Male Incarceration Rate^{rsat-3}		0.7912***		0.4925***
		(0.1867)		(0.0959)
Male Incarceration Rate^{rsat-4}		1.0123***		0.5264***
		(0.1762)		(0.0937)
Male Incarceration Rate^{rsat-5}		1.2047***		0.5402***
		(0.1297)		(0.0729)
Male Incarceration Rate^{rsat-6}		1.3731***		0.5482***
		(0.1036)		(0.0545)
Male Incarceration Rate^{rsat-7}		1.5219***		0.5641***
		(0.1460)		(0.0637)
Male Incarceration Rate^{rsat-8}		1.6558***		0.6022***
		(0.2021)		(0.0866)
Male Incarceration Rate^{rsat-9}		1.7795***		0.6764***
		(0.2267)		(0.0994)
Male Incarceration Rate$^{rsat-10}$		1.8975***		0.8008***
		(0.2069)		(0.0959)
Male Incarceration Rate$^{rsat-11}$		2.0145***		0.9894***
		(0.2071)		(0.1053)
Male Incarceration Rate$^{rsat-12}$		2.1351***		1.2562***
		(0.3995)		(0.1942)
Male Incarceration Rate$^{rsat-13}$		2.2638***		1.6152***
		(0.7970)		(0.3734)
Year controls?	yes	yes	yes	yes
State controls?	yes	yes	yes	yes
Age group controls?	yes	yes	yes	yes
Observations	21,051	21,018	21,051	20,359

Bootstrapped standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%

Columns 2 and 4 estimate constrained 13-yr distributed lag models, using a third-order polynomial to represent the lag weights. These models include the same series of lagged female incarceration rates as shown for men--coefficient estimates on these variables are suppressed in the Table. All regressions are weighted by cell frequency.

Table 6. Regression Models Estimating the Effects of Male Incarceration Rates on AIDS Infection Rates among Men and Women, Complete Specification

Explanatory Variables	Dependent variable: AIDS^{rsat} (any source)		
	Men: 1982-1996	**Women: 1982-1996**	
	Male Incarceration Rate^{rsat}	**Male Incarceration Rate^{rsat}**	**Female Incarceration Rate^{rsat}**
Contemporaneous Year (t)	0.7772	0.0703	1.6798
	(0.9110)	(0.4169)	(1.0931)
Lag Year t-1	0.0689	0.5001**	0.9577*
	(0.4196)	(0.2146)	(0.5656)
Lag Year t-2	-0.1575	0.8317***	0.4994
	(0.3351)	(0.1784)	(0.5104)
Lag Year t-3	0.0138	1.0802***	0.2468
	(0.3990)	(0.2010)	(0.5548)
Lag Year t-4	0.4986	1.2611***	0.1421
	(0.3909)	(0.2013)	(0.5103)
Lag Year t-5	1.2128***	1.3894***	0.1274
	(0.3105)	(0.1790)	(0.4051)
Lag Year t-6	2.0720***	1.4804***	0.1448
	(0.2234)	(0.1602)	(0.3511)
Lag Year t-7	2.9922***	1.5494***	0.1363
	(0.2398)	(0.1700)	(0.4325)
Lag Year t-8	3.8891***	1.6116***	0.0441
	(0.3372)	(0.2003)	(0.5538)
Lag Year t-9	4.6785***	1.6824***	-0.1897
	(0.4086)	(0.2217)	(0.6064)
Lag Year t-10	5.2763***	1.7768***	-0.6231
	(0.4024)	(0.2130)	(0.5422)
Lag Year t-11	5.5983***	1.9103***	-1.3139***
	(0.3452)	(0.1820)	(0.4741)
Lag Year t-12	5.5602***	2.0979***	-2.3201***
	(0.5037)	(0.2325)	(0.8753)
Lag Year t-13	5.0778***	2.3551***	-3.6995**
	(1.0635)	(0.4656)	(1.8204)
Year controls?	yes	yes	
Sex Market Fixed Effect: Race*State*AgeGroup	yes	yes	
Race-specific Year Effect: Year*Race	yes	yes	
Age group-specific Year Effect: Year*AgeGroup	yes	yes	
State-specific Year Effect: Year*State	yes	yes	
Observations	21,018	21,018	

Bootstrapped Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%

We estimate constrained a 13-yr distributed lag model, using a third-order polynomial to represent the lag weights. This model includes the same series of lagged female incarceration rates as shown for men. The regression is weighted by cell frequency.

Table 7. Regression Models Estimating the Effects of Male Incarceration Rates on AIDS Infection Rates by Race/Ethnicity and Gender

	Men: 1982-1996			Men: 1982-1996			Women: 1982-1996			Women: 1982-1996		
	AIDS^{rsat} (any source)			Homosexually-contracted AIDS^{rsat}			AIDS^{rsat} (any source)			Heterosexually-contracted AIDS^{rsat}		
	White	Black	Hispanic	White	Black	Hispanic	White	Black	Hispanic	White	Black	Hispanic
Male Incarceration Rate^{rsat}	6.1553***	1.6006	7.9098***	5.2239***	1.6889***	4.5360***	1.4881***	2.6465***	1.3014**	0.8288***	1.2035***	0.2189
	(1.5533)	(1.5684)	(1.4236)	(1.8336)	(0.3420)	(0.7656)	(0.3024)	(0.6282)	(0.5243)	(0.1458)	(0.2532)	(0.2361)
Male Incarceration Rate^{rsat-1}	0.6542	-0.9210	4.0287***	0.1227	0.4600*	1.8621***	0.9195***	1.5304***	1.4432***	0.5415***	0.8696***	0.3644***
	(1.0804)	(0.8526)	(0.8455)	(1.0355)	(0.2445)	(0.3479)	(0.1440)	(0.3530)	(0.3206)	(0.0786)	(0.0946)	(0.1205)
Male Incarceration Rate^{rsat-2}	-2.1757**	-2.1212***	1.7454**	-2.4392***	-0.0813	0.3272	0.5288***	0.7980***	1.5474***	0.3299***	0.6330***	0.4779***
	(1.0965)	(0.5550)	(0.7430)	(0.9338)	(0.3049)	(0.3276)	(0.0866)	(0.1883)	(0.3048)	(0.0661)	(0.1156)	(0.1168)
Male Incarceration Rate^{rsat-3}	-2.8387***	-2.2405***	0.7827	-2.9526***	-0.0812	-0.2802	0.2896***	0.3917***	1.6209***	0.1843***	0.4797***	0.5649***
	(1.0915)	(0.5464)	(0.7714)	(0.9963)	(0.3178)	(0.3751)	(0.1003)	(0.1253)	(0.3238)	(0.0712)	(0.1435)	(0.1347)
Male Incarceration Rate^{rsat-4}	-1.8392**	-1.5193***	0.8637	-1.9085**	0.3142	-0.1715	0.1755	0.2538**	1.6705***	0.0948	0.3961***	0.6306***
	(0.9317)	(0.5459)	(0.7340)	(0.9360)	(0.2628)	(0.3348)	(0.1072)	(0.1121)	(0.3066)	(0.0678)	(0.1276)	(0.1313)
Male Incarceration Rate^{rsat-5}	0.3183	-0.1983	1.7111***	0.2022	0.9585***	0.4420*	0.1602*	0.3268***	1.7033***	0.0516	0.3685***	0.6805***
	(0.6775)	(0.4596)	(0.6362)	(0.7690)	(0.1636)	(0.2357)	(0.0945)	(0.0922)	(0.2561)	(0.0554)	(0.0810)	(0.1114)
Male Incarceration Rate^{rsat-6}	3.1296***	1.4823***	3.0479***	2.8887***	1.7055***	1.3487***	0.2173***	0.5528***	1.7262***	0.0449	0.3830***	0.7197***
	(0.5223)	(0.3034)	(0.5603)	(0.6329)	(0.0886)	(0.1947)	(0.0741)	(0.0582)	(0.2014)	(0.0431)	(0.0458)	(0.0936)
Male Incarceration Rate^{rsat-7}	6.0902***	3.2817***	4.5969***	5.6601***	2.4091***	2.3372***	0.3206***	0.8744***	1.7461***	0.0648	0.4260***	0.7537***
	(0.6683)	(0.1677)	(0.5845)	(0.6896)	(0.1613)	(0.3009)	(0.0677)	(0.0411)	(0.1841)	(0.0439)	(0.0908)	(0.0977)
Male Incarceration Rate^{rsat-8}	8.6958***	4.9595***	6.0812***	8.0254***	2.9229***	3.1962***	0.4436***	1.2338***	1.7699***	0.1016*	0.4838***	0.7878***
	(0.9215)	(0.2609)	(0.6758)	(0.8748)	(0.2585)	(0.4280)	(0.0830)	(0.0704)	(0.2112)	(0.0556)	(0.1456)	(0.1171)
Male Incarceration Rate^{rsat-9}	10.4420***	6.2753***	7.2236***	9.4938***	3.1008***	3.7142***	0.5600***	1.5734***	1.8046***	0.1453**	0.5424***	0.8274***
	(1.0677)	(0.4364)	(0.7367)	(1.0065)	(0.3088)	(0.4898)	(0.0989)	(0.0969)	(0.2408)	(0.0640)	(0.1736)	(0.1297)
Male Incarceration Rate$^{rsat-10}$	10.8244***	6.9886***	7.7470***	9.5744***	2.7965***	3.6797***	0.6435***	1.8356***	1.8571***	0.1863***	0.5883***	0.8776***
	(0.9934)	(0.5611)	(0.7092)	(0.9620)	(0.2839)	(0.4436)	(0.0968)	(0.0932)	(0.2440)	(0.0592)	(0.1590)	(0.1220)
Male Incarceration Rate$^{rsat-11}$	9.3386***	6.8587***	7.3743***	7.7763***	1.8638***	2.8813***	0.6676***	1.9627***	1.9343***	0.2145***	0.6077***	0.9440***
	(0.6625)	(0.5950)	(0.6698)	(0.6995)	(0.1871)	(0.3191)	(0.0702)	(0.0457)	(0.2478)	(0.0394)	(0.1098)	(0.1072)
Male Incarceration Rate$^{rsat-12}$	5.4803***	5.6454***	5.8284***	3.6087***	0.1565	1.1077**	0.6062***	1.8970***	2.0433***	0.2203***	0.5869***	1.0319***
	(0.6758)	(0.5562)	(0.9847)	(0.6649)	(0.2657)	(0.5071)	(0.0778)	(0.0869)	(0.3747)	(0.0530)	(0.1772)	(0.1679)
Male Incarceration Rate$^{rsat-13}$	-1.2550	3.1079***	2.8322	-3.4195**	-2.4718***	-1.8526	0.4328**	1.5810***	2.1908***	0.1937	0.5120	1.1465***
	(1.8972)	(0.6379)	(1.8678)	(1.7001)	(0.6864)	(1.1843)	(0.2053)	(0.2832)	(0.6988)	(0.1402)	(0.4309)	(0.3432)
Sex Market Fixed Effect: Race*State*AgeGroup	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
Race-specific Year Effect: Race*State*Year	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
Observations	5,265	5,139	5,265	5,265	5,139	5,265	5,265	5,139	5,265	5,265	5,139	5,265

Bootstrapped Standard errors in parentheses; * significant at 10%; ** significant at 5%; *** significant at 1%

All columns estimate constrained 13-yr distributed lag models, using a third-order polynomial to represent the lag weights. These models include the same series of lagged female incarceration rates as shown for men--coefficient estimates on these variables are suppressed in the Table. All regressions are weighted by cell frequency.

Table 8. Regression Models of Effects of Male Incarceration on AIDS Infections among Black Women via Heterosexual Transmission vs. via Intravenous Drug Use

| | Black Women: 1982-1996 | |
| | AIDS^{rsat} | |
Variables:	Heterosexually-contracted	Contracted via IV-Drug Use
	(1)	(2)
Male Incarceration Rate^{rsat}	-0.0089	0.3977
	(0.5265)	(0.5679)
Male Incarceration Rate^{rsat-1}	-0.1890	0.3988
	(0.3280)	(0.2798)
Male Incarceration Rate^{rsat-2}	-0.0716	0.2904
	(0.2849)	(0.2235)
Male Incarceration Rate^{rsat-3}	-0.0724	0.1275
	(0.2909)	(0.2854)
Male Incarceration Rate^{rsat-4}	-0.0876	-0.0284
	(0.2290)	(0.2627)
Male Incarceration Rate^{rsat-5}	-0.0650	-0.1503
	(0.2116)	(0.2220)
Male Incarceration Rate^{rsat-6}	0.1673	-0.1321
	(0.1954)	(0.2065)
Male Incarceration Rate^{rsat-7}	0.3803	-0.1541
	(0.2329)	(0.1843)
Male Incarceration Rate^{rsat-8}	0.5071*	-0.5615**
	(0.3044)	(0.2838)
Male Incarceration Rate^{rsat-9}	0.7867***	0.0187
	(0.2868)	(0.3349)
Male Incarceration Rate$^{rsat-10}$	0.9533***	-0.4150
	(0.3031)	(0.3028)
Male Incarceration Rate$^{rsat-11}$	1.3727***	-0.2045
	(0.2955)	(0.3757)
Male Incarceration Rate$^{rsat-12}$	1.9256***	0.6036
	(0.5787)	(0.5195)
Male Incarceration Rate$^{rsat-13}$	1.5225**	0.0081
	(0.7319)	(0.5499)
Sex Market Fixed Effect: Race*State*AgeGroup	yes	yes
Race*State-specific Year Efx: Year*State*Race	yes	yes
Race*AgeGrp-specific Yr Efx: Year*Age*Race	yes	yes
Observations	5,244	5,244

Bootstrap standard errors in parentheses

* significant at 10%; ** significant at 5%; *** significant at 1%

Note: We estimate a (constrained) 13-yr distributed lag model, using a third-order polynomial to represent the lag weights. Interpolation of incarceration rates for non-Census years are based on BJS aggregated race*state-specific time pattern. This model includes the same series of lagged female incarceration rates as shown for men, and allows a linear trend in the effects of the incarceration rate variables over the course of the AIDS epidemic. The coefficients of the linear trend in the effects of the incarceration variables are suppressed in the table to conserve space. The results reveal that the effects of the male incarceration variables increase in magnitude over the course of the epidemic. The coefficients presented in this table represent the estimated lag structure by the end of the period, year 1996. The regression is weighted by cell frequency.

Table 9. Regression Model Using State-Year Level Panel and Incorporating the Contemporaneous and Lagged Effects of Prison Admissions, Prison Releases, and Crack Cocaine Usage Prevalence

	1982-1996			
	Dependent variable: $\mathbf{AIDS^{st}}$ (any source)			
Explanatory Variables	Prison Pop Size	Annual Prison Admissions	Annual Prison Releases	Crack Cocaine Index
Contemporaneous Year (t)	-24.1524	-0.0550*	0.0508	2.3093***
	(19.1980)	(0.0299)	(0.0314)	(0.5209)
Lag Year t-1	-9.7688	-0.0094	0.0026	1.8291***
	(8.4641)	(0.0234)	(0.0258)	(0.2241)
Lag Year t-2	2.4583	0.0158	-0.0208	1.4767***
	(10.1042)	(0.0270)	(0.0301)	(0.2832)
Lag Year t-3	12.6802	0.0232	-0.0235	1.2160***
	(12.7866)	(0.0291)	(0.0317)	(0.3400)
Lag Year t-4	20.8434	0.0157	-0.0092	1.0108***
	(13.3183)	(0.0291)	(0.0299)	(0.3153)
Lag Year t-5	27.0436**	-0.0040	0.0182	0.8248***
	(12.8741)	(0.0295)	(0.0280)	(0.2459)
Lag Year t-6	31.2615**	-0.0330	0.0547*	0.6219***
	(13.2449)	(0.0330)	(0.0300)	(0.2053)
Lag Year t-7	33.5380**	-0.0684*	0.0964***	0.3659
	(14.9591)	(0.0395)	(0.0369)	(0.2513)
Lag Year t-8	33.9159**	-0.1075**	0.1394***	0.0205
	(16.8596)	(0.0469)	(0.0459)	(0.3311)
Lag Year t-9	32.3682*	-0.1473***	0.1796***	-0.4504
	(17.3950)	(0.0535)	(0.0544)	(0.3988)
Lag Year t-10	29.0753*	-0.1852***	0.2132***	-1.0831**
	(15.5189)	(0.0589)	(0.0616)	(0.4860)
Lag Year t-11	23.8832*	-0.2183***	0.2364***	-1.9137***
	(12.7773)	(0.0652)	(0.0692)	(0.7073)
Lag Year t-12	17.0909	-0.2439***	0.2452***	-2.9785**
	(18.4934)	(0.0784)	(0.0822)	(1.1679)
Lag Year t-13	8.4094	-0.2590**	0.2360**	-4.3136**
	(37.5058)	(0.1051)	(0.1078)	(1.9090)
F-test (p-value) for joint significance of: Lagged Vars	30.16 (.0072)	29.75 (.0082)	22.61 (.0669)	129.34 (.0000)
State Fixed Effects	yes			
Year Fixed Effects	yes			
Observations	765			

Boostrapped Standard errors in parentheses

* significant at 10%; ** significant at 5%; *** significant at 1%

Note: This table contains coefficient estimates of a single model of AIDS incidence, including 13-year lagged variables of incarceration levels, annual prison admissions, annual prison releases, and crack cocaine prevalence. The lagged coefficient estimates of each of these sets of variables in the 13-yr constrained distributed lag model uses a third-order polynomial to represent the lag weights, and allows a linear trend in the effects of the prison variables over the course of the AIDS epidemic. The coefficients of the linear trend in the effects of the prison variables are suppressed in the table to conserve space. The results reveal that the effects of all of the prison variables increase in magnitude over the course of the epidemic. The coefficients presented in this table represent the estimated lag structure by the end of the period, year 1996. The regression is weighted by population size.

Table 10: Prison Overcrowding Litigation Status 1985-1993
(States w/Entire Prison System Under Court Order)

	Final Decision	Comparison States
Alaska	90-93	NV, GA, AZ
Delaware	88-91	AK, GA, AZ
New Mexico	90	ID, SD, MS
South Carolina	91-93	MO, MI, AZ
Texas	85-91	CA, OR, GA

Nearest neighbor matching estimator for comparison states based on 5-yr average levels and 5-yr averages changes of incarceration rate, annual prison-release rates, and annual prison admission rates in yrs preceeding court order

Table 11
Matching Estimates of the Effects of a Court Order to Relieve Prison Overcrowding on Prisoners per 100,000, Prison Releases per 100,000, and Prison Admissions per 100,000

	Year Relative to Court Mandate Regarding Prison Overcrowding Relief (Year 0 Corresponding to Year of Court Decision)			
	-4 through -1	0 through 3	4 through 7	8 through 10
Panel A: Prisoners per 100,000				
Effected states	318.66 (23.81)	361.77 (26.25)	435.89 (33.23)	531.53 (45.27)
Comparison states	256.75 (11.87)	318.70 (12.17)	392.53 (12.52)	455.56 (15.49)
Difference	61.90 (24.71)[b]	43.07 (25.91)[d]	43.37 (28.86)	75.96 (37.25)[c]
Adjusted Difference[a]	61.91 (14.32)[b]	43.07 (13.91)[b]	43.36 (16.68)[b]	78.96 (32.66)[c]
Panel B: Admissions per 100,000				
Effected states	158.11 (7.52)	199.67 (15.52)	229.43 (18.27)	246.38 (31.99)
Comparison states	143.00 (5.42)	183.36 (5.52)	211.49 (5.78)	219.62 (12.52)
Difference	15.11 (10.35)	16.31 (13.06)	17.94 (14.46)	26.75 (28.26)
Adjusted Difference[a]	15.11 (7.21)[c]	16.31 (9.66)[d]	20.29 (11.78)[d]	26.75 (27.96)
Panel C: Releases per 100,000				
Effected states	138.39 (6.76)	186.39 (11.36)	206.98 (16.77)	211.80 (38.62)
Comparison states	122.60 (5.05)	163.23 (5.04)	183.89 (5.38)	191.38 (11.87)
Difference	15.78 (9.58)	23.16 (10.89)[c]	23.09 (13.36)[d]	20.42 (29.93)
Adjusted Difference[a]	15.78 (7.26)[c]	23.17 (7.69)[b]	24.97 (10.76)[c]	20.42 (29.57)

Standard errors are in parentheses.
  a. Differences are adjusted for comparison-specific fixed effects.
  b. Statistically significant at the one percent level of confidence.
  c. Statistically significant at the five percent level of confidence.
  d. Statistically significant at the ten percent level of confidence.

Table 12
Matching Estimates of the Effects of Prison Releases on Subsequent Average Annual
New AIDS Cases per 100,000, Overall and by Race and Gender

	Year Relative to Court Mandate Regarding Prison Overcrowding Relief (Year 0 Corresponding to Year of Court Decision)			
	-4 through -1	0 through 3	4 through 7	8 through 10
Panel A: All new AIDS cases				
Effected states	4.26 (0.74)	13.43 (1.54)	20.70 (2.63)	16.84 (1.44)
Comparison states	5.57 (0.64)	14.15 (1.12)	17.27 (1.32)	13.42 (1.44)
Difference	-1.31 (1.19)	-0.73 (2.13)	3.43 (2.73)	3.41 (3.05)
Adjusted Difference[a]	-1.32 (0.84)	-0.73 (1.81)	3.43 (2.19)	3.41 (2.05)
Panel B: New AIDS cases among whites				
Effected states	3.63 (0.56)	10.44 (0.97)	12.86 (1.49)	8.62 (2.08)
Comparison states	5.35 (0.61)	12.15 (0.97)	13.15 (1.23)	9.08 (1.29)
Difference	-1.73 (1.11)	-1.71 (1.78)	-0.28 (2.30)	-0.45 (2.54)
Adjusted Difference[a]	-1.73 (0.85)[c]	-1.71 (1.64)	-0.28 (1.72)	-0.45 (1.42)
Panel C: New AIDS cases among blacks				
Effected states	4.83 (1.77)	25.05 (5.28)	56.58 (13.91)	45.85 (12.99)
Comparison states	9.31 (1.32)	27.17 (2.96)	39.84 (3.48)	34.76 (3.46)
Difference	-4.47 (2.51)	-2.12 (5.91)	16.73 (9.79)[d]	11.09 (9.47)
Adjusted Difference[a]	-4.47 (1.72)[b]	-3.73 (4.51)	18.02 (8.53)[c]	12.54 (6.92)[d]
Panel D: New AIDS cases among men				
Effected states	7.55 (1.26)	23.28 (2.36)	33.90 (4.05)	26.48 (4.76)
Comparison states	10.35 (1.19)	25.62 (1.98)	29.92 (2.33)	22.29 (2.51)
Difference	-2.80 (2.19)	-2.34 (3.70)	3.98 (4.66)	4.18 (5.14)
Adjusted Difference[a]	-2.80 (1.55)	-2.34 (3.17)	3.98 (3.57)	4.18 (3.07)
Panel E: New AIDS cases among women				
Effected states	0.77 (0.28)	3.42 (0.83)	7.38 (1.49)	7.06 (1.75)
Comparison states	0.69 (0.11)	2.60 (0.31)	4.53 (0.45)	4.47 (0.52)
Difference	0.08 (0.25)	0.82 (0.72)	2.84 (1.15)[b]	2.59 (1.34)[d]
Adjusted Difference[a]	0.08 (0.21)	0.82 (0.60)	2.84 (1.04)[b]	2.59 (1.23)[c]

Standard errors are in parentheses.
    a. Differences are adjusted for comparison-specific fixed effects.
    b. Statistically significant at the one percent level of confidence.
    c. Statistically significant at the five percent level of confidence.
    d. Statistically significant at the ten percent level of confidence.