A Tale of Two Crises: Did HIV/AIDS Help Fuel the Crack Epidemic? [Working Title]

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Abstract

Crack cocaine is viewed by many academics and policy-makers as the worst drug epidemic in U.S. history. We hypothesize that a contributing factor for its severity was substitution from intravenous (e.g., heroin) to non-intravenous drugs (e.g., crack cocaine) as a result of the HIV/AIDS epidemic, which pre-dated the crack epidemic by several years. We document a strong correlation between the geographic areas in which HIV/AIDS appeared first and most intensely and those which experienced the brunt of the crack epidemic. This correlation is robust to the controls commonly used by past researchers studying the advent of crack, choices of functional form, and the level of geographic aggregation. While it is impossible to eliminate all omitted variables to conclude the relationship is causal, this strong correlation suggests that past estimates of the effect of crack on neighborhood outcomes might better be viewed as the joint effect of crack and HIV.

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1 Introduction

The crack epidemic is often called the worst drug epidemic in U.S. history. Though there is disagreement as to whether the social ills associated with the epidemic were due to drug use itself or chiefly through the "war on drugs" that followed, there is little disagreement that the use of cocaine increase substantially in the 1980s. For example, Fryer *et al.* (2012) document that the per capita number of Emergency Room related to cocaine increased by a factor of eleven from 1980-1985 to 1985-2000. Evans *et al.* (2012) find that the number of death certificates noting cocaine-dependence as a contributing factor increased from eight in 1981 to 523 in 1989.

Why did crack cocaine become so popular, especially among marginalized populations? At first, crack was believed to be more addictive than others drugs, a theory that has generally been debunked (XX find cite). Researchers have generally viewed crack as a technological advance that decreased the effective price of using cocaine. We offer a new—though certainly not mutually exclusive—hypothesis: that drug abusers substituted from intravenous drugs (chiefly heroin) to crack as a reaction to HIV/AIDS, which predates the arrival of crack by several years. Put differently, part of the crack's attraction is that its consumption did not require needles and thus did not expose consumers to HIV/AIDS, a key advantage during the late 1980s and early 1990s when the disease claimed roughly 30,000 American lives a year.

Consider the following facts. First, the rough timing in heroin trends supports this story. Though there is limited data on drug use from the 1970s, the consensus in the literature among ethnographers and public health researchers is that heroin use peaked in the early 1980s (see Inciardi *et al.*, 1998 for a review). In March of 1983, the Center for Disease Control first announced that HIV could be transmitted through sharing needles and identified IV drug users as the largest risk group after men who have sex with men (MSMs).

Second, the so-called "heroin junkies" of the late 1970s and early 1980s was of the same demographic as the so-called "crack fiend" of the late 1980s. In both cases, the majority of drug sales are to hard-core users who are disproportionately male, minority and poor. As **?** write: "From its first appearance, crack has always been used heavily by the same population that has always used heroin heavily: the urban poor."

Third, and we believe novel, is that DESCRIBE EVIDENCE. This result holds when

we.... **IK to fill in.** xx

We emphasize upfront that we do not believe that substitution from IV drugs explains the entire crack epidemic. Though data are limited, what data we have suggest that the pool of IV drug users of the late 1970s and early 1980s is substantially smaller than the pool of crack users from the peak of the Crack Era. Even if all individuals who would have initiated as heroin users instead initiate as crack users in the 1980s, we suspect that a fair amount of total crack use in the late 1980s and early 1990s would need to be explained by other factors. Another point to note is that while the demographics of heroin and crack users are very similar, as crack is a stimulant and heroin a depressant, it is possible that the motivations for consumption could be different.

Another major challenge for this paper and the literature on illicit activities more generally is data quality. Survey data on self-reported cocaine or crack use are not readily available at the state or local level, so our analysis follows past work and relies on proxies, which could be correlated to HIV prevalence even absent a substitution story. As a concrete example of a potential concern, both our explanatory variables as well as some of our dependent variables rely on medical diagnoses (for HIV/AIDS in the case of the explanatory variable and for cocaine dependence in the case of the dependent variable). If the medical community in certain cities are more aware about recent medical trends, they might code a higher share of diagnosis as HIV in the early 1980s and crack-related in the late 1980s (indeed, one could imagine that some medical professionals might *over*code for these diagnoses if they are overly sensitive to the latest trend). Indeed, while deaths related to cocaine in mortality files skyrocket in the late 1980s, national level data on survey measures of cocaine use suggests that cocaine use among high schools seniors as well as the twelve and over population peaked in 1985 and fell substantially in the second half of the 1980s, highlighting the slipperiness of using noisy proxies. We propose some checks to see whether reporting issues drive our results [STILL NEED TO DO.]

Our paper is related to the literature on how individuals respond to health shocks and information. Agüero and Beleche (2014) find that the outbreak of the deadly H1N1 ("swine") flu in 2009 may have led to a decrease in diarrhea cases in Mexico, as the swine flu acted as a "natural nudge" to improve personal hygiene as protection against infection. Margolis *et al.* (2014) finds that while patients undergoing percutaneous coronary intervention (PCI) versus coronary artery bypass graft (CABG) appear similar ex ante, ex post CABG patients are

significantly more likely to quit smoking, which they attribute to the more invasive CABG providing a more salient and lasting health shock. In the context of HIV, Philipson and Posner (1994) find that the first major news item on AIDS in 1981 led to a decrease in male homosexual gonorrhea cases at a San Francisco city clinic, presumably because individuals responded to the news with safer sexual practices that positively spilled over to other diseases.

Our results suggest that reactions to informational health socks may have ambiguous health effects if they induce substitution toward other risky activities. In that sense, they are closely related to the literature on whether the decline in smoking has led to an increase in obesity.[1] More recent work on substitution among drugs argues that medical marijuana legalization may have led to a decline in (the arguably more dangerous) prescription pain-killers (Powell *et al.*, 2015).

As HIV increased the effective price of consuming heroin, our work is also related to the literature on the demand elasticity of drugs. This literature has focused mainly on the demand elasticities for marijuana, and to a lesser degree, cocaine; these estimates range broadly, from essentially zero to about -1.0.[2] On heroin use, Dave (2006) finds a small but significant price elasticity of demand, using ER visits to proxy for consumption. Using data from the 1920s and 1930s, Van Ours (1995) finds elasticities just under one for opium in the Dutch East Indies.

Finally, our paper is related to the surprisingly small economics literature on the effect of AIDS in the US. Given the disproportionate impact of the disease in developing countries, it is not surprising that most economists have focused their attention there. But, as we document in the next section, HIV had an enormous impact on disadvantaged populations (which we will typically proxy by race) in the US, accounting for the majority of the slow-down in the black-white mortality convergence in the 1980s. Francis and Mialon (2010) has modeled the spread of HIV in the US, finding evidence that tolerant attitudes towards homosexuality slows its advance. Francis (2008) documents perhaps an even more dramatic avoidance mechanism than we propose: he finds that local AIDS prevalence leads to more men reporting heterosexual behavior and more women reporting homosexual behavior, arguably to avoid sex with men, the gender that served as the far more virulent HIV transmission

[1]The results on this question are mixed, likely due to the challenge of isolating exogenous variation in smoking status. See, e.g., Gruber and Frakes (2006) and Courtemanche *et al.* (2015).

[2]See, for example, Grossman and Chaloupka (1998).

vector. [THERE IS PROBABLY STUFF WE ARE MISSING?] In the conclusion, we suggest future lines of work on the effect of HIV/AIDS on disadvantaged groups in the US.

The paper proceeds as follows. In the next section, we provide the relevant background on HIV, crack cocaine, and reasons the two epidemics could interact. In Section **??** we document the populations affected by HIV, demonstrating that HIV was such a large, negative shock to the health of disadvantaged communities in the US that avoidance behavior would be plausible, and that the demographics of the 1970s and early 1980s heroin addict were very similar to those of the late 1980s crack addict, and distinct from heavy consumers of other substances such as alcohol and marijuana. In Section **??** we describe our data and empirical strategy and in Section **??** present our results.

2 Background on HIV and crack cocaine

2.1 Brief history of AIDS in the US

The current scientific consensus hypothesizes that HIV(human immunodeficiency virus) was brought to the US from Haiti sometime in 1969 (Gilbert *et al.*, 2007). The Center for Disease Control (CDC) first documented a case of what would become known as AIDS (acquired immune deficiency syndrome), the cluster of diseases caused by HIV, in its Mortality of Morbidity Weekly Report (MMWR) of June 5, 1981 and within weeks dozens of other cases were identified. All early cases identified by the CDC were homosexual males, and in fact the center initially refereed to the disease as gay-related immune deficiency (GRID) until they retired that name in 1982 when other risk groups emerged.

As is well known today, HIV is spread via contact with bodily fluids such as blood or semen, making sharing needles a key transmission pathway. While men who have sex with men (MSM) were initially the main risk group, as early as 1984 over one-fourth of new diagnoses involved IV drug use as a risk factor.[3] In fact, the time between diagnosis and death was typically much shorter for IV drug users (IDUs) than MSMs, though the reason—later diagnosis, a more virulent form of the disease, or worse pre-infection health—remains unclear. But the high death rate among IDUs and their marginalized position have lead public health agencies to conclude that their number, especially in the 1980s, were

[3]Our calculations from online CDC data (`http://wonder.cdc.gov/aids-v2002.html`) though this result is noted in other papers as well.

significantly undercounted.[4]

While initial infection is sometimes accompanied by mild flu-like symptoms, the virus is then asymptomatic for five to ten years, meaning the first AIDS cases emerging in the early 1980s were due to HIV infections from the mid 1970s. Indeed retrospective testing of blood samples of individuals who entered IV drug treatment centers in the late 1970s indicate substantial levels of HIV infection. For example, Des Jarlais *et al.* (1989) found that between 1978 (the earliest sample they had) and 1980, the share who tested positive for HIV grew from nine to 38 percent. Between 1981 and 1983 the share testing positive in their sample was fifty percent. While individuals entering drug treatment are a selected sample of the universe of all IDUs (though it is not clear the sign of the selection bias with respect to HIV infection) their substantial level of HIV infection suggests a widespread prevalence of the disease in the general population of IV drug users before 1980. Des Jarlais *et al.* (1989) speculates that bisexual IV drug users served as the "bridge group" between the MSM—the original vector of the disease—and the IDU populations.

The March 4, 1983 MMRW first reported IDUs as a risk group for AIDS and as early as 1982 both the *Wall Street Journal* and the *New York Times* listed them as risk group for this mysterious, new disease. **?** report that at least in New York City the connection between AIDS and needles was understood by IV drug users themselves by that year. By 1984, 59 percent of the heroin addicts undergoing methadone treatment surveyed by Friedman *et al.* (1987) said they had made some form of behavioral change because of AIDS.

While European countries as well as some U.S. cities embraced "harm reduction" strategies such as needle exchanges, the federal response to HIV stressed a more zero-tolerance approach toward heroin consumption (Wormer, 1999). While certainly not meant to be an exhaustive collection, the posters we have found online generally seem to echo this distinction. The campaigns from Maryland and DC (Appendix Figures A.1) emphasize harm reduction, the first urging heroin users to quit but also highlighting clean needles as the next best alternative and the second focusing entirely on safer IV drug practices. The ads from federal agencies in Appendix Figure A.2 engage in stigmatization of drug users, warning women (presumably assuming most IV drug users were male) not to have sex with them. In sum, the real and perceived effective price of IV drug use in the early 1980s substantially rose, whether because of the associated cost of acquiring a fatal disease, the extra time and

[4]See, e.g., Alcabes and Friedland (1995).

resources required to reduce this risk with safer practices, or the increased stigmatization.

A final point relevant to the study at hand is the geographic concentration of the disease, especially in its early days, our focus. Table **??** shows summary statistics (please focus on the first column for now) for the top twenty PMSAs ordered by per capita HIV cases by 1985. The distribution is log normal, with San Francisco and New York being large outliers. New Jersey, Florida and Texas also contains PMSA with relatively high HIV rates, whereas the Midwest is largely spared during the early days of the disease.

2.2 Background on heroin and crack cocaine

Heroin is a derivative of morphine, a compound found in the opium poppy, and has been rather commonly used in the US since the 1800s, initially viewed as a "miracle drug" for a diverse set of ailments. Intravenous transmission becomes popular in the twentieth century, leading to two commonly acknowledged epidemics: the first in the 1920s, and the second beginning in the 1970s and peaking in the early 1980s (with some scholars arguing that the epidemic was intensified by heroin initiation among military personnel during the Vietnam War). [xxx Cites?]

Crack cocaine is a form of cocaine that can be smoked. The exact arrival of crack cocaine in U.S. cities is impossible to pinpoint precisely, but the general consensus points to the mid-1980s. Fryer *et al.* (2012) write that crack was "virtually unheard of prior to the mid-1980s" but spread quickly after that, peaking in 1989. Grogger and Willis (2000) surveys police chiefs to conclude that crack arrived in Los Angeles in 1984, and in New York and San Francisco in 1985.[5] Evans *et al.* (2012) find slightly earlier arrival dates by examining death certificates that list cocaine as a contributing factor (e.g., 1982 for Miami, LA and New York), though they also find arrival dates clustered around 1985 to 1988 for the majority of cities.

There has been limited work on why the severity of the crack epidemic varied across the U.S. with respect to place and time. As Fryer *et al.* (2012) write, "crack is an important technological innovation in many regards" in that it can be smoked (a more effective and convenient transmission route that results in a more powerful high), can be sold in small units, and is incredibly addictive. To the extent that papers have tried to model its geographic

[5]There appear to be two outliers: Atlanta in 1981 and Kansas City in 1982. No other city's police chief documents an arrival date before 1984.

impact, Evans *et al.* (2012) proposed essentially a technology diffusion model, using distance from New York, LA and Miami (where they argue crack was first introduced) as a predictor for where crack would arrive next. Instead, the focus has been more on the potential impact of the crack epidemic on educational and health outcomes.

[XXX Will add more XXX]

2.3 Connections between HIV and crack cocaine

To the best of our knowledge the hypothesis that HIV may have increased demand for non-injectable drugs such as crack has not been unexplored. However, among practitioners, there has at least been mention of the idea. In a meeting of the House Select Committee on Narcotics Abuse and Control in 1991, Dr. Herbert Kleber of the Office of National Drug Control Policy testified that "part of the allure of crack cocaine is that it is convenient to use because it's smokable. No dirty needles, no risk of AIDS." Outside of that reference, we have found very little previous speculation related to our proposed hypothesis.

Given the five to ten year gap between infection and symptoms (and thus, at least in the early years, diagnosis), the onset of HIV clearly pre-dates the arrival of crack cocaine, yet the research that exists between the connection between crack and HIV focuses entirely on how the former causes the latter. Ethnographers and public health researchers have documented how crack cocaine led to unsafe sexual behavior—often women trading sex for access to the drug—and thus the spread of HIV (see, e.g., Edlin *et al.*, 1994; Booth *et al.*, 1993). For this reason, we will typically avoid using any AIDS data from the 1990s as an explanatory variable, given that the crack-to-HIV causal chain would render later AIDS rates potentially endogenous to local crack consumption. However, even AIDS deaths from as late as 1990 would suggest an HIV infection date between 1980 and 1985, generally pre-dating the crack era.

2.4 The characteristics of at-risk populations

In this section, we document the demographic groups most affected by the heroin, HIV and crack cocaine epidemics. While the "face" of HIV tended to be white (e.g., the Gay Men's Health Coalition or the 13-year-old hemophiliac Ryan White), black mortality rates from AIDS were roughly five times that of whites in the first decade of the disease, mirroring

blacks overrepresentation among heroin and crack consumers.

Table 1 uses vital statistics mortality data to explore background characteristics of those for whom a death certificate mentions heroin dependence (col. 1), cocaine dependence (col. 2), AIDS (col. 3) and, as a comparison to another drug-dependent group, alcohol dependence (col. 3). AIDS deaths are listed in col. (4). We limit heroin deaths to the pre-crack era and for the most part pre-AIDS era (1979-1983), cocaine dependence to the peak of crack era (1987-1995), though in practice demographics are stable across the sample period. Those whose deaths are related to cocaine or heroin have very similar demographics: blacks, the never-married, the young and urban are substantially over-represented, relative not only to all deaths (not shown) but also among deaths due to alcohol abuse (col. 3). As col. (4) shows, AIDS deaths exhibit very similar demographics to those related to heroin and cocaine, though are somewhat more male and more urban. The totals at the bottom of the table show that AIDS deaths vastly swamp deaths related to cocaine and especially heroin. Between 1987 and 1995, over 260,000 Americans die of AIDS, compared to 5,000 for cocaine over the same period and only about 200 for heroin (albeit only from 1979 to 1983).

Given how rare among the general population heroine abuse was, even at its peak, we prefer to examine the universe of death certificates in order to observe a large enough sample of hard-core users, but in the Appendix Table A.1 we also show self-reported use in the past month of heroin, crack cocaine, cocaine excluding crack (e.g., powder cocaine used for snorting), and marijuana. Again, the racial breakdown of the first two groups shows a much larger minority share compared to the second two. In general, both mortality and self-report data support the conclusion of Kinlock *et al.* (1998), in their review of heroin use in the US: "the predominance of the poor black urban resident among the heroin user population became evidence during the 1960s and 1970s and continues in the present day."

Heroin users of the 1970s and crack users of the 1980s had similar criminal propensities as well. While Caulkins (1995) estimates that 45 percent of arrestees from major U.S. cities had cocaine (assumed to be mostly from crack) in their systems at the height of the crack epidemic, earlier work showed similar shares for heroin in the 1970s: twenty percent of arrestees in Miami, 45 percent in New York, and fifty percent in the District of Columbia (Research Triangle, 1976).

We close this section by emphasizing just how large an impact AIDS had on the health and mortality of black Americans. To give some context for the magnitude of the HIV mor-

tality effects for blacks, we compare them to homicide rates during the same period, an era famous for historically high murder rates. In fact, the contribution of HIV in explaining changes in white-black mortality gaps in the 1980s swamps that of homicide. As documented by Kochanek *et al.* (1994), since 1900, life expectancy for both whites and blacks had steadily increased, with generally larger gains for blacks than whites, leading to significant convergence. Between 1984 and 1989, for the first time in the twentieth century the trend in life expectancy for blacks reversed, with black males and females losing 0.730 and 0.178 years of life, respectively (the corresponding figures for whites were gains of 0.831 and 0.499). For black males, the leading contributor to the reversal in life expectancy were HIV and homicide, but the effect of HIV was twice as large as the effect of homicide. For black females, HIV was again the leading contributor to the reversal, dwarfing the effect of homicide which does not crack the list of top five contributors. For black males, HIV lead to a loss of 0.664 years of life expectancy between 1984 and 1989, compared to 0.329 years from homicide.[6]

The above paragraph does not suggest that in *levels* HIV was a larger contributor to black mortality than homicide, but that *changes* in the 1980s are better explained by HIV than homicide (as the former essentially did not exist before 1980 whereas the latter was obviously at a substantial level). However, even in levels HIV came very close to homicide as a cause of death for young black men during the period of study. In Appendix Figure A.3 we document mortality rates due to homicide and AIDS for black men. Note that death certificates begin standard recording of AIDS as a cause of death only in 1987 (and even then with substantial under-reporting, as we note below), so AIDS deaths before this date will (incorrectly) appear as zero. While homicide swamps AIDS for men in their twenties (subfigures a and b), the reverse pattern obtains for men in their thirties (subfigures c and d). The graphs suggest that twin public health crises—the introduction of HIV alongside the rise in homicides—struck the black community beginning in the mid- to late-1980s.[7]

[6]Of course, during this time there were factors that, all else equal, increased life expectancy for blacks (particularly gains in heart disease), so the 0.664 and 0.329 loss of years due to HIV and homicide were partially counteracted by some positive developments. Interestingly, the largest contributor to the increasing *gap* between blacks and whites during this period was heart disease—for both groups, it was a contributing factor to increased life expectancy, but gains for whites swamped those for blacks.

[7]We are not the first to compare AIDS and homicide mortality rates, though existing work tends to focus on the 1990s, slightly later than our focal period. For example, Elo and Drevenstedt (2004) finds that between 1993 and 1995 homicide and HIV were the two largest causes of death for black males age 15-39, accounting for 108.06 and 87.85 deaths per 100,000 persons (and 47.3 and 30.4

In addition, there are two reasons why mortality statistics likely understate the impact of HIV relative to homicide. First, even after the introduction of a standardized ICD-9 code for AIDS, AIDS deaths were not uniformly recorded, so many individuals dying of the opportunistic diseases associated with AIDS were not recorded as AIDS victims. As late as 1990, Chang *et al.* (1993) finds that outside of New York City, New York state death certificates missed 13 percent of AIDS diagnoses. Secondly, unlike homicide, a period of extended and severe morbidity precedes death by AIDS. As just one example, Selwyn *et al.* (1989) track 18 HIV-positive heroin addicts in 1987, recording 48 hospitalizations among this cohort that year.

3 Data and empirical strategy

3.1 Data

Measures of AIDS cases. We rely on the CDC for data on AIDS. State-level data on AIDS cases documented by can be found online.[8] The majority of our analysis is at the PMSA level, as the CDC has posted an online data tool that allows disaggregation to this level of geographic disaggregation.[9] AIDS cases are available for 101 PMSAs. From these data, we create per capita measures of all AIDS cases reported by 1986 using Census population data. Note that because we include in these measures those who died before 1986, they are not true prevalence rates.

While HIV/AIDS *cases* are not broken down geographically beyond the PMSA level, we can get county-level data on AIDS *deaths* beginning in 1987 from vital statistics mortality data. While the later starting point is not ideal, we benefit from far greater geographic detail. We generally focus on AIDS deaths between 1987 and 1990, as, given the five to ten year gap between HIV infection and AIDS death, these deaths are very likely to have arisen from HIV infections in the pre-crack period. While we will entirely miss deaths occurring before

percent of the white-black mortality gap), respectively. For black men ages 40 to 64, HIV accounted for 135.06 deaths per 100,000 persons whereas homicide fell substantially to 37.06.

[8] See, e.g., `http://www.cdc.gov/hiv/topics/surveillance/resources/reports/pdf/surveillance84.pdf` and `http://www.cdc.gov/hiv/topics/surveillance/resources/reports/pdf/surveillance85.pdf` for 1984 and 1985, respectively [xx Let's include for 1986].

[9] See `http://wonder.cdc.gov/aids-v2002.html`. We document in greater detail how we extracted these data in Appendix XXX.

1987, arguable key for the substitution story we propose, it is likely that deaths between 1987 and 1990 are a strong proxy for deaths between 1980 and 1985.

Measures of crack cocaine use. For data on crack cocaine, we rely on the excellent work of past researchers, either directly borrowing their published data or using their methodology to calculate their measures of crack intensity at the PMSA level. Fryer *et al.* (2012) developed a state- and city-level crack index—based on factors such homicide rates, cocaine arrest rates, per capita cocaine-related emergency department measures, cocaine seizures by the Drug Enforcement Agency, and the number of local newspaper articles mentioning crack cocaine. They published these indices for 1985, 1989, 1993, 1997 and 2000. We generally focus on 1989 and 1993, as they fall during the peak period of the crack era. The crack index cities and the CDC AIDS cases PMSAs yield an overlap of 77 localities.

Evans *et al.* (2012) developed a measure for the year crack arrived in a geographic area by noting the point in time when for two consecutive years a death certificate in the geographic area mentioned cocaine addiction. They develop this measure at both the state and MSA level. We directly use their state-level measures and replicate their methodology for the PMSA level, the level of aggregation used in the CDC AIDS data.[10]

Beyond crack arrival, we are also interested in the intensity of cocaine use in a given locality as opposed to the drug's exact arrival date. As such, using the Evans et al. insight, we also total cocaine deaths from the mortality data for 1987 to 1995 and dividing by 1990 population, for a measure of the magnitude of the crack epidemic in a locality during the height of the crack era. We choose 1987 and 1995 as cut-off points to have two years on either side of our main crack index years of 1989 and 1993, but in practice the exact cutoffs make little difference to the results.

Grogger and Willis (2000) surveyed police chiefs in 1991 about the date they "first encountered crack," receiving replies from 25 cities. We use this measure sparingly given the small sample size. Grogger and Willis (2000) came up with a supplementary measure by estimating breaks in annual time series data on ER visits involving cocaine. They asked graduate students not associated with the project to pick the year the break seems to occur. This measure is available for 22 cities.

[10]For example, San Francisco and Oakland are in the same MSA but not the same PMSA. Evans et al. helpfully present total numbers of cocaine deaths for illustrative years in their paper, which we took care to replicate before adapting their definition to our needs.

Table **??** presents summary statistics for the top thirty localities ranked by 1986 HIV rate. New York and San Francisco are the top two, as one might expect, though some localities are more surprising (e.g., Seattle,

3.2 Empirical strategy

Our estimation strategy is very straightforward and is essentially a cross-sectional regression, subjecting the correlation between early 1980s AIDS measures in a locality to measures of its crack consumption five to ten years later to an increasing number of controls and to different levels of geographic aggregation.

We will most often use as our key explanatory variable the log of total AIDS cases documented by 1986 in locality i per 100,000 people in locality i (based on 1980 population numbers), where i can be either a state or PMSA (we describe county-level analysis later). We focus on 1986 for three reasons, though show that results are robust to other years as well. First, given the five to ten year lag between infection with HIV and AIDS symptoms, we feel comfortable that essentially all cases documented by 1986 would have been contracted *before* the arrival of crack cocaine in the US (even if one assumes some of the early outlier arrival years are correct), and thus not susceptible to the crack-to-HIV pathway discussed earlier. Second, given the well documented under-reporting of AIDS cases in the early days of the epidemic, using earlier CDC data would likely miss many cases that IV drug users and other risk groups may well have been responding to on the ground. Third, and related, the strongly log normal distribution of per capita AIDS cases suggests using a log specification, and we thus wish to avoid distributions with many observations miscoded as zero.

Our controls again borrow from past work on the crack cocaine literature. We follow the Evans et al. methodology and use 1970 and 1980 IPUMS data to generate pre-period controls at the locality level: share black, share white, share black at different education levels, share black in poverty, share black unemployed. We also include the the Fryer et al. controls not contained in the previous list (e.g., per capita income from the city-county data book and share Hispanic).

In addition to these controls, we add some of our own. Evans et al. suggest that crack diffusion can be predicted the shortest distance to Miami, New York or LA, where they argue crack first originated in the US. We also control for pre-crack homicide rates (1982-1984),

meant to account for the fact that certain localities may have been more susceptible to drug epidemics or other negative outcomes, independent of our hypothesized channel.[11]

4 Results

We first present simple, unconditional scatter plots and then present regression analysis, varying controls to demonstrate the robustness of the underlying correlation.

4.1 Graphical results

We begin at the state level. Figure 1 shows the 1989 Fryer et al. crack index as a function of our standard dependent variable, (logged) per capita AIDS cases documented by 1986. We weight each point by state population in 1980. The correlation has an R-squared value of over 0.65 (0.553 unweighted). Appendix Figures **??** and **??** show that the same general result holds if we use AIDS cases documented by 1985 or by 1984, respectively. A nearly equally strong relationship emerges in Figure 2 with the 1993 crack index or in Figure 3 with crack "arrival" as dependent variables (though in the latter the relationship is negative). A somewhat noisier relationship emerges between cocaine-related deaths from 1987-1995 and our AIDS measure (Figure 4).

Results at the PMSA level (Figures 5 - **??**) tell the same story, though in each case are somewhat noisier than their state-level counterparts.

4.2 Regression results

We again begin with state-level analysis using the 1989 crack index as the outcome, in Table **??**. We tend to prefer weighted estimates (in terms of understanding the public health consequences of our proposed hypothesis, it is more important for the relationship to hold true in the most populous areas), though in every case anytime we show a regression table, the unweighted analogue appears in the Appendix and in no case does the general story change (results that appear strong when weighted continue to appear strong unweighted, and results that are weaker are also weak in both cases).

[11] County identifiers before 1982 are harder to merge to FIPS codes, so we try to avoid pre-1982 vital statistics data when possible.

The first column of Table **??** merely shows the results from Figure 1 in regression form: the weighted, unconditional correlation between the 1989 crack index and our AIDS measure. Col. (2) adds all of the standard controls in Evans et al. (with the exception of one, which we discuss below). Col. (3) drops these controls and instead includes the Fryer et al. controls. Our result is robust to either set of controls as well as their union (col. 4). Adding these varying controls barely changes the coefficient of interest. In col. (4), for example, a one-unit increase in the explanatory variable (going from, say, Houston to Miami) is predicted to increase the 1989 crack index by 0.993, or a 40 percent increase from the (weighted) sample mean.

In col. 5 we keep the union of controls as well add the diffusion control (the minimum distance of the locality from Miami, New York or Los Angeles) proposed by Evans et al., confirming that this variable indeed predicts the magnitude of the crack epidemic (as measures by the 1989 crack index). However, adding our AIDS rate variable to this set of covariates in col. (6) shows that it retains much of its significance (declines by only 28 percent) conditional on this distance variable.

In col. 7 we control for pre-crack-era homicide rates, which has little effect on the coefficient of interest. Finally, we show that our result holds up even after conditional on the nine Census division fixed effects. Despite only within-division variation in AIDS cases remaining to identify the coefficient, its magnitude and significant remain large unchanged (whereas the minimum distance variable loses its explanatory power).

In Table **??**, we show that using the 1993 crack index as our explanatory variable yields very similar, if not slightly stronger, results. Table **??** also show a strong (but negative) relationship between early AIDS cases and the arrival of crack, as proxied by the Evans et al. measure of consecutive years of at least one cocaine death in the state. By far the weakest relationship (evident as well in the scatter plots) is between AIDS cases and cocaine deaths between 1987 and 1995. The relationship remains positive of meaningful economic significance regardless of which controls we use (e.g., even our smallest estimate suggests that a ten percent increase in 1986 aids cases leads to a 1.4 percent increase in cocaine deaths), it loses its statistical significance. Interestingly, the relationship is greater in magnitude and regains significance when division fixed effects are added in the final column, suggesting perhaps that unobserved regional differences could mask the positive *ceteris paribus* relationship between AIDS cases and cocaine deaths.

Appendix Tables A.2 through A.5 show the unweighted analogues to each of the regression tables discussed thus far.

Tables **??** through **??** are identical to the four tables discussed so far but are at the PMSA level. In terms of the stability of the coefficient of interest as it is subjected to different sets of controls, the PMSA crack index results are, if anything, stronger than their state-level counterparts, though in most specifications the coefficient magnitudes are slightly smaller. PMSA-level results for cocaine arrival are not as strong as those at the state-level, with significance being lost once the minimum-distance, homicide and division fixed effects are added. As with the state-level results, total cocaine deaths is always positive correlated with AIDS deaths no matter which of our controls we condition on, but is not always significant.

The unweighted analogues of the PMSA regressions appear as Appendix Tables A.6 through A.9 and in no case do results appreciably change without population weights.

4.3 County-level results

As noted earlier, the most disaggegated geographic unit for which the CDC reports HIV/AIDS cases is the PMSA, but vital statistics data reports AIDS *deaths* at the county-level beginning in 1987. We use vital statistics data to calculate cocaine deaths at the county level (aggregating, as before, from 1987 and 1995).

Table **??** shows the county-level analogue to Tables **??** and **??**. Excluding for the moment the final column, the unconditional correlation decreases by less than thirty percent as we successively add more covariates. In the final column, instead of merely controlling for Census district fixed effects, we control for *state* fixed effects, a more demanding specification where only differences across counties within the same state identifies our coefficients.

In sum, the relationship between the crack index in 1989 and 1993, the peak years of the crack era, and AIDS cases documented by 1986 remains strong across varying controls and whether we aggregate at the state or PMSA level. The statistical significant of the relationship between AIDS cases and crack arrival and total cocaine deaths depends more on the level of aggregation, but even when not significant the relationship is always positive and sizable. Especially for crack arrival, small sample size may limit our ability to reject that even a sizable coefficient is equal to zero.

[Still to do: We could map the Fryer city-level crack index to the county-level and do a

similar analysis. Presumably could do arrival by county, but I feel less interested in predicted the year that a county had two consecutive cocaine deaths than explaining magnitude of cocaine mortality rate.]

5 Conclusion

We have documented a strong, positive correlation between deaths in a locality due to AIDS in the 1980s and proxies for cocaine abuse in that locality from the late 1980s and early to mid 1990s. While ethnographers have documented a strong association between drug addition and the risky behaviors that could lead to HIV infection, given that all of our measures of AIDS are based on infections that would have occurred, at least, in the early 1980s, this channel cannot be responsible for our result. Instead, we argue that a causal pathway from AIDS to crack cocaine use may have existed during this period, as the risk of AIDS made non-IV drugs such as crack especially attractive.

We hope that a contribution of this paper is highlighting the disproportionate impact of HIV/AIDS on minorities relative to others groups in the US, a fact we feel has been overlooked by economists in the literature on the evolution of black-white outcome gaps. Future work might also examine the local public economics of HIV/AIDS. Initially, the federal government had little role in blunting the fiscal impact of AIDS, outside of automatic stabilizers such as matching payments to those HIV/AIDS patients who qualified for Medicaid.[12] Not until life expectancies increased in 1990s did AIDS patients qualify for Medicare (as previously they would rarely survive the two-year-waiting period required if one obtains Medicare via disability status) and thus federal support was rather limited. Given the large share of uninsured AIDS patients, uncompensated care for AIDS patients was a fiscal huge challenge to local hospitals (Andrulis *et al.*, 1987, Solomon *et al.*, 1991). The use of foster care is another potential outcome....

XXX More ideas....

[12]Today, all AIDS patients with T-cell counts below 200 qualify for Medicaid. XX Need to examine evolution of this policy.

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Figure 1: Relationship between 1989 crack index and 1986 AIDS cases, by state

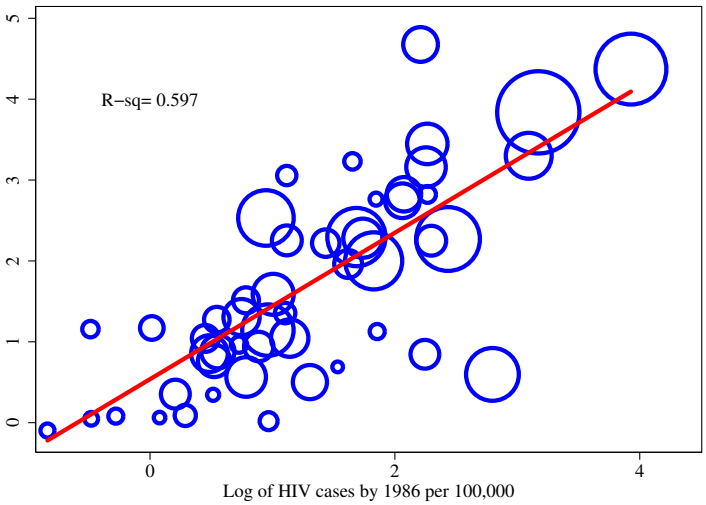


Table 1: Characteristics of individuals with heroin, cocaine, alcohol or AIDS listed on the death certificate

	(1) Heroin (1979-83)	(2) Cocaine (1987-95)	(3) Alcohol (1979-95)	(4) AIDS (1987-95)
Black	0.424	0.570	0.229	0.346
Male	0.741	0.746	0.785	0.867
Age	38.26	37.49	53.40	38.96
Never married	0.459	0.452	0.212	0.672
Resident of city w pop. at least 250K	0.405	0.412	0.285	0.511
Resident of city w pop. at least 500K	0.312	0.267	0.204	0.398
Observations	205	5864	399890	263199

Figure 2: Relationship between 1993 crack index and 1986 AIDS cases, by state

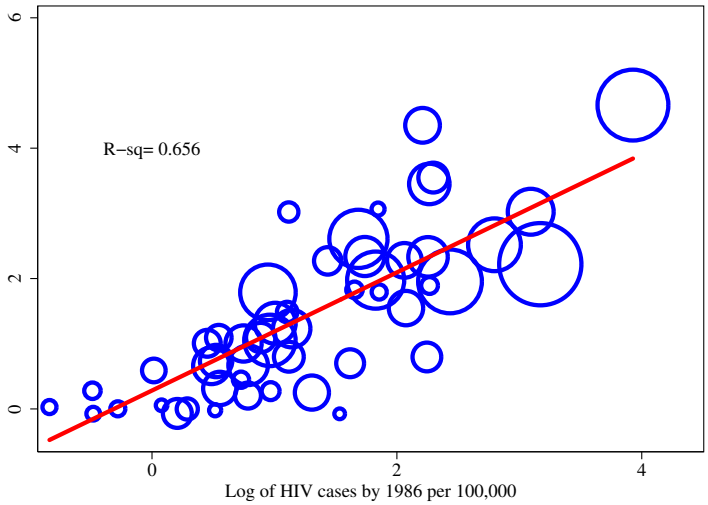


Figure 3: Relationship between crack arrival and 1986 AIDS cases, by state

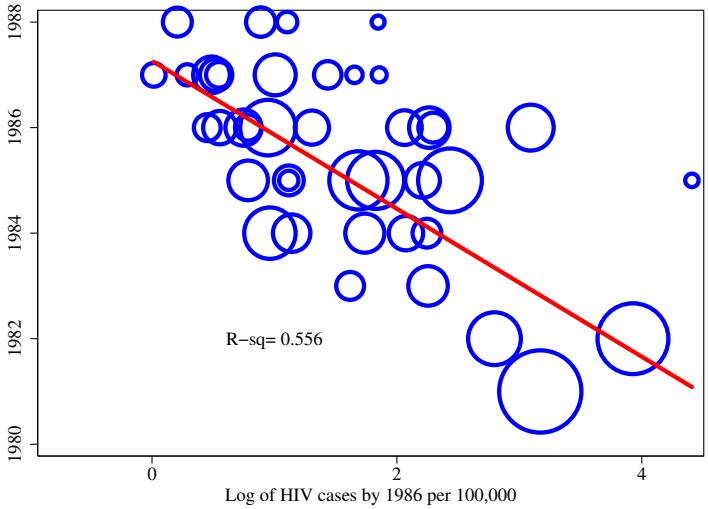


Figure 4: Relationship between 1987-1995 cocaine deaths and 1986 AIDS cases, by state

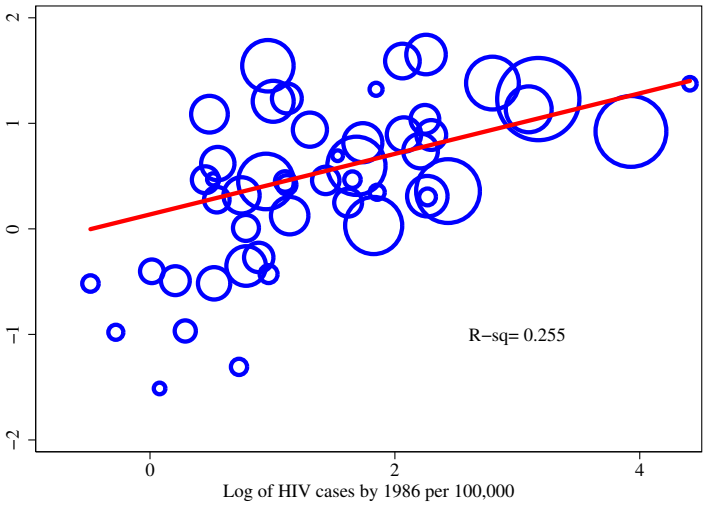


Figure 5: Relationship between 1989 crack index and 1986 AIDS cases, by PMSA

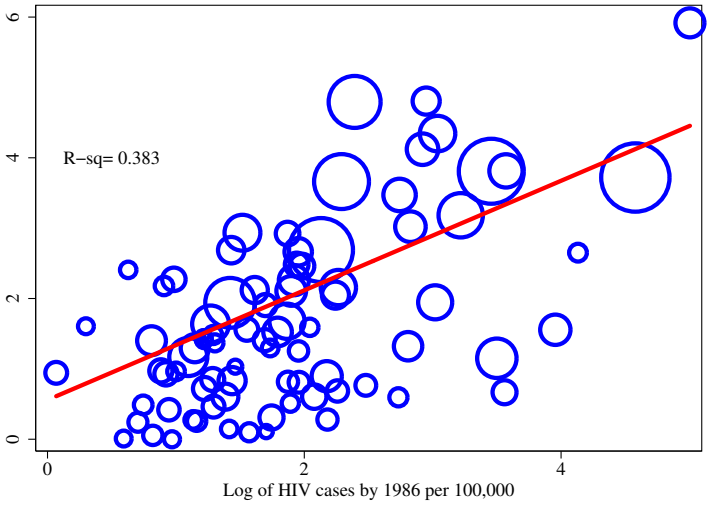


Figure 6: Relationship between 1993 crack index and 1986 AIDS cases, by PMSA

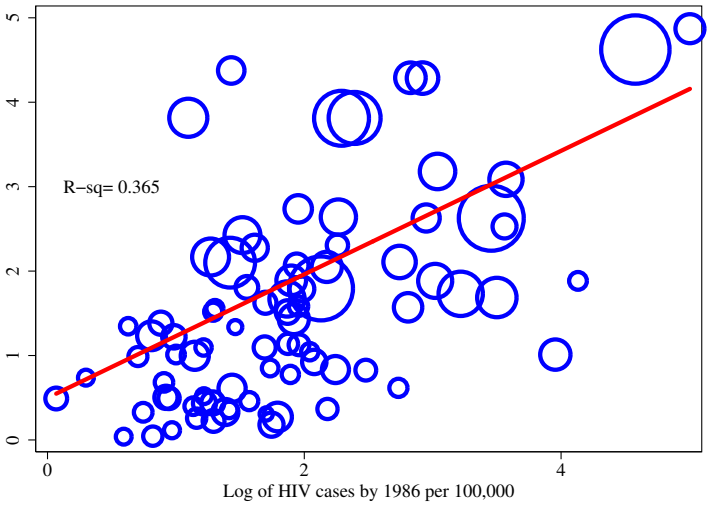


Figure 7: Relationship between crack arrival and 1986 AIDS cases, by PMSA

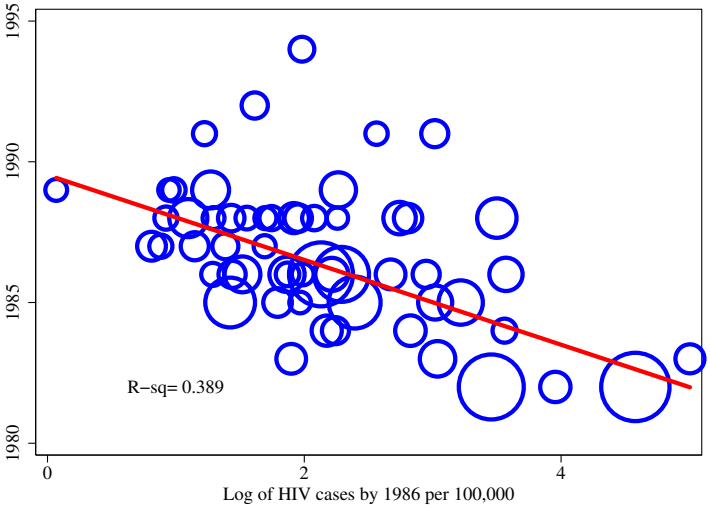


Figure 8: Relationship between 1987-1995 cocaine deaths and 1986 AIDS cases, by PMSA

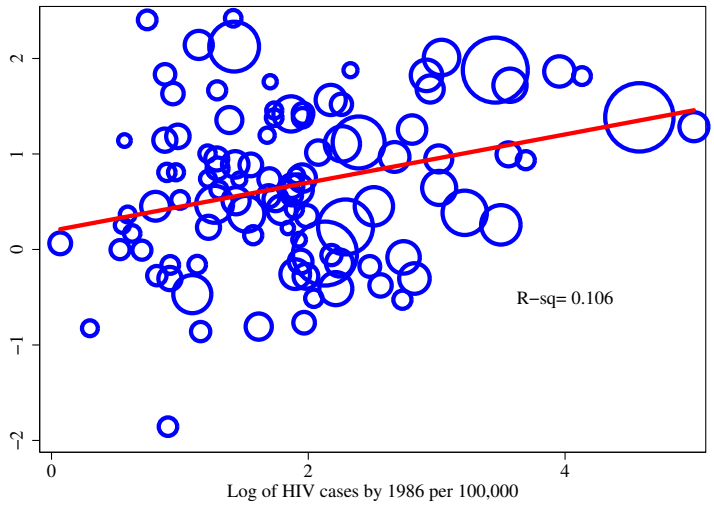


Table 2: Summary statistics for thirty PMSAs with respect to AIDS cases by 1986

	(1) 1985 HIV cases	(2) 1993 CI	(3) Arrival	(4) Coke deaths	(5) Arr. (chiefs)
New York, NY	199.6801	4.624	1985	3.953756	1985
San Francisco, CA	93.09067	4.871	1987	1.933056	1985
Jersey City, NJ	34.65165	1.884	1989	5.423984	
Miami, FL	28.17108	1.011	1983	5.059124	
West Palm Beach, FL	20.97552		1983	1.73708	
Newark, NJ	19.5071	3.087	1987	4.958433	1985
Los Angeles, CA	18.56235	2.626	1983	5.201303	1984
Fort Lauderdale, FL	15.81222	2.529	1985	2.230209	
Houston, TX	15.13994	1.687	1989	1.023472	
Washington, DC	14.20408	1.731	1986	.8760538	1986
Bergen-Passaic, NJ	11.36917		1992	2.503051	
Atlanta, GA	10.16422	3.18	1984	5.50685	1981
Dallas, TX	9.828574	1.884	1986	1.008875	1986
New Orleans, LA	9.817456	2.629	1987	3.267796	1986
Oakland, CA	9.649446	4.286	1987	4.512908	1985
Austin, TX	8.204413	.617	1985	.4726864	
Seattle, WA	8.053202	4.29	1985	.3442923	
San Diego, CA	7.626839	2.108	1989	.5604448	1984
Denver, CO	7.488613	1.571	1989	2.895908	1986
Middlesex, NJ	7.445991		1992	.5883304	
Mean for reg. samp.	2.415385	1.553641	1990.487	1.308162	1985.963

Table 3: Relationship (weighted) between 1989 crack index and 1986 AIDS cases (state-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.905***	0.899***	0.835***	0.993***		0.709***	0.729***	0.647***
1986 per 100,000	[0.122]	[0.151]	[0.230]	[0.194]		[0.187]	[0.184]	[0.213]
Min(Dist to NY, Dist					-0.139***	-0.0891***	-0.0749***	-0.0170
to MIA, Dist to LA)					[0.0226]	[0.0197]	[0.0220]	[0.0443]
Log of 1982-1984							-0.568	-0.981*
homicide rate							[0.370]	[0.552]
Mean, dep. var.	2.240	2.240	2.240	2.240	2.240	2.240	2.240	2.240
R-sq	0.597	0.764	0.608	0.774	0.752	0.829	0.840	0.873
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	50	50	50	50	50	50	50	50

Table 4: Relationship (weighted) between 1993 crack index and 1986 AIDS cases (state-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.906***	1.024***	1.183***	1.132***		1.022***	1.030***	0.753***
1986 per 100,000	[0.166]	[0.122]	[0.163]	[0.149]		[0.162]	[0.162]	[0.209]
Min(Dist to NY, Dist					-0.106***	-0.0345	-0.0290	0.0127
to MIA, Dist to LA)					[0.0340]	[0.0240]	[0.0240]	[0.0422]
Log of 1982-1984							-0.222	-0.450
homicide rate							[0.369]	[0.518]
Mean, dep. var.	1.985	1.985	1.985	1.985	1.985	1.985	1.985	1.985
R-sq	0.656	0.810	0.741	0.822	0.654	0.831	0.832	0.865
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	50	50	50	50	50	50	50	50

Table 5: Relationship (weighted) between crack arrival date and 1986 AIDS cases (state-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	-1.402***	-0.812**	-0.787**	-0.981***		-0.941***	-0.878***	-1.331**
1986 per 100,000	[0.280]	[0.300]	[0.345]	[0.290]		[0.313]	[0.308]	[0.476]
Min(Dist to NY, Dist					0.0963	0.0152	0.0566	0.0650
to MIA, Dist to LA)					[0.0671]	[0.0648]	[0.0619]	[0.119]
Log of 1982-1984							-1.380**	-2.039**
homicide rate							[0.626]	[0.717]
Mean, dep. var.	1984.6	1984.6	1984.6	1984.6	1984.6	1984.6	1984.6	1984.6
R-sq	0.556	0.784	0.655	0.796	0.742	0.797	0.820	0.909
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	41	41	41	41	41	41	41	41

Table 6: Relationship (weighted) between 1990s cocaine mortality and 1986 AIDS cases (state level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.287***	0.267*	0.203	0.250		0.143	0.143	0.323*
1986 per 100,000	[0.0832]	[0.134]	[0.175]	[0.177]		[0.151]	[0.153]	[0.188]
Min(Dist to NY, Dist					-0.0447*	-0.0345*	-0.0375*	-0.0304
to MIA, Dist to LA)					[0.0222]	[0.0180]	[0.0219]	[0.0477]
Log of 1982-1984							0.126	-0.0341
homicide rate							[0.394]	[0.634]
Mean, dep. var.	0.683	0.683	0.683	0.683	0.683	0.683	0.683	0.683
R-sq	0.256	0.449	0.389	0.496	0.518	0.532	0.535	0.581
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	49	49	49	49	49	49	49	49

Table 7: Relationship (weighted) between 1989 crack index and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.779***	0.577***	0.563**	0.664***		0.572***	0.617***	0.617***
1986 per 100,000	[0.113]	[0.189]	[0.213]	[0.216]		[0.213]	[0.206]	[0.206]
Min(Dist to NY, Dist					-0.0839**	-0.0552	-0.0435	-0.0435
to MIA, Dist to LA)					[0.0364]	[0.0359]	[0.0355]	[0.0355]
Log of 1982-1984							-0.229	-0.229
homicide rate							[0.321]	[0.321]
Mean, dep. var.	2.365	2.393	2.393	2.393	2.393	2.393	2.393	2.393
R-sq	0.384	0.546	0.517	0.552	0.505	0.574	0.578	0.578
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	82	79	79	79	79	79	79	79

Table 8: Relationship (weighted) between 1993 crack index and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.731***	0.633***	0.774***	0.934***		0.844***	0.919***	0.919***
1986 per 100,000	[0.146]	[0.160]	[0.159]	[0.151]		[0.147]	[0.149]	[0.149]
Min(Dist to NY, Dist					-0.0958**	-0.0535*	-0.0339	-0.0339
to MIA, Dist to LA)					[0.0379]	[0.0287]	[0.0291]	[0.0291]
Log of 1982-1984							-0.383	-0.383
homicide rate							[0.271]	[0.271]
Mean, dep. var.	2.195	2.217	2.217	2.217	2.217	2.217	2.217	2.217
R-sq	0.365	0.594	0.584	0.673	0.533	0.695	0.706	0.706
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	82	79	79	79	79	79	79	79

Table 9: Relationship (weighted) between 1990s cocaine mortality and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.252***	0.245**	0.226	0.223		0.188	0.119	0.119
1986 per 100,000	[0.0815]	[0.111]	[0.153]	[0.157]		[0.149]	[0.157]	[0.157]
Min(Dist to NY, Dist					-0.0291	-0.0197	-0.0383	-0.0383
to MIA, Dist to LA)					[0.0249]	[0.0235]	[0.0306]	[0.0306]
Log of 1982-1984							0.365	0.365
homicide rate							[0.252]	[0.252]
Mean, dep. var.	0.774	0.785	0.785	0.785	0.785	0.785	0.785	0.785
R-sq	0.107	0.240	0.215	0.250	0.237	0.258	0.290	0.290
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	102	98	98	98	98	98	98	98

Table 10: Relationship (weighted) between crack arrival date and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	-1.507***	-0.598	-1.053***	-0.715*		-0.590	-0.381	-0.381
1986 per 100,000	[0.250]	[0.395]	[0.366]	[0.400]		[0.382]	[0.401]	[0.401]
Min(Dist to NY, Dist					0.114	0.0898	0.174	0.174
to MIA, Dist to LA)					[0.0937]	[0.0946]	[0.113]	[0.113]
Log of 1982-1984							-1.275	-1.275
homicide rate							[1.010]	[1.010]
Mean, dep. var.	1985.9	1985.8	1985.8	1985.8	1985.8	1985.8	1985.8	1985.8
R-sq	0.389	0.530	0.487	0.534	0.530	0.549	0.574	0.574
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	57	56	56	56	56	56	56	56

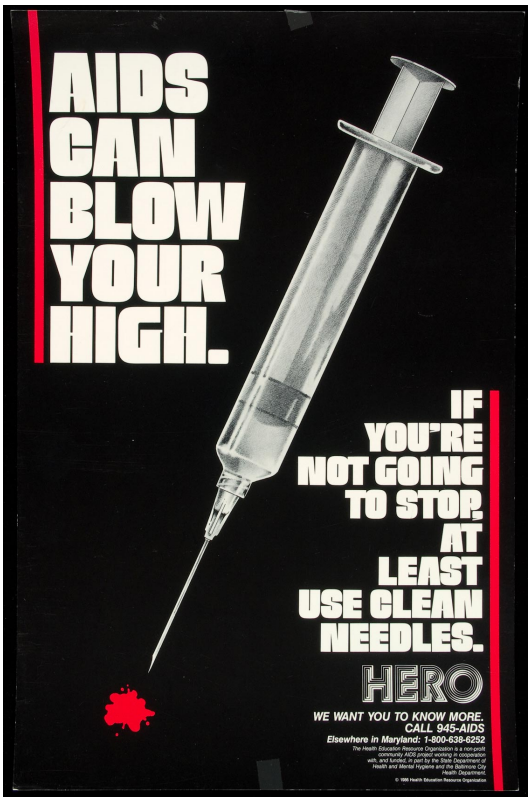
Table 11: Relationship (weighted) between cocaine deaths and 1987-1990 AIDS deaths (county-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of 1987-90 AIDS	0.416***	0.446***	0.297***	0.421***		0.382***	0.294***	0.223**
deaths per 100,000	[0.0566]	[0.100]	[0.0963]	[0.100]		[0.0962]	[0.101]	[0.0948]
Min(Dist to NY, Dist					-0.383	-0.260	-0.424	0.259
to MIA, Dist to LA)					[0.289]	[0.260]	[0.290]	[0.787]
Log of 1982-1984							0.298**	0.464***
homicide rate							[0.134]	[0.144]
Mean, dep. var.	0.734	0.761	0.761	0.761	0.753	0.753	0.753	0.753
R-sq	0.201	0.347	0.299	0.368	0.303	0.366	0.382	0.552
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
State FE?	No	No	No	No	No	No	No	Yes
Obs	391	301	301	301	301	299	299	299

Appendix A. Supplementary figures and tables noted in the text

Appendix Figure A.1: Posters emphasizing harm reduction

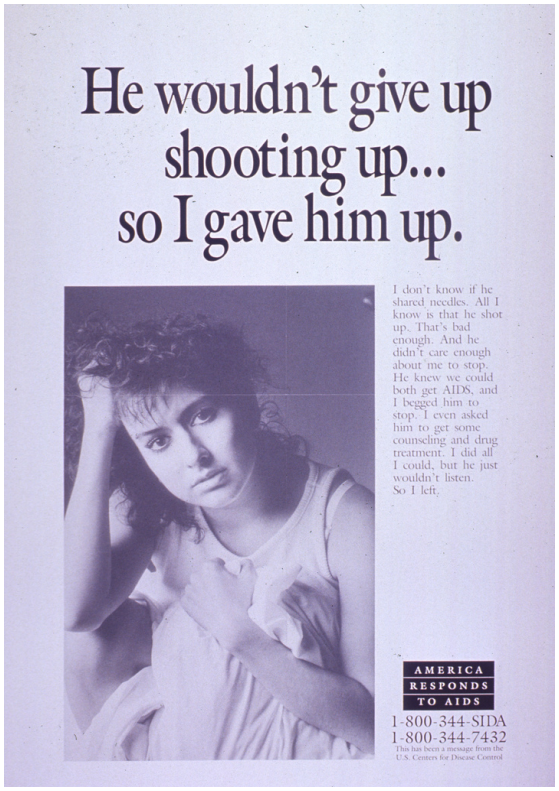
(a) Poster from (Maryland) State Department of Health and Mental Hygiene and Baltimore City Health Department



(b) Poster from Department of Human Services, Washington DC

Appendix Figure A.2: Posters emphasizing zero-tolerance, stigmatization

(a) Poster from U.S. Center for Disease Control

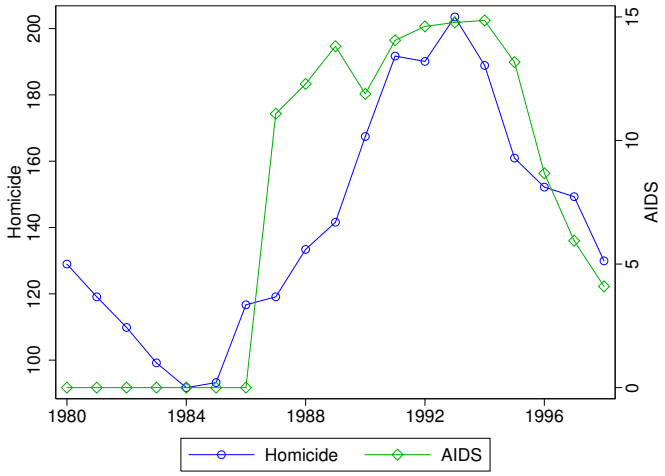


(b) Poster from CDC

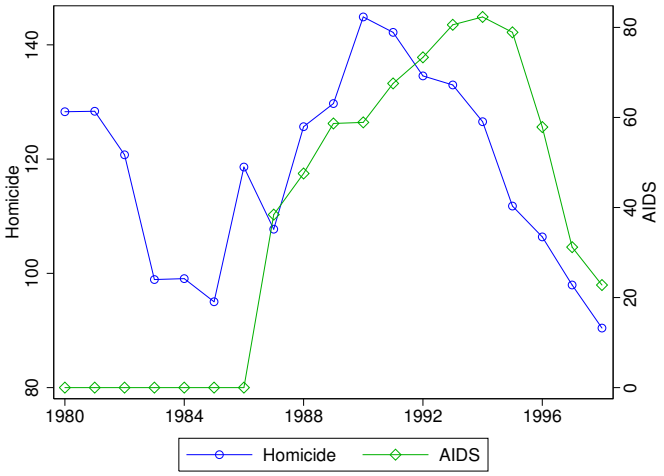
(c) Poster from U.S. Department of Health and Human Services

Appendix Figure A.3: Mortality rates for black males from homicide and AIDS, by age group

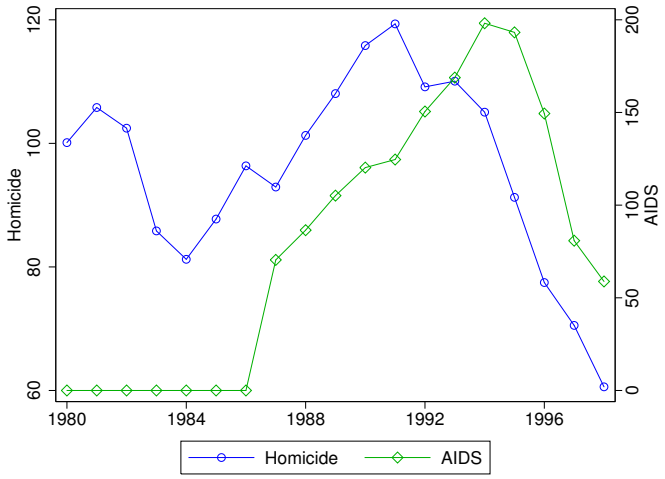
(a) Poster from U.S. Center for Disease Control



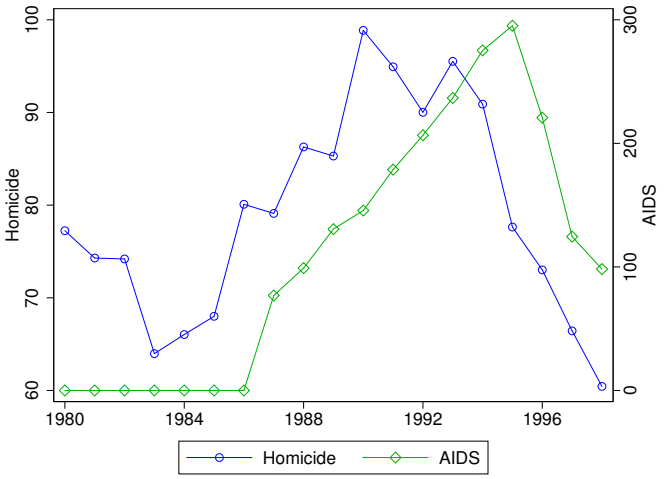
(b) Poster from U.S. Center for Disease Control



(c) Poster from CDC



(d) Poster from CDC



Appendix Table A.1: Demographics of "current" (i.e., use in past month) drug users

	Heroin 1979-1985	Crack 1988-1995	Cocaine (ex. crack) 1979-1985	Marijuana 1979-1995
White	0.552	0.482	0.802	0.760
Black	0.423	0.395	0.117	0.147
Hispanic	0.0248	0.121	0.0684	0.0719

Source: NSDUH (author's calculations).

Appendix Table A.2: Relationship (unweighted) between 1989 crack index and 1986 AIDS cases (state-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.894***	0.848***	0.836***	0.912***		0.602**	0.602**	0.336
1986 per 100,000	[0.115]	[0.171]	[0.211]	[0.239]		[0.250]	[0.254]	[0.337]
Min(Dist to NY, Dist					-0.124***	-0.0749**	-0.0649**	-0.0554
to MIA, Dist to LA)					[0.0297]	[0.0283]	[0.0301]	[0.0469]
Log of 1982-1984							-0.277	-0.459
homicide rate							[0.226]	[0.407]
Mean, dep. var.	1.651	1.651	1.651	1.651	1.651	1.651	1.651	1.651
R-sq	0.553	0.656	0.585	0.684	0.674	0.723	0.729	0.772
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	50	50	50	50	50	50	50	50

Appendix Table A.3: Relationship (unweighted) between 1993 crack index and 1986 AIDS cases (state-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.943***	0.884***	0.944***	0.952***		0.752***	0.752***	0.460
1986 per 100,000	[0.0973]	[0.143]	[0.158]	[0.176]		[0.191]	[0.195]	[0.300]
Min(Dist to NY, Dist					-0.109***	-0.0482	-0.0438	-0.0288
to MIA, Dist to LA)					[0.0294]	[0.0290]	[0.0313]	[0.0491]
Log of 1982-1984							-0.123	-0.335
homicide rate							[0.249]	[0.348]
Mean, dep. var.	1.407	1.407	1.407	1.407	1.407	1.407	1.407	1.407
R-sq	0.616	0.702	0.639	0.748	0.687	0.764	0.765	0.783
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	50	50	50	50	50	50	50	50

Appendix Table A.4: Relationship (unweighted) between crack arrival date and 1986 AIDS cases (state-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	-0.987***	-0.715**	-0.557*	-0.698*		-0.670	-0.626	-1.389**
1986 per 100,000	[0.265]	[0.307]	[0.307]	[0.343]		[0.394]	[0.390]	[0.525]
Min(Dist to NY, Dist					0.0575	0.00911	0.0214	-0.0291
to MIA, Dist to LA)					[0.0559]	[0.0634]	[0.0622]	[0.114]
Log of 1982-1984							-0.677	-1.430**
homicide rate							[0.573]	[0.567]
Mean, dep. var.	1985.5	1985.5	1985.5	1985.5	1985.5	1985.5	1985.5	1985.5
R-sq	0.327	0.678	0.611	0.686	0.655	0.686	0.700	0.835
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	41	41	41	41	41	41	41	41

Appendix Table A.5: Relationship (unweighted) between 1990s cocaine mortality and 1986 AIDS cases (state level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.466***	0.417***	0.382**	0.389**		0.400**	0.397**	0.704***
1986 per 100,000	[0.0791]	[0.128]	[0.159]	[0.169]		[0.191]	[0.193]	[0.222]
Min(Dist to NY, Dist					-0.0275	0.00274	0.00762	0.0206
to MIA, Dist to LA)					[0.0207]	[0.0219]	[0.0237]	[0.0293]
Log of 1982-1984							-0.144	-0.334
homicide rate							[0.274]	[0.300]
Mean, dep. var.	0.422	0.422	0.422	0.422	0.422	0.422	0.422	0.422
R-sq	0.401	0.566	0.506	0.569	0.510	0.569	0.573	0.669
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Division FE?	No	No	No	No	No	No	No	Yes
Obs	49	49	49	49	49	49	49	49

Appendix Table A.6: Relationship (unweighted) between 1989 crack index and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.762***	0.581***	0.452**	0.536***		0.509***	0.534***	0.534***
1986 per 100,000	[0.144]	[0.177]	[0.184]	[0.187]		[0.187]	[0.192]	[0.192]
Min(Dist to NY, Dist					-0.0383	-0.0182	-0.0126	-0.0126
to MIA, Dist to LA)					[0.0312]	[0.0295]	[0.0297]	[0.0297]
Log of 1982-1984							-0.123	-0.123
homicide rate							[0.256]	[0.256]
Mean, dep. var.	1.683	1.723	1.723	1.723	1.723	1.723	1.723	1.723
R-sq	0.334	0.505	0.479	0.514	0.453	0.517	0.518	0.518
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	82	79	79	79	79	79	79	79

Appendix Table A.7: Relationship (unweighted) between 1993 crack index and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.704***	0.530***	0.505***	0.595***		0.544***	0.567***	0.567***
1986 per 100,000	[0.119]	[0.135]	[0.131]	[0.147]		[0.149]	[0.162]	[0.162]
Min(Dist to NY, Dist					-0.0563**	-0.0348	-0.0296	-0.0296
to MIA, Dist to LA)					[0.0264]	[0.0239]	[0.0234]	[0.0234]
Log of 1982-1984							-0.113	-0.113
homicide rate							[0.248]	[0.248]
Mean, dep. var.	1.551	1.578	1.578	1.578	1.578	1.578	1.578	1.578
R-sq	0.335	0.572	0.504	0.576	0.504	0.589	0.590	0.590
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	82	79	79	79	79	79	79	79

Appendix Table A.8: Relationship (unweighted) between crack arrival date and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	-1.271***	-0.421	-1.150***	-0.647		-0.524	-0.303	-0.303
1986 per 100,000	[0.236]	[0.392]	[0.375]	[0.462]		[0.453]	[0.532]	[0.532]
Min(Dist to NY, Dist					0.0973	0.0782	0.147	0.147
to MIA, Dist to LA)					[0.0868]	[0.0880]	[0.108]	[0.108]
Log of 1982-1984							-1.125	-1.125
homicide rate							[0.973]	[0.973]
Mean, dep. var.	1986.7	1986.7	1986.7	1986.7	1986.7	1986.7	1986.7	1986.7
R-sq	0.243	0.397	0.324	0.412	0.411	0.425	0.447	0.447
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	57	56	56	56	56	56	56	56

Appendix Table A.9: Relationship (unweighted) between 1990s cocaine mortality and 1986 AIDS cases (PMSA-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of HIV cases by	0.210***	0.213**	0.231**	0.204*		0.190*	0.158	0.158
1986 per 100,000	[0.0752]	[0.0909]	[0.104]	[0.113]		[0.113]	[0.119]	[0.119]
Min(Dist to NY, Dist					-0.0178	-0.0107	-0.0169	-0.0169
to MIA, Dist to LA)					[0.0210]	[0.0214]	[0.0238]	[0.0238]
Log of 1982-1984							0.168	0.168
homicide rate							[0.161]	[0.161]
Mean, dep. var.	0.673	0.697	0.697	0.697	0.697	0.697	0.697	0.697
R-sq	0.0580	0.216	0.168	0.231	0.212	0.234	0.243	0.243
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
FE?	No	No	No	No	No	No	No	Yes
Obs	102	98	98	98	98	98	98	98

Appendix Table A.10: Relationship (unweighted) between cocaine deaths and 1987-1990 AIDS deaths (county-level)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log of 1987-90 AIDS	0.323***	0.298***	0.271***	0.300***		0.290***	0.235***	0.257***
deaths per 100,000	[0.0365]	[0.0605]	[0.0599]	[0.0617]		[0.0658]	[0.0712]	[0.0793]
Min(Dist to NY, Dist					-0.0804	0.0505	-0.0492	0.902*
to MIA, Dist to LA)					[0.161]	[0.159]	[0.172]	[0.528]
Log of 1982-1984							0.214**	0.313***
homicide rate							[0.0983]	[0.112]
Mean, dep. var.	0.609	0.634	0.634	0.634	0.636	0.633	0.633	0.633
R-sq	0.151	0.316	0.309	0.334	0.281	0.332	0.344	0.485
EGM controls	No	Yes	No	Yes	Yes	Yes	Yes	Yes
FHLM controls	No	No	Yes	Yes	Yes	Yes	Yes	Yes
State FE?	No	No	No	No	No	No	No	Yes
Obs	391	301	301	301	301	299	299	299