"The White/Black Educational Gap, Stalled Progress, and the Long Term Consequences of the Crack Epidemic"

William N. Evans
Department of Economics
University of Notre Dame
437 Flanner Hall
Notre Dame, IN 46556
P: (574) 631 7039
E: wevans1@nd.edu

Craig Garthwaite
Kellogg School of Management
Northwestern University
2001 Sheridan Road
Evanston, IL 60208
P: (202) 746 0990
E: c-garthwaite@kellogg.northwestern.edu

Timothy J. Moore
Department of Economics
University of Maryland
College Park, MD 20742
P: (301) 442 1785
E: moore@econ.umd.edu

Abstract
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We propose the rise of crack cocaine as an explanation for the end to the convergence in black-white educational outcomes beginning in the mid-1980s. After constructing a measure of the arrival of crack arrival in cities and states, we first show there are large increases in incarceration and murder rates after the arrival of the drug. We show that the emergence of crack accounts for between 39 and 71 percent of the fall in black male high school graduation rates. The results suggest that, in line with human capital theory, educational investments declined in response to decreased returns to schooling.

I. Introduction

Historically, there are persistent differences between the high school graduation rates and standardized test scores of white and black students.[1] These differences narrowed between the mid-1960s and the late-1980s as the educational outcomes of black students improved dramatically. Then, for reasons that previous academic and policy researchers have been unable to explain, this progress stopped. We argue that the introduction and spread of crack cocaine in the 1980s and 1990s explains a large fraction of the "stalled progress" in black educational outcomes.

The trends for high school graduation rates are shown in Figure 1. To produce data on high school graduation rates we use data from the pooled 2005-2009 American Community Survey (ACS) (Ruggles et al., 2010).[2] We place U.S. born, non-Hispanic white and black ACS respondents into cohorts based on the year they turned age 18. Figure 1a contains the race-specific high school graduation rates for cohorts turning 18 between 1967 and 2004, while Figure 1b shows the white-black difference in these rates. A number of facts in these figures are of note. First, the gap in graduation rates halved between 1967 and 1986, falling from 9.2 to 4.4 percentage points. Second, almost all of the convergence is due to rising black achievement—the graduation rates of whites changed little over this period. Third, the convergence ends in 1986 and white-black graduation rates diverge until 2003 when the gap is 7.8 percentage points. Fourth, as with the convergence, the divergence is almost entirely driven by black graduation rates, which declined by 4.4 percentage points between 1986 and 2003. This wiped out much of the previous progress: the black cohort that turned 18 in 2003 had a graduation rate similar to the 1972 cohort.

The decline in graduation rates for blacks is primarily driven by the outcomes for males. We report the high school graduation rates in the ACS by race and sex in Figure 1c and the sex-specific white-black difference in these rates in Figure 1d. Between the 1967 and 1986 cohorts, high school graduation rates increased by 4.7 and 6.7 percentage points for black females and males, respectively.

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[1] For data on the test score gap, see Jencks and Phillips (1998), Neal (2006) and Magnuson and Waldfogel (2008). For data on the high school graduation gap, see Rivkin (1995), Heckman and LaFontaine (2010).

[2] High school dropout rates for the population 16-24 are reported over time in the *Digest of Education Statistics*. These estimates are calculated from the October School Enrollment Supplement to the *Current Population Survey* which is the only annual series that provides information on school enrollment and attainment for teenagers back to the 1960. This survey is limited in that there are roughly 200 blacks per age group. The high school completion rates in our ACS sample are higher than in the October CPS for two reasons. First, for many years in the CPS, GEDs were not counted as high school graduates but they are in the ACS. Second, as a cohort ages, it has more opportunity to obtain a GED so high school graduation rates tend to rise in the years with cohort age. See Neal (2006) for a comparison of white-black differences for high school graduation rates with and without counting the GED.

Between 1986 and 2003, graduation rates for black males fell by 5.7 percentage points while, over the same period, the rate for black females fell by one percentage point.

A number of hypotheses have been put forward to explain the convergence in educational outcomes through the mid-1980s, including improved parental education (Armor, 1992), reduced segregation (Jayne and Williams, 1989; Grissmer et al., 1998), federal spending on education (Koretz, 1987), and changes in school spending (Boozer et al., 1992). Cook and Evans (2000) estimated that factors such as rising parents' education and school characteristics explain only 25% of the convergence in standardized test scores for 13 year olds, and suggest that 75% of the convergence occurred in racially-integrated schools. Chay, Guryan and Mazumder (2009) found that the convergence was due to improvements by black students in the South, and provided suggestive evidence that these improvements were due to increased access to health care early in life.

Relatively less attention has been paid to the end of this convergence in the 1980s and this small literature has to date failed to provide solid evidence explaining the stalled progress of black educational outcomes. Neal (2006) used data from the ACS, the National Assessments of Education Performance Long Term Trend Assessments (NAEP-LTT), and a variety of other sources to document the stalled progress. He then considers a number of possible explanations, including changes in the race-specific education-wage relationship, income and other shocks to black families, school factors, and the role of culture. None provide an adequate explanation and Neal concludes: "It is not clear why the process of black-white skill convergence appeared to stop around 1990" (p. 570).

Magnuson and Waldfogel (2008) edited a nine-chapter volume on the potential sources of stalled progress. Contributors concluded that while factors such as changing family income, rising income inequality, relative changes in parental education, changes in school segregation, and changes in teacher quality may account for a slowing convergence, none provide a comprehensive explanation. In the final chapter, Ferguson (2008, p. 321) noted that "…researchers, parents, activists, and policy makers still seek to understand why progress stalled in 1990."

A previously unexplored cause for the stalled progress is the widespread emergence of crack cocaine markets. Crack cocaine was an innovation that cut the price of cocaine and, as a result, dramatically expanded the market size and profits from drug dealing. The drug first appeared in Miami, New York, and Los Angeles around 1982, and then spread to cities across the nation over the next decade. Intense competition between entrepreneurial suppliers of crack cocaine produced unprecedented violence and incarceration. Young black males were most adversely impacted by the

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crack epidemic, with a tripling of their homicide and incarceration rates. Selling crack cocaine also provided an opportunity for black males to earn money without formal educational credentials.

We hypothesize that decreased life expectancy, increased likelihood of imprisonment, and new source of informal earnings lowered individuals' expected returns to human capital and subsequent investment in education. We are not the first to propose this possible connection. Neal (2006) suggested that future research should consider the crack cocaine epidemic as a possible explanation for the lack of continued convergence in educational outcomes. Fryer et al. (forthcoming) also mentioned that part of the destructiveness of the crack cocaine epidemic may have been that increased returns to drug dealing reduced educational investments. However, neither of these works documented a relationship between the crack epidemic and educational outcomes.

The changing mortality risks over this time were large in absolute terms. To better understand the magnitude of these changes consider the case of 15-year-old black males—students who are likely contemplating investing in a high school diploma. One way these individuals may calculate their chance of dying by the age of 30 is to examine the age-specific mortality rates of black males aged between 15 and 29 years who are living in the same city.[3] Figure 2 shows these mortality calculations between 1980 and 2000 for the District of Columbia (DC), Dallas, Saint Louis, and New Orleans, using the National Center for Health Statistics' Multiple Cause of Death data. In DC, the expectation that a 15-year-old black male would die before age 30 based on contemporaneous death rates was 4% prior to 1985, rises steadily to 12% by 1993, and then declines to 6% by 2000. For Dallas, Saint Louis and New Orleans, three representative cities in other parts of the U.S., the figures for a young black male's perceived probability of death by age 30 show increases between the early 1980s and the early-to-mid 1990s of 3.5 to 8 percentage points. These changes in perceived mortality risks are broadly similar to those resulting from the AIDS epidemic in Africa (Chicoine, 2012). In contrast, the changes in perceived mortality risks for white males, white females, and black females in DC over this period were less than one percent.

Ethnographic accounts and survey data document shifts in the perceived mortality risks of young black males during this time. Surveys suggest that a large percentage of school age children were keenly aware of the violence associated with the drug trade. High school students in Washington, DC who were not selling drugs estimated that the 12-month risk of death or severe

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[3] That is, the expected mortality rate by age 30 is the sum the contemporaneous mortality rates of 15 year olds, 16 year olds (conditional on living to 16), 17 year olds (conditional on living to 17), and so on, up to and including those aged 29.

injury of drug dealers was 61% (Reuter et al., 1990). In a study of criminal offenders in Boston and Los Angeles, Hoffman (2004) found that young black males accepted the possibility of an early death as normal or expected. Similarly, Anderson (1994, p. 94) said, "[m]any are uncertain about how long they are going to live and believe they could die violently at any time. They accept this fate." Numerous studies in the adolescent mental health literature have found that expectations of premature death among school age children are correlated with lower adult socioeconomic status including lower levels of education (Nguyen et al., 2012), worse adult health outcomes (Borowsky et al., 2009; Duke et al. 2011), higher adolescent violence (Stoddard et al, 2011) and increased self-destructive behaviors (Duke et al., 2009; McDade et al. 2011).

We exploit city- and state-level variation in the timing and severity of the emergence of crack cocaine markets to examine their impact on the white-black education gap. Using cocaine-related deaths recorded in the Multiple Cause of Death data, which were rare before the introduction of crack cocaine, we date the arrival of crack markets in 57 cities and 41 states. Our dates correspond with newspapers and law enforcement reports, as well as those used by Grogger and Willis (2000) in their study of the crack cocaine epidemic and urban crime. Importantly, we show that these arrival dates appear to have more to do with geography and population size than local socioeconomic indicators, demonstrating the crack markets did not systematically emerge in areas that were already trending negatively on educational outcomes or other socioeconomic characteristics.

The arrival of crack cocaine is strongly predictive of increases in both the murder rate of young black males and decreases in their high school graduation rates. This latter relationship is shown in Figure 3a, where we report the average graduation rate for black and white non-Hispanic males based on the year crack arrived in their MSA in comparison to the when they turned age 18 years. The zero on the horizontal axis is the year crack cocaine arrived.[4] Consistent with the previously discussed convergence of white-black outcomes prior to the late 1980s, racial differences in male graduation rates narrowed before crack cocaine arrived. Beginning approximately two years after the emergence of crack markets, the 18-year-old black male graduation rate falls. In Figure 3b, we see a similar pre-crack increase in graduation rates for non-Hispanic black females, but a much less pronounced drop following the arrival of crack.

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[4] This data is taken from the IPUMS version of the 2000 Census 5% Public Use Micro Samples (Ruggles et al., 2010). The respondents are residents in 2000 of the 57 largest MSA used in Table 3. When the cohorts turn 18 is calculated as (2000- current age +18).

There could be a concern that the arrival of crack was coincident with other changes in cities impacting educational outcomes. For example, a declining manufacturing sector or race-based changes in school quality are potential confounding factors. However, if these alternative events were the actual cause of the stalled progress in educational outcomes there should be a similar decline in human capital investment for both females and younger black males. Instead, our estimated impacts of crack markets on black female high school graduation rates are small in magnitude and generally lack statistical precision. Furthermore, as we document below, standardized test scores for nine year old black males continued to improve over this time period, a result in stark contrast to the test scores of those at older ages. An additional concern may be that the use of crack by students, rather than changes in the costs and returns of human capital caused the decline. Survey evidence demonstrates that the use of crack by school-age children was low in absolute terms and similar across races, making it unlikely that the use of crack explains the halted convergence.

In addition to geographic variation in the timing of the introduction of crack, there were also large differences in the virulence of the epidemic across cities and states. We develop two geographic and time-varying measures of the severity of the epidemic based on city and state level murder and incarceration rates. First, we correlate the local murder rates of young adults during the period each cohort was aged 16 to 18 years with cohort-specific high school graduation rates. When we do this for race- and sex-specific cohorts, we find that a higher murder rate is connected to a decrease in the high school graduation probability of males but not females. Second, using state-level data from the Bureau of Justice Statistics' National Corrections Reporting Program, we calculate the annual race-specific fractions of males aged 20 to 24 years that entered prison in the year each cohort turned 18 years old. We correlate this alternate measure of the virulence of local crack markets with male high school graduation rates. The percentage of 20- to 24-year-olds entering prison when a cohort turns 18 is also a strong predictor of high school graduation rates. A model containing both incarceration and murder rates finds that both are independently predictive of changes in the educational outcomes for black males. Depending on the assumption about the continued progress of black males, the combined effect of these two factors accounts for between 39 and 71 percent of the change in graduation rates during this period.

By 2004, the murder rate for young black males had receded from its peak in the 1990s. At this time, the pace of the decline in black male high school graduation rates also dramatically slowed but did not return to the pre-crack trend of white-black convergence. There are two reasons why this

is likely the case. First, the white-black difference in incarceration rates remained large over this time. Second, and perhaps more importantly, the emergence of crack markets delivered a shock to the stock of individuals in the community with a high school diploma. Given the intergenerational and cultural transmission of education (Sacerdote, 2007; Bjorklund et al., 2006), it may not be reasonable to expect an immediate return to the pre-crack convergence following such a large shock.

Beyond the primary question of understanding changes in the differential educational outcomes between white and black students, these estimates provide insights into whether factors that limit the benefits of education lower human capital investments. Previous tests of this hypothesis have exploited shocks to the length of life that should be unrelated to other factors in the human capital investment decision. Jayachandran and Lleras-Muney (2009) found increased female education in post-World War II Sri Lanka following a sharp drop in maternal mortality. Fortson (2010) showed that areas of sub-Saharan Africa with the largest increases in mortality from the HIV epidemic subsequently experienced the greatest decline in educational attainment. Finally, Oster et al. (2012) estimated that individuals who learn that they possess the genetic mutation responsible for the fatal Huntington's disease acquire lower levels of education and participate in less job training.

The emergence of crack has three primary impacts on young black males: an increased probability of being murdered, an increased risk of incarceration, and a potentially higher income from outside the legal labor market. Each of these factors limits the benefits of education. Our results demonstrate that the crack epidemic explains a majority of the stalled progress in black male educational outcomes. This analysis provides further evidence that individuals are investing in human capital in a manner consistent with the models of Becker (1964) and Ben-Porath (1967).

II. The Crack Cocaine Epidemic

Crack cocaine was introduced from the Caribbean to the U.S. around 1981, first in Miami and soon after in Los Angeles and New York. The drug is made by cooking powder cocaine with baking soda, which can then be broken up and smoked once it cools and hardens. Although cocaine was already available in other forms, crack cocaine was easier to produce than other types of smokeable cocaine and produced a more intense, shorter high than intranasally ingested powder cocaine (Agar, 2003).[5] Crack cocaine users report that smoking the drug yields a high lasting 20 minutes, which is

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[5] Crack was not the first means for drug users to realize the intense, albeit brief, high that comes from smoking cocaine. Previously, wealthier drug users transformed cocaine into a smokeable form through "freebasing," which required users to heat powdered cocaine

followed by a sharp crash and an intense drive to get high again (Fagan and Chin, 1989). The drug proved to be popular with existing and new users of cocaine, and could be extremely profitable for dealers. Adler (1995) reported an individual earning $10,000 from converting one ounce of powder cocaine into crack and selling it in Detroit over the 1984 July 4th weekend. Police estimated that a $5,000 investment in cocaine could yield $125,000 in crack sales (GAO, 1989).

The simple technology and small quantities of cocaine required to make crack meant that it could be locally cooked and distributed by small and decentralized organizations.[6] Simon and Burns (1997, p. 63) described the crack market in Baltimore as "a freelance market with twenty-year-old wholesalers supplying seventeen-year-old dealers. Anyone could ride Amtrak or the Greyhound to New York and come back with a package." This was unlike existing drug markets that required large amounts of capital or expertise, and were primarily controlled by long established organized criminal enterprises. As would be expected in any industry, the combination of low entry barriers and high profit potential led to large amounts of entry and fierce competition (Fagan and Chin, 1990). For example, qualitative data from 1,500 crack cocaine sellers in New York suggest that the majority of low-level dealers were entrepreneurs who owned the drugs (Caulkins et al., 1999).

The potential for violence resulted partly from the type of customers demanding crack and its usual place of sale. The relatively low price of an individual sales unit of crack made hard drugs affordable to a population that was shut out of the market for existing products such as powder cocaine and heroin. Blumstein (1995, p. 30) said, "an important feature of crack is its low price, which brought into the cocaine market many low-income people who could buy it one 'hit' at a time; this significantly increased the number of transactions in those drug markets—both by the number of new buyers and the number of transactions each buyer engaged in per week." This stood in contrast to the higher-income powder cocaine buyers who had the funds necessary to reduce the number of illegal transactions by making larger and less frequent drug purchases.

When crack first appeared in a new market it was distributed though "crack houses"—which offered both a retail sales location and a place to immediately consume the purchases. As the popularity of the drug increased, dealers moved to open air markets on street corners where they used

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and mix it with a volatile substance such as ether (Agar, 2003). In addition to the inherent danger of this practice, freebasing required the purchase of large amounts of cocaine, an expense that kept it out of reach of most eventual crack users (Fagan and Chin, 1990). Witkin (1991) said "cocaine powder required an investment of at least $75 for a gram, but a hit of crack cost as little as $5."

[6] According to Massing (1989): "A typical crack organization would have no more than seven or eight people - a street seller or two, a steerer to direct customers, a guard to protect the merchandise, a police lookout, a weigher (known as a "scale boy"), a manager and a 'Mr. Big' to count the profits."

violence to deter entry and establish the local monopolies necessary to support elevated prices (Hamid, 1990). Felson and Bonkiewicz (2011, p. 3) said "[c]rack markets were more dangerous than marijuana markets because they were more likely to be small localized markets involving repeat sales from a small number of users. These markets increased the competition among traffickers."

This violence was primarily perpetrated using firearms (Hamid, 1990; Felson and Bonkiewicz, 2011). A diffusion of guns for self-defense further increased violence in areas with crack markets. This resulted in large increases in the homicide rates of young black males that extended into the community beyond crack cocaine users and distributors (Blumstein, 1995).

Surveys suggest that a large percentage of school age children were keenly aware of this violence. High school students in Washington DC who were not selling drugs estimated that the 12-month risk of death or severe injury of drug dealers was 61% (Reuter et al., 1990). A 1992 study of inner-city children aged 7 to 19 years in Birmingham, Alabama, found that 43% had witnessed a homicide or a body after a homicide (Morganthau, 1992). Similar figures were reported among inner-city children in other cities.[7] Some school children also carried guns. A Department of Health and Human Services survey of a nationally representative sample of 12,000 students found that 4% of respondents, and 21% of black male respondents, reported carrying a gun at least once in the previous 30 days (U.S. Department of Health and Human Services, 1991).

a. The Spread of Crack Cocaine

The availability of crack cocaine spread across the United States over the course of the 1980s and early-1990s. The three cities where the drug first appeared—Los Angeles, Miami and New York City—remained the key distribution points for cocaine over this time, and strongly influenced where crack markets later developed. While within any local area crack markets experienced large amounts of entrepreneurial entry and subsequent violence, according to law enforcement agencies and ethnographic accounts the initial emergence and establishment of crack markets in a city was primarily driven by the decisions of national trafficking groups such as the Jamaican "posses" based in New York and Miami, and "The Bloods" and "The Crips" African-American street gangs of Los Angeles (Witkin, 1991; Cooper, 1998). A 1989 *New York Times* feature on the spread of crack

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[7] Schubiner, Scott, and Tzelepis (1993) found that, among 14-23 year old black youths in Detroit, 42% had witnessed a shooting and 22% had seen someone killed. Among inner city Baltimore residents age 12-24, 42% had seen a shooting, 25% had witnessed a stabbing, and 23% saw someone murdered (Gladtstein, Rusonis, and Heald, 1992). A parental survey in a New Orleans housing project found that 71% of children had reported seen a weapon used and 39% had seen dead bodies" (Osofsky et al., 1993).

cocaine focused on the distribution networks emanating from New York City and Miami that were dominated by Jamaican "posses"—gangs formed during the violent 1980 Jamaican elections and later re-organized in the United States into armed and violent drug distributors (Massing, 1989).

As competition in these three cities became intense, enterprising groups moved to new areas where they could sell crack at higher prices (Massing, 1989; Witkin, 1991). Traffickers initially looked for cities that were easily accessible from their distribution hubs. From New York City, for example, such cities included Philadelphia and Washington DC. Crack markets were also established in many easily accessible smaller cities. The arrival of these national organizations often led to violent confrontations with the local entrepreneurs. These conflicts were exacerbated when multiple national organizations simultaneously entered an area.

b. Identifying the Arrival of Crack Cocaine

In order to estimate long run impacts of the crack epidemic on educational outcomes, we require a measure of the year that crack markets emerge in cities and states. Despite numerous ethnographic accounts of crack markets, there is no existing systematic measure of the arrival of crack across a large number of cities and states.[8] The normal difficulties associated with measuring sales and consumption in illicit drug markets are compounded here, because crack cocaine was initially not distinguished from powder cocaine in survey and administrative datasets.

Our approach is to use cocaine-related deaths to estimate when crack arrived in specific locations. Few deaths in the United States had a cocaine-related cause prior to the introduction of crack, with reported cocaine-related deaths numbering 13 in 1979, nine in 1980, and eight in 1981.[9] The number of cocaine-related deaths increased dramatically thereafter, with 67 deaths in 1985, 523 in 1989, 1,075 in 1994, and 1,497 in 1998. Given that the consumption of powder cocaine did not change substantially over this period, it seems reasonable to attribute this increase to the use of crack

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[8] Existing measures of when crack markets emerged, like those in Grogger and Willis (2000), are for fewer than 30 cities. Other measures, such as Fryer et al. (forthcoming), are indices that do not pinpoint when crack arrives.

[9] These tabulations are from the public-use versions of the National Center for Health Statistics' (NCHS) Multiple Cause of Death (MCOD) data files. They contain a unique record of each death in the United States, including information about each decedent's age, race, gender, place of residence, and cause of death. The public-use files are provided by the National Bureau of Economic Research: http://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html. Deaths in the United States between 1979 and 1998 are coded according to the 9th Revision of the International Classification of Diseases system (ICD-9) system. The relevant ICD-9 codes are 304.2 (Cocaine dependence) and 305.6 (Cocaine abuse).

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cocaine (Boggess and Bound, 1997).[10] While the cocaine-mortality relationship is not direct enough to use death records to measure the intensity of crack cocaine use (Derlet and Albertson, 1989), multiple cocaine-related deaths in a location is a consistent and widely available measure that likely indicates the presence of crack cocaine. We create consistent mortality counts from 1980 to 1998 for the 60 largest Metropolitan Statistical Areas (MSA) in terms of their 1980 population, which is every MSA with a population over 800,000 in 1980. We then define the arrival of crack as the first of two consecutive years where cocaine-related deaths are reported.

These MSAs and the estimated years that crack cocaine arrived are listed in Table 1. The years of crack arrival span 1982 to 1994. The three MSAs with the earliest arrival of crack under this measure are Los Angeles/Long Beach, Miami and New York, which matches reports on where crack cocaine first appeared in the U.S.. While it is possible this reflects powder cocaine deaths, none of these MSAs would have met the same test of consecutive cocaine-related deaths in 1980 (by having deaths in 1980 and 1981) or in 1981 (by having deaths in 1981 and 1982).[11]

The dates match closely those in Grogger and Willis (2000). These authors sent surveys to police chiefs in 1991 and asked, among other things, when they first became aware of the existence of crack. They received responses from 25 cities. In 22 cities they also used data from the Drug Abuse Warning Network (DAWN), a surveillance system that monitors drug-related visits to hospital emergency departments to identify when there is an increase in reports of smoking cocaine. The correlation between our measure and the police survey is 0.43, and between our measure and the DAWN measure is 0.37. These correlations are reasonably high, particularly given that the correlation across the two Grogger and Willis (2000) measures is only 0.32.

Our dates are also generally within a year of the earliest dates given in newspaper reports and ethnographies for larger cities.[12] The only city where our measure misses the arrival of crack by more than that is Baltimore. We place the arrival of crack there in 1989. In contrast, media reports in

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[10] Data from the 1982 National Household Survey on Drug Abuse (NHSDA) indicates that the past-year cocaine use rate was 6.4%, which was most likely all powder cocaine use. The same survey for 1995 indicates a one-year use rate of 1.7% which includes use of powder and crack cocaine. The one-year crack use rate from the 1995 survey was 0.5%.

[11] Only the Nassua/Suffolk MSA (NY) consistently reports cocaine-related deaths prior to 1981; it is omitted from the sample. These cocaine deaths are likely driven by individuals with sever cocaine addiction and problems prior to the introduction of crack. There is evidence of individuals in drug treatment for cocaine problems in the 1970s (Copemann and Shaw, 1975).

[12] Our date for Los Angeles is 1982, compared to 1981 in the *Los Angeles Times* and 1983 in a *Newsweek* article. Our date for New York is 1982, the same year as Johnson, Golub and Fagan (1995) report crack use and one year earlier than reports cited in Agar (2003). Our date for Detroit is 1985, one year later than crack dealing being reported by Adler (1995). A Kansas City police taskforce targeting a crack distributor was set up in 1986, one year after our date. The *Washington Post* first covers crack's arrival in that city in 1986, while we date its arrival in 1985. These news reports suggest that our dating measure is generally consistent with contemporaneous accounts.

the *Baltimore City Paper* and the *New York Times* discuss the presence of Baltimore crack markets in 1984 and 1987 respectively.[13]

A similar approach is taken for timing the arrival of crack cocaine in states (and the District of Columbia). The main measure is again two consecutive years where cocaine-related deaths are reported in a location; in the three most populous states of California, New York and Texas, the measure used is more than two deaths two years in a row. The crack arrival dates for 41 states are presented in Table 2.[14] The earliest crack arrival dates are 1981 for California and 1982 for Florida and New York. Overall, the state level dates are more compressed, with all 41 states having crack cocaine by 1988. The dates are roughly in accord with other evidence of when crack appeared in particular locations.[15]

c. Factors Affecting the Spread of Crack Markets

We have already noted that some media articles and ethnographies emphasize the importance of being close to the major cocaine distribution hubs of New York, Miami and Los Angeles in the development of crack cocaine markets (Massing, 1989; Cooper, 1998). Witkin (1991, p. 52) said, "America was caught in a pincer movement; Los Angeles street gangs moved east and Jamaican posses move west from the East Coast, and between them, by the end of the decade, they had introduced much of the rest of the country to crack." If true, the introduction of crack cocaine in particular cities should be related to proximity to these three cities and less influenced by existing social and economic trends that may separately influence educational outcomes. More importantly, this would suggest that crack's arrival and impact is not necessarily a function of the levels or trends in other socioeconomic characteristics of cities.

Tabulating the minimum driving distance to New York, Miami and Los Angeles by the years that crack arrives suggests there is some relationship. For cities estimated to get crack markets in 1983 and 1984, the median minimum distance from New York, Miami, and Los Angeles is 340

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[13] To understand the sensitivity of our results to these measurement issues, we also use an alternate measure: the first year of a three-year period where cocaine-related deaths are reported in at least two of the years. Under this measure, crack cocaine is estimated to arrive in Baltimore in 1985, which is much closer to the date documented elsewhere. Such differences are relatively rare, however, and the year of arrival is the same for approximately 70 percent of the MSAs and the alternate measure produces similar results.

[14] Ten states are omitted because they have small black populations and do not provide enough observations for analyzing education outcomes.

[15] Again, the alternate measure of first year of a three-year period where cocaine-related deaths are reported in at least two of the years provides largely similar dates and results.

miles, compared to 424 miles for cities with arrival dates of 1985 and 1986, 559 miles for cities with arrival dates of 1987 and 1988, and 638 miles for cities with arrival dates after 1988.

We can also examine the role of distance together with other factors using a Weibull duration model, with the dependent variable equal to the number of years after 1980 that crack cocaine is estimated to arrive. In addition to the minimum distance to New York, Miami or Los Angeles, we include the natural log of population, percentage white, percentage black, percentage of blacks in poverty, percentage of blacks who are unemployed, percentage of blacks who are high school dropouts, percentage of blacks who are high school graduates, and the percentage of blacks who have some college education. We estimate an initial model using values in the 1980 Census, and then a second model using the changes in these variables between the 1970 and 1980 Census.[16]

The results of these models are presented in Table 3. Hazard rates and 95% confidence intervals for the first model are presented in Column (1), which shows that only the log 1980 population and the minimum distance from New York, Miami, and Los Angeles are predictive of the length of time it takes for crack markets to emerge in a city. Larger cities were more likely to see crack markets emerge in earlier years, while cities that were a greater distance from the three distribution hubs were less likely to get crack in early years. The model based on changes between 1970 and 1980, which is shown in Column (2), shows a similar pattern except that the estimated coefficient on the minimum distance variable is only statistically significant at a p-value of 0.10.[17] The evidence in Table 3 demonstrates that the timing of the spread of crack markets was not strongly related to the pre-existing levels or trends in key economic variables at the city level.

III. The Introduction of Crack and Youth Murder Epidemic

In this section, we use the dates constructed in the previous section to demonstrate that the epidemic of youth murder outlined above was triggered by the introduction of crack.

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[16] The sample for this analysis contains the 50 MSAs that are identified in our crack arrival data and the 1970 and 1980 5% PUMS.

[17] It is possible that the timing of the crack epidemic was unrelated to key economic variables but the differential severity across cities was driven by socioeconomic characteristics. To explore this question, we estimated an OLS model with the same explanatory variables as the survival model and a dependent variable equal to the city specific difference in the minimum murder rate in the 1980s and the maximum murder rate in the 1990s for black males aged 20 to 24 for a sample containing the 104 MSAs jointly identified in the 1970 PUMS, 1980 PUMS, and the MCOD data. For explanatory variables defined as either levels or pre-existing trends from 1970 to 1980, the change in murder rate is negatively correlated to population and positively correlated to the minimum distance at a p-value of 0.05. No other variables are statistically significant at this p-value. For the levels specification, a one standard deviation change in the minimum distance variable was associated with an increased in the murder rate of 29 per 100,000. The negative estimate for log 1980 population corresponds to the graphical evidence in Figure 5d showing that smaller cities had the largest increases in their murder rate during this time period. For a specification with explanatory variables in levels and a dependent variable equal to change in the murder rate from 1973 to 1980 the estimate (standard error) for the coefficient on the minimum distance variable was 0.0013 (0.034).

a. The Rapid Rise in Murder Rates in the 1980s

Several measures of crime and violence increased sharply from the mid-1980s, especially for young black males. Consider the homicide rate which is defined as deaths per 100,000 residents.[18] The aggregate homicide rate in 1980 was 10.7. This rate declined to 8.3 in 1985, before increasing to 10.4 in 1991. After remaining at around 10 over the next three years, the murder rate declined and was 5.9 in 2000.

While the aggregate homicide rate is reasonably stable between 1980 and 1995, there are large changes in the homicide rate among younger individuals. The homicide rate for different demographic groups is shown in Figure 4. Panel A shows the rates for four age groups: individuals aged less than 15 years; those aged 15-24 years; those 25-39 years; and individuals 40 year of age and older. What is most striking is the large increase among 15-24 year olds, where the rate increases by 94 percent from 1985 to 1993. There is a 10 percent increase in the homicide rate among 25-39 year olds over the same period, while the rates for those under 15 are small and reasonably flat. The homicide rate for those 40 and over declines over throughout the period. The homicide rate among 15-24 year olds is lower than 25-39 year olds at the beginning of the period, but by 1993 it is 50 percent higher than the 25-39 age group and several times higher than other age groups.

To understand which subgroups of 15-24 year olds account for these changes, the homicide rates for 15-24 year old black males, black females, white females and white females are presented in Panel B of Figure 4. Note that because the black male homicide rate is many times higher than the rates for the other groups, it is measured on the right vertical axis while the other three groups are plotted in reference to the left vertical axis. Starting in the middle of the 1980s, there is a large increase in the homicide rate for black males aged 15-24 years. Their rate increased from 66 in 1985 to 166 in 1993, an increase of 150 percent over an eight year period. Over the same period, the homicide rates of white males and black females increased from 11 to 17 and from 14 to 22, respectively. The homicide rates for all three demographic groups decline between 1993 and 2000 by 42 to 51 percent. The homicide rate for white females is the lowest of the four groups, and is flat or declining throughout the sample period. Despite the fact that black males represent only about seven

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[18] Homicides are taken from the public-use versions of the National Center for Health Statistics' (NCHS) Multiple Cause of Death (MCOD) data files. These files contain a unique record of each death in the United States, including information about each decedent's age, race, gender, place of residence, and cause of death. The public-use files are provided by the National Bureau of Economic Research: http://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html. Population data is Census data that is compiled by Cancer SEER.

percent of the 15-24 year old population, the change in homicides for this group accounts for more than 70 percent of the total homicide change among 15-24 year olds between 1985 and 1993.

Crack related violence is often thought of as a problem limited to the inner cities of America. However, the MCOD data shows that the impact on murder from the emergence of crack markets around the nation stretched well beyond the often discussed large urban centers. The five cities with the largest trough to peak murder rate change in the 1980s and 1990s are Youngstown, OH, New Orleans, LA, Gary, IN, Shreveport, LA, and Chattanooga, TN. Each of these cities experienced an absolute increase in their murder rate greater than 300 per 100,000 residents. Other notable cities (change in murder rate, rank) are Washington, DC (280, 14), Toledo, OH (260, 19), Mobile, AL (202, 36), Los Angeles-Long Beach, CA (182, 44), New York, NY (135, 63), Cleveland, OH (121, 70), and Atlanta, GA (92, 85). A striking fact from even a casual examination of these changes is that the cities experiencing the largest absolute and relative increases in murders were located far from the coastal cities anecdotally associated with the worst crack-related violence. This fact corresponds to earlier duration model estimates and law enforcements accounts which suggest that crack-related violence was worst when multiple national organizations competed for territory (Massing, 1989; Witkin, 1991).

This pattern of large murder increases outside of the large cities can also be seen in Panel C of Figure 4, which depicts the change in the murder rate since 1980 by year and city size. The largest increase in the murder rate was for cities with a population between 250,000 and 500,000, following by cities with a population between 100,000 and 250,000. The smallest relative increase in murders was for cities with greater than 500,000 residents. By the year 2000, these cities saw their murder rates decline to roughly their 1980 level. In contrast, cities with between 250,000 and 500,000 residents had a murder rate in 2000 that was nearly twice their 1980 level.

Figures 4a-c show large increases in the homicides of individuals aged 15-24 over a period in the late 1980s and early 1990s where there was little change in the rates of other age groups. This change was primarily driven by an increase in the black male homicide rate and occurred in settings beyond the large cities typically thought of as the primary players in the crack epidemic.

b. Increased Incarceration

Beyond increasing violence, the arrival of crack was followed by large increases in incarceration. Following the widespread emergence of crack the national incarceration rate more

than doubled—with the largest increases occurring for drug offenses. Similar to the murder rates, young black males bore the brunt of this increase. From 1980 to 1996, the number of sentenced black male prisoners increased by 261 percent. Over the same time period the number of sentenced white male prisoners increased by 185 percent. These relative increases obscure the large differences in the absolute incarceration rates for these two groups. In 1996, the incarceration rate for black males was 1,574 per 100,000 compared to 193 per 100,000 for white males (Blumstein and Beck, 1999). Among the birth cohort turning 18 between 1983 and 1987, 20 percent of black males compared to 3 percent of white males will serve time in prison by age 30 (Petit and Western, 2004).[19]

These national figures also obscure heterogeneity across states. To examine state-based differences in the probability of incarceration by age and race, we construct a prison intake measure from restricted use data provided by the Bureau of Justice Statistics National Corrections Reporting Program (NCRP) from 1983 to 2000. The NCRP tracks the entry and exit from prisons and the data contain information on the admission date, state of residence, and basic demographic data.

Ideally, we would hope to construct a measure of first time incarcerations by race, sex, age, and geography. Unfortunately, the NCRP data do not report previous incarcerations for those entering prison. Therefore, we are only able to generate a measure of the percentage of each demographic group that enters prison in a particular year.[20] A further concern is that the NCRP data only begin in 1983 and there is inconsistent participation by states during each subsequent year. Although 36 states participate in an average year, only 17 states contain intake data for each year from 1983 to 1999.[21] The lack of data prior to 1983 and the small number of states reporting in the mid-1980s means that for many states there are very few years of intake data prior to the arrival of crack.

Despite these limitations, the NCRP data provide important insight into the general increase in imprisonment in the 1980s and the differences in this change by racial groups across states and over time. Panel D of Figure 4 contains the prison intake rate for males aged 20 to 24 by sex from 1983 to 1999 for the 17 states reporting data in each year. In 1983, the percentage of black males entering

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[19] Part of the increase in incarceration rates is due to the 1986 Anti-Drug Abuse Act (PL 99-570, 100 Stat. 3207) that established mandatory minimum penalties for trafficking 500 grams of powder cocaine and 5 grams of crack cocaine. Given that crack sellers were much more to be black than powder cocaine sellers, authors have pointed to the disparate levels for mandatory minimum sentences as a key reason for the increase in black male incarceration rates (Blumstein, 2003).

[20] We exclude individuals that enter because of a transfer from another prison, are there for a court commitment, returned from appeal or bond, returned escapees, or illegal entry into prison.

[21] The states participating in each year are AL, CA, CO, IL, KY, MD, MI, MN, MS, MO, NE, NH, ND, OR, TN, WV, and WI. An additional 6 states (NJ, NY, NC, NC, OH, SC, and WA) are included in 16 of the 17 years.

prison was higher than white males but the rates for both groups were generally low. From 1983 to 1984, a time period where crack was only present in a small number of areas, there was essentially no change in these aggregate prison rates. However, over the next 5 years the prison intake rate for black males increased by 2.4 percentage points compared to a 0.4 percentage point increase for white males. By 1999, the original 0.5 percentage point gap grew to 3.2 percentage points.

These national averages obscure a large amount of state-based variation that increased over time. In 1984 the state-level prison intake rates for black males aged 20 to 24 ranged from 0 to 1.4% per annum. A decade later, this range was 0.3 to 6.9% per annum. This state-based variation results from factors such as state drugs laws, sentencing policies, and the virulence of the crack epidemic.

c. The Arrival of Crack Cocaine and Youth Homicide

In the remainder of this section we use our measure of the year crack arrives in geographic areas to demonstrate the impact of the drug on the murder rates for young black males. Figure 5 documents the evolution in murder rates for different age and demographic groups over the eight years before and after the arrival of crack in the 57 MSAs for which we can date the year when crack markets first emerged.[22] Panel A contains data by age group for black males. Prior to the year crack arrived in the MSA, the murder rate for 15 to 24 year olds was relatively flat. Two years after the first appearance of crack, this murder rate begins to increase dramatically and peaks four years later. Over the six years after crack first arrived in an MSA, the murder rate doubled from 102.6 to 207.1 per 100,000. Black males between the ages of 25 and 39 also experienced a slight 12 percent increase that began three years after crack arrived. In contrast, black males aged 40 and older were unaffected by the development of crack cocaine markets in their MSAs.

Panel B displays similar murder rates for white males. Similar to their black counterparts, the 15 to 25 year old cohort saw a murder rate increases three years after crack arrives in the MSA. By 8 years after the emergence of the drug, the murder rate increased by 51 percent. It is important to note that despite this increase, the peak rate for this cohort was only 25.7 per 100,000—approximately one eighth the rate experienced by their black counterparts. Murder rates for white males aged 25 and older continued to decline at a slow steady pace even after the introduction of crack.

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[22] The MSA sample for the reduced-form analysis contains 57 rather than 60 MSAs. This reflects differential coding of MSAs in the PUMS and MCOD data. In the MCOD data that city pairs of Dallas and Fort Worth, Houston and Brazeria, and San Francisco/Oakland and Vallejo are defined as separate MSAs. In order to match the PUMS, where these cities are combined into single areas, MCOD observations are aggregated for these city pairs.

The estimates for females of both races are contained in panels C and D. There are several things of note in these graphs. Neither race is affected to the degree as males. Both 15-24 and 25-39 year old black females experienced small increases in the murder rate in the years after crack arrives. However for both of these age cohorts the murder rate eight years after crack arrives is generally similar to the rate eight years before crack's emergence. White females in the two oldest age groups have far lower murder rates and appear to be unaffected by the emergence of crack.

Despite this striking graphical evidence of the impact of crack on the murder rate of young black males, it could be that other MSA specific factors or trends are driving some portion of the increase. Therefore, we estimate the impact the arrival of crack on murder rates using a difference-in-difference regression framework. In order to control for potential confounding factors influencing murder rates over this time period, we require a comparison group that was unaffected by the emergence of crack but would be exposed to other factors affecting overall murder rates. The results in Figure 5 demonstrate that those 40 years of age and older were unaffected by the arrival of crack and for each race/gender group, they serve as the comparison group in our model.[23]

Many of the MSAs in the data have multiple years where certain demographic groups had zero counts for murders. Given these characteristics, we utilize a negative binomial count data model to generate the differences-in-differences estimate of the impact of crack on murder rates. Covariates in the model include city and year effects as well as city-specific time trends and a dummy for the control group (those 40 and above). We also include the natural log of population for the group and fix the parameter to be one so the parameter estimates can be thought of as roughly equivalent to estimates from an OLS regression with the natural log of the mortality rate as the outcome of interest.

Table 4 contains these maximum likelihood negative-binomial estimates. The key covariates are a series of dummy variables that equal 1 if the year is 0-2, 3-5, and 6-8 years after crack arrives in an MSA interacted with whether the observation is from the treatment group. The comparison group for all of the models is the race and sex specific cohort that is aged 40 and up.

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[23] Grogger and Willis (2000) analyzed the impact of crack on crime within a differences-in-differences framework where they used black males residing in suburban areas to control for the level of crime experienced by blacks living in the central city of the MSA. While this was likely appropriate for an analysis of crime locations, which were often concentrated in the center city, we are interested in the impact of murders and crack on human capital decisions. Therefore, individuals who reside in a suburban area but are killed in an urban area are still critically important to our analysis. Contrary to many commonly held perceptions of crack, there were significant impacts on murder outside of central areas. Figure 5c shows the change in the 15 to 24 year old black male murder rate based on a city's population. It is evident from this figure that there were large changes in the black male murder rate outside large cities suggesting that an identification strategy comparing suburban and urban areas is not appropriate in this context.

Panel A contains the estimates for male MSA residents. The first row of Table 4 contains the estimates for black males aged 15 to 24. In each time period after crack there is a statistically significant increase in murders. This impact peaks 6 to 8 years after the arrival of crack when the murder rate increased approximately 100% compared to the change for blacks males aged 40 and older over the same time period. The magnitude of this estimate corresponds closely to the unadjusted graphical evidence in Figure 5. The third row contains estimated coefficients for white males aged 15 to 24, who experience an approximately 49 percent increase in the murder rate 6 to 8 years after crack's arrival compared to white males aged 40 and older. This relative change is approximately one half the size of the estimate for similarly aged blacks and the pre-crack murder rates for whites are approximately one-fifth the size of blacks.

Estimates for females are displayed in Panel B. For both races, there are statistically significant increases in murder rates of younger age groups compared to their equivalent demographic group aged 40 and older. However, these estimates are uniformly small in magnitude. For example, the third row of results is for white females aged 15 to 24. In the six to eight years after crack, this group experiences an 18.1 percent increase in murder rates. Given the pre-crack murder rates for this group, this suggests a crack related increase in the murder rate of 1 per 100,000.

The graphical evidence and the regression estimates confirm the general validity of our measure of the entry of crack markets into an MSA. Soon after the MCOD overdose data indicates crack arrives in a city, there is a marked increase in the murder rates for most demographics. Black males aged 15 to 24 bore the largest burden from this increase. This amounts to a double insult for this population. Prior to crack they had the highest murder rate of any demographic and as a result of crack they experienced the largest absolute and relative surge in violence. These estimates also document important patterns in violence as crack markets emerge in an area. Our overdose measure of the arrival of crack documents the first emergence of the drug in an area. These MSA level graphs and regression estimates demonstrate that it takes a couple of years before the violence from these markets causes large increases in murder rates. It is important to keep these patterns in mind as we document the relationship between educational outcomes and the emergence of crack markets.

IV. The Arrival of Crack Cocaine and High School Graduation Rates

If crack is responsible for the stalled progress in the closing of the black-white education gap, then its emergence in each MSA or state should be followed by worsening black educational

outcomes. To investigate this relationship we use data from the 2000 Census 5% Public Use Microdata Samples (PUMS) and exploit differential timing of the arrival of crack in MSA and states. The ideal data for our purposes would describe educational outcomes and the place of residence during high school. Unfortunately, no national datasets consistently provide this information. Within our state-based PUMS sample we generate definitions for the geography of high school attendance using the state of birth and the state of residence. Across these two definitions we receive similar estimates suggesting that this data issue is not a problem for our analysis.

The first sample we construct is a MSA sample that contains PUMS respondents currently residing in an MSA who turned 18 between the years 1975 and 1999. While the MSA-level indicator of the emergence of crack markets is likely to be more precise than the state-based indicator, we must assume that the MSA of residence is the MSA where an individual attended high school. Among the respondents in this sample, 80% resided in the MSA they lived in five years previously.

Our second sample contains all individuals in the PUMS whose age cohort turned 18 between the years 1975 and 1999. The benefit of this sample is that it allows us to use all of the observations in the PUMS, including those for individuals living outside of metropolitan areas.[24] As discussed above, despite crack cocaine being commonly thought to have only impacted these metropolitan areas, black males in non-metropolitan areas experienced dramatic increases in murders over this time period. While we do not know the state where respondents attended high school, we can define this as either the state of current residence or the state of birth. In the 1990 Census 5% PUMS, 75% of high school students were living in the state where they were born, suggesting that state of birth is a reasonable proxy for the state of high school attendance.

Figures 3a and 3b were introduced previously and, in these graphs, we use data from the MSA-based PUMS sample to examine differential changes in black and white educational outcomes in relation to when crack arrives in cities. The horizontal axis describes the number of years before or after crack first arrives in a city based on our cocaine deaths indicator. The left vertical axis contains the percentage of white individuals that report a high school degree, while the right vertical axis provides the same information for blacks. Figure 3a describes the educational outcomes for males. The results are consistent with those in Figure 1 in that they show a convergence in white-black outcomes prior to the arrival of crack with the narrowing in the graduation rates due to

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[24] Given the focus on differences in educational attainment between whites and blacks, the sample only includes respondents of these two races.

increasing graduation rates for black males. Three years after crack arrives, the graduation rate for 18 year old black males starts to fall precipitously. This pattern corresponds to the earlier estimates of the impact of crack on violence across different cities, given that murder rates for young black males began to increase two years after crack arrived in a city. Figure 3b contains a similar analysis for females. Following the arrival of crack the graduation rates of black females relative to whites, although this decline is smaller than the male decline. Figure 3c and 3d contain the reduced-form analysis using the state-based PUMS sample and provides similar patterns to those at the MSA level.

The impact of the arrival of crack markets can be more rigorously examined in a regression context. The model must be specified in a way that captures a number of key time series features in the data, such as the convergence in test scores prior to the arrival of crack and the possible divergence in outcomes after the arrival of the drug. Since there are persistent differences in outcomes across geographic regions, races, and cohorts, the model must also control for these dimensions of the data. A specification that captures these characteristics is described by the model:

(1) $y_{icg} = \mu_c + \lambda_{rg} + Black_{icg} * CappedTrend_{cg}\beta_1 + Black_{icg} * YearsAfterCrack_{cg}\beta_2 + \varepsilon_{icg}$

where y_{icg} is an indicator equal one if person i from cohort c and geographic area g graduated high school and zero otherwise, μ_c represents fixed effects for each birth-year cohort, the variable $Black_{icg}$ is an indicator equal to one if the respondent is black and zero otherwise, and λ_{rg} are geographic-specific fixed effects that we allow to vary by race r. The variable $CappedTrend_{cg}$ measures trends in high school graduation before the arrival of crack cocaine. It equals one for the cohort that turns 18 in 1973, two for the 1974 cohort, and so on, until the year that crack arrives in a geographic area when it is held constant at that value throughout the rest of the sample. For example, for residents of Los Angeles, Miami, and New York who have a crack arrival date of 1982, the value of $CappedTrend_{cg}$ is set to ten for all years after 1982. The coefficient β_1 from the interaction of this variable and the race indicator $Black_{icg}$ captures the convergence in white-black high school graduation rates across cohorts prior to the arrival of crack markets.

There is a second trend variable named $YearsAfterCrack_{cg}$, which also varies by cohort and location. It equals zero in all years prior to the arrival of crack, then one in the year after crack arrives, two the next year, etc. The coefficient β_2 from the interaction of this trend variable and the race indicator $Black_{icg}$ describes the deviation from trend in white-black graduation rates after the

arrival of crack.[25] Since crack markets emerge at different times in each MSA, the cohort effects

control for age-specific secular changes in these outcomes. Under the assumption the arrival of the

drug is not correlated with the location-specific progress in graduation rates, β_2 represents the causal

impact of crack if there had been no further convergence in graduation rates. We also estimate a

specification of equation (1) that replaces the $YearsAfterCrack_{cg}$ variable with nine indicator variables

that identify the year crack arrives and the eight years afterwards.

  To better understand the role of the trend variables, define a variable $Trend_c$ that equals 1 for

the oldest cohort, 2 for the second, etc. If the arrival of crack had no race-specific impact on high

school completion rates, then β_1 (the pre-crack trend) will equal β_2 (the post-crack trend) and

$Black_{icg}CappedTrend_{cg}+Black_{icg}YearsAfterCrack_{cg}=Black_{icg}Trend_c$. Therefore, we have specified the

model to be flexible enough to measure the continued improvements for successive black cohorts if

crack had no impact on high school graduation rates.

  Table 5 contains the coefficients from both specifications of equation (1) for males and

females. The first column of Panel A contains the estimates using the MSA PUMS sample for males,

and shows that for each year prior to the arrival of crack the percentage of blacks with a high school

degree increased by a statistically significant 0.165 percentage points (p-value <0.001) compared to

their white counterparts. The estimated coefficient on $YearsAfterCrack_{cg}$ shows that after crack

arrives black males had 0.265 percentage point annual decline in high school graduation rates relative

to white males. This estimate is statistically significant at a p-value of 0.01.[26] The second column

contains similar estimates for females using the MSA sample. Black female graduation rates also

showed progress compared to whites before the arrival of crack. However, the estimated annual

impact of crack on the probability of obtaining a high school degree for females is approximately

one-third the magnitude of the estimate for males and is not statistically significant.

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[25] Because crack enters cities at different times, larger values of $YearsAfterCrack_{cg}$ will combine two effects. One is the long-term impact of crack and the other is the changing composition of cities. We have at most 8 years of post-crack graduation rates for all areas and therefore we delete observations for cohorts if they are 9 or more years after the arrival of crack in their MSA.

[26] There could be a concern that the estimated coefficients in this context are affected by changes in the educational attainment of white males. Therefore, we estimated a specification of the model for a sample including white females and black males. White females were essentially unaffected by the emergence of crack cocaine, but they are also unaffected by other factors that influence the educational attainment rates of all males regardless of race. For the MSA sample, the estimated coefficient (standard error) on the capped trend and post-crack index from this model is 0.0015 (0.0004) and -0.0027 (.0009) respectively. An alternate comparison group contains black females, who are exposed to similar non-crack related race-specific shocks but are not as affected by crack as black males. The estimate for the capped trend using black females as the comparison group is 0.00015 (0.0004). The small size and lack of statistical significance for this pre-trend should not be surprising since both groups demonstrated similar progress in graduation rates over this time period.   The post-crack index estimate (standard error) is -0.0016 (0.00078), which is smaller in magnitude than the estimate using either white males or females, but still statistically significant at a p-value of 0.05.

The last four columns of Panel A contain equivalent estimates from the state PUMS sample, with results in the third and fourth columns using state of birth to define high school attendance and the final two columns using current state of residence to define high school attendance. The estimated effect across both samples is remarkably similar. The estimated annual decreases in graduation rates after crack for males is 0.33 percentage points across both samples. The impact for females is statistically insignificant and approximately one-sixth the size of the male estimate.

Panel B of Table 5 contains results for the specification of equation (1) with indicator variables for each year after crack arrives. The estimates for males in the MSA sample is in the first column, and shows large and consistently statistically significant decreases in high school graduation rates that begin four years after the emergence of crack in an MSA. This pattern should not be surprising. Those individuals first experiencing the negative impacts from the arrival of the drug on graduation rates are members of the cohort that were approximately high school freshman when the drug first emerged in their city. A similar pattern is seen for the state-level male results, irrespective of the method used to assign high school location. The estimates for females in the MSA sample are small in magnitude and not statistically significant at conventional levels. Only the coefficients for the first and seventh year after crack arrives are statistically significant. The year-by-year estimates for females in the state sample are small, and those that are statistically significant are positive— indicating continued post-crack progress for black females compared to their white counterparts.

Recall from above that Grogger and Willis (2000) also attempted to measure how the introduction of crack cocaine in a geographic area altered crime. To examine the robustness of our findings, we estimated a version of equation (1) using the dates from their survey of police chiefs. The male estimate (standard error) for β_2 using these dates is a statistically significant -0.002 (0.0011) percentage points.[27] The corresponding estimate (standard error) for women is -0.0006 (0.0009).

V. The Local Severity of the Crack Epidemic and the Educational Attainment of Black Males

The results above document the link between the arrival of crack and the educational outcomes of young black males. In order to estimate the potential mechanisms underlying this stalled progress, we construct measures of the impact of crack on different demographic groups that vary across geographic areas—murder and prison intake rates.

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[27] Estimating a similar model using the DAWN data arrival dates, the coefficient on the index of post crack years for males is -0.0014 (0.0009). The estimate for females is -0.0003 (0.0005).

a. The Rapid Rise in the Murder Rate

If the perceived mortality risk following the introduction of crack is partly driving the reduced-form results then the educational outcomes of young black males should be correlated with their race- and sex-specific murder rate. As a measure of perceived mortality risk we construct a three-year moving average of the murder rate an individual witnesses during their high school career. For example, for an individual in the cohort that turns 18 in 1980 the perceived mortality risk would be constructed by the murder rate from 1978 to 1980. In order to avoid a potential mechanical correlation between educational outcomes and homicides, we use the murder rate of individuals between the ages of 20 and 24.[28]

Table 6 contains the estimates from a model that regresses high school graduation rates on the moving average of the race and sex specific murder rate during the high school career, cohort fixed effects, and sex- and race-specific geographic area effects. The first panel of Table 6 contains the estimates at the MSA level. Results are provided for two samples—the 57 MSA sample used for the reduced-form results and a broader sample of the 171 largest MSAs in the United States.[29] The first two columns contain estimates for both sexes from these two samples and the number in brackets is the marginal effect for blacks from 1983 to 1993 when the murder rate for blacks aged 20 to 24 increased from 54.8 to 107.1. Within the 57 MSA sample, this increase was associated with a 0.5 percentage point decrease in black male graduation rates. The broader sample of MSAs returns a similar effect of 0.6 percentage points.

Recall from above that the aggregate black murder rate masks a great deal of sex-based heterogeneity. The third and fourth column contains results for only males. From 1983 to 1993 the 20 to 24 year old black male murder rate increased from 91.6 to 192.1 per 100,000 residents. In the 57 MSA sample, this increase was associated with an approximately 0.9 percentage point decrease in high school graduate rates. For the broader sample the estimated effect of crack was a 1.1 percentage point decline. The consistently larger impact for the full MSA sample likely results from the greater impact on the black male murder rate from crack in smaller MSAs. The final columns of the top panel of Table 6 contain the estimates for females who experienced a must smaller murder rate change. The impact of crack on women is small in magnitude and statistically insignificant.

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[28] The results are robust to using the murder rate for individuals aged 15 to 24.
[29] The sample of 171 MSAs comprises all cities that can be consistently identified in the PUMS and the MCOD data over the entire time period of the analysis.

The second panel of Table 6 contains estimates for the state PUMS sample. Estimates are provided for two samples based on the respondent's state of birth and their state of residence. The estimated marginal effect of the murder rate on educational outcomes for both sexes was 0.9 percentage points in both samples. The marginal effect for males in both samples is a statistically significant (p-value <0.01) 2 percentage point decline. The increase in size and precision compared to the MSA results should not be surprising. The state sample has approximately 1.2 million more observations, and many of these non-metro areas experienced larger increases in their black male murder rate. The estimated effect for women in the third column of Table 6 was positive and statistically significant, but given the small change in the murder rate, the marginal effect for females was only 0.3 percentage points in both samples.

b. Increased Incarceration of Black Males

Recall from above that crack's emergence in a community also significantly changed the probability of incarceration rates for this cohort—a factor that further limits the expected benefits from investments in human capital (Geller, Garfinkel, and Western, 2006). We analyze the impact of the changing incarceration risk on educational attainment using the state PUMS sample with high school attendance defined as the state of birth. Table 7 contains estimates from a regression of male graduation rates on the race specific NCRP prison intake rate of 20 to 24 year olds when a respondent turned 18, state by race fixed effects, and cohort fixed effects.[30] The first panel contains the estimate from the full sample of states reporting NCRP prison intake data in any year. The marginal effect for black males in brackets in the first column suggests that the change in average intake rate from 1983 to 1993 is responsible for a 2.7 percentage point decrease in high school graduation rates. This estimated effect is slightly larger than the murder rate estimates in the previous table. The estimates in the second panel are from data for the states providing data in each year, where the marginal effect is a 4.3 percentage point decrease—approximately 60 percent larger than the full sample estimate.[31]

In order to determine the independent impacts of murder and prison rates, the third column of Table 7 contains a specification with both of these indices included as independent variables. Both of

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[30] For the murder rate we used a three year moving average of the murder rate when a cohort turns 18. Because we have so few years of data on prison entry rates, we use as the covariate the prison entry rate the year a cohort turned 18.

[31] The second column of estimates replicates the murder rate regressions from Table 7 for the sample of states in the NCRP. In the NCRP sample the marginal effect of the murder rate is a 2.4 percentage point change in graduation rates—slightly larger than the earlier estimates. Similar to the prison intake estimates, the murder rate estimate from the sample containing consistent NCRP reporting states is higher.

these factors are independently predictive of changes in the high school graduation rate. In the full sample of states, the marginal effects for black males suggests that the combined impact of these factors is a 3.4 percentage point decline in high school graduation rates, with a larger impact from the prison intake variable than the murder rate variable.

c. An Alternative Measure of the Local Severity of the Crack Epidemic

Fryer et al. (forthcoming) generated an index of the severity of crack in 144 cities across the nation. [32] This index is composed of a variety of factors such as homicide arrests, DEA cocaine seizures, and newspaper articles discussing the drug.[33] The purpose of this index is to provide a summary measure of the growth of crack across different cities and then correlate this measure with outcomes such as murder rates and child health outcomes, but not educational attainment.

We used the PUMS MSA sample to estimate an OLS model that regresses an indicator variable for high school graduation status on the Fryer et al. index interacted with the race indicator variable. We merge their index into the data based on the year a cohort turned 18, so a black respondent from New York that turns 18 in 1986 received the index number for that year. We include fixed effects for cohorts and MSA by race effects. The estimated coefficient (standard error) on this index for such a model for males is -0.0081 (0.0018). The estimate for females is -0.0013 (0.0011)—nearly one-seventh the magnitude of the male estimate and statistically insignificant at conventional levels. From 1983 to 1993 the Fryer et al. index increased by 1.85 points, suggesting that crack was associated with roughly a 1.5 percentage point decrease in black male graduation rates.

This is smaller than our estimate (2.3 percentage points) of the impact of crack eight years after its arrival. The larger estimated impact of crack from our arrival dates compared to those using the Fryer et al. index likely results from the fact that most cities experience a large increase in their index values in 1986—a degree of uniformity that is not found in our measure or in a wide variety of ethnographic accounts.[34] This appears to be driven by a surge in media coverage of the crack epidemic beginning in that year. The increased attention appears to result from several factors occurring in 1986 such as the CBS documentary *48 Hours on Crack Street,* the overdose death of

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[32] Among the 57 MSAs in our reduced-form analysis, 52 are included in the Fryer et al. index.

[33] Newspaper articles are included in this measure if they contain the name of city along with both the terms crack and cocaine.

[34] To demonstrate this point we estimated an OLS model of the 1980 to 1999 city-level index on a full set of city and year indicator variables using a sample of the 51 cities jointly identified for our measure and the index. The estimated coefficients (standard errors) from a model on the indicator variables for the years 1983, 1984, 1985, 1986, and 1987 are 0.124 (0.04), 0.2 (.045), 0.309 (0.051), 0.966 (0.1113), and 1.167 (0.118) respectively.

basketball star Len Bias, and President Ronald Reagan's re-declaration of a war on drugs.[35] Therefore, the inclusion of media coverage in the index may explain why many cities experience a large jump in their index values in 1986 despite crack having not reached all areas at that time. If multiple cities have large changes in their index values prior to the emergence of local crack markets, then the estimated impact of crack on educational outcomes would be biased downward.

VI. Potential Alternative Mechanisms Impacting Black Male Educational Attainment

There were other effects of the crack epidemic that may have impacted educational attainment. For example, using and selling crack cocaine by school-age youths or changes in family structure and school quality could have affected educational attainment. We consider each of these and show that while they may have some effect on white-black educational differences, it seems unlikely they are the primary mechanisms through which the crack epidemic affected educational outcomes.

a. Differential Crack Cocaine Use

A potential mechanism behind the changes in educational attainment is differential rates of crack cocaine consumption among school-age teens. This is an unlikely explanation for several reasons. First, crack use by individuals under the age of 18 was low over this period. According to the 1990 Monitoring the Future Survey, 1.2% of high school seniors reported crack cocaine use in the past year and 0.7% reported use in the previous year (Johnston, O'Malley, and Bachman, 1991).

Second, there is no evidence that blacks in this age range used crack at differentially higher rates than whites. According to data on drug utilization by age from the National Household Survey of Drug Abuse in 1995, the lifetime crack use for 12 to 15 year old blacks was 6.2% compared to 6% for similarly-aged whites. This pattern is reversed for 16 to 17 years olds, where the lifetime use rate among whites is 5.1% compared to 4.2% for blacks at similar ages. While participation rates may mask underlying differences in cocaine dependence, treatment of teenagers for smokeable cocaine was relatively rare.[36] The low utilization rates and no differential use by race suggest that crack use of crack is not a confounding factor driving the results above.

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[35] In the seven months following *48 Hours on Crack Street* (which was watched by 15 million viewers), NBC ran over 400 separate news stories discussing crack cocaine (Reinarman and Levine, 1989).
[36] According to the Substance Abuse and Mental Health Services Administration's (SAMHSA) Treatment Episode Dataset for 1992-1995 (the first years where treatment data are available), the rate of crack abuse requiring treatment was less than 1% for children aged 17 years or less and a further 2% for those aged 18 or 19 (SAMHSA, 1997).

b. Teen Drug Dealing

It is particularly difficult to understand who sells crack cocaine and what sort of returns they earn. There are, however, several regularities across available studies that make it unlikely that the link between the arrival of crack and decreased educational attainment for black males resulted directly from school-age youths selling crack cocaine. These studies most commonly use self-reports obtained from individuals intersecting with the criminal justice system or who were recruited from an area known to have intense drug activity. They also make use of criminal justice records and school-based surveys (Reuter et al., 1990), financial records from a gang selling crack cocaine (Levitt and Venkatesh, 2000), and ethnographic observations of drug markets (Johnson et al., 2000).

The most important regularity across these different sources is that crack dealing was predominantly a part-time activity that did not conflict with formal employment or, presumably, high school attendance. Johnson et al. (2000) observed 300 participants in crack markets in New York, and found most used it as a sporadic way to earn a limited income. Levitt and Venkatesh (2000) reported that members of a Chicago crack-selling gang most commonly worked four times a week for four hours each time, that the pay was low, and that many held legitimate jobs. Reuter et al. (1990) examined arrest records in the DC between 1987 and 1989 and found that the employment rates of individuals arrested for drug selling were similar to the rates of those arrested for non-drug offenses. In a follow-up survey, they found that individuals working more intensively in the formal labor market were more active in selling crack and that their income from dealing drugs was limited.

Second, studies reporting the ages of crack sellers suggest most were of post-school age. Johnson et al. (2000) reported that many inner-city youths did not sell crack and those that did were less skilled and more likely to be arrested. The General Accounting Office (1989) found that crack dealers were most commonly in their early 20s, while Reuter et al. (1990) found that arrests for drug selling in DC peak at 24 years of age. The murder rate changes among 15-24 year old black males are mainly due to the murder rate changes of 19-24 year olds, so to the extent murders reflect participation in crack markets then this indicates more activity among post-school age children.

c. Changing Family Structure and School Quality

Another possibility is that, rather than reacting to increased mortality or prison risk, these changes in educational outcomes reflect changes in family structure or differential school quality. If changes in family structure were actually responsible for the decreased educational outcomes among

older black males than results of similar magnitude should be found for females and younger black males. Both the reduced-form and the murder rate results above show larger and more precise effects for males than for females. The lack of an effect for females limits the potential for changes in school quality or family structure to account for educational patterns after the arrival of crack.

There could be a remaining concern that the impact of absent fathers or other family-level changes could differentially affect males as compared to females. If this were the case, then we should expect decreases in educational attainment for black males at all ages, but this is not the case. Some evidence on this point can be found in patterns for the NAEP-LTT standardized test scores. These math and reading tests have been given to 9, 13, and 17 year olds every two to five years since the early 1970s, and tests have not changed over this period.[37] In Figures 6a and 6b we present the percentage differences in white and black scaled scores by age for reading and math, respectively. The filled year markers represent the years in which NAEP tests were administered. Among 17-year-olds, the reading scores of whites were 22% higher than of blacks in 1971.[38] This gap fell to 7% in 1988, and then rose to 11% by 2004. For this same age group, the gap in math test scores halved from 14% to 7% between 1978 and 1990, and then rose to 10% by 2004.[39] The trends for 13-year-old reading and math scores are similar, with white and black test scores converging until the late 1980s and diverging after. The trends for nine year olds are different, however, with the gap mainly decreasing throughout the entire time period. This is particularly evident for math scores. The lack of a similar impact in test scores for younger black students than is observed for older black males suggests that changes in family structure are not primary drivers of the stalled progress.

VII.   Conclusion

The emergence of crack cocaine markets in an area generates three primary impacts for young black males: an increased murder rate, an increased prospect of incarceration, and an increased opportunity for employment outside of the formal sector. Each of these factors lowers the potential return from an investment in human capital and therefore our estimates of reduced schooling provide additional evidence of individuals investing in education as discussed in Becker (1964).

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[37] More information about the NAEP LTT is available at http://nces.ed.gov/nationsreportcard/ltt/.

[38] Although we do not show it here, all of the convergence in test scores though the 1980s is due to rising black test scores. Scores for white students have changed little over this period (Neal,2006; Magnuson and Waldfogel, 2008).

[39] Only those attending school are tested, so the 17 year old sample is missing individuals who have dropped out of high school. The decreasing high school graduation rates of blacks relative to whites in the 1990s should remove 17 year old black students of below-average ability and bias the results away from finding a widening test score gap.

From 1970-1985 the percentage of black males who received a high school degree increased by an average of 0.4 percentage points per year. As a testament to the pace of white-black convergence prior to the arrival of crack, if black males had continued this historic progress by 1996 their high school attainment rate would have been roughly equal to the rate for white males. The results above show that the introduction of crack cocaine to a city reversed a large portion of this progress and that the responses to changing murder and prison intake rates reduced the percentage of black males with a high school degree by 3.4 percentage points. Over the period of greatest post-crack decline in black high school graduation rates, 1986 to 1996, there was a 4.8 percentage point decrease in the percentage of black males with a high school degree. Depending upon assumptions about the continuation of the historical convergence, the widespread violence, incarceration, and other factors associated with the crack cocaine epidemic explains between 39 and 71 percent of the decline in educational achievement among black males between 1986 and 1996.

By the late 1990s and early 2000s, much of the violence associated with crack markets had subsided. Law enforcement accounts suggest that this was the result of crack turning into a stable drug market where property rights were relatively well established amongst the new entrants. It is important to note that this decline in the murder rate was not matched by a similar decrease in the prison intake rate. In 1999, 3.5 percent of black males between the ages of 20 to 24 in our data entered a federal or state prison facility. This demonstrates that a continued disruption from some portion of the crack cocaine markets. Panel C of Figure 1 contains the high school attainment rate by race and sex from 1967 to 2004. This time period of reduced violence in the late 1990s was matched by a slowing of the decline in the educational attainment of black males. From 1986 to 1996 the high school attainment rate of black males fell by an average of 0.6 percentage points a year. However, from 1996 to 2004 this rate fell by only 0.097 percentage points a year. While this was not a return to the historical growth throughout the 1970s, it certainly was a break from the declining trend during the height of the crack epidemic. The failure to return to growth could be a sign of a new equilibrium that could result from factors such as decreased intergenerational transfers in education from parents, siblings, or other children to relatives. Further research is necessary to determine the factors underlying the current trends in black educational attainment.

References

Adler, William M. 1995. *Land of Opportunity: One Family's Quest for the American Dream in the Age of Crack*. New York: Atlantic Monthly Press

Agar, Michael. 2003. "The Story of Crack: Towards a Theory of Illicit Drug Trends." *Addiction Research and Theory* 11 (1): 3-29.

Armor, David J. 1992. "Why is Black Educational Achievement Rising?" *Public Interest* 108(Summer): 65-80.

Anderson, Elijah. 1994. "The Code of the Streets." *Atlantic Monthly,* 273: 81-94.

Becker, Gary S. 1964. *Human Capital*. New York: Columbia University Press.

Ben-Porath, Yoram. 1967. "The Production of Human Capital and the Life Cycle of Earnings." *Journal of Political Economy* 75(4):

Bjorkland, Anders, Mikael Lindahl, Erik Plug. 2006. "The Origins of Intergenerational Associations" Lessons from Swedish Adoption Data." *Quarterly Journal of Economics* 121(3): 999-1028.

Blumstein, Alfred. 1995. "Youth Violence, Guns, and the Illicit-Drug Industry." *The Journal of Criminal Law and Criminology* 86 (1): 10-36.

Blumstein, Alfred. 2003. "The Notorious 100:1 Crack:Powder Disparity – The Data Tell Us that it is Time to Restore the Balance." *Federal Sentencing Reporter* 16(1), 87-92.

Blumstein, Alfred, and Allen J. Beck. 1999. "Population Growth in U.S Prisons, 1980-1996." University of Chicago Press, *Crime and Justice* 26: 17-61.

Blumstein, Alfred. 2002. "Youth, Guns, and Violent Crime." *The Future of Children* 12 (2): 38-53.

Boggess, Scott, and John Bound. 1997. "Did Criminal Activity Increase During the 1980s? Comparisons across Data Sources." *Social Science Quarterly* 78 (3): 725-39.

Borowsky, Iris, Marjorie Ireland, and Michael Resnick. 2009. "Health Status and Behavioral Outcomes for Youth Who Anticipate a High Likelihood of Early Death," *Pediatrics,* 124(1): e81-e87.

Boozer, Michael A., Alan B. Krueger, and Shari Wolkon. 1992. "Race and School Quality Since Brown v. Board of Education." *Brookings Papers on Economic Activity – Microeconomics,* 269-326.

Caulkins, John P., Bruce Johnson, Angela Taylor, and Lowell Taylor. 1999. "What Drug Dealers Tell Us About Their Costs of Doing Business." *Journal of Drug Issues* 29(2), 323-340.

Chay, Kenneth Y., Jonathan Guryan, and Bhashkar Mazumder. 2009. "Birth Cohort and the Black-White Achievement Gap: The Roles of Access and Health Soon After Birth." Federal Reserve Bank of Chicago Working Paper 08-20.

Chicoine, Luke. 2012. "AIDS Mortality and its Effect on the Labor market: Evidence from South Africa." *Journal of Development Economics* 98(2): 256-69.

Cook, Michael D., and William N. Evans. 2000. " Families or Schools? Explaining the Convergence in White and Black Academic Performance." *Journal of Labor Economics* 18 (4): 729-54.

Cooper, Edith Fairman. 1998. "The Emergence of Crack Cocaine Abuse in the United States: A Pubic Health Perspective." *CRS Report for Congress* 1-78.

Copemann, Chester D., and Paula L. Shaw. 1975. "Selected demographic characteristics and patterns of drug abuse among treated addicts from a suburban community." *British Journal of Addiction to Alcohol & Other Drugs* 70(2): 205-12.

Derlet, Robert W., and Timothy E Albertson. 1989. "Emergency Department Presentation of Cocaine Intoxication." *Annals of Emergency Medicine* 18(2): 182-186.

Duke, Naomi, Carol Skay, Sandra Pettingell, and Iris Borowsky. 2009. "Adolescent Perception of Premature Risk for Death: Contributions from Individual and Environmental Contextual Factors," *Academic Pediatrics,* 9(4): 256-262.

Duke, Naomi, Iris Borowsky, Sandra Pettingell, Carol Skay, and Barbara McMorris. 2011. "Adolescent Early Death Perception: Links to Behavioral and Life Outcomes in Young Adulthood," *Journal of Pediatric Health Care*, 25(4): 224-234.

Fagan, Jeffrey. 1989. "The Social Organization of Drug Use and Drug Dealing Among Urban Gangs." *Criminology* 27 (4): 633-69.

Fagan, Jeffrey, and Ko-Lin Chin. 1989. "Initiation Into Crack and Cocaine: A Tale of Two Epidemics." *Drug Problems* 16: 579-617.

Fagan, Jeffrey, and Ko-Lin Chin. 1990. "Violence as Regulation and Social Control in the Distribution of Crack." en De la Rosa, Mario, Elizabeth Y. Lambert, and Bernard Gropper, eds. *Drugs and Violence: Causes, Correlates, and Consequences*. National Institute on Drug Abuse Research Monogram Series 103: 8-43.

Felson, Richard B., and Luke Bonkiewicz. 2011. "Guns and Trafficking in Crack-Cocaine and other Drug Markets." *Crime and Delinquency* 20 (10): 1-25. http://cad.sagepub.com/content/early/2011/02/10/0011128711398023

Fortson, Jane G. 2011. "Mortality Risk and Human Capital Investment: The Impact of HIV/AIDS in Sub-Saharan Africa." *The Review of Economics and Statistics* 93 (1): 1-15.

Fryer, Ronald G. Jr., Paul S. Heaton, Steven D. Levitt, and Kevin M. Murphy. Forthcoming. "Measuring Crack Cocaine and Its Impact." *Economic* Inquiry.

Geller, Amanda, Irwin Garfinkel, Bruce Western. 2006. "The Effects of Incarceration on Employment and Wages: An Analysis of the Fragile Families Survey." Center for Research on Child Wellbeing, Working Paper 2006-01-FF.

Gladstein, Jack, Elisa Rusonis, Felix Heald. 1992. "A Comparison of Inner-City and Upper-Middle-Class Youth's Exposure to Violence." *Journal of Adolescent Health* 13(4): 275–280.

Grissmer, David W., Sheila Nataraj Kirby, Mark Berends, and Stephanie Williamson. 1994. *Student Achievement and the Changing American Family.* Santa Monica, CA: RAND.

Grogger, Jeff, and Michael Willis. 2000. "The Emergence of Crack Cocaine and the Rise in Urban Crime Rates." *The Review of Economics and Statistics* 82 (4): 519-29.

Hamid, Ansley. 1990. "The Political Economy of Crack-Related Violence." *Contemporary Drug Problems* 17: 31-78.

Heckman, James J. and Paul A LaFontaine. 2010. "The American High School Graduation Rate: Trends and Levels." *The Review of Economics and Statistics* 92(2): 244-262.

Hoffman, Joan. 2004. *Youth Violence, Resilience, and Rehabilitation.* New York: LFB.

Jencks, Christopher, and Meredith Phillips, eds. 1998. *The Black-White Test Score Gap.* Washington, DC: Brookings Institution.

Jayachandran, Seema, and Andriana Lleras-Muney. 2009. "Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines." *Quarterly Journal of Economics* 349-97.

Jaynes, Gerald David, and Robin Murphy Williams, eds. 1989. *A Common Destiny: Blacks and American Society.* Washington, DC: National Academy Press.

Johnson, Bruce D., Andrew Golub and Jeffrey Fagan. 1995. "Careers in Crack, Drug Use, Drug Distribution, and Nondrug Criminality." *Crime & Delinquency* 41(3): 275-295

Johnson, Bruce D., Eloise Dunlap and Sylvie C. Tourigny. 2000. "Crack distribution and Abuse in New York." In: Mangai Natarajan and Mike Hough, eds. *Illegal Drug Markets: From Research to Prevention Policy.* Monsey, NY: Criminal Justice Press: 19–57.

Johnston, Lloyd, Patrick O'Malley, and Jerald Bachman. 1991. *Drug Use Among American High School Seniors, College Students, and Young Adults.* National Institute on Drug Abuse, US Department of Health and Human Services.

Koretz, Daniel. 1987. *Educational Achievement: Explanation and Implications of Recent Trends,* Washington, DC: Congressional Budget Office.

Magnuson, Katherine A., and Jane Waldfogel, eds. 2008. *Steady Gains and Stalled Progress: Inequality and the Black-White Test Score Gap.* New York: Russell Sage Foundation.

Massing, Michael. 1989. "Crack's Destructive Sprint Across America." *New York Times Magazine,* November 27, 2011. http://nytimes.com/1989/10/01/.../crack-s-destructive-sprint-across-america.html

McDade, Thomas, Laura Chyu, Greg Duncan, Lindsay Hoyt, Leah Doane, and Emma Adam. 2011. "Adolescents' Expectations for the Future Predict Health Behaviors in Early Adulthood," *Social Science and Medicine*, 391-398.

Morganthau, Tom. 1992. "It's Not Just New York …" *Newsweek,* Mar 8 1992.

Neal, Derek. 2006. "Why Has Black-White Skill Convergence Stopped?" *Handbook of the Economics of Education* 1: 512-76.

Nguyen, Quynh, Jon Hussey, Carolyn Halpern, Andres Villaveces, Stephen Marshall, Arjumand Siddiqi, and Charles Poole. 2012. "Adolescent Expectations of Early Death Predict Young Adult Socioeconomic Status," *Social Science and Medicine*, 74 (2012): 1452-1460.

Osofsky, Joy D., Sarah Wewers, Della M. Hann, and Ana C. Fick. 1993. "Chronic Community Violence: What is Happening to Our Children?." *Psychiatry* 56 (1): 36-45.

Oster, Emily, Ira Shoulson, and Ray Dorsey. 2012. "Limited Life Expectancy, Human Capital and Health Investments: Evidence from Huntington Disease. Working Paper, University of Chicago Booth School of Business.

Pettit, Becky, and Bruce Western. 2004. "Mass Imprisonment and the Life Course: Race and Class Inequality in U.S Incarceration." *American Sociological Review* 69: 151-69.

Reinarman, Craig and Harry Levine. 1989. "Crack in context: politics and media in the making of a drug scare." *Contemporary Drug Problems* 16(4): 535-577.

Reuter, Peter, Robert MacCoun, Patrick Murphy, Allan Abrahamse, and Barbara Simon. 1990. "Money from Crime. A Study of the Economics of Drug Dealing in Washington D.C." *RAND: Drug Policy Research Center* 1-172.

Rivkin, Steven G. 1995. "Black/White Differences in Schooling and Employment." *Journal of Human Resources* 30(4): 826-852.

Ruggles, Steven, J. Trent Alexander, Katie Genadek, Ronald Goeken, Matthew B. Schroeder, and Matthew Sobek. Integrated Public Use Microdata Series: Version 5.0 [Machine-readable database]. Minneapolis: University of Minnesota, 2010.

Sacerdote, Bruce. 2007. "How Large are the Effects from Changes in Family Environment? A Study of Korean American Adoptees." *Quarterly Journal of Economics,* 122(1): 119-157.

Schubiner, Howard, Richard Scott, and Angela Tzelepis. 1993. "Exposure to Violence Among Inner-city Youth." *Journal of Adolescent Health* 14: 214-219.
eries 110: 1-344.

Simon, David and Edward Burns. 1997. *The Corner: A Year in the Life of an Inner-City Neighborhood*. New York: Broadway Books.

Stoddard, Sarah, Susan Henly, Renee Sieving, and John Bolland. 2011. "Social Connections, Trajectories of Hopelessness, and Serious Violence in Impoverished Urban Youth," *Journal of Youth and Adolescence,* 40: 278-295.

Substance Abuse and Mental Health Services Administration, Office of Applied Studies. *National Admissions to Substance Abuse Treatment Services (TEDS) 1992-1995*, Advance Report No. 12, SAMHSA, Rockville, MD, 1997.

U.S. Department of Health and Human Services. 1991. "Morbidity and Mortality Weekly Report: Weapon-Carrying Among High School--United States, 1990." Washington, D.C.: Department of Health and Human Services, Public Health Service.

U.S. General Accounting Office. 1989. Nontraditional Organized Crime: Law Enforcement Officials' Perspective on Five Criminal Groups. Washington, DC: U.S. Government Printing Office.
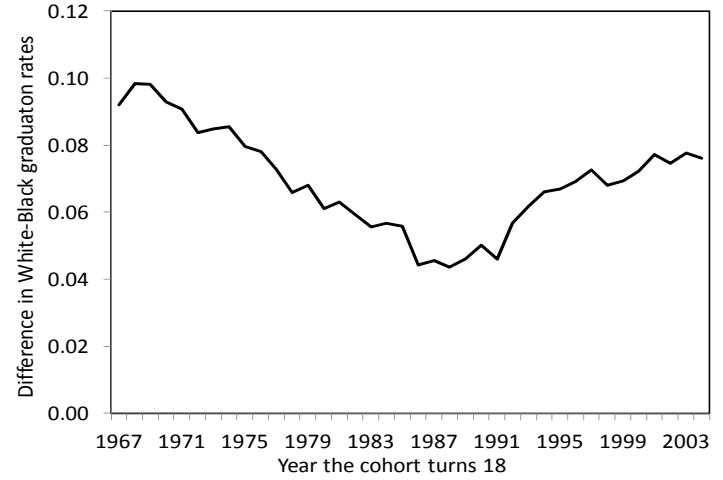
Witkin, Gordon, and Maudi Mukenge. 1991. "The Men Who Created Crack." *U.S News and World Report* 111 (8): 44-60.

Figure 1 High School Completion Rates by the Year each Cohort Turns 18
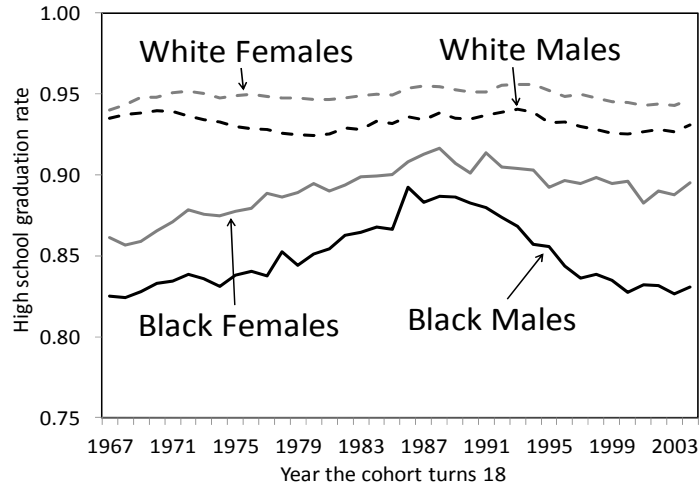By Race and Sex, 2005-2009 American Community Survey
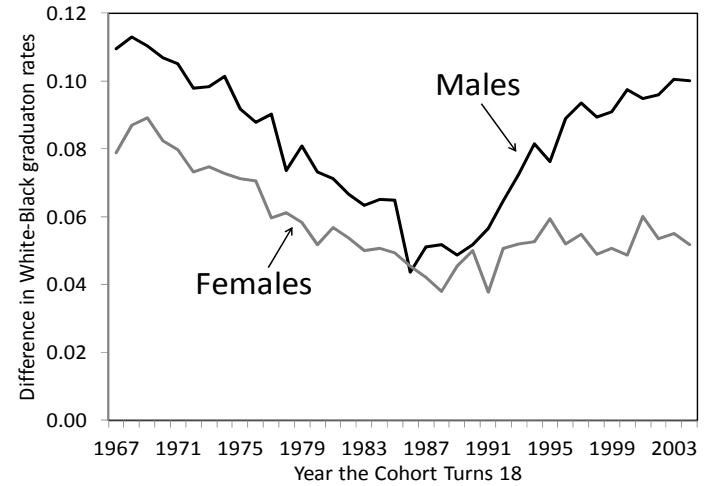
A: High School Graduation Rates, by Race and Cohort
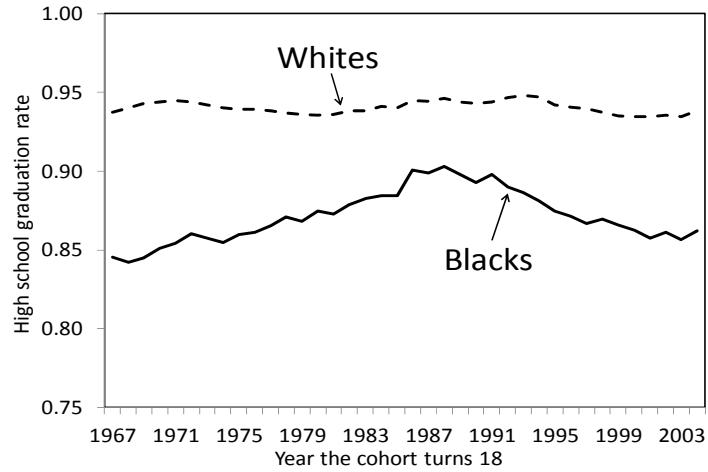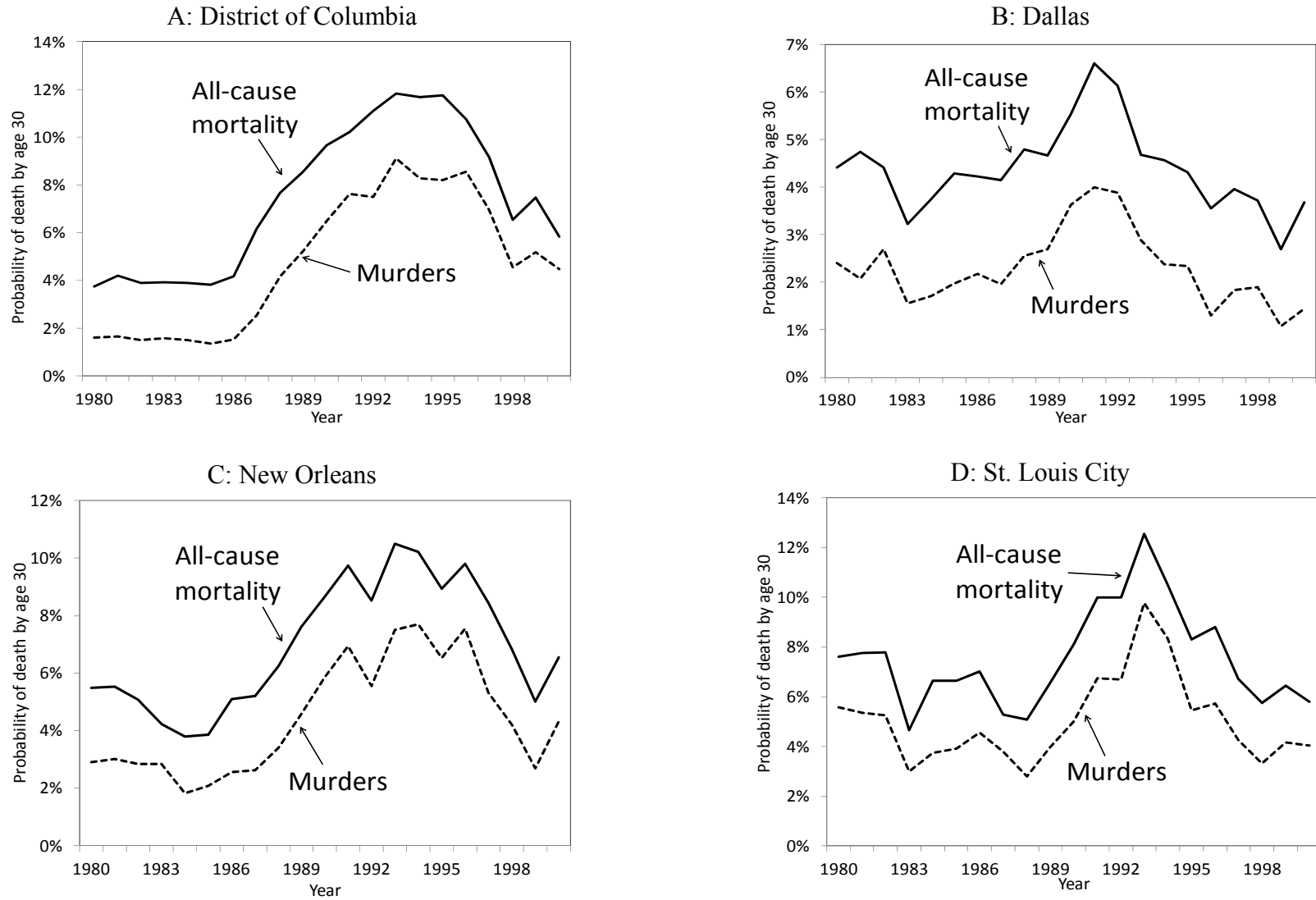

B: Difference in White-Black Graduation Rates
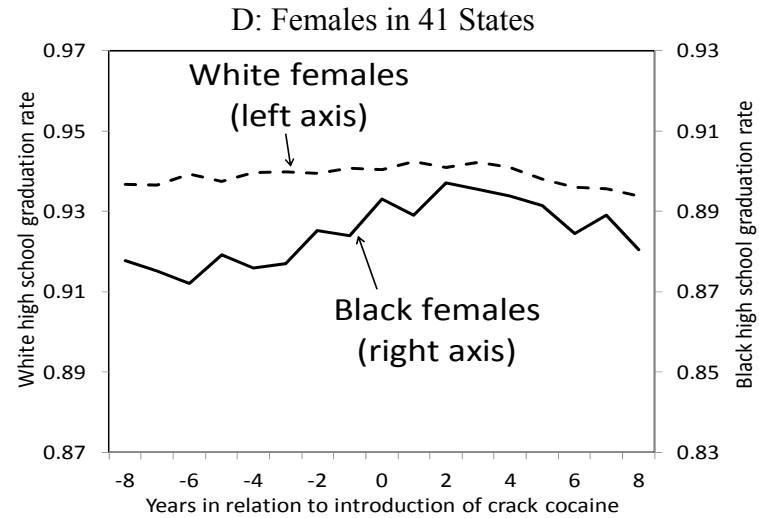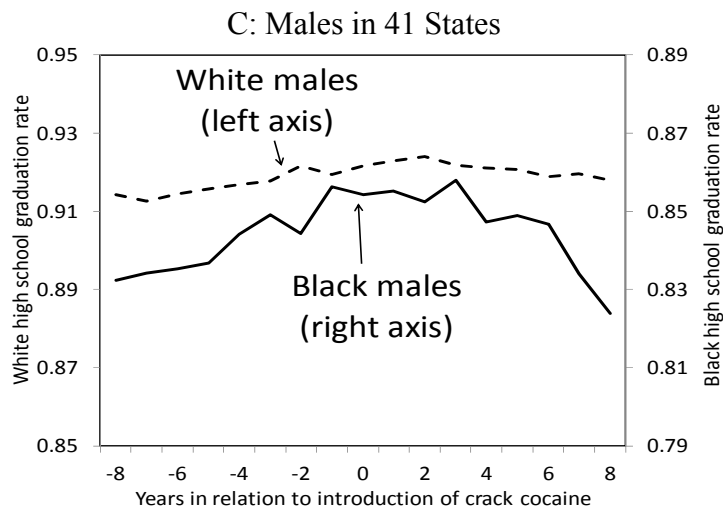

C: High School Graduation Rates, by Sex, Race and Cohort

D: Differences in White-Black Graduation Rates, by Sex

Figure 2 Expectations of Death by Age 30 for a Black Males at Age 15, Based on Contemporaneous Age-specific Mortality Rates

A: District of Columbia

B: Dallas

C: New Orleans

D: St. Louis City

Figure 3 High School Graduation Rates by when Cohorts Turn Age 18 in Relation to When Crack Cocaine Entered MSAs and States, by Race and Sex, 2000 Census 5% PU

A: Males in 57 Large MSAs

B: Females in 57 Large MSAs

C: Males in 41 States

D: Females in 41 States
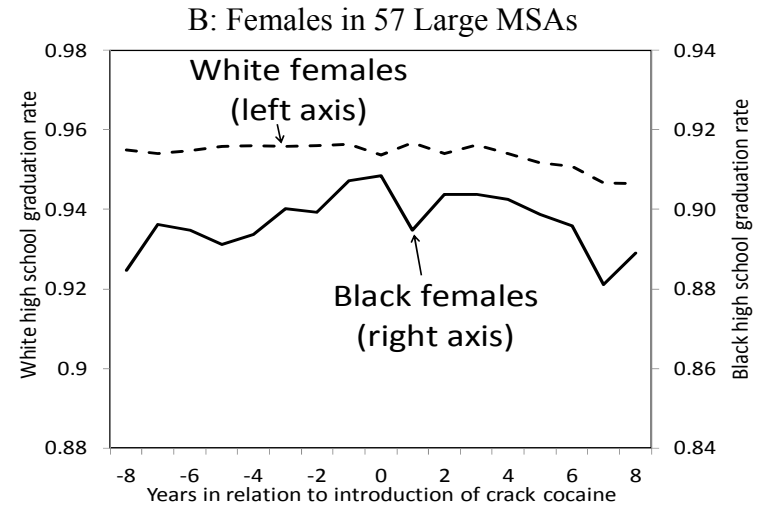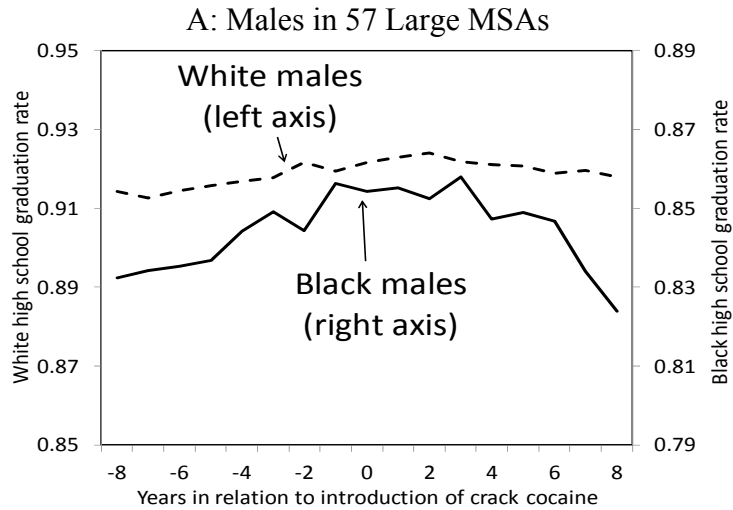
Figure 4 Murder Rates (Deaths/100,000 pop.) and Prison Intake Rates for Various Groups, 1980-2000

A: Murder Rates for Various Age Groups

B: Murder Rate for Those Aged 15-24, By Age and Sex
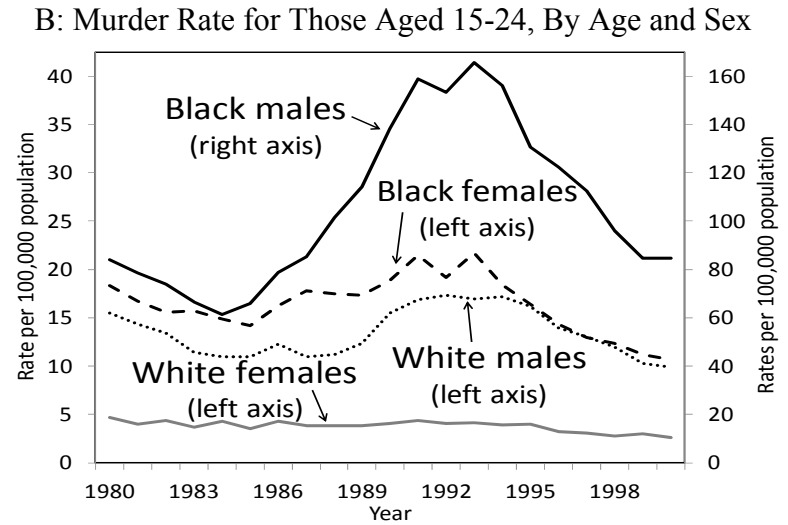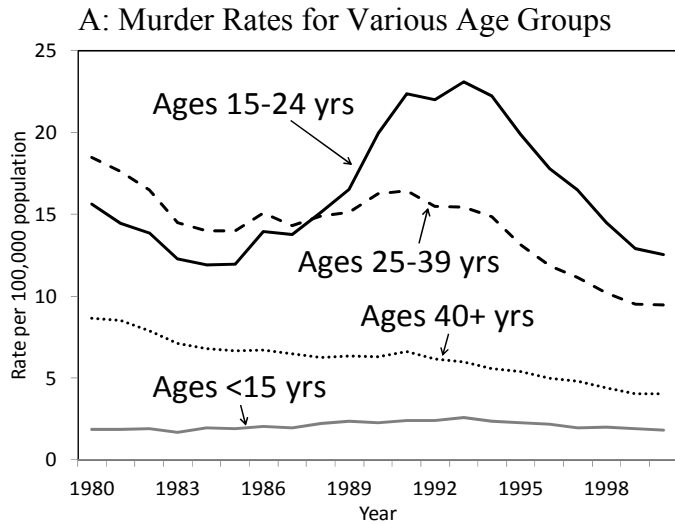
C: Change in Murder Rates of Black Males Aged 15-24,
By Area Population Size
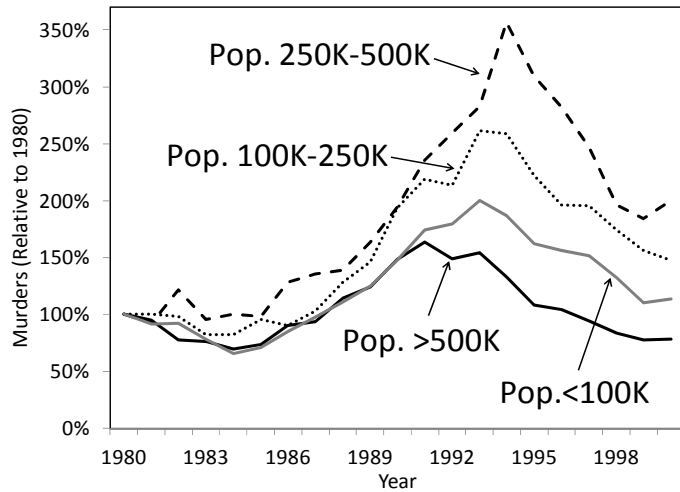
D: Prison Intake Rates for Males Aged 20-24 (17 States).
By Race
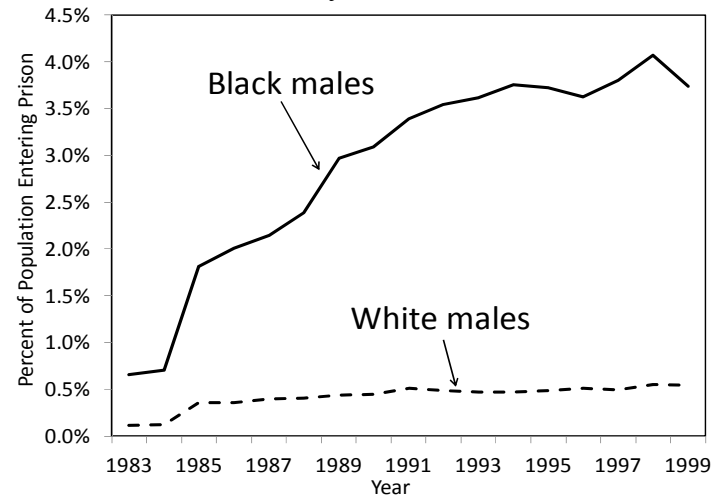
Figure 5 Murder Rates (Murders/100,000 People), Before and After the Introduction of Crack in MSAs, By Race and Sex

A: Black Males

B: White Males

C: Black Females

D: White Females
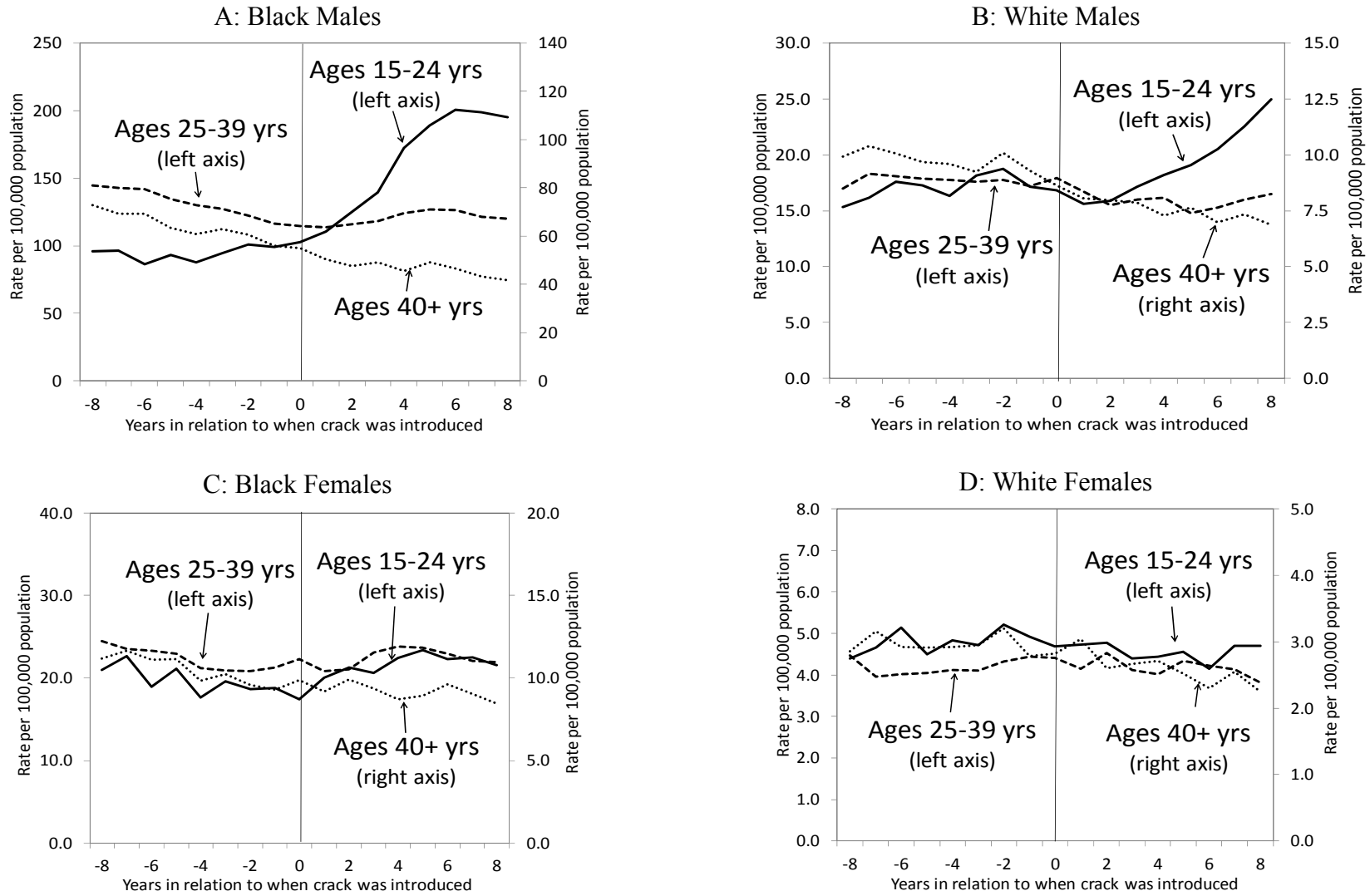
Figure 6 White/Black Test Score Gaps for 9, 13 and 17 year olds

A: Percentage Differences in White/Black NAEP-LTT Reading Scores

B: Percentage Differences in White/Black NAEP-LTT Math Scores

Table 1 The Year Crack Arrives in the 57 Largest Metropolitan Statistical Areas (by 1980 Population) Based on Cocaine-related Deaths in Two Consecutive Years, Multiple Cause of Death Data, 1981-1998

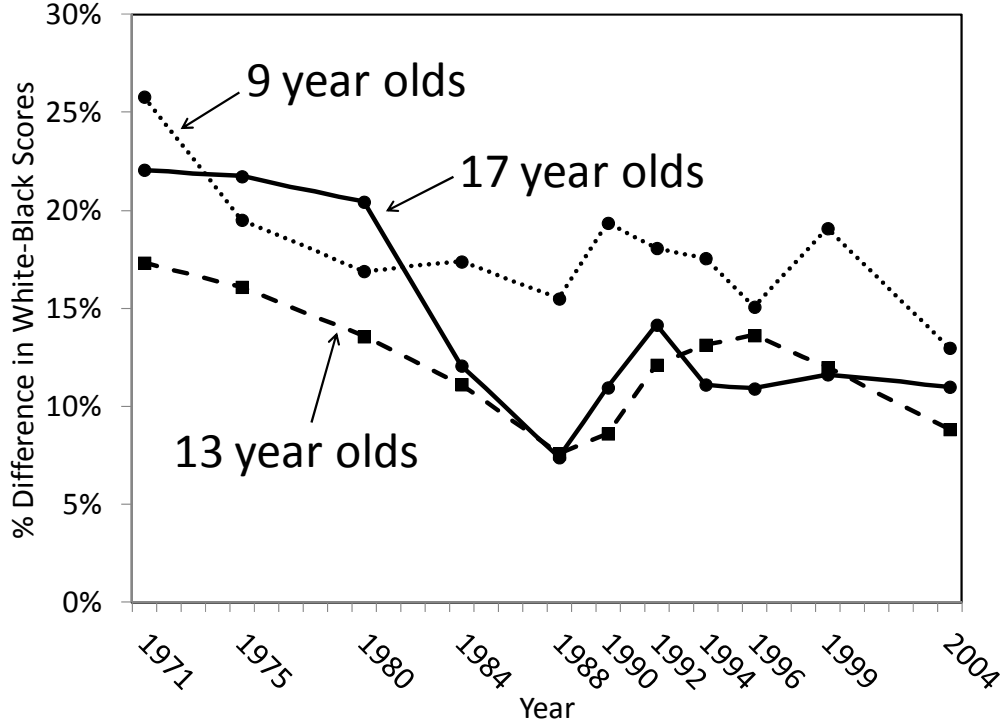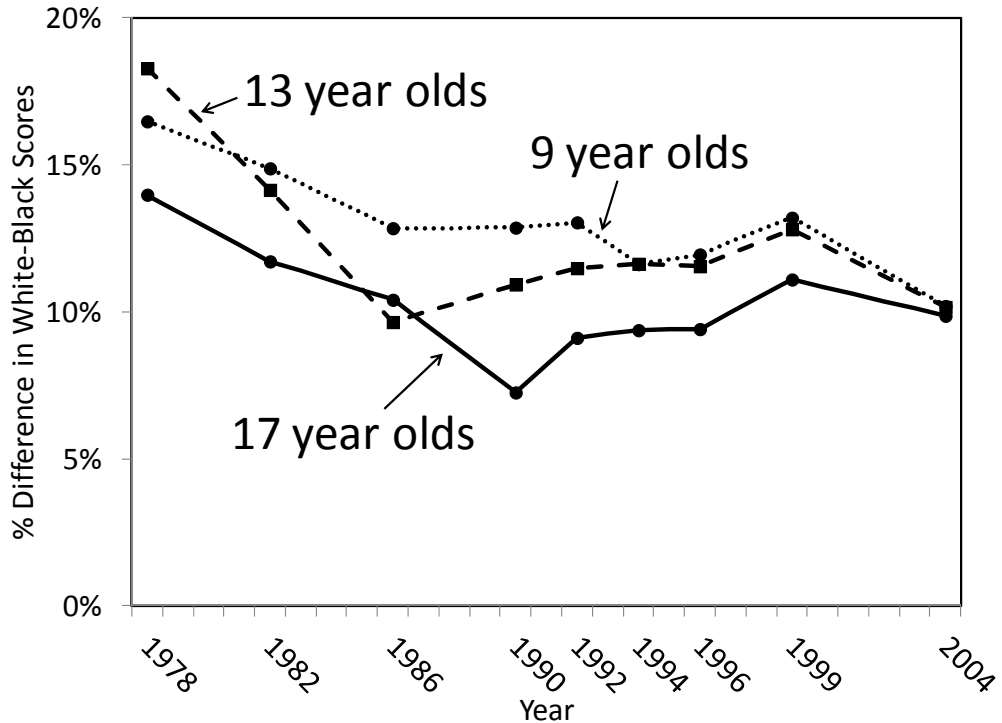| Year	Metropolitan Statistical Area
1982	Los Angeles/Long Beach; Miami; New York
1983	Atlanta; Riverside/San Bernadino; San Francisco/Oakland
1984	Ft. Lauderdale; San Jose; Seattle/Bellevue/Everett; Tampa/St. Petersburg/Clearwater
1985	Albany/Schenectady/Troy; Dallas/Fort Worth/Arlington; Detroit; Kansas City; Philadelphia; Washington, DC
1986	Boston; Chicago; Cleveland; Indianapolis; Memphis; Minneapolis/St. Paul; Monmouth-Ocean, NJ; Newhaven/Bridgeport; New Orleans; Newark, NJ; Orange Co. CA; Sacramento
1987	Cincinnati; Greensboro/Winston Salem/High Point; Milwaukee/Waukesha Norfolk/VA Beach/Newport News; Providence
1988	Buffalo/Niagara Falls; Denver; Hartford, CT; Houston; Louisville; Nashville; Oklahoma City; Orlando; Phoenix/Mesa; Pittsburgh; Portland, OR/Vancouver, WA; Rochester; Salt Lake City/Ogden; San Diego
1989	Baltimore; Birmingham; Charlotte, NC/Gastonia, NC/Rock Hill, SC; Grand Rapids/Muskegeon/Holland, MI; St. Louis, MO
1991	Bergen/Passaic; Dayton/Springfield; Middlesex/Somerset/Hunterdon, NJ
1992	Columbus
1994	San Antonio

Notes: The Nassua/Suffolk MSA is omitted because cocaine-related deaths were present in multiple years prior to 1981.

Table 2 The Year Crack Arrives in a State based on Cocaine-related Deaths in Two Consecutive Years, Multiple Cause of Death Data, 1981-1998

Year	State
1981	California
1982	Florida, New York
1983	Arizona, Georgia
1984	Colorado, Michigan, Missouri, Virginia, Washington
1985	District of Columbia, Illinois, Indiana, Maryland, New Mexico, Pennsylvania, South Carolina, Texas
1986	Alabama, Connecticut, Indiana, Kansas, Louisiana, Massachusetts, Minnesota, Mississippi, New Jersey, Ohio, Tennessee
1987	Arkansas, Kentucky, Nebraska, Nevada, North Carolina, Oregon, Rhode Island, West Virginia, Wisconsin
1988	Delaware, , Oklahoma, Utah

Notes: Ten states with small black populations are omitted (HI, AK, ID, ME, MT, NH, ND, SD, VT, WY). The criterion for the three largest states (CA, NY, and TX) is two or more deaths in two consecutive years.

Table 3 Maximum Likelihood Weibull Duration Model Estimates of the Relationship Between MSA Characteristics and When Crack Cocaine Arrives, 50 Largest MSAs

Covariates	Hazard Ratio 1980 Levels [95% CI]	Hazard Ratio Changes 1970-80 [95% CI]
Min. Distance NY/MIA/LA (per 100 miles)	0.908** [0.828, 0.996]	0.919* [0.831, 1.015]
Log 1980 Population	2.657*** [1.593. 4.429]	2.873*** [1.776. 4.648]
% White	1.021 [0.975. 1.068]	1.0302 [0.882. 1.204]
% Black	1.012 [0.938. 1.091]	1.073 [0.801. 1.438]
% Black Poverty	0.946 [0.835. 1.072]	0.948 [0.873. 1.029]
% Black Unemployment	0.963 [0.848. 1.094]	0.928 [0.700. 1.230]
% Black HS Dropout	0.951 [0.798. 1.132]	0.920 [0.805. 1.051]
% Black HS Graduate	0.878 [0.728. 1.059]	0.892 [0.757. 1.051]
% Black Some College	0.983 [0.770. 1.255]	0.875 [0.718. 1.067]
N	50	50

* p<0.10, ** p<0.05, *** p<0.01
Standard errors in parentheses. Covariates are constructed from the 1970 and 1980 Census 5% PUMS.

Table 4 Maximum Likelihood Estimates of Difference-in-Difference
Negative Binomial Model for Murder Counts, Eight Years Before and
Nine Years After Crack Arrives, 57 Large MSAs

Treatment Group	In Treatment Group x			p-value, After Coefs.=0	Mean Murder Rate, Treated Group, Year Before Crack Arrives
	0-2 Years After Crack	3-5 Years After Crack	6-8 Years After Crack		
Panel A: Male MSA residents					
Black, 15-24	0.252***	0.703***	1.000***	<0.0001	99.9
	(0.064)	(0.082)	(0.056)		
Black, 25-39	0.062**	0.198***	0.329***	<0.0001	118.0
	(0.028)	(0.037)	(0.060)		
Whites, 15-24	0.042	0.185***	0.491***	<0.0001	17.9
	(0.050)	(0.054)	(0.029)		
Whites, 25-39	0.119***	0.075***	0.152***	<0.0001	17.8
	(0.024)	(0.024)	(0.029)		
Panel B: Female MSA residents					
Blacks, 15-24	0.030	0.103	0.216***	<0.0001	19.0
	(0.058)	(0.068)	(0.057)		
Blacks, 25-39	-0.003	0.053	0.114***	0.0058	21.4
	(0.047)	(0.048)	(0.035)		
Whites, 15-24	-0.017	-0.023	0.181***	0.012	5.1
	(0.064)	(0.068)	(0.064)		
Whites, 25-39	0.116**	0.149**	0.209***	<0.0001	4.2
	(0.048)	(0.050)	(0.045)		

* $p<0.10$, ** $p<0.05$, *** $p<0.01$
Standard errors in parentheses allow for arbitrary correlation in observations within an MSA over time.
Each model has 1,938 observations – 2 groups x 17 years x 57 cities. The comparison group in each
model is residents in the same race/sex cell that are aged over 40. Observations are weighted by the
population within each cell. Other covariates include a dummy variable for the comparison sample, MSA
fixed effects, year fixed effects, and MSA-specific time trends.

Table 5 Linear Probability Estimates of High School Graduation Models,
Cohorts that Turned 18 1976-1999, 2000 5% PUMS

| | 57 MSAs | | 41 states | | | |
| | | | State of Birth | | Current State of Residence | |
Covariate	Males	Females	Males	Females	Males	Females
Panel A: Model (1)						
Black*Pre-Crack Time Trend	0.0017***	0.0013***	0.0033***	0.0017***	0.0036***	0.0020***
	(0.0004)	(0.0003)	(0.00067)	(0.0004)	(0.0007)	(0.0004)
Black* (Years after crack≥0)	-0.0027**	-0.0009	-0.0033***	-0.0006	-0.0033***	0.0002
	(0.0008)	(0.0005)	(0.0006)	(0.0004)	(0.0007)	(0.0004)
R^2	0.0202	0.0174	0.0203	0.0152	0.0221	0.0161
Panel B: Model (2)						
Black*Pre-Crack Time Trend	0.0016***	0.0012***	0.0032***	0.0008**	0.0036***	0.0013***
	(0.0005)	(0.0003)	(0.0007)	(0.0003)	(0.0008)	(0.0004)
Black* 1(Years after crack=0)	-0.0053	0.0049	-0.0036	0.0096***	-0.0066	0.0072**
	(0.0049)	(0.0034)	(0.0040)	(0.0023)	(0.0053)	(0.0025)
Black*1(Years after crack=1)	-0.0013	-0.0073*	-0.0036	0.0047	-0.0028	0.0026
	(0.0043)	(0.0038)	(0.0050)	(0.0031)	(0.0048)	(0.0036)
Black*1(Years after crack=2)	0.0020	0.0018	-0.0077**	0.0111***	-0.0060	0.0119***
	(0.0047)	(0.0043)	(0.0035)	(0.0028)	(0.0044)	(0.0033)
Black*1(Years after crack=3)	-0.0030	0.0012	-0.0031	0.0088**	-0.0050	0.012**
	(0.0056)	(0.0036)	(0.0060)	(0.0034)	(0.0056)	(0.0041)
Black*1(Years after crack=4)	-0.0092**	0.0006	-0.0139*	0.0078**	0.0101**	0.0063*
	(0.0045)	(0.0037)	(0.0053)	(0.0035)	(0.0050)	(0.0037)
Black*1(Years after crack=5)	-0.0151**	0.0002	-0.0119**	0.0075**	-0.0161**	0.0067*
	(0.0063)	(0.0049)	(0.0050)	(0.0030)	(0.0049)	(0.0036)
Black*1(Years after crack=6)	-0.0175**	-0.0019	-0.0133*	0.0010	-0.0114*	0.0021
	(0.0071)	(0.0045)	(0.0069)	(0.0032)	(0.0065)	(0.0039)
Black*1(Years after crack=7)	-0.0168**	-0.0134**	-0.0256***	0.0055	-0.0256**	0.0050
	(0.0074)	(0.0046)	(0.0063)	(0.0049)	(0.0076)	(0.0040)
Black*1(Years after crack=8)	-0.0232**	-0.0054	-0.0335**	-0.0009	-0.0339**	0.0011
	(0.0072)	(0.0067)	(0.0057)	(0.0048)	(0.0050)	(0.0051)
R^2	0.0202	0.0174	0.0203	0.0152	0.0221	0.0161
Observations	738,497	773,829	1,315,608	1,360,257	1,313,879	1,359,403
Mean of dep. Variable for blacks the year before crack arrives	0.879	0.908	0.856	0.884	0.858	0.886

* p<0.10, ** p<0.05, *** p<0.01
Standard errors allow for arbitrary correlation in errors within geographic region (state or MSA). All models include cohort and geographic fixed effects plus a dummy variable for race.

Table 6 OLS Estimates of the Impact of Crack Intensity on High School Graduation

	Sample 1: Current MSA Residents 2000 5% PUMS, Cohorts that turned 18 over 1976-1999					
	Both Sexes, 20-24		Males, 20-24		Females, 20-24	
	57 MSA Sample	171 Largest MSAs	57 MSA Sample	171 Largest MSAs	57 MSA Sample	171 Largest MSAs
Sex/race specific murder rate during HS (x 1000)	-0.0916** (0.0380) [-0.005]	-0.1070*** (0.0316) [-0.006]	-0.0861** (0.0370) [-0.009]	-0.1080*** (0.0320) [-0.011]	0.1442 (0.1012) [0.0007]	0.0824 (0.6820) [0.0004]
R^2	0.025	0.023	0.280	0.031	0.021	0.022
Observations	1,616,728	2,290,892	789,761	1,122,021	826,967	1,168,871

	Sample 2: All State Residents, 2000 5% PUMS, Cohorts that turned 18 over 1976-1999					
	Both Sexes, 20-24		Males, 20-24		Females, 20-24	
	State of Birth	State of Residence	State of Birth	State of Residence	State of Birth	State of Residence
Sex/race specific murder rate during HS (x 1000)	-0.1863** (0.0695) [-0.009]	-0.1907** (0.0670) [-0.009]	-0.201** (0.0760) [-0.020]	-0.2101** (0.0710) [-0.021]	0.6095*** (0.1800) [0.003]	0.6432*** (0.1670) [0.003]
R^2	0.024	0.025	0.026	0.027	0.016	0.018
Observations	3,460,871	3,463,148	1,708,369	1,708,931	1,752,502	1,754,217

* $p<0.10$, ** $p<0.05$, *** $p<0.01$

Standard errors are in parentheses, and allow for arbitrary correlation in errors within a geographic area (state or MSA). The square brackets contain the estimated effect of the change in the black murder rate on graduation rates (e.g., the value of 0.005 in the top panel of the first column represents a 0.5% decrease in graduation rates). Each model contains fixed effects for a full set of FIPS x sex x race interactions, and for cohort.

Table 7 OLS Estimates of the Impact of Crack Intensity on the High School Graduation Rates of Black Males, Based on the Separate Impacts of Murder and Prison

In sample:	(1)	(2)	(3)
	Sample 1: All States With Any Prison Data		
Race specific murder rate during HS (x 1000)		-0.237**	-0.130**
		(0.077)	(0.061)
		[0.024]	[0.013]
Race Specific Prison Intake Rate	-0.961***		-0.721**
	(0.247)		(0.251)
	[-0.0284]		[-0.021]
R^2	0.027	0.026	0.027
Mean of dependent variable			
Observations	971,749	971,749	971,749
	Sample 2: Only States with Prison Data for Every Year		
Race specific murder rate during HS (x 1000)		-0.385***	-0.193**
		(0.071)	(0.057)
		[-0.039]	[0.020]
Race Specific Prison Intake Rate	-1.497***		-1.020**
	(0.262)		(0.263)
	[-0.043]		[-0.030]
R^2	0.024	0.024	0.024
Mean of dependent variable			
Observations	453,111	453,111	453,111

* p<0.10, ** p<0.05, *** p<0.01
Standard errors are in parentheses, and allow for arbitrary correlation in errors within a state. The square brackets contain the estimated effect of the change in the black murder rate on graduation rates (e.g., the value of 0.005 in the top panel of the first column represents a 2.7% decrease in graduation rates). Each model contains fixed effects for a full set of FIPS x sex x race interactions, and for cohort.