"The White/Black Educational Gap, Stalled Progress, and the Long-Term Consequences of the Emergence of Crack Cocaine Markets"

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Abstract

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We propose the rise of crack cocaine markets as an explanation for the end to the convergence in black-white educational outcomes that began in the mid-1980s. After constructing a measure to date the arrival of crack markets in cities and states, we show large increases in murder and incarceration rates after these dates. Black high school completion rates also decline, and we estimate that factors associated with crack markets can account for between 36 and 73 percent of the fall in black male high school completion rates. We argue that the primary mechanism is reduced educational investments in response to decreased returns to schooling.

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I. Introduction

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

Using data from 2000 Census Five Percent Public Use Micro Samples (PUMS) (Ruggles et al., 2010), Figure 1 shows the trends for high school completion rates for U.S.-born, non-Hispanic white and black respondents.[2] Figure 1a contains the race-specific high school completion rates for cohorts turning 18 between 1965 and 1997, while Figure 1b shows the white-black difference in these rates. Several features of the data are remarkable. First, the gap in graduation rates fell by 37 percent between 1965 and 1986, falling from 15.3 to 9.6 percentage points. Second, almost all of the convergence is due to rising black achievement—the graduation rates of whites changed little over this period, rising from 90.4 percent to 91.0 percent over the same period. Third, the convergence ends around 1986 and white-black graduation rates diverge until 1997, when the gap was 14.4 percentage points. Fourth, as with the convergence, the divergence is driven almost entirely by black graduation rates, which declined by 7.3 percentage points between 1986 and 1997. This wiped out much of the previous progress:

[1] For data on the white/black 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] These graduation rates are higher than estimates one would obtain from sources such as the October School Enrollment Supplement to the Current Population Survey (CPS) because the ACS does not distinguish between regular high school graduates and those obtaining a degree via the GED. Since individuals acquire the GED over time, older cohorts have had more time to acquire the degree. Neal (2006) finds the GED narrows the white-black gap in high school graduation but does not change the trends, while Heckman and Lafontaine (2010) find progress in narrowing black-white differences is overstated by the inclusion of the GED.

the black cohort that turned 18 in 1997 had a graduation rate similar to the 1965 black cohort.

We display these high school completion rates for males and females in Figure 1c, and the sex-specific white-black difference in these rates in Figure 1d. Between the 1965 and 1986 cohorts, the high school completion rates of black males and females increased by 5.4 and 7.1 percentage points, respectively. Both sexes halt their convergence after 1986, with males suffering more of a decline than females. Between 1986 and 1997, completion rates for black males fell by approximately 9.4 percentage points while, over the same period, the rate for black females declined by roughly 5.1 percentage points.

A number of hypotheses have been put forward to explain the convergence in educational outcomes between the 1960s and the mid-1980s, including improved parental education (Armor, 1992), reduced segregation (Jaynes and Williams, 1989), increases in school spending (Boozer et al., 1992), and better access to health care (Chay, Guryan and Mazumder, 2009). Less attention has been paid to the end of this convergence, and two studies that did examine it have not identified why it occurred. After considering a host of potential explanations such as returns to education, income shocks to black families, school factors, and cultural changes, Neal (2006, p. 570) concluded that "[i]t is not clear why the process of black-white skill convergence appeared to stop around 1990." The chapters in Magnuson and Waldfogel (2008) examined factors such as changing family income, rising income inequality, relative changes in parental education, changes in school segregation, and changes in teacher quality as possible explanations for these trends. They concluded that while these factors may account for a slowing convergence, none explain the stalled progress.[3]

We examine the widespread emergence of crack cocaine markets as a potential

[3] In the final chapter of Magnuson and Waldfogel (2008), Ferguson (p. 321) notes that "…researchers, parents, activists, and policy makers still seek to understand why progress stalled in 1990."

explanation for the stalled progress in black high school completion rates.[4] Crack cocaine was an innovation that cut the price of cocaine and dramatically expanded the market size and potential profits from drug dealing. The drug first appeared in Miami, New York, and Los Angeles around 1982, and then spread to cities and towns across the nation over the next decade.

Crack is easier to make than other types of smokeable cocaine, and produces a more intense high than intra-nasally ingested powder cocaine (Agar, 2003). It was affordable to a low-income population priced out of the powder cocaine market, especially as it could be purchased one "hit" at a time (Blumstein, 1995). It quickly became a popular and heavily-used drug: National Household Survey on Drug Abuse estimates suggest that 4.2 million individuals had ever used crack cocaine by 1991, while treatment for smokeable cocaine addictions rose from 0.5 percent of all drug admissions in 1980 to 33 percent in 1992 (SAMHSA, 1997).

Retail crack cocaine markets had additional effects on both market participants and individuals living close to these markets, even if they were not necessarily participating in the market. Dealers initially operated though "crack houses," which also offered a place to use the drug. They then moved to open-air sales, where widespread violence was used to both establish and protect local markets (Fagan and Chin, 1989; Reuter et al., 1990).

The negative effects of crack markets were particularly devastating for black families. This is because crack cocaine use rate for adults was much higher among blacks than whites; because black males (and primarily black criminal gangs) were heavily involved in crack markets; and because these markets commonly operated in predominately black neighborhoods. Together, these facts suggest that the emergence of crack markets could explain some of the stalled progress in educational attainment for both black males and females.

[4] Neal (2006) and Fryer et al. (2013) have previously suggested a potential connection between crack cocaine markets and educational progress, although neither paper empirically examines this relationship.

In addition, the specifics of the crack trade and its associated violence differentially affected young black males. As we outline below, the murder rates of young black males doubled after the emergence of crack cocaine, while their incarceration rates tripled. These factors potentially help explain why the decline in high school completion rates for black males between 1986 and 1997 was nearly twice as large as the decline for black females.

The changes in violence and incarceration for young black males were large enough to have substantially altered their expectations about their future, and hence their interest in obtaining more education. Consider how their risk of dying changed between the 1980s and 1990s. Suppose that 15 year olds estimate their chance of dying before age 30 by examining the contemporaneous, age-specific mortality rates of older individuals living in the same city and who share the same sex and race. For example, a 15 year old black male in the District of Columbia (DC) uses the annual death rates of each cohort of older black males in DC to judge his cumulative risk of dying.[5] Figure 2 shows this calculation from 1980 to 2000 for black males in DC, Dallas, Saint Louis, and New Orleans.[6] In 1980, a 15 year old black male in DC making this calculation would have estimated his risk of dying before age 30 to be four percent. This increases sharply after 1985, and by 1993 the cumulative risk of death by age 30 is 12 percent. It declines to six percent by 2000. In Dallas, New Orleans and St. Louis, cities in different parts of the U.S., 15 year old black males' risk of death by age 30 increased by between 3.5 and 8 percentage points from the mid-1980s to the early-to-mid 1990s. For comparison, these changes in perceived mortality risks are broadly similar to the mortality changes resulting from the AIDS epidemic in South Africa (Chicoine, 2012). In contrast, the changes in the equivalent mortality

[5] The expected mortality rate by age 30 is the sum of 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 years.
[6] The mortality data come from the National Center for Health Statistics' Multiple Cause of Death data, while population data is from the Census. These sources are described in detail later in the paper.

risk for white males, white females, and black females over this period were generally less than one percentage point in these cities. As we show in Figures 3a and 3b, and discuss in detail later, the increase in murders was concentrated among teenagers and young adults.

As we document in Figure 3c and later in the paper, large increases in youth murder rates occurred outside of major cities during this period. Major cities have often been the focus of reporting and research into crack markets, and this has sometimes created an impression that crack cocaine could not explain stalled progress in other locations (Neal, 2006). In reality, crack markets were established in many small cities and towns. For example, both the U.S. Department of Justice (1989) and Massing (1989) document crack markets in towns with as few as 1,000 to 3,000 residents.[7] These facts help explain a potential widespread connection between crack markets and educational outcomes.

Ethnographic accounts and surveys indicate that violence became a fact of life for school-age children after the arrival of crack cocaine markets. Children frequently reported witnessing shootings and murders.[8] Many also carried guns. A nationally-representative survey of 16,000 high school students in 1993 found that 21 percent of black male respondents reported carrying a gun at least once in the previous 30 days. The corresponding number for all other students was seven percent.[9] Anderson (1994, p. 94) found that inner-city youth were "uncertain about how long they are going to live and believe they could die violently at any time. They accept this fate." Such views may alter behavior; studies in the adolescent health literature find suggestive

[7] Massing (1989) provides specific examples of the presence of crack houses and crack selling in numerous small towns, including Newburgh (1990 population of 26,000) and Kingston (24,000) in New York State; Martinsburg (13,000) and Charles Town (3,000) in West Virginia; and Chambersburg (15,000) in Pennsylvania. A 1989 Drug Enforcement Agency report also mentions the availability of crack cocaine in small towns, including Evergreen (1990 pop. of 4,000), Foley (4,000), and Selma (27,000) in Alabama; Holmes County (16,000), Malone (1,000), Marianna (6,000), and Walton County (28,000) in Florida; Minden (14,000) in Louisiana, and Cambridge (12,000) and Salisbury (21,000) in Maryland (U.S. Department of Justice, 1989)

[8] For example, Schubiner, Scott, and Tzelepis (1993) found that, among 14-23 year old black youths in Detroit, 42 percent had witnessed a shooting and 22 percent had seen someone killed. Other researchers report similar statistics among inner-city youth in Baltimore (Gladstein, Rusonis and Heald, 1992) and New Orleans (Osofsky et al., 1993).

[9] Authors' calculations from the 1993 National Youth Risk Behavioral Survey.

evidence that an expectation of premature death among children is correlated with lower levels of education and lower adult socioeconomic status(Nguyen et al., 2012); worse adult health outcomes (Borowsky, Ireland and Resnick, 2009); higher adolescent violence (Stoddard et al., 2011); and self-destructive behavior (McDade et al., 2011).

Previous work has established that rapid declines in expected lifespan can have a negative causal impact on human capital investments. Fortson (2011) showed that areas of sub-Saharan Africa with the largest increases in AIDS mortality have the largest decline in schooling. Oster, Shoulson and Dorsey (2013) found that individuals who learn they possess the genetic mutation responsible for the fatal Huntington's disease have lower educational attainment, and are less likely to participate in job training. Jayachandran and Lleras-Muney (2009) found increased female education in post-World War II Sri Lanka following a sharp drop in maternal mortality.

Beyond an increased risk of death, the emergence of crack cocaine markets was associated with a large increase in the incarceration rate for young black males. Blumstein and Beck (1999) document a tripling of the incarceration rate between 1980 and 1996, with a ten-fold increase in the numbers incarcerated for drug offenses and a large increase in incarceration for violent crimes. The overall growth in incarceration disproportionately impacted black males. Bonczar (2003) estimated that black males' lifetime chance of going to prison based on age-specific incarceration rates changed from 13 to 31 percent between 1979 and 1997. Pettit and Western (2004) found that black males who turned 18 during the start of crack epidemic (those born between 1965 and 1969) actually faced a cumulative probability of incarceration by age 30-34 of 20.5 percent. This is twice the rate of black males born between 1945 and 1949, and approximately seven times the rate for white males born between 1965 and 1969. The increase in

the prison population resulted from both an increase in the level of criminal activity and a shift in national drug policy in response to public concerns about crack cocaine. In 1986, Congress passed a series of mandatory minimums for drug offenses, and in 1988 extended the application of these sentences to relatively low-level market participants, including lookouts and other individuals not physically possessing crack cocaine.

Using data from the National Corrections Reporting Program (NCRP), we calculate the percentage of race- and sex-specific cohorts that enter federal or state prisons in a particular year. Figure 3d contains these rates for males between the ages of 20 and 24. There is a dramatic increase in the rate for black males beginning in 1985, with a peak of approximately four percent in 1997. Unlike the murder rate, the prison intake rate does not decline during our sample period. The annual intake rate for 20 to 24 year old black males is close to four percent through to 2000, which shows that incarceration remains a salient risk for young black males throughout our sample period. The same rate for white males age 20 to 24 years is much lower over this period, at approximately 0.5 percent per annum.

We exploit city- and state-level variation in the introduction and severity 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 but prevalent afterwards, we date the arrival of crack markets in 57 large cities and 41 states. Our dates correspond well with other published estimates. We also show that these dates appear to have more to do with geography and population size than local socioeconomic indicators, suggesting that crack markets did not systematically emerge in response to changing educational or other socioeconomic characteristics.

We then show that the arrival of crack cocaine is strongly predictive of increases in

young black males' murder rates and of decreases in their high school completion rates. The nature of the relationship between crack cocaine markets and high school completion rates is shown in Figures 4a and 4b, where we report average graduation rates for U.S.-born black and white non-Hispanic males and females based on the year crack markets arrived inMetropolitan Statistical Areas (MSA). The zero on the horizontal axis is the year crack cocaine arrived.[10] Figure 4a shows that differences in male graduation rates narrowed before crack cocaine arrived, in line with the literature on convergence. The 18-year-old black male graduation rate starts to fall two years after crack markets emerge. In Figure 4b, we see a similar pre-crack increase in graduation rates for black females in the 57 MSAs, with a less pronounced drop after the arrival of crack markets. We find similar patterns in high school completion rates in a sample of comparably-defined respondents who live in the 41 states with the largest black populations.

We develop two location-specific measures of the impact of crack markets. The first is the murder rate of young adults aged 20-24 that a sex- and race-specific birth cohort experienced when they were 16 to 18 years old. We find that a higher murder rate among an older reference group lowers the probability of graduating high school for males but not females. However, it is important to note that there were only small changes in murder rates for women over this time period, which makes it difficult to detect such a relationship. These relationships are similar after adding time-varying race-specific variables at the state level that are designed to account for general economic events. The second measure is the annual race-specific fraction of 20-24 year old males entering prison in the year each cohort turned 18. A higher prison entry rate decreases the high school completion probability of males. Murder and incarceration rates are independently predictive of changes in males' high school completion in a regression containing

[10] This data is taken from the IPUMS version of the 2000 Census Five Percent Public Use Micro Samples (Ruggles et al., 2010). The sample is residents of the 57 largest MSA used in Table 3. Cohorts are based on when they turn 18.

both measures. We also note that these two factors have roughly the same impact on high school completion. Depending on assumptions about the continued progress of black males, the combined effect of the murder and incarceration risks account for 36 to 73 percent of the change in black male graduation rates between the mid-1980s and the mid-1990s.

We conduct three exercises to assess whether these relationships are symptoms of another factor, like urban decay. First, we re-estimate the relationship between high school completion and murder rates for the time period prior to 1982, before the arrival of crack cocaine in the United States. We find a similar estimated coefficient on the race-specific murder rate, suggesting that the relationship between perceived mortality risk and educational attainment is not solely present in the crack cocaine era.

In a second set of models, we replace murder rates with overall mortality rates. We note that forward-looking teens should respond to any salient change in mortality, regardless of the cause. In these models, we find similar coefficients on overall mortality to those using the murder rate. Strikingly, we find a coefficient on the overall death rate for females that is similar in magnitude to the coefficient for males. Although variation in the murder rate for females is too limited to identify an effect in our main results, we find it reassuring that females and males respond similarly to changes in their overall mortality risk.

Finally, we estimate a model where we allow the effect of the overall death rate to vary by race. Here, we find no statistically significant race-specific differences in the relationship between mortality risk and high school completion for either males or females. Taken together, these additional exercises suggest that our main results with respect to changes in the murder rate are not specific to the time period surrounding the emergence of crack cocaine markets, the cause of mortality, or a person's race. Instead, they result from changes in the mortality risk of black

males.[11]

In the final section, we consider other potential mechanisms that might have led to the widening white-black educational gap since the mid-1980s. Current and future income opportunities that arose from dealing crack cocaine could have also played a role in declining educational attainment among black males, and was likely to be more important than differential use of crack cocaine. We also examine the AIDS epidemic, changes in school quality, and the changing socio-economic circumstances of black families. We find that none of them can account for the observed sex- and race-specific differences in high school completion rates over this period. As we document, none of these variables are moving enough to explain the large fall in black high school completion rates.

In the late 1990s and early 2000s, the pace of the decline in black male high school completion rates slowed dramatically, but did not return to the pre-crack trend of white-black convergence. While murder rates declined during this period, our estimates suggest that the mortality risk represents only half of the effect of crack markets on high school completion. The remaining effect is a result of the high incarceration rate for young black males, which remains high throughout our sample. Since successive cohorts of black males face this continued incarceration risk, it is not surprising that we do not observe a return to convergence. The number of federal and state prisoners declined in 2010, which was the first decrease since 1972 (Guerino, Harrison and Sabol, 2011). It will take some time to see whether such declines lead to further convergence in black-white educational outcomes. [12]

[11] While we would like to conduct a similar set of exercise for the effect of incarceration, we lack data on the prison intake rate for the years before the emergence of crack cocaine markets and the incarceration rate for females is too low to provide a useful source of variation.

[12] There is also evidence for the intergenerational and cultural transmission of education (Bjorklund, Lindahl and Plug, 2006; Sacerdote, 2007), which could also have an effect on how education differences change over time.

II. Crack Cocaine Markets in the United States

Crack cocaine was introduced to the U.S. from the Caribbean 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 and water and then letting it cool and harden. Crack was easier to produce than other types of smokeable cocaine, and produced a more intense high than intra-nasally ingested powder cocaine (Agar, 2003). Crack cocaine users report that smoking it yields a high lasting 20 minutes, 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, who could buy a "hit" of crack for as little as $5 (Witkin, 1991). Selling it could be highly profitable, with police estimating that a $5,000 investment in powder cocaine could yield $125,000 in crack sales (U.S. GAO, 1989).

a. The Spread of Crack Cocaine Markets

Retail crack cocaine markets spread across the United States over the course of the 1980s and early-1990s. The emergence of organized crack markets in cities across America was primarily driven by the decisions of loosely-organized 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). Traffickers initially looked for large cities that were easily accessible from their distribution hubs. Retail markets were also established in many nearby smaller cities (Massing, 1989). Therefore, proximity to the three original cities where the drug emerged – New York, Miami and Los Angeles – was a critical determinant of when crack markets emerged in a particular city or state.

The structure of these markets differed greatly from most existing hard drug markets. Initially, national organizations entered new cities and established "crack houses," which both

offered a place to use the drug and served as a means of certifying the quality of the new product (Mieczkowski, 1992). Dealers established markets on street corners as the popularity of the drug increased, and used violence to establish and protect local monopolies (Fagan and Chin, 1989; Reuter et al., 1990). This often involved using intimidation and widespread violence to clear out any existing competitors in a city that were manufacturing and selling small amounts of crack. The violence often affected individuals not participating in illicit activities. Witkin (1991, p. 52) provides an example: "on August 4, 1985 a feud between two [Jamaican] posses resulted in a frenzied shootout at an Oakland, N.J., picnic attended by some 5,000 Jamaicans… posse members fired well over 700 rounds; three people were killed and thirteen wounded."

Selling in these open-air crack markets did not require large amounts of capital or expertise.[13] Attracted by the high profits earned by national organizations, small and decentralized organizations later attempted to enter the newly-established local markets, resulting in fierce competition and additional violence (Fagan and Chin, 1989).[14] When competition in the initial cities intensified, the national organizations moved to new areas where they could establish organized retail markets and continue to sell crack at marked up prices (Massing, 1989; Witkin, 1991). 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 … they had introduced much of the rest of the country to crack."

The potential for violence from competition within organized retail crack markets was exacerbated by both the type of customers buying crack, and the open-air sales locations. Crack

[13] This was different to other illicit drug markets. For example, Mieczkowski (1992) documents the existence of open air street sales of heroin in Detroit in the late 1970s. However, the entry barriers to these heroin markets limited the competition and subsequent violence in these open air heroin markets. Such barriers did not exist for retail crack cocaine markets.

[14] According to Massing (1989), a typical small 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."

cocaine was affordable to a low-income population for which powder cocaine was too expensive. Buyers would often purchase one "hit" at a time, which generated many transactions and opportunities for violence than existing drug markets. This violence was primarily perpetrated with firearms (Blumstein, 1995). Importantly for causing a widespread impact on educational outcomes, a diffusion of guns for self-defense further increased violence in areas with crack markets, resulting in large increases in the homicide rates of young black males that extended well beyond crack cocaine users and distributors (Blumstein, 1995).

b. Identifying the Arrival of Crack Cocaine

In order to estimate the long-run impacts of the crack epidemic on educational outcomes, we require a measure of when organized crack markets first emerged in cities and states. Despite numerous ethnographic accounts of crack markets, there is a lack of systematic measures of the arrival dates of crack markets across a large number of cities and states.[15] The difficulties associated with measuring illicit drug markets are compounded by surveys and administrative data not initially distinguishing crack cocaine from powder cocaine.

Our approach is to use cocaine-related deaths to estimate when crack markets emerged 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.[16] 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

[15] Existing measures of when crack markets emerged, like those in Grogger and Willis (2000) and Cork (1999), are for fewer than 30 cities. Other measures, such as Fryer et al. (2013), are indices that do not pinpoint when crack arrives.

[16] 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).

consumption of powder cocaine did not change substantially over this period, it seems reasonable to attribute this increase to the use of crack cocaine (Boggess and Bound, 1997).[17] 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), cocaine-related deaths provide a consistent and nationally-available measure that likely indicates the presence of crack. We create consistent mortality counts from 1980 to 1998 for every MSA with a 1980 population over 800,000. We 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 markets arrived are listed in Table 1.[18] The crack arrival years span 1982 to 1994. The three MSAs with the earliest arrival of crack are Los Angeles/Long Beach, Miami and New York, which matches reports on where crack cocaine first appeared. 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 or 1981.[19] Our dates are generally within a year of the earliest dates given in newspaper reports and ethnographies for larger cities.

Our dates are also broadly similar to the ones generated for a smaller set of cities by Grogger and Willis (2000) and Cork (1999). Grogger and Willis (2000) surveyed police chiefs in 25 cities in 1991 about when they first became aware of the existence of crack. 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

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

[18] Columns (2) to (4) of Appendix Tables A1 contain, respectively, the dates for cocaine deaths in two out of three consecutive years, three consecutive years, and a population-based measure that has higher thresholds for larger cities. The dates are similar across the different measures.

[19] Only the Nassua/Suffolk MSA (NY) consistently reports cocaine-related deaths prior to 1981; it is omitted from the sample. See the online appendix for more details.

increase in reports of smoking cocaine. The correlation coefficient between our measure and the police survey dates is 0.43, and between our measure and the DAWN measure is 0.37. These correlations are higher than the correlation across their two sets of dates, which is 0.32. Cork (1999) used murder and drug arrests to calculate a date for the arrival of crack markets. Our dates have a correlation coefficient of greater than 0.3 for each of these measures.

We adopt a similar approach for dating the arrival of crack cocaine in states and the District of Columbia. The crack arrival dates for 41 states, again using a measure based on two consecutive years, are presented in Table 2.[20] The earliest 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 the arrival of crack markets.

c. Factors Affecting the Spread of Crack Markets

We noted above that many observers and law enforcement officials have emphasized the importance of proximity to the major cocaine distribution hubs of New York, Miami and Los Angeles in the development of organized crack cocaine markets (Massing, 1989). If true, the spread of crack cocaine should be related to proximity to these three places, and less influenced by existing social and economic trends that may separately influence educational outcomes.

Tabulating the distances 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 driving distance to New York, Miami, or Los Angeles is 340 miles, compared to 424 miles for cities with arrival dates of 1985 and 1986, 559 miles for cities with

[20] In the three most populous states of California, New York and Texas, we use more than two deaths two years in a row. Ten states with small black populations are omitted because they do not have enough observations to analyze education outcomes.

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 Censuses.[21]

The results of these models are presented in Table 3. Hazard rates and 95 percent confidence intervals for the models 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 are more likely to get crack markets earlier, while cities farther from the three distribution hubs were likely to get crack later. The model based on changes in covariates between 1970 and 1980, shown in Column (2), displays a similar pattern except the estimated coefficient on the minimum distance variable is only statistically significant at a p-value of 0.10. These results suggest the spread of crack markets was not strongly related to differences in the economic characteristics of MSAs; further support for this comes from examining the relationship between murder rate changes and socioeconomic characteristics.[22]

[21] We use data from the 1970 One Percent Metro PUMS and the 1980 5-percent PUMS. The sample contains the 50 MSAs identified in both our crack arrival data and our PUMS samples.

[22] 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 further explore this question, we estimated an OLS model with the same

III. The Introduction of Crack Markets and the Murder and Incarceration of Youths

In this section, we provide more details about the rise in youth murder and incarceration rates discussed above, and consider their connection to crack markets. While we focus on these two outcomes because they are well measured for different demographic groups and across many locations, there is evidence that related outcomes like assaults and gunshot wounds also increased after the emergence of crack cocaine markets (Boggess and Bound, 1997; Fryer et al., 2013).

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

The aggregate annual murder rate was stable between 1980 and 1995, at between eight to ten deaths per 100,000 residents, before declining to six deaths per 100,000 by 2000.[23] However, this masks enormous differences across age groups. Figure 3a shows the murder rates for four age groups: less than 15 years; 15-24 years; 25-39 years; and 40 years and older. What is most striking is the large increase among 15-24 year olds, where the murder 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 aged less than 15 years are small and

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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. These estimates do not reflect a pre-existing relationship between murder rates and distance to LA, NYC, and Miami. 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 (0.0340).

[23] Homicides are taken from the public-use versions of the National Center for Health Statistics' 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.

reasonably flat. The homicide rate for those over 40 years old declines 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 the rates for other age groups.

To understand which subgroups of 15-24 year olds account for these changes, Figure 3b shows the homicide rates of 15-24 year old black males, black females, white females and white females. 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 reference 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.

We can also look at how these changes differed by location. Figure 3c shows the relative change since 1980 in the murder rate of black males aged 15-24 years for different city sizes. The largest increase was in cities with 250,000 – 500,000 residents, followed by cities with 100,000 – 250,000 residents. The smallest relative increase in murders was for cities with greater than 500,000 residents. This indicates that the increase in murder rates occurred outside of major cities. This fact is reinforced by noting that, of the 100 MSAs with the largest black population, the five cities with the largest absolute change in the murder rate of black males aged 15-24 years between the 1980s and 1990s were: Youngstown, OH; New Orleans, LA; Gary, IN;

Shreveport, LA; and Chattanooga, TN. Each city experienced an absolute increase in their murder rate of over 300 per 100,000 residents in this demographic group (or 0.3 percent). Their locations are also consistent with suggestions that competition for territory between national criminal enterprises in the middle of the country led to the worst crack-related violence (Massing, 1989; Witkin, 1991).

b. Increased Incarceration

The number of federal and state prisoners more than doubled from 1980 to 1996, driven by a nine-fold increase in incarceration numbers for drug offenses (Blumstein and Beck, 1999). 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 1999. The NCRP tracks the entry and exit from prisons, and includes basic demographic information and where the sentence was imposed. The NCRP data do not report previous incarcerations for those entering prison, so we are not able to identify first-time incarcerations. We instead calculate the percentage of each demographic group that enters prison in a particular year. There is inconsistent participation in the NCRP by states over time. Although 36 of our 41 states participate in any year, only 15 of our 41 states provided intake data for each year from 1983 to 1999.[24] The lack of data prior to 1983 and the small number of states reporting in the mid-1980s means there is little intake data prior to the arrival of crack markets.

Despite these limitations, the NCRP data provide insights into the general increase in

[24] In total there were 17 states participating in each year (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. New Hampshire and North Dakota are not in our 41 state sample.

imprisonment in the 1980s and the differences in this change by racial groups across states and over time. Figure 3d contains the prison intake rate for males aged 20 to 24 from 1983 to 1999 for the 17 states reporting data in each year. In 1983, the fraction of black and white males entering prison was 0.86 percent and 0.12 percent, respectively. Over the next 16 years, the prison intake rate for black males increased by 3.3 percentage points, compared to a 0.3 percentage point increase for white males. By 1999, the original 0.7 percentage point racial gap in incarceration rates grew to 3.6 percentage points. It is important to note that none of these figures include jails, which generally hold around 40 percent as many people as state and federal prisons (Caulkins and Chandler, 2006).

The increase in the prison intake rate is likely driven by both a change in criminal activity and a shift in federal policy that results, in part, from the emergence of crack cocaine. In the 1980s, Congress passed two major pieces of federal legislation attempted to address to the rising violence related to drug markets. The Anti-Drug Abuse Act of 1986 created a set of mandatory minimums for drug offenses. Two years later, the Omnibus Anti-Drug Abuse Act of 1988 created much harsher penalties for the possession of crack cocaine compared to powder cocaine. In addition, this law applied stringent penalties to all conspirators in a criminal organization, greatly increasing the probability of lengthy federal incarceration for low-level participants in the drug trade. Mascharka (2001, p.941) said that "this measure—designed to catch drug kingpins, who rarely have large quantities of drugs in their possession—has been criticized for being more routinely used against low-level drug dealers, look-outs, and peripheral conspirators such as the girlfriends of drug dealers." The broad application of mandatory minimum sentences in this period increased the risk of incarceration far beyond high-level dealers and traffickers.

In addition to the large national increase in incarceration, there was substantial variation

across states. In 1984, the state-level prison intake rates for black males aged 20 to 24 ranged from 0 to 1.4 percent. A decade later, this range was 0.3 to 6.9 percent. This variation is useful for identifying an effect of incarceration risk on educational attainment. However, there could be a concern that the variation is solely driven by changes in state policies and not the level of activity and violence in local crack markets. To the degree that these policy changes are a response to crack cocaine, they should still be considered an effect of emergence of these markets. For example, many states passed their own mandatory minimum statutes intended to deter and control crack cocaine markets.[25]

The rise in the prison intake rate does not appear to be related to increased sentence length brought about by the rising prevalence of "three strikes" laws that mandated long prison terms for habitual offenders, or "truth in sentencing" laws that required criminals to serve a certain percentage of their sentence. The vast majority of states adopting these laws did so after 1993 (Sabol et al., 2002). More importantly, these type of laws affect sentencing lengths, which directly changes the percentage of the population that is incarcerated, but can only indirectly impact the fraction that enter prison in a given year .

c. The Arrival of Crack Cocaine and Youth Homicide

In this section we examine how murder rates changed after the arrival of crack.[26] Figure 5 shows the murder rates of different age and demographic groups for the eight years before and

[25] California was one of the first states to enact the Anti-Drug Abuse Act in 1986, which made crack cocaine count for between two and four times the multiple of its actual weight in sentencing, depending on whether a court determined if the offender had intent to distribute. The next state to enact the law was Michigan which introduced one of the largest sentencing disparity laws in 1989 with a ratio of sentencing to actual weight of 75 to 1. In 1990, Maryland introduced a sentencing disparity between crack cocaine and powder cocaine at a ratio of 9 to 1, while North Dakota and Alabama set a 10 to 1 ratio in the same year. New Hampshire enacted a sentencing disparity of 28 to 1 in 1994, and a year later a sentencing disparity of 10 to 1 was introduced in Ohio.

[26] Given the limited amount of NCRP data prior to the emergence of crack markets it is not feasible to conduct a similar analysis for changes in incarceration rates.

nine years after the arrival of crack in the 57 MSAs for which we have arrival dates.[27] Panel A shows the rates for black males aged 15-24 years, 25-39 years, and 40 years and older. The 15-24 year old murder rate is initially flat, and then rises considerably after the arrival of crack markets. Six years after the arrival of crack, murder rates for young black males are twice what they were the year before the arrival of crack. Black males aged of 25 to 39 experienced a 12 percent increase after crack arrival, while there is no change in the trend for black males aged 40 and older.

Panel B of Figure 5 displays the murder rates of white males for the same three age groups. The 15-24 year old murder rate increases three years after crack arrives in MSAs, and increases by 51 percent eight years after crack arrives. It is important to note that the peak murder rate for this group was approximately one-eighth the rate of their black counterparts. Murder rates for white males aged 25 years and older are unchanged by the introduction of crack. Murder rates for black females are in Panel C. The respective rates for black females aged 15-24 and 25-39 had small increases in the years after crack arrives, although they are not affected to the same degree as males. The rates for white females are in Panel D; all age groups appear to be unaffected by the emergence of crack markets.

We estimate the impact the arrival of crack markets on murder rates using a difference-in-difference model, where individuals aged 40 and older serve as the comparison group. As the results in Figure 5 show, these individuals were largely unaffected by the emergence of crack but should be exposed to other factors driving changes in overall murder rates.[28] We estimate

[27] The results are similar if we re-organize murder rates in terms of the arrival dates for the 41 states.

[28] 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

separate models for 15-24 year olds and 25-39 year olds in each sex-race group.

We use a negative binomial count data model because there are zero murders in some MSA/year/group cells, especially in the female subsamples. The effect of crack markets is estimated from the interaction between a treatment group identifier and dummy variables spanning 0-2, 3-5, and 6-8 years after crack arrives. Covariates in the model include the treatment group main effect, plus city and year fixed effects. We also include the natural log of population for each demographic group and fix the parameter to be one, so the parameter estimates are roughly equivalent to estimates from an OLS regression with the natural log of the mortality rate as the outcome of interest. Observations are weighted by population values, and we allow for arbitrary correlation in observations within an MSA.

Table 4 contains the estimates for this model, with Panel A showing results for males. In comparison to black males aged 40 years and older, there is an increase in the murder rate of black males aged 15-24 in each period after crack markets arrive, with a near doubling of their murder rate six to eight years after the arrival of crack. These estimates closely match the unadjusted graphical evidence in Figure 5. Black males aged 25 to 39 are estimated to have a 30 percent higher murder rate six to eight years after the introduction of crack markets. The third row contains estimated coefficients for white males aged 15 to 24, who experience a 51 percent increase in their murder rate six to eight years after crack arrives. The estimates for white males aged 25-39 years, presented in the fourth row, are roughly one-third of this size. Virtually all the estimates in this panel are statistically significant at the five percent level.[29]

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crack, there were significant impacts on murder outside of central areas. Figure 3c 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 our context.

[29] In all models except for those for black females we can reject the null that the over-dispersion parameter equals 0. In the model for black males aged 15-24, this parameter (standard error) is 1.85 (0.36), meaning we can reject the null with a p-value <

Estimates for females are shown in Panel B. Compared to black females aged 40 plus, black females aged 15-24 and 25-39 years have statistically significant increases in their murder rates six to eight years after crack arrives of 20 percent and 10 percent, respectively. Coefficients on the earlier periods are smaller and statistically insignificant. White females aged 25-39 years have statistically significant increases for all three periods, although the increases never translate into an increase in the annual murder rate of more than one per 100,000 people.

IV. Crack Cocaine Markets and High School Completion Rates

If crack is responsible for the stalled progress in closing the black-white education gap, then its emergence in each MSA or state should be followed by worsening black educational outcomes. We investigate this using data from the 2000 Census Five Percent PUMS, and exploit differential timing in the arrival of crack markets. While we would like to know each individual's place of residence during high school, we only know their MSA and state of residence, and their state of birth. We will show that using current residence or place of birth in the state sample produces similar estimates, suggesting migration concerns are not having a strong impact on the results.

Each sample contains U.S.-born, non-Hispanic respondents who turned 18 between the years 1975 and 1998. The first includes individuals currently residing in the 57 MSAs. Among the respondents in this sample, 80 percent resided in the MSA they lived in five years previously.[30] Our second and third samples are based on the 41 states with larger black populations. The second sample contains all current residents of those states, while the third

0.01. The estimates are not sensitive to the use of the negative binomial distribution. When we estimate the model from Table 4 assuming that counts are a Poisson distribution, the coefficients (standard errors) on 0-2, 3-5 and 6-8 years after crack variables are 0.260 (0.069), 0.704 (0.083), 1.004 (0.056), which are within 3 percent of the equivalent estimates in Table 4.

[30] A specific concern may be that families move to get away from crack-related violence. Cullen and Levitt (1999) find migration in response to crime occurs by families, but they tend to stay within the same MSA.

contains respondents born in them. In addition to addressing migration concerns, a benefit of these state samples is that it allows us to include individuals living outside of metropolitan areas. As discussed above, black males in non-metropolitan areas experienced dramatic increases in murders over this time period.

Figures 4a and 4b were introduced previously. In these graphs, we use data from the MSA-based Five Percent 2000 PUMS sample to examine changes in black and white high school completion rates in relation to when crack arrives in cities. Figure 4a shows the completion rates for males, and is consistent with Figure 1 in that it shows a convergence in white-black outcomes prior to the arrival of crack. Two years after crack arrives, the graduation rate for 18 year old black males starts to decrease. 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 4b contains a similar analysis for females. The graduation rate of black females declines relative to whites after the arrival of crack, although this decline is smaller than for males. Figures 3c and 3d contain similar completion rates using the PUMS sample based on state of birth, and display similar patterns to those at the MSA level.

We also use a regression to examine the impact of the arrival of crack markets on completion rates. 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 emergence of these illicit markets. Since there are persistent differences in outcomes across geographic regions, races, and cohorts, the model must also control for these dimensions of the data. To capture these characteristics we use the following specification:

(1) $$y_{icg} = \mu_c + \lambda_g + \eta_1 Black_{icg} + Black_{icg} * CappedTrend_{cg}\beta_1 + Black_{icg} * YearsAfterCrack_{cg}\beta_2 + \varepsilon_{icg}$$

where y_{icg} is an indicator equal to one if person *i* from cohort *c* and geographic area *g* graduated high school, and zero otherwise. A complete set of fixed effects for each birth-year cohort is represented by μ_c, a full set of geographic-specific fixed effects are given by λ_g, $Black_{icg}$ is an indicator equal to one if the respondent is black and zero otherwise, and ε_{icg} is an idiosyncratic error term that allows for arbitrary correlation in errors within a geographic area. We also estimate a specification with the addition of a full set of race-specific cohort fixed effects. Estimates are weighted using Census person weights.

The variable $CappedTrend_{cg}$ measures trends in high school completion 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, after which it is held constant. For example, we estimate that crack arrives in Los Angeles, Miami, and New York in 1982, so $CappedTrend_{cg}$ is set to ten for residents in those MSAs for all years after 1982. The coefficient β_1 results from the interaction of this variable and the black indicator $Black_{icg}$. It measures the convergence in white-black graduation rates across cohorts prior to the arrival of crack markets, so we expect β_1 to be positive.

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. In order to maintain an equal set of post-crack years, we drop all observations for a city that are greater than 8 years after crack arrives. In the online appendix we report results without these sample restrictions that are qualitatively similar to our main results. The coefficient β_2 from the interaction of this trend variable and the race indicator

$Black_{icg}$ measures the trend in white-black graduation rates after the arrival of crack.[31] 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 that the arrival of crack markets 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.

To better understand the role of the trend variables, define a variable $Trend_c$ that equals one for the oldest cohort, two for the second, etc. Notice that, by design, $Trend_c = CappedTrend_{cg} + YearsAfterCrack_{cg}$. If the arrival of crack had no race-specific impact on the time series of 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 markets had no impact on high school completion rates. In our reduced form results below, we report a test for the equality of the coefficients on these two trends. If the emergence of crack cocaine markets decreased black educational outcomes compared to their white counterparts, β_2 would be smaller and statistically different from β_1.

We also estimate a specification of equation (1) that replaces the $CappedTrend_{cg}$ and $YearsAfterCrack_{cg}$ trends with a series of indicator variables that identify the eight years before crack arrives and the eight years afterwards. Given the number of coefficients from this specification, we graphically summarize the results in a series of event study figures.

Table 5 contains the key coefficients of interest from estimating equation (1) using

[31] 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.

several different samples. The first column of Panel A contains estimates from the MSA sample of males. For each year prior to the arrival of crack, the percentage of blacks with a high school degree increased by a statistically significant 0.23 percentage points (p-value<0.01) compared to their white counterparts. The estimated coefficient on $Black_{icg}$*$YearsAfterCrack_{cg}$ suggests that, after crack arrives, black males had a statistically significant (p-value<0.05) average annual decline in relative high school completion rates of 0.3 percentage points. The p-value for the equality of these coefficients is less than 0.01.

The first column of Panel B contains similar estimates for females using the MSA sample. While black female graduation rates also showed progress compared to whites before the arrival of crack, the estimated annual impact of crack on the probability of obtaining a high school degree for females is approximately forty percent smaller than the estimate for males and only statistically significant at a p-value of 0.10. The p-value on the test of equality for the pre- and post-crack trends for females is 0.05.

One concern with our reduced form results is that given the aggregate changes in educational outcomes, many sets of dates chosen for the arrival of crack cocaine markets in a city or state might result in a negative and statistically significant estimate on $Black_{icg}$*$YearsAfterCrack_{cg}$. To see if our set of dates provides unique and important information about changes in high school completion rates, we conduct an exercise where we allow the set of arrival dates to randomly vary across an interval of four years before or after the date used in our main results, and then estimate equation (1) using these random dates. Doing this exercise 2,000 times we find that the estimate using our dates is at the fifth percentile of all estimates.[32] This

[32] The relatively narrow band in this test means that it contains a good deal of information from our methodology. If our dates of the emergence of crack markets impart unique information about the change in black high school complete, then increasing the size of the band should result in the estimate from our dates being more distinct. For example, if we draw random crack

provides clear evidence that our dates for the emergence of crack cocaine markets provide particularly important information about the effect of these markets on the educational outcomes of black males.

The third and fifth columns of Panel A contain equivalent estimates from the state of birth and current state of residence PUMS samples. The coefficients across both samples are remarkably similar. The estimated annual decrease in black male graduation rates after the arrival of crack is 0.32 percentage points the state of residence sample and 0.4 percentage points for the state of birth sample. The post crack decline in high school achievement for females is again smaller than for males, but the test of the quality of the pre-crack and post-crack coefficients is statistically significant at a p-value of 0.05.

Columns 2, 4, and 6 contain the estimates from a specification of equation (1) where we add race-specific cohort fixed effects. This specification absorbs nearly all of the residual variation in the data. For example, the R^2 from a regression of $YearsAfterCrack_{cg}$ on the set of geographic and race-by-cohort fixed effects is 0.9944. A similar regression of the post-crack trend on the race, cohort, and geographic fixed effects used in our main specification has an R^2 of only 0.4816. Although the covariates in this new specification leave little variation in the data for identifying the effect of crack cocaine markets on educational outcomes, estimates in the state-based samples still provide evidence of a break from the pre-existing convergence in educational outcomes. While the estimated coefficient for males on the post-crack trend is no longer negative and statistically significant, in both the state-of-birth and state-of-residence samples the p-value on the test of equality of the pre- and post-crack coefficients is statistically significant at a p-value of 0.05. Similarly, for females in the state of birth sample, the p-value on

introduction dates that are 8 years on either side of our date, the estimates from our dates is at the one half percentile of all estimates.

the test of equality for the pre- and post-crack trends is 0.04.

Figure 6 contains event study graphs from a specification of equation (1) that replaces the trend variables with indicator variables for each year before and after the arrival of crack. The omitted year is the one in which crack arrives in the MSA or state. In general, these figures provide a very similar picture to Figure 4. This is most apparent in the male state-of-birth results in Panel C. In the years prior to the emergence of crack cocaine markets, the relative high school completion rate of black males is trending upwards. However, it declined precipitously in the years following the establishment of crack cocaine markets. Similar to the results in Table 5, the estimated coefficients for females in Panel D show a far smaller impact of crack cocaine markets on female graduation rates.

While the location fixed effects in equation (1) control for time-invariant differences across areas, it is possible that changing economic and community characteristics affected graduation rates. To account for the potential impact of these factors, we estimated a specification of equation (1) that includes covariates that vary by state, race and year, such as the educational status of mothers, the percentage of children without a father present in the home, median family income, and the state unemployment rate.[33] These covariates are generally available starting in 1978 when the CPS started to identify all states in the sample. We also include race-specific measures of the school environment, including current expenditures per student, the racial fractions in schools, and the within-district Gini coefficient in family income.[34] The full set of results from this specification is available in the online appendix. The inclusion of covariates decreases the magnitude of the male pre-trend estimate for males by

[33] The first three variables were calculated from the March CPS while the state unemployment rate is calculated from all monthly CPS samples.

[34] The variables are calculated from the data in Corcoran and Evans (2010) which is a panel data set of unified school districts from 1970/1980/1990/2000. To get race-specific state level averages, we aggregate data from the district to the state level using the number of whites and blacks in the district as the weight. We interpolate data between census dates.

approximately 30 percent, which is similar to the results in Cook and Evans (2000) who found that school and demographic factors can explain 25 percent of the convergence in test scores. The estimated impact of crack arrival on black male graduation rates is still a statistically significant -0.2 percentage points per annum, and the p-value on the test of equality with the pre-crack trend is less than 0.01. After the inclusion of covariates, the estimate on the post-crack trend for females in MSAs falls to -0.0002 (0.0007) and the p-value on the test of equality between the pre- and post-crack trend coefficients is 0.07. These additional results suggest that the estimates in Table 5 are unlikely to be solely due to changing socioeconomic and school-level characteristics.

To further examine the robustness of our findings, we examine the effect of crack cocaine on high school completion using the alternative versions of our dating methodology and the dates generated by previous authors. The first four columns of Appendix Table A5, contain estimates from equation (1) using the alternate dates for the arrival of crack cocaine markets defined in Appendix Table A1. The pattern across all of these dating measures is qualitatively similar, with the population-based threshold measure showing the largest post-crack decline in black male high school completion. Columns (5) and (6) contain estimates using the two sets of arrival dates in Cork (1999). Both sets of dates show a statistically different pre and post crack trend at a p-value of 0.10. Finally column (7) and (8) contain estimates for the two sets of dates in Grogger and Willis (2000). For the dates obtained from changes in emergency room admissions there is a statistically different pre- and post-crack trend, however the estimates for the police chief survey lack the precision to detect a difference in the pre- and post-crack trends.

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

The results above show a link between the arrival of crack market and educational outcomes. In order to understand the potential mechanisms underlying this stalled progress, we next construct two measures of the intensity of problems associated with these markets that are based on murder and prison intake rates, and then examine their relationship with educational outcomes.

a. The Rapid Rise in the Murder Rate

If the perceived mortality risk following the introduction of crack is affecting the educational outcomes of young black males, then graduation should be correlated with the murder rates of a reference group of older black males. As a measure of perceived mortality risk, for each individual we calculate the average murder rates of 20-24 year olds when they were aged 16 to 18 years. We make these rates specific to location, sex and race. For example, a black male in Denver is assigned the average murder rate of 20-24 year old black males in Denver in the years he was 16, 17 and 18. The use of 20-24 year old rates avoids a potential mechanical correlation between educational outcomes and homicides.[35]

Table 6 contains the estimates from a model that regresses high school completion rates on the average murder rates during high school, plus controls that are described below. Given the disparities in murder rates by gender, we estimate models separately by sex. The top panel of Table 6 contains estimates for males, while the bottom panel shows the results for females. We utilize four different samples. In columns (1) and (2) we generate estimates for the 57 MSAs that match the cities used in the reduced-form analysis form Table 5. In columns (3) and (4) we expand this to include all 176 MSAs that we can define consistently in the MCOD data and

[35] The results are robust to using the murder rate for individuals aged 15 to 24.

match to data in the PUMS. In columns (5) and (6), we merge in data based on the respondents' current state of residence using the 41 states with the largest black populations, while in the final two columns we use data based on the respondents' state of birth for these same 41 states. For each sample, we estimate two models. In the odd-numbered columns, our controls are an indicator variable for being black, plus cohort and geographic area fixed effects. In the even-numbered columns, we provide estimates from a specification with the addition of race-specific cohort effects. In all models, we calculate standard errors allowing for an arbitrary correlation in errors within the geographic area, and use Census person weights. For each model, we report the coefficient on the murder rate (x 1000) and the standard error.

The estimates for the 57 larger MSAs suggest that, as murder rates for the reference group increase, there is a statistically significant decline in high school completion rates. Importantly, the inclusion of race-specific cohort effects in column (2) does not qualitatively change the estimates. Changing the sample from the 57 larger MSAs to all 176 MSAs results in similar estimates, as can be seen by comparing columns (1) and (3). Adding race-specific cohort effects reduces the coefficient on the race-specific murder rate by 23 percent in the 176 MSA sample but the estimate remains statistically significant at demanding levels.

Moving to samples based on state of residence or birth, we note two important features of the data. First, the sample sizes increase considerably as we now include residents in smaller geographic areas. Second, these smaller towns tended to have a much larger increase in murder rates over the 1980s and early 1990s. Given these facts, it is no surprise then that we obtain a larger coefficient on the race-specific murder rate. The results in column (5) and (7) are 85 and percent and 43 percent larger than the comparable estimates for the sample of 176 MSAs from column (3). The inclusion of race-specific cohort effects reduces the results in the state of

residence sample by 17 percent, and the results in the state of birth sample by 30 percent. However, even after including this extensive set of controls, the results are still statistically significant at demanding levels, and represent qualitatively important effects.

In all specifications for females, the coefficient on the race-specific murder rates is negative, about one tenth the size of the coefficient for males, and statistically insignificant. Given the small relative change in murder rates for black females, and the small estimated coefficient on the race-specific murder rates, we are not able to detect a relationship between violence and the black female high school completion rate.

b. Increased Incarceration of Black Males

Changes in the probability of incarceration would further limit the expected benefits from investments in human capital.[36] We analyze the impact of incarceration on male educational attainment using the state PUMS sample, with high school attendance defined as the state of birth.[37] Table 7 shows the estimates from two samples. We first use the 15 states that are both reporting NCRP data each year and among the 41 states with the largest black populations. Panel A of Columns (1) and (2) show coefficients from the murder rate regression used to produce the results in Table 6. While these estimates are approximately twice the magnitude of the corresponding results in Table 6, the estimated coefficient of interest is not statistically different from previous ones at the five percent level. To put these numbers in perspective, below these estimates in square brackets is the total estimated effect on black high school completion using the change in the relevant black murder rates from 1983 to 1993. They suggest that rising murder

[36] Incarceration potentially has direct effects on high school graduation as measured here, as approximately one-tenth of GEDs are obtained in prison (Heckman and Lafontaine, 2010). This may lead to an understatement of any negative relationship between incarceration rates and high school graduation.

[37] As for the results we have presented so far, similar results are obtained using state of current residence.

rates can explain a four percentage point drop in the high school completion rate of black males.

The second sample is the 36 states that report intake data in at least one year. The results for this sample are shown in Panel B in the equivalent columns of Table 6. They show a relationship between murder rates and high school completion rates that explains an approximately two percentage point decline in the high school completion rates of black males in these states. The relative decline in black male high school graduation rates between the 1980s and 1990s was smaller in these 36 states than in the 15 state sample.

Columns (3) and (4) of Table 7 contain 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.[38] The main specification includes state, cohort, and race fixed effects. We also report results with a full set of race-specific cohort fixed effects. The marginal effects suggest that changes in average intake rates from 1983 to 1993 is responsible for a decline in black males completion rates of four to six percentage points in the 15-state sample, and two to three percentage points in the 36-state sample.

In order to determine the independent impacts of murder and prison rates, columns (5) and (6) contain the estimates from a specification with both the murder rate and prison intake rate included as independent variables. In the sample of 15 states, both of these factors are independently predictive of changes in the high school completion rate. The marginal effects for black males suggests that the combined impact of these factors is a decline in male high school completion rates of six to seven percentage points, with a roughly similar impact from the two variables. In the sample of 36 states, the p-value on the estimate of the prison intake rate in column (5) is only 0.14. The lack of precision in the 36 state sample for the incarceration

[38] 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.

estimates likely results from the fact that for many of the additional states included in this larger sample we lack data detailing the large increase in the prison intake rate that occurred during the mid-1980s. That being said, the point estimates suggest a combined marginal effect of four to five percentage points.

Across both samples, including the prison intake rate as an independent variable has a small effect on the magnitude of the estimated relationship between the mortality rate and high school completion. For the 15-state sample, accounting for the prison intake rate decreases the coefficient on the murder rate by 28 percent in the main estimate and 15 percent in the specification including race-specific cohort effects. In the 36-state sample, the estimated coefficient falls by no more than seventeen percent across the two specifications. This suggests that the relationship between mortality and high school completion is not strongly influenced by changing incarceration over this period.

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

Fryer et al. (2013) constructs an index of the severity of crack in 144 cities and in states.[39] It is composed of factors such as homicides, cocaine arrests, cocaine-related emergency department measures, cocaine seizures by the Drug Enforcement Agency, and newspaper articles discussing crack cocaine.[40] The index provides a summary measure of social indicators that may have been affected by crack markets, together with indicators of crack market activity and awareness. While index values are not separated by race, and may therefore be influenced by the racial composition of a city or state, it does provide an alternative measure of crack market severity that can be used to check the robustness of our results.

[39] Among the 57 MSAs in our reduced-form analysis, 52 are included in the Fryer et al. index.
[40] Newspaper articles are included in this measure if they contain the name of city along with both the terms crack and cocaine.

We used the PUMS MSA sample to estimate the relationship between the high school completion of blacks and the Fryer et al. index. We merge their index into the data based on the year each cohort turned 18, so a respondent from New York that turns 18 in 1986 received the index number for that year. We interact the index with the black identifier, and include fixed effects for cohorts, MSA and race. The estimated coefficient (standard error) on the interaction term for such a model is -0.0091 (0.0029) for males and -0.0038 (0.0026) for females. From 1983 to 1993, the Fryer et al. index increased by 1.85 points, suggesting that crack was associated with roughly a two percentage point decrease in black male graduation rates.

d. Concerns about Internal and External Validity

The emergence of organized crack cocaine markets is a unique event and, as a result, there are two important questions surrounding the results in Tables 6 and 7. The first is the interval validity of the numbers, and whether the unique aspects of the event raise concerns that the results are driven by an omitted variables bias. The second issue is external validity, and whether the uniqueness of the event conveys any information about a more general economic process. In this section, we address both of these concerns.

There is the possibility that the results in Tables 6 and 7 are driven by an omitted variables bias, where the rise of murder and incarceration rates following the emergence of crack cocaine markets and the fall in the educational attainment of black males are actually symptoms of a third unmeasured factor like urban decay, and hence are spuriously correlated. If true, this would suggest that the large negative coefficients we obtain for the race-specific murder rate in Table 6 are unique to the period under study. We address this concern by noting that the general link between young adult murders and educational attainment should, to some degree, be present

in other time periods. We therefore estimate the main model from Table 6 using data for cohorts that turned 18 prior to the presence of crack cocaine in the U.S. If the results in Table 6 are driven by some omitted factor, the relationship between murder rate changes and high school completion should be weaker in this restricted sample.

We report the results for cohorts turning 18 between 1975 and 1981 in column (2) of Table 8, using data for the 176 MSAs and the model that contains fixed effects for race, cohort and MSA. The specification is analogous to the specification in column (3) of Table 6, and for reference we report those coefficients in column (1) of Table 8. The top half of the table contains the estimates for males, and the results for females are in the bottom half. The estimates in column (2) of Table 8, based on the pre-crack era, are virtually identical to those in column (1) that include cohorts that turned 18 before and after the arrival of crack. The coefficient on the race-specific murder rate for males in the pre-crack sample is statistically significant at the one percent level, even though the sample size has been cut by two-thirds.[41] We cannot reject the null that the results in columns (1) and (2) are the same. These estimates suggest that our main results are not driven by a general unmeasured variable coterminous with the emergence of crack cocaine markets.

In the bottom half of the table, columns (1) and (2) contain the equivalent results for females. Interestingly, with the smaller sample and lower murder rates in this group, we now estimate a coefficient on the race-specific murder rate for females that is roughly the same magnitude as the companion estimate for males. The p-value on the null hypothesis that this coefficient equals zero is 0.11.

[41] Murder rates for young black males were in excess of 100 per 100,000 prior to the arrival of crack, and there is tremendous time series and cross sectional variation in the race-specific murder rate even during this period. Given this, it is not surprising that we can generate a statistically significant coefficient in the smaller pre-crack cocaine sample

Although we use the race-specific homicide rate as the covariate of interest, there is no reason that human capital investments should only respond to this cause of death. Forward-looking teens should respond to any salient change in mortality, regardless of the cause. In a second set of models, we consider the external validity of our estimates by replacing the murder rate with aggregate death rates. In this case, we estimate models identical to those in columns (1), but replace the race-specific murder rate with the aggregate death rate for young adults aged 20-24 for cohorts when they are aged 16 to 18. These estimates are reported in columns (3).

The coefficient on the race-specific death rate is virtually identical in both the male and female equations and, in both cases, the estimate is statistically significant. Interestingly, the coefficient is almost the same as the coefficient for the homicide rate for males reported in column (1). One might be concerned that the only changes in aggregate death rates over this period is driven by murder rates, but in column (4) we replace the aggregate death rate with the non-homicide deaths rate and find coefficients on those variables close to those for the aggregate death rate. While we are not able to detect a murder risk-education gradient for females in the main estimates in Table 6, this is likely a result of relatively limited variation in murder rates for women over this time period. We are reassured, however, by the similar aggregate mortality risk-education gradient across the sexes in Table 8.

Finally, we consider whether there is heterogeneity across races in the impact of the murder and death rates on human capital accumulation. In columns (5) and (6), we allow the coefficient on the race-specific murder and death rates to vary by race, and find no statistically significant difference in the mortality-education gradient by race for either males or females.

Taken together, we believe the results from these additional models demonstrate that our main estimates are not specific to the time period around the emergence of crack cocaine markets

or the result of an unobserved trend coterminous with the murder rate throughout our main sample. Instead, our estimates result from the combination of a more general mortality-education gradient and a shock to black male murder rates.

VI. Understanding the Mechanisms

In this section, we consider the explanatory power of changes in human capital investment, and whether other crack-related changes may have also affected educational attainment. We show it is plausible that changing returns to education account for a substantial part of the observed decline in the black high school completion rates. We then consider alternative explanations 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 emergence of crack cocaine markets affected educational outcomes.

a. The Potential Role of Human Capital Investment

We start with the relationship between homicides and high school completion. The various estimates suggest that a 0.1 percentage point increase in the annual risk of 20-24 year old black males being murdered generally results in a decline in the high school completion of black males of 1–2 percentage points. It is important to note two things when it comes to interpreting changes in annual homicide rates. The first is that annual changes lead to much larger cumulative effects. While the homicide risk for black males aged 20-24 in both samples increased by 0.1 percentage points per annum between 1983 and 1993, the cumulative risk that a 20 year old black male would be murdered before reaching 25 years of age increased by 0.44 percentage

points, and their chance of dying from any cause increased by 0.54 percentage points.[42] Mortality risks were also changing among those outside of the 20-24 age range, as shown in Figures 2 and 4. It is informative to again consider 15 year old black males assessing cumulative risks in terms of contemporaneous death rates of older black males. Their probability of dying by age 30 increased by 1.6 percentage points between 1983 and 1993. If life expectancy is censored at 65, as a measure of changes to working life, then between 1983 and 1993 their estimated work life declined by 0.9 years (1.5 percent), from 58.1 to 57.2 years.[43]

A second point is that, during periods of increased gun homicides, there should be increased rates of non-fatal gunshots, which may have distinct effects on future earnings.[44] Cook (1985) estimates that the death rate from gun assaults is approximately 15 percent, which suggests there are approximately 5.7 non-fatal shootings for each fatality. Many result in hospitalizations, and some lead to long-term impairments like brain trauma and spinal cord injuries. Studies have estimated that aggregate earnings losses from non-fatal gunshot wounds are between five and 40 percent as large as the losses associated with gun-related fatalities (Max and Rice, 1993; Miller and Cohen, 1997; Corso et al. 2007).

When the 1.5 percent decline in expected working life due to an increase in murders is scaled to account for the additional effect of gunshot wounds, then expected working life may have declined by 1.5-2.1 percent during this period. The implied of elasticity of high school completion to this changing expectancy of working life is in the range of 0.5 to 1.3. This calculation is useful, as it is the same one made by Jayachandran and Lleras-Muney (2009) in

[42] As before, this is basing risk on the contemporaneous age-specific mortality rates for an individual's same demographic group.
[43] For white males, over the same period the contemporaneous risk of dying by age 30 decreased by 0.1 percentage points, from 2.2 to 2.1 percent, 65-censored life expectancy increased by 0.09 years, or 0.16 percent.
[44] Between 1983 and 1993, there is a correlation of 0.98 between the annual number murders with firearms and the annual number assaults with firearms recorded in the Uniform Crime Reports (http://bjs.ojp.usdoj.gov/content/glance/tables/guncrimetab.cfm).

understanding the relationship between maternal mortality declines in Sri Lanka and human capital accumulation (measured in terms of schooling years and literacy). Their implied elasticity is between 0.6 and 1.

In total, these exercises indicate the possibility that the magnitude of the relationships identified through the murder rate are consistent with changes in expectations about the returns to educational attainment. The labor market impacts of incarceration also suggest the estimates presented in Table 7 may be largely related to lowering returns to education. The direct incapacitation effects of incarceration obviously prevent work, and increasing sentence lengths were the primary driver of the growth in incarceration between 1980 and 1996 (Blumstein and Beck, 1999). There is also evidence that ex-prisoners have long-term difficulties in finding employment and slower earnings growth. For black males, recent studies have found incarceration lowers subsequent employment by approximately 12 to 25 percent (Western and Beckett, 1999; Western, 2006; Raphael, 2007). Job quality is also affected, with Western (2002) estimating that incarceration lowers wage growth by approximately 30 percent. Bonczar (2003) estimates that black males' lifetime risk of going to prison changed from 13 to 31 percent between 1979 and 1997.[45] The magnitude of the increases in incarceration, and the labor market consequences of these changes, suggest it is plausible that the high school completion rates of black males declined by 2-3 percentage points between the early 1980s and mid 1990s.

b. What is the Role of Income Generation through Drug Dealing while of School Age?

One possible pathway through which the rise of crack markets could impact education attainment is if young males were exiting school in order to participate full time in the drug

[45] These figures, and the dataset we use, do not include offenders in jail. Caulkins and Chandler (2006) estimate that including jailed drug offenders increases incarceration estimates by 40 percent.

trade. Unfortunately, detailed information about the income-generating effects of crack cocaine markets is not available, so we must rely on insights from the literature on the returns to drug dealing to understand its likely role in our estimates. [46] Studies use a variety of data collection methods including 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, Dunlap and Tourigny, 2000). While these studies do not necessarily provide a representative picture of crack cocaine dealing, several regularities across studies suggest that it is unlikely that students are dropping out of school to be involved in the drug trade.

One is that crack dealing was predominantly a part-time activity that did not conflict with formal employment or high school attendance. Johnson, Dunlap, and Tourigny (2000) observed 300 participants in crack markets in New York and found most used it as a sporadic way to earn 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 DC between 1987 and 1989, and found the employment rates of individuals arrested for drug selling were similar 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.

[46] Per-capita emergency department (ER) mentions of cocaine are a noisy measure of MSA-level consumption, which should also be related to the income-generating opportunities from being involved in distributing crack. These have been published in Drug Abuse Warning Network reports, the surveillance system that Grogger and Willis (2000) used to create one of their set of crack arrival dates. We located reports covering 1978-1999 and used them to calculate rates of cocaine ER mentions for 20 of the 57 MSAs in our sample. Appendix Table A7 reports the estimates from estimates from a regression of educational attainment on ER mentions including race, cohort, and city fixed effects. We find a negative but statistically insignificant relationship between cocaine ER mentions and male high school graduation when we include both ER mentions and the murder rate as independent variables, while the magnitude of the negative relationship between murder rates and high school completion is unchanged. The only difference is that, with only 20 MSAs, the coefficients are only statistically significant at the 10 percent level; this is the case with or without the inclusion of cocaine ER episodes as an additional variable.

This is not to say that participation in the drug trade did not have an impact. Being involved in the drug trade would certainly increase the risk of homicide mortality and/or arrest. Reuter et al. (1990) found that among adolescents in DC, half though that dealing drugs for a year would lead to serious injury or death, while 38 percent felt it would lead to an arrest. Being involved in the drug trade therefore increased risks that would lower the expected returns to education. That said, dropping out of school was neither a necessary nor a sufficient reason to be involved in such activity.

c. Differential Crack Cocaine Use as an Alternate Mechanism

The use of crack cocaine by school-age teens is unlikely to be an important mechanism behind the changes in educational attainment, 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 percent of high school seniors reported crack cocaine use in the past year (Johnston, O'Malley and Backman, 1991). Second, what data exists suggest that blacks in this age range used crack at rates similar to that of whites. According to data on drug utilization by age from the National Household Survey of Drug Abuse in 1995, the lifetime crack use for 16 to 17 years olds was 5.1 percent for whites compared to 4.2 percent for blacks. While participation rates may mask underlying differences in cocaine dependence, treatment of teenagers for smokeable cocaine was relatively rare.[47]

d. The Roles of Changing Family Structure and School Quality

[47] 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), children aged 17 years or less accounted for less than one percent of all crack cocaine treatment episodes. Individuals aged 18 and 19 accounted for two percent of episodes (SAMHSA, 1997).

High school completion rates are higher among children from families that that have more educated parents, higher incomes, and both biological parents (Astone and McLanahan, 1991; Sandefur, McLanahan and Wojtkiewicz, 1992; Haveman, Wolfe and Spaulding, 1991; Evans and Schwab, 1995). Another possibility is that, rather than reacting to increased mortality or prison risk, the changes in educational outcomes outlined above reflect changes in family structure or differential school quality. Some of the possible explanations can be dismissed without much discussion. For example, Berends and Penaloza (2008) show that among children in school from 1972 to 2004, parental education increased for black children relative to white children, indicating this input cannot explain the differential trends we detect.

In general, we would need to identify a change in a determinant of high school completion that a) moved massively against blacks relative to whites and b) differentially impacted males compared to females. For a variety of reasons, we suspect these concerns are not the case. The nine chapters in Magnuson and Waldfogel (2008) round up the "usual suspects" (e.g., changing income levels and inequality, school inputs, changes in family structure, etc.) in an attempt to explain the stalled progress among blacks in test scores. Their conclusion is that few of the inputs to test scores they consider moved adversely enough against black students compared to their white counterparts to explain stall progress in test scores. Neal (2006) also examines similar factors as possible explanations but finds no strong connection. It is therefore unlikely these inputs moved enough to explain the massive decline in high school completion rates experienced by black males outlined in Figure 1.

Perhaps more importantly, although the crack epidemic generated a wave of violence and incarceration, the changes in observed characteristics for families that are typically predictive of high school completion rates did not move appreciably for black families with children when

compared to white families with children. In Table 9, we provide descriptive information about changes in the family characteristics and economic conditions for black and white families with children from the early 1980s to the 1990s. This table includes estimates from a variety of samples including the March Current Population Survey.[48]

The results in Table 9 provide at best mixed evidence of deteriorating conditions for black families over our sample period. While there is some decline in basic conditions among families with young black children (e.g., more out-of-wedlock births, higher poverty rates, lower fraction of families with a birth father present), many outcomes are improving (e.g., lower fraction of families on food stamps, smaller fraction of teen births, higher families where the birth mother is present, higher rates of return to education, lower unemployment rates and more school spending). Moreover, the absolute and relative changes in these variables are modest. These factors do not seem to explain the collapse in high school completion rates for black youths.

In addition, if the small observed changes in these outcomes were actually responsible for the decreased educational outcomes among older black males, then we might expect results of similar magnitude 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 far smaller effect for females limits the potential for changes in school quality or family structure to

[48] In the top half of Table 9, we generate descriptive information about families from the 1980 and 1990 March Current Population Survey (King et al., 2010) for families with children aged 0-14. In the next block of results in Table 9, we use data from the 1981 and 1991 March CPS, reduce the sample to those aged 25-44 with a high school degree and estimate the rate of return to a college degree. We restrict the sample to males with 30 or more hours per week and 40 or more weeks per year and estimate a Mincer-type human capital earnings function. The dependent variable in the model is the natural log of weekly earnings and key covariate is a dummy variable for whether the respondent has at least a four year degree. We control for a complete set of dummies for age, type of marital status, pus controls for race and veteran's status. In the table we report the rate of return to a college degree for males and its associated standard error. We estimate separate models for each year and allow the rate of return to vary by race. In the next row we use the monthly CPS to construct the annual unemployment rate for black and white non-Hispanics. In the final row of the table, we report the average current expenditures in K-12 education for a randomly selected black and white student from Corcoran and Evans (2010).

solely 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 nine, 13, and 17 year olds every two to five years since the early 1970s, and tests have not changed over this period.[49] In Figures 7a and 7b, 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 percent higher than of blacks in 1971.[50] This gap fell to seven percent in 1988, and then rose to 11 percent by 2004. For this same age group, the gap in math test scores halved from 14 to seven percent between 1978 and 1990, and then rose to 10 percent by 2004.[51] 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 generally 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 is not a primary driver of stalled progress.

VII. Conclusion

[49] More information about the NAEP LTT is available at http://nces.ed.gov/nationsreportcard/ltt/.
[50] 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).
[51] 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.

The emergence of crack cocaine markets generated three primary impacts: an increased murder rate, an increased prospect of incarceration, and an increased opportunity for employment outside of the formal sector. These factors disproportionately affected black youth, and particularly black males. Each of these factors lowers the potential return from an investment in human capital, and therefore our estimates of reduced schooling provide suggestive evidence of individuals investing in education as outlined in Becker (1964) and Ben-Porath (1967).

From 1965 to 1985, the percentage of black males who received a high school degree increased by an average of approximately 0.3 percentage points per year. The results above show that the introduction of crack cocaine reversed this progress. Over the period of greatest post-crack decline in black high school completion rates, 1986 to 1996, there was an approximately eight 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 change in murder and incarcerations risks explains between 37 and 73 percent of the decline in educational achievement among black males between 1986 and 1996.[52]

By the late 1990s and early 2000s, the murder rate 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. Despite a general belief that this marked an end of crack epidemic beginning in the mid-1990s, it is important to note that the decline in mortality risk was not matched by a similar decrease in the prison intake rate. According to our data, in each year from 1995-1999 over four percent of black males aged 20-24 entered a federal or state prison facility. The rate for similarly-aged white males over this time

[52] This estimate is based on a historical trend of convergence in male black-white high school completion of 0.3 percentage points per year from 1965 to 1985. The magnitude of the combined effect of mortality and prison on black male educational outcomes comes from column (5) of Panel A and B of Table 7.

period never exceeded 0.5 percent a year. Given that this represents the flow of new groups of young black males into prison, this demonstrates a large and continued disruption in the lives of black males throughout our entire sample.

To examine the net effect of declining murder rates and continued incarceration risk on the high school attainment of black males, we use the 2009-2011 American Community Survey to document the white-black high school completion gap in more recent years. Panel A contains the high school completion rate for black males by the year they turn 18. As would be expected, prior to 1997 these data show a generally similar pattern to the trends in the 2000 PUMS data shown in Figure 1. However, the pace of the decline in graduation rates for cohorts turning 18 in the late 1990s was far slower. From 1987-1997 black male graduation rates fell by 0.4 percentage points per year. However, in the next five years the rate only fell by 0.06 percentage points per year, for a total decline of 0.3 percentage points over the five year period.

While this was not a return to the historical growth throughout the 1970s, it certainly was a clear break from the declining trend during the height of the crack epidemic. The failure to fully return to trend is at least partly a function of the continued incarceration risk documented in Figure 3d. Recall that we estimate the morality and incarceration risk played equal roles in the decline in educational attainment during the peak of crack market activity.

In addition, the lack of a rebound in graduation rates for black males could be a sign of a new equilibrium resulting from factors such as decreased intergenerational transfers in education from parents, siblings, or other relatives. Further research is necessary to determine the factors underlying the current trends in black educational attainment.

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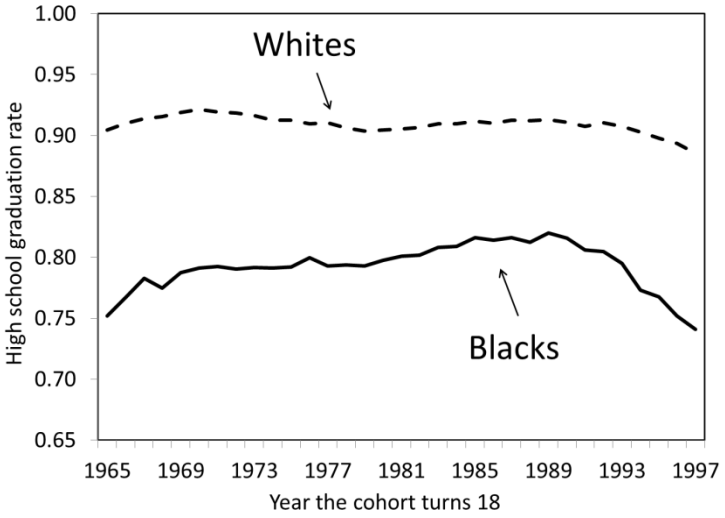
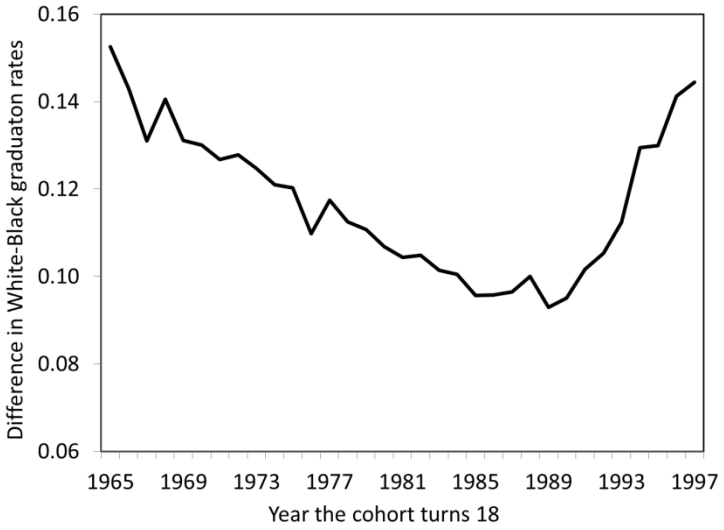
Figure 1 High School Completion Rates by the Year each Cohort Turns 18, By Race and Sex, 2000 Census 5% PUMS

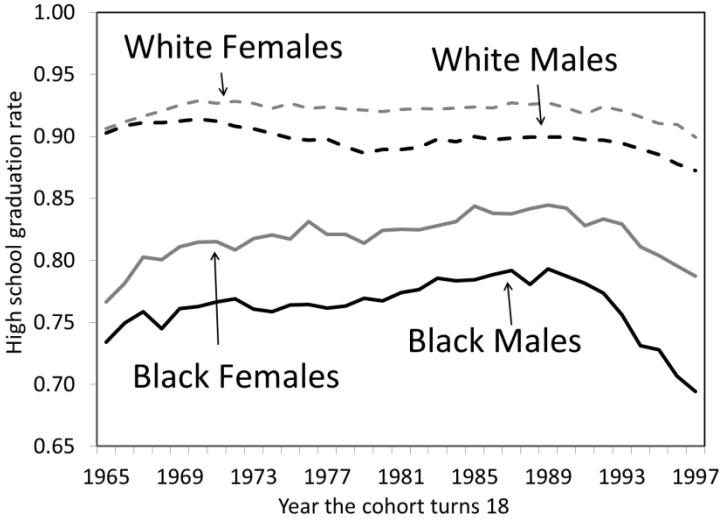
A: High School Graduation Rates, by Race and Cohort



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

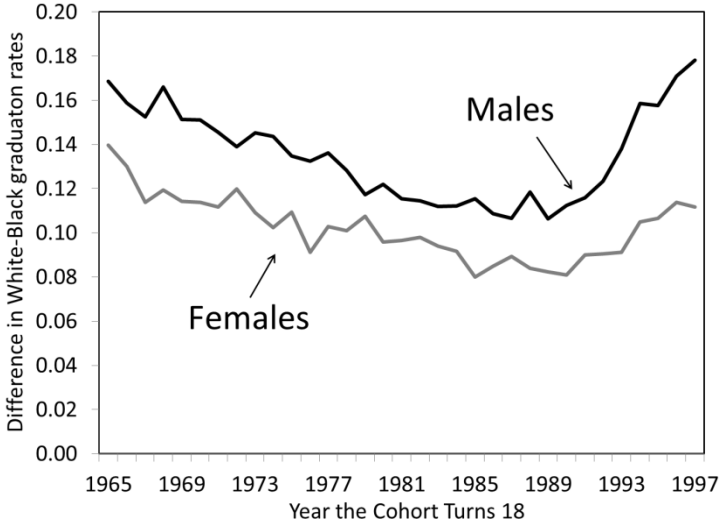


Figure 2 Expectations of Death by Age 30 for a Black Males at Age 15, Based on Contemporaneous Age-Specific Mortality Rates

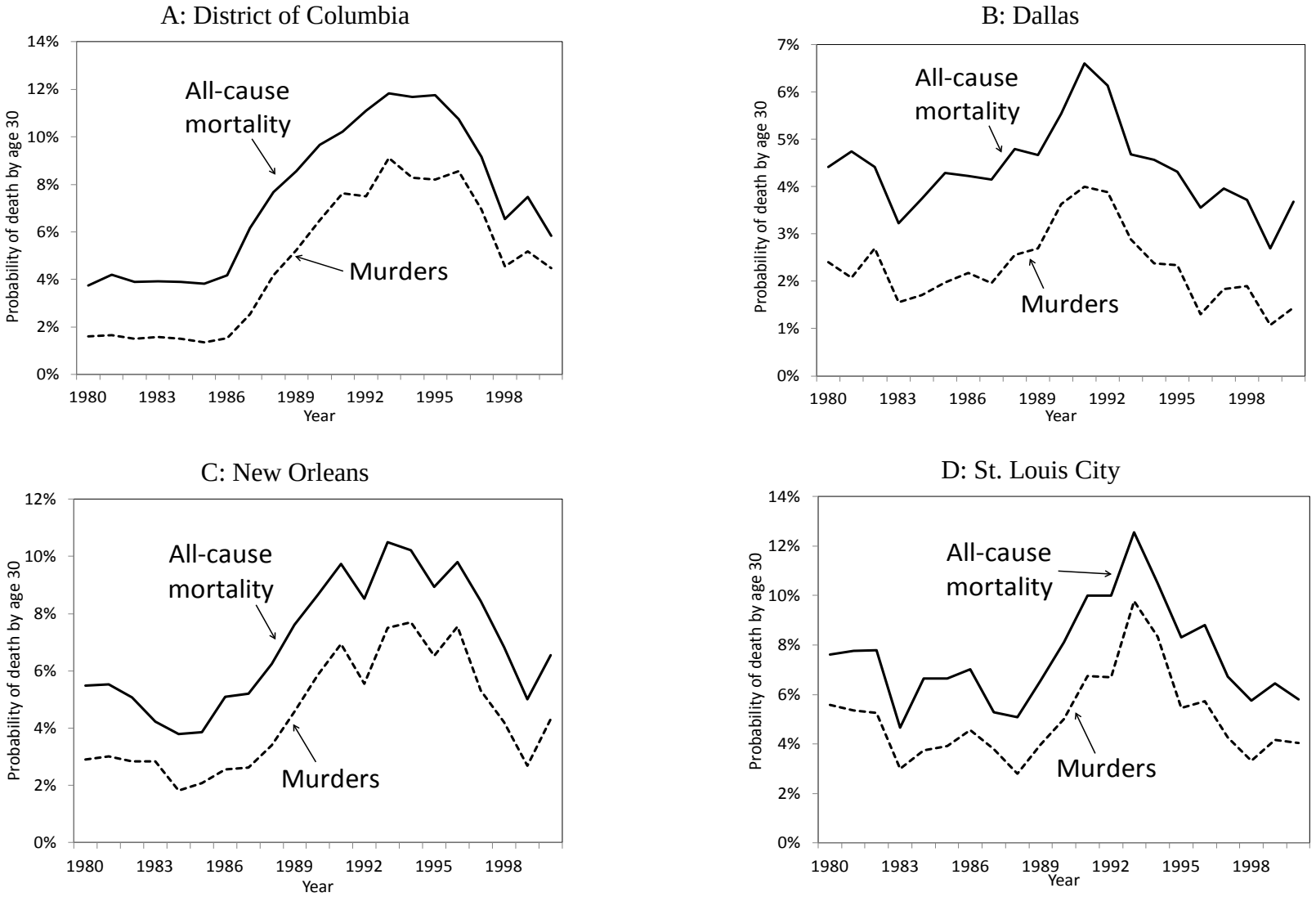


Figure 3 High School Completion Rates by when Cohorts Turn Age 18 in Relation to When Crack Cocaine Entered MSAs and States (Based on State of Birth), by Race and Sex, 2000 Census 5% PUMS

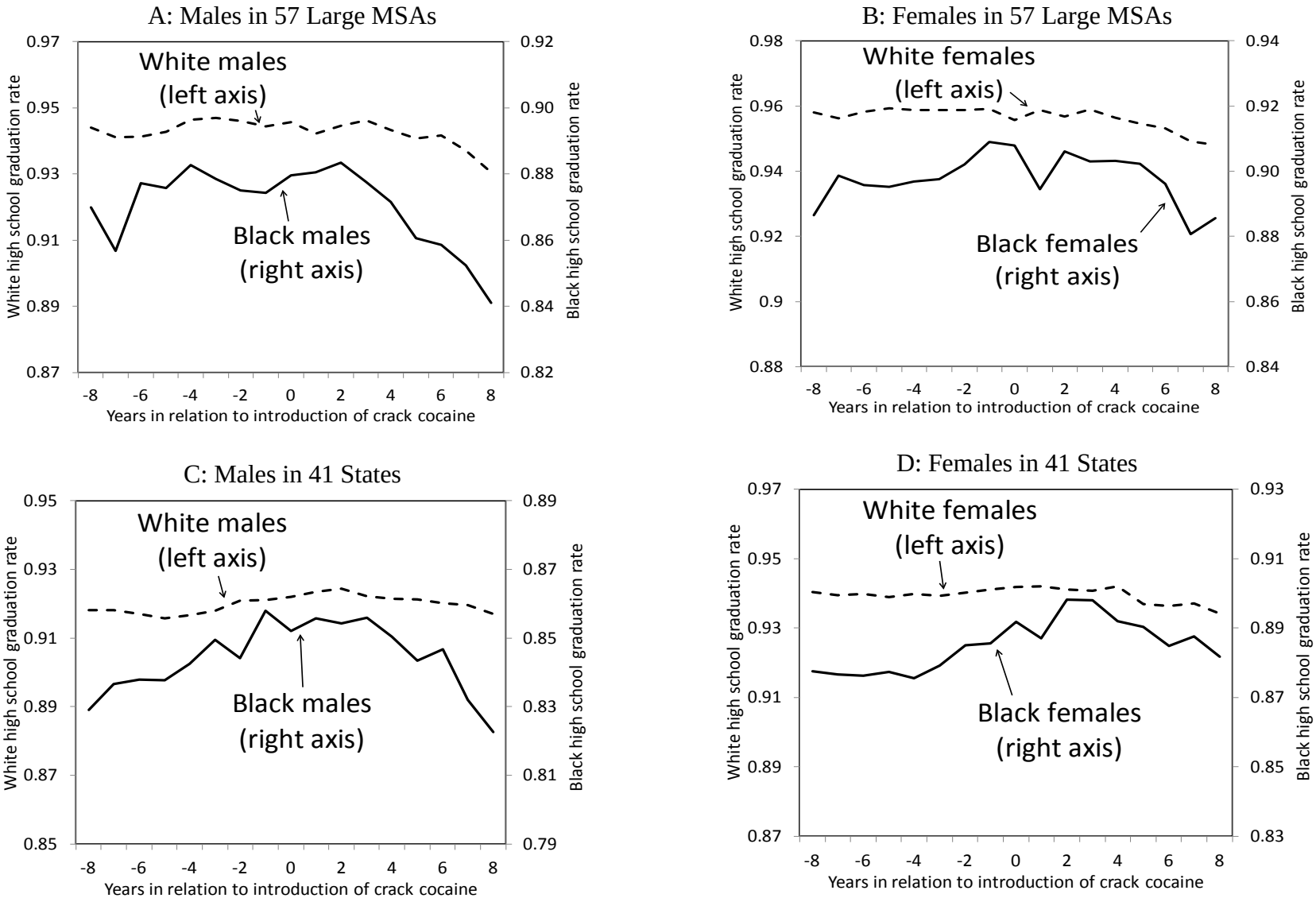
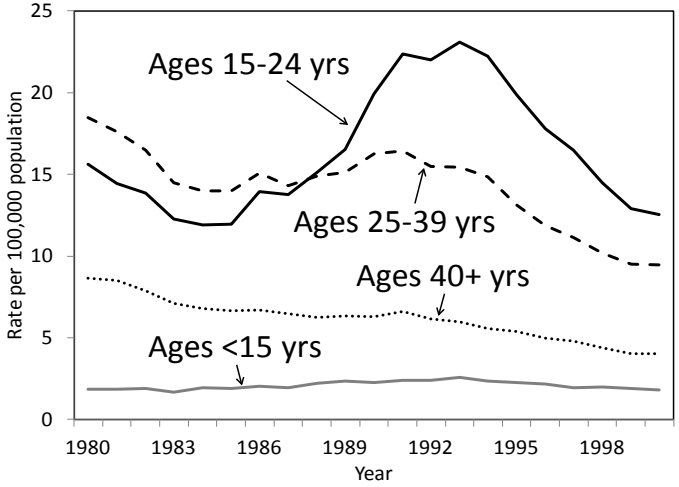
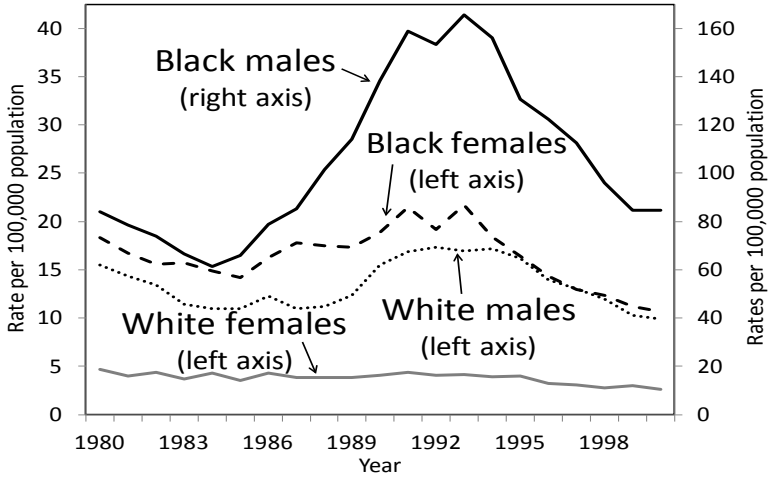


Figure 4 Murder Rates (Deaths/100,000 Pop.) and Prison Intake Rates for Various Groups, 1980-2000

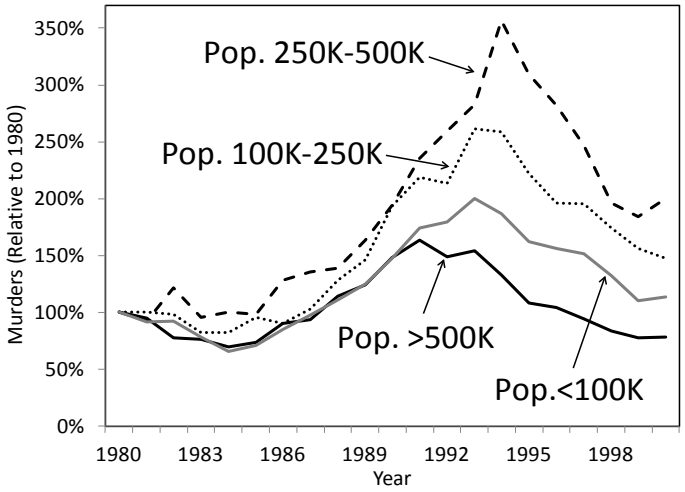
A: Murder Rates for Various Age Groups



B: Murder Rates 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

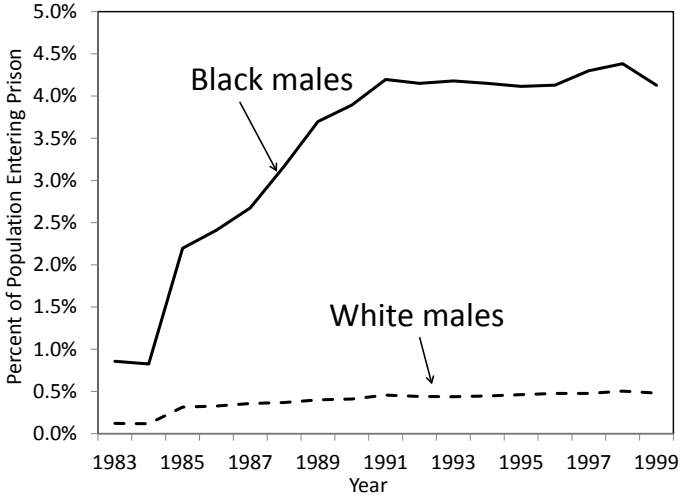


Figure 5 Murder Rates (Deaths/100,000 Pop.), Before and After the Introduction of Crack in 57 Large MSAs, By Race and Sex

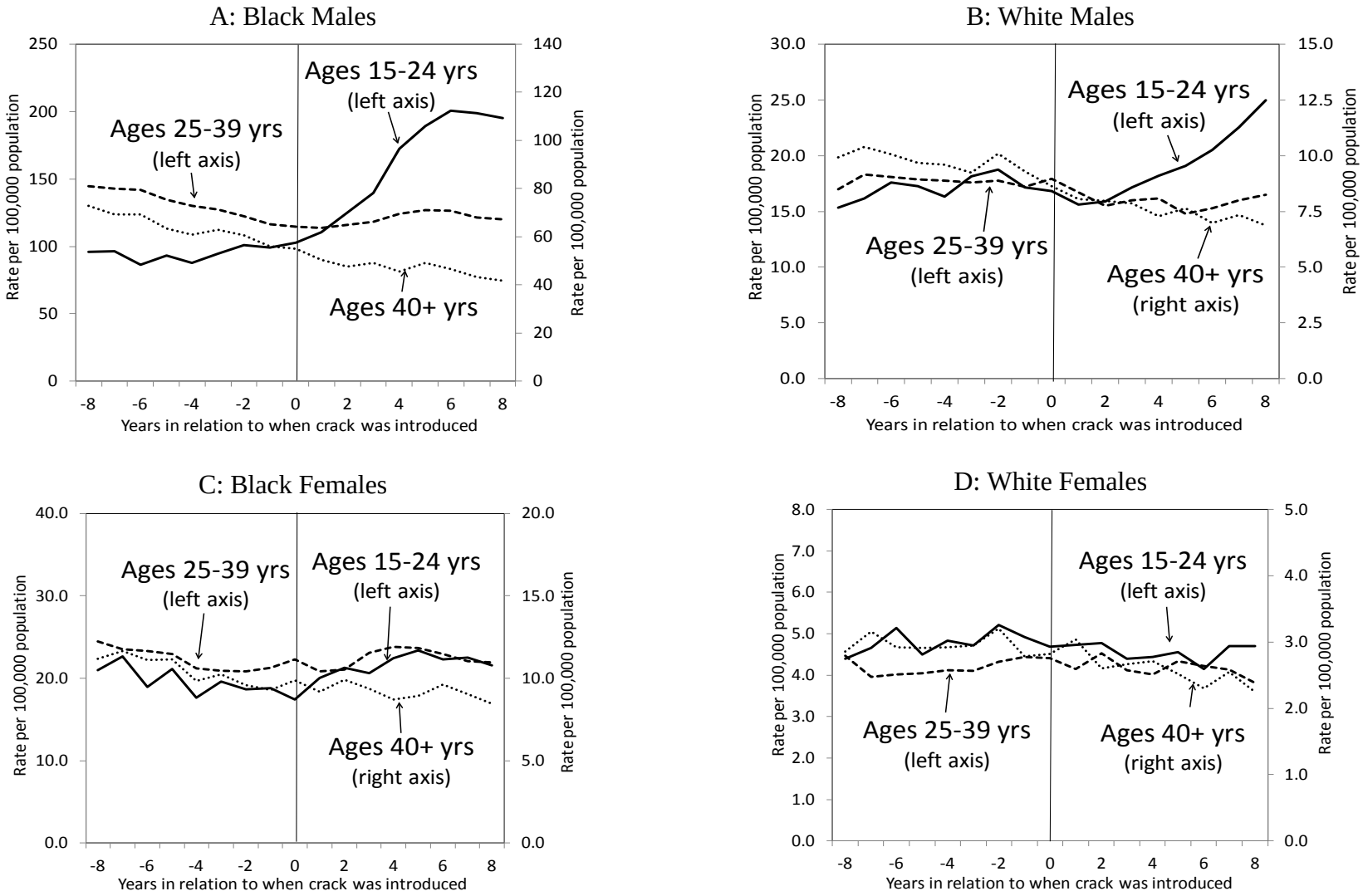
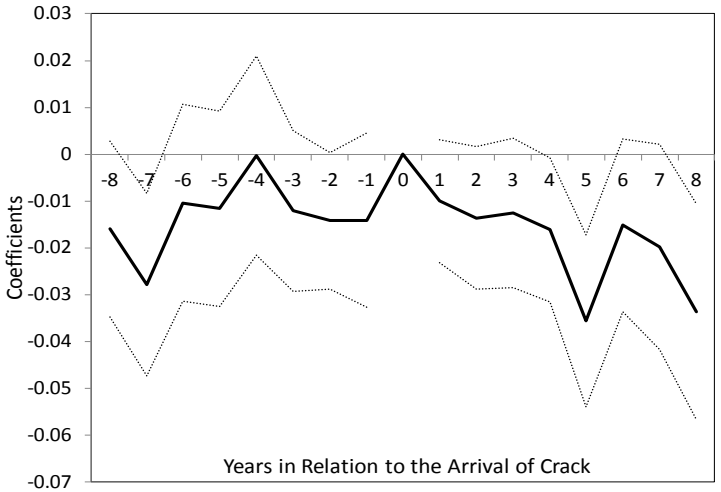
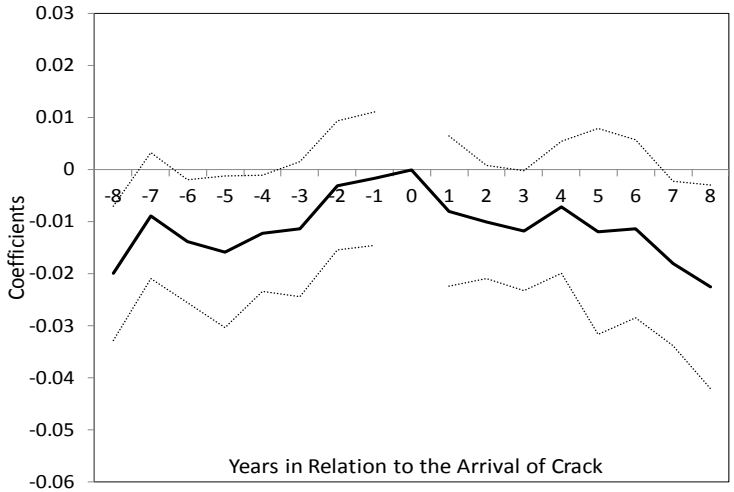


Figure 6 Linear Probability Estimates (and 95% Confidence Intervals) of High School Completion Relative to the Arrival of Crack Markets, Based on Cohorts that Turned 18, 1973-1999

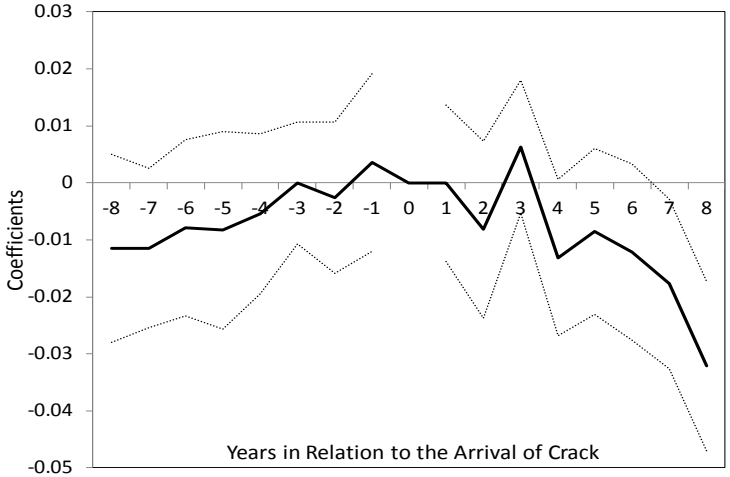
A: Males, Current MSA Residence



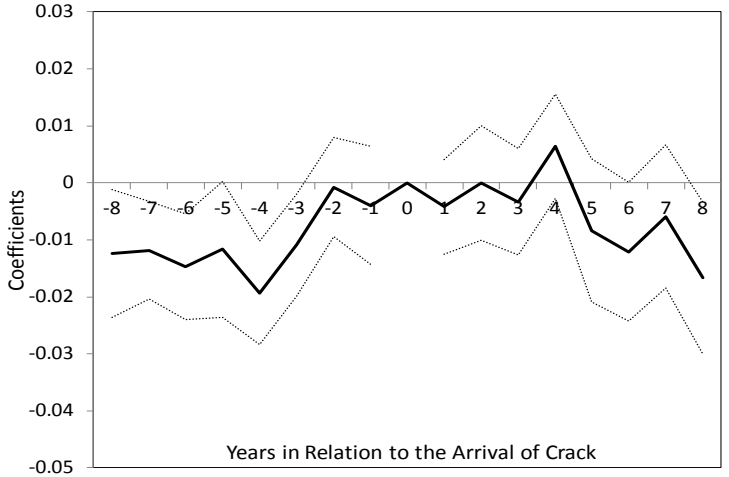
B: Females, Current MSA Residence



C: Males, State of Birth



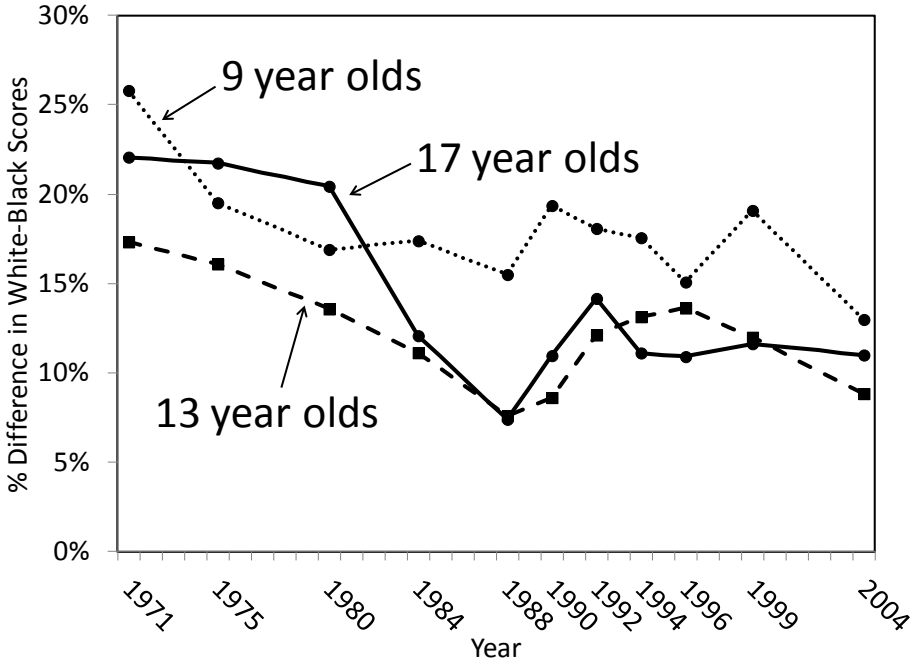
D: Females, State of Birth



These coefficients are from regressions similar to those used to produce the results in Table 4. See the text for more details.

Figure 7 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

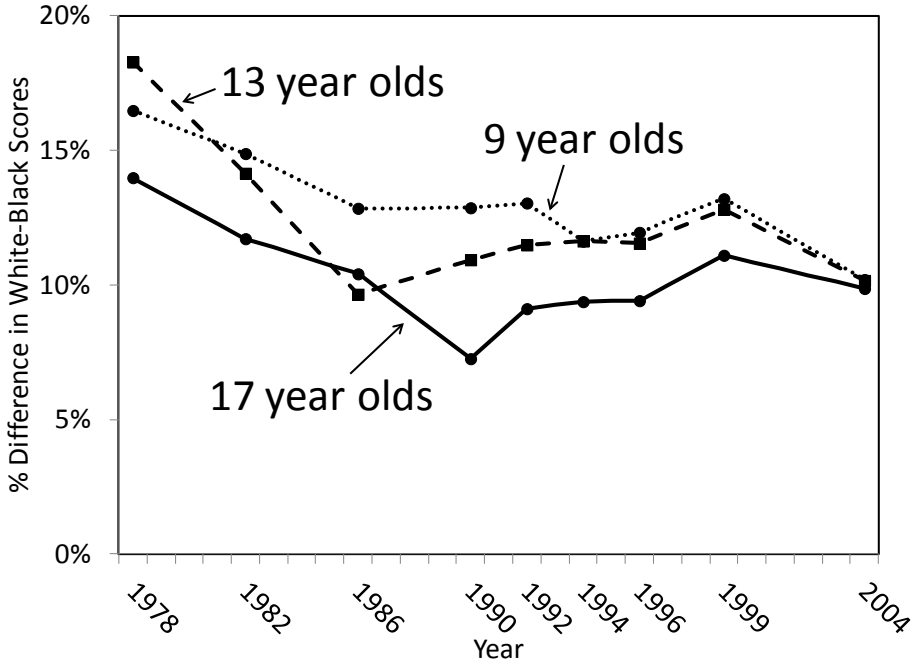
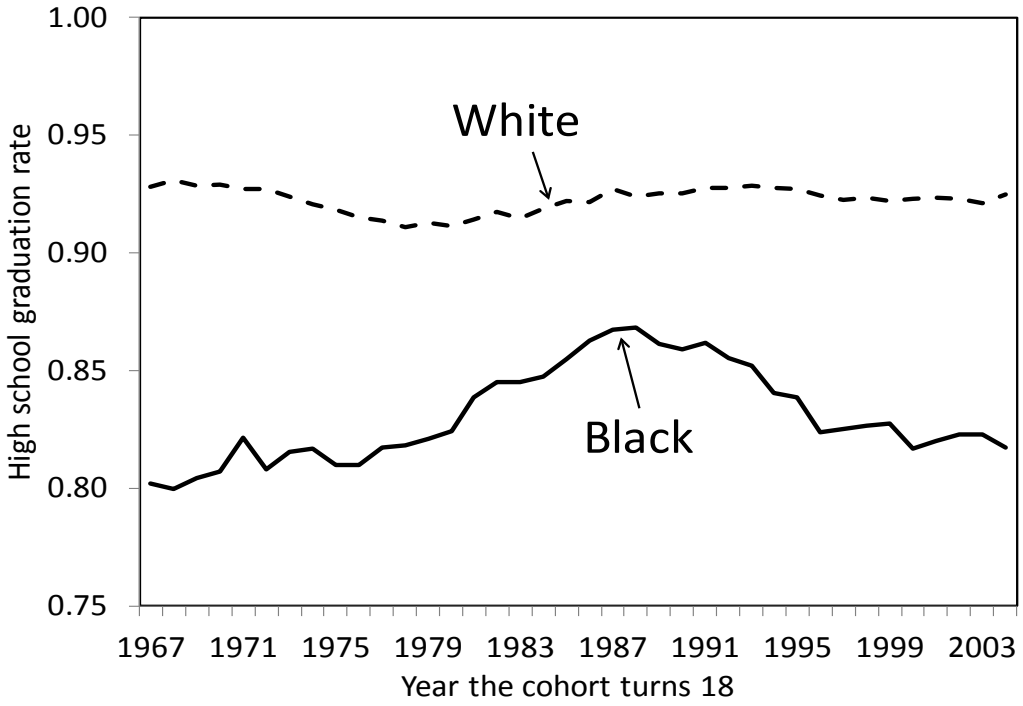


Figure 8 1 High School Completion Rates by the Year each Cohort Turns 18, By Race and Sex
2009-2011 American Community Survey

A: Males



B: Females

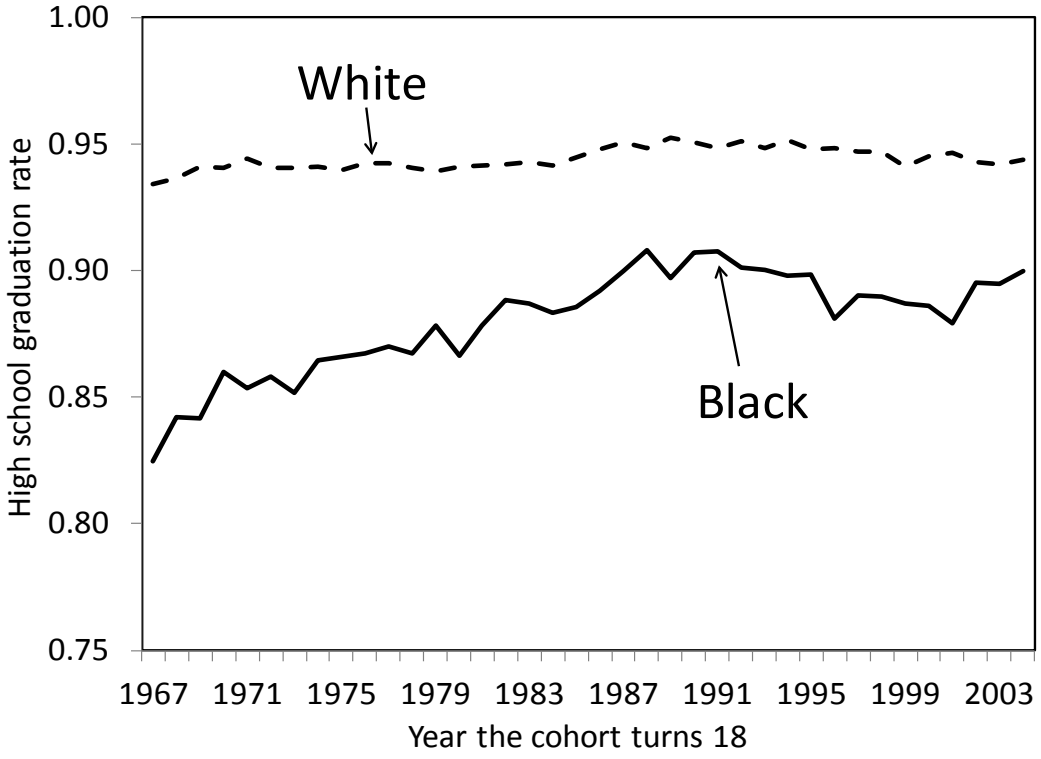


Table 1 Year Crack Arrives in the 57 Largest Metropolitan Statistical Areas
Based on Cocaine-related Deaths in Two Consecutive Years, 1981-1998

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

The Nassua/Suffolk MSA is omitted because cocaine-related deaths were present in multiple years prior to 1981. The size of the MSA is based on 1980 population.

Table 2 Year Crack Arrives in a 41 States
Based on Cocaine-related Deaths in Two Consecutive Years, 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

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, TX) is two or more deaths in two consecutive years.

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

Covariates	Hazard Ratio 1980 Levels [95% CI] (1)	Hazard Ratio Changes 1970-80 [95% CI] (2)
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]	1.030 [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

The parentheses contain the 95 percent confidence intervals. Covariates are constructed from the 1970 and 1980 Census five percent 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 Largest MSAs

Treatment Group	In Treatment Group x: 0-2 Years After Crack	In Treatment Group x: 3-5 Years After Crack	In Treatment Group x: 6-8 Years After Crack	p-value, After Coefs.=0	Mean Murder Rate, Treated Group, Year Before Crack Arrives
			Males		
Black, 15-24	0.297***	0.720***	0.942***	<0.01	101.8
	(0.062)	(0.078)	(0.060)		
Black, 25-39	0.075**	0.201***	0.302***	<0.01	116.4
	(0.026)	(0.037)	(0.044)		
Whites, 15-24	0.024	0.180***	0.507***	<0.01	15.9
	(0.041)	(0.053)	(0.033)		
Whites, 25-39	0.080***	0.058**	0.183***	<0.01	17.2
	(0.028)	(0.025)	(0.023)		
			Females		
Blacks, 15-24	0.040	0.103	0.199***	<0.01	18.3
	(0.055)	(0.065)	(0.054)		
Blacks, 25-39	0.009	0.058	0.096***	0.06	21.3
	(0.042)	(0.046)	(0.036)		
Whites, 15-24	-0.002	-0.019	0.154**	0.03	4.5
	(0.067)	(0.072)	(0.061)		
Whites, 25-39	0.126***	0.147***	0.174***	<0.01	4.4
	(0.048)	(0.049)	(0.060)		

* 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, and complete sets of MSA and year fixed effects.

Table 5 Linear Probability Estimates of High School Completion Models, Cohorts that Turned 18 from1973-1998

	57 MSAs		41 States			
			State of Residence		State of Birth	
	(1)	(2)	(3)	(4)	(5)	(6)
			Males			
(1) Black*Pre-Crack Time Trend	0.0023***	0.0106	0.0039***	0.0101	0.0037***	0.0171*
	(0.0008)	(0.0114)	(0.0010)	(0.0130)	(0.0007)	(0.0099)
(2) Black* (Years after crack≥0)	-0.0030**	0.0076	-0.0032***	0.0016	-0.0040***	0.0072
	(0.0012)	(0.0097)	(0.0008)	(0.0100)	(0.0008)	(0.0077)
P-value of test (1) = (2)	<0.01	0.31	<0.01	0.05	<0.01	0.01
R^2	0.030	0.030	0.027	0.027	0.027	0.027
Observations	679,067	679,067	1,562,331	1,562,331	1,564,393	1,564,393
Mean of dep. var. for blacks the year before crack arrives	0.805	0.805	0.786	0.786	0.789	0.789
			Females			
(1) Black*Pre-Crack Time Trend	0.0013**	0.0096	0.0022***	0.0059	0.0019***	0.0030
	(0.0006)	(0.0085)	(0.0007)	(0.0083)	(0.0004)	(0.0067)
(2) Black* (Years after crack≥0)	-0.0019*	0.0078	-0.0007	0.0008	-0.0011*	-0.0023
	(0.0011)	(0.0069)	(0.0008)	(0.0062)	(0.0006)	(0.0053)
P-value of test (1) = (2)	0.05	0.50	0.02	0.13	<0.01	0.04
R^2	0.027	0.027	0.021	0.021	0.021	0.021
Observations	713,912	713,912	1,617,164	1,617,164	1,618,387	1,618,387
Mean of dep. var. for blacks the year before crack arrives	0.851	0.851	0.836	0.836	0.834	0.834
Race x Cohort Fixed Effects		X		X		X

* 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 race, cohort and geographic area fixed effects.

Table 6 OLS Estimates of Impact of Murder Rates on High School Completion, Cohorts that Turned 18 from 1975-98

	57 Larger MSAs		176 MSAs		41 States: State of Residence		41 States: State of Birth	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
				Males				
Sex/race-specific murder rate in HS (x 1000)	-0.183***	-0.149***	-0.171***	-0.131***	-0.317***	-0.264***	-0.245***	-0.171**
	(0.053)	(0.056)	(0.039)	(0.041)	(0.080)	(0.092)	(0.080)	(0.082)
R^2	0.035	0.036	0.035	0.036	0.030	0.031	0.030	0.030
Observations	704,988	704,988	1,025,361	1,025,361	1,720,256	1,720,256	1,719,950	1,719,950
				Females				
Sex/race-specific murder rate in HS (x 1000)	-0.019	-0.082	-0.046	-0.082	-0.128	-0.205	0.020	-0.088
	(0.206)	(0.208)	(0.112)	(0.112)	(0.278)	(0.289)	(0.026)	(0.272)
R^2	0.030	0.030	0.029	0.029	0.022	0.023	0.022	0.022
Observations	740,661	740,661	1,070,704	1,070,704	1,769,373	1,769,373	1,767,901	1,767,901
Race x Cohort Fixed Effects	No	Yes	No	Yes	No	Yes	No	Yes

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

Standard errors are in parentheses, and allow for arbitrary correlation in errors within a state/MSA. All models include race, cohort and geographic area fixed effects.

Table 7 OLS Estimates of the Impact of Murder and Prison Entry on the High School Completion, Based on the Separate Impacts of Murder and Prison, Cohorts that Turned 18 from 1983-1998

	Murder Rates Only		Prison Rates Only		Murder & Prison Rates	
	(1)	(2)	(3)	(4)	(5)	(6)
	A: 16 States Reporting Intake Every Year [Obs.=405,507]					
Race-specific murder rate during HS (x 1000)	-0.451***	-0.374***			-0.323***	-0.318**
	(0.103)	(0.121)			(0.079)	(0.101)
	[-0.047]	[-0.039]			[-0.034]	[-0.033]
Race-specific prison intake rate (x 1000)			-1.788***	-1.322**	-1.039**	-0.827*
			(0.494)	(0.596)	(0.360)	(0.403)
			[-0.059]	[-0.044]	[-0.035]	[-0.027]
R^2	0.031	0.031	0.031	0.033	0.031	0.033
	B: 36 States With Intake Data [Obs.= 869,938]					
Race-specific murder rate during HS (x 1000)	-0.299***	-0.237***			-0.254***	-0.221**
	(0.085)	(0.087)			(0.086)	(0.087)
	[-0.025]	[-0.020]			[-0.022]	[-0.019]
Race-specific prison intake rate (x 1000)			-0.955***	-0.570*	-0.522	-0.332
			(0.344)	(0.334)	(0.349)	(0.341)
			[-0.031]	[-0.018]	[-0.017]	[-0.011]
R^2	0.032	0.033	0.032	0.033	0.032	0.033
Race x Cohort Fixed Effects		X		X		X

* 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 total effects that changes in the murder and incarceration rates between 1983 and 1993 had on black graduation rates (e.g., the value of -0.047 in the first column represents a 4.7 percentage point decrease in graduation). All models include a dummy variable for race, and a complete set of cohort and state fixed effects.

Table 8 OLS Estimates of Impact of Death Rates of Those Aged 20-24 on High School Completion, 176 MSA Sample, Cohorts that Turned 18 from 1975-98

	(1)	(2)	(3)	(4)	(5)	(6)
			Males			
Cohorts	All	Pre-Crack	All	All	All	All
Death rate definition	Murders	Murders	All cause	Non-murder causes	Murders	All cause
Death Rates in HS (x 1000)	-0.171***	-0.163***	-0.140***	-0.174***		
	(0.039)	(0.048)	(0.026)	(0.035)		
(2) Black-specific Death Rates in HS (x 1000)					-0.171***	-0.140***
					(0.039)	(0.028)
(3) White-specific Death Rates in HS (x 1000)					-0.015	-0.135***
					(0.026)	(0.044)
P-value of test (2) = (3)					0.55	0.91
R^2	0.035	0.030	0.035	0.035	0.035	0.035
Observations	1,025,361	351,646	1,025,361	1,025,361	1,025,361	1,025,361
			Females			
Cohorts	All	Pre-Crack	All	All	All	All
Death rate definition	Murders	Murders	All cause	Non-murder causes	Murders	All cause
Death Rates in HS (x 1000)	-0.046	-0.185	-0.138***	-0.177***		
	(0.112)	(0.112)	(0.048)	(0.056)		
(2) Black-specific Death Rates in HS (x 1000)					-0.035	-0.143**
					(0.114)	(0.056)
(3) White-specific Death Rates in HS (x 1000)					-0.140	-0.123*
					(0.255)	(0.074)
P-value of test (2) = (3)					0.68	0.82
R^2	0.029	0.027	0.029	0.029	0.029	
Observations	1,070,704	368,013	1,070,704	1,070,704	1,070,704	1,070,704

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

Standard errors are in parentheses, and allow for arbitrary correlation in errors within an MSA. Results in columns (2) and (6) are based on murder rates from 1975 to 1981. All models include race, cohort and geographic area fixed effects.

Table 9 Family and School Characteristics for Black and White Children, 1980 and 1990

		Black, Non-Hispanics			White, Non-Hispanics			
Outcome	Source	1980	1990	Δ	1980	1990	Δ	ΔΔ
% Children aged 0-15:								
In poverty	March CPS	42.4%	45.0%	2.6%	10.9%	11.9%	1.0%	1.6%
In HH receiving food stamps	March CPS	41.2%	39.3%	-1.9%	9.2%	8.7%	-0.5%	-1.4%
Without father present	March CPS	55.4%	58.4%	3.0%	14.2%	15.7%	1.5%	1.5%
Without mother present	March CPS	13.9%	12.2%	-1.7%	3.0%	4.3%	1.3%	-3.0%
Without both present	March CPS	12.0%	8.5%	-3.5%	1.5%	1.4%	-0.1%	-3.4%
Rate of return to college, males, aged 25-44,	March CPS	0.247 (0.038)	0.372 (0.035)	0.125	0.200 (0.009)	0.361 (0.010)	0.161	-3.6%
Unemployment rate, males, ages 16-44	Monthly CPS	17.2%	13.7%	-3.7%	7.6%	5.6%	-2.0%	-1.7%
Real per pupil K-12 spending (2002 $)	Evans and Corcoran	$4,515	$6,456	$1,941	$4,438	$6,181	$1,743	$198

Standard errors are in parentheses.