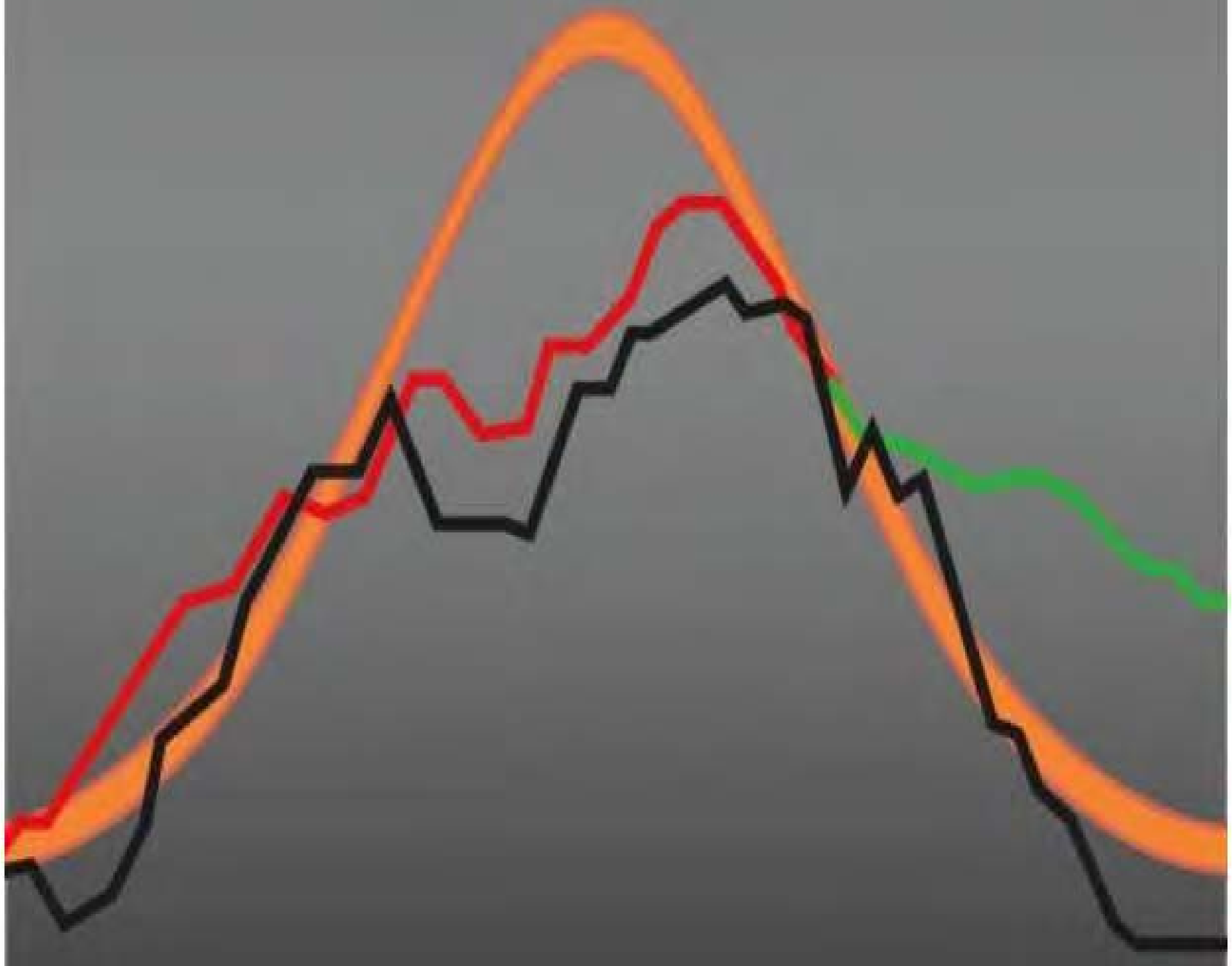
LUCIFER CURVES

THE LEGACY OF LEAD POISONING



RICK NEVIN

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Rick Nevin

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Dedication

For Janice,

The co-author of every accomplishment and noble effort in my life

Contents

List of Lucifer Curves and other Figures

and Under Property Crime
and Vandalism Arrest Rates

I. Just Call Me Lucifer

"Pleased to meet you
Hope you guessed my name
But what's confusing you
Is just the nature of my game

Just as every cop is a
criminal
And all the sinners, saints
As heads is tails
Just call me Lucifer
'Cause I'm in need of some
restraint"[1]

(Jagger/Richards, 1968)

Although "Lucifer" is commonly thought to be another name for the devil, some Bible scholars contend

that this association is an artifact of misunderstood scripture translation.[2] The Vulgate Bible, used by the Catholic Church for almost 1000 years, translated the Hebrew word heylel as "Lucifer", the Latin word for Venus when it appeared as the morning star. The Vulgate text also translated the Greek word phosphorus as "Lucifer". The King James Bible retained the word Lucifer in Isaiah 14:12 but translated other references to "Lucifer" and "phosphorus" as "morning star" or "day star", including references to Christ. This title for Christ is used in a scriptural context that clearly conveys reverence, whereas "Lucifer" in Isaiah 14:12 was used as a sarcastic taunt to a Babylon

king who compared himself to the Babylonian god Helel, the morning star - Venus.

Of course, we now know that Venus is not a god or a star. It is a planet that shines like a bright star, in part because of its proximity to Earth, but also because its atmosphere is a toxic mix of carbon dioxide and sulfuric acid clouds that reflect sunlight.[3] The atmosphere also traps heat, so temperatures on Venus are hot enough to melt lead. Lead sulfide is vaporized on the planet surface, rises as a mist, condenses in the cooler Venus clouds, and settles as a "shiny, metallic frost on the tops of the mountains", making the Venus highlands more reflective

than lower elevations.[4]

Lucifer is not the name of the devil, and does not refer to anything innately evil. Lucifer is just the Latin name for a reflected image caused by a toxic lead-contaminated environment.

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II. Lead Poisoning is the Master of Horror

> "The association we observe may be one new to science or medicine and we must not dismiss it too light-heartedly as just too odd. As Sherlock Holmes advised Dr. Watson, 'when you have eliminated the impossible, whatever remains, *however improbable*, must be the truth'."[5] (Hill, 1965)

In a 2015 article in *The Atlantic*, Ta-Nehisi Coates examined the causes of mass incarceration and its

impact on black families, including the personal story of Odell Newton.[6] That article featured lead poisoning in the dual role that Alfred Hitchcock often played in his classic horror films. There was a brief cameo appearance in a scene about Odell almost dying from lead poisoning as a young child, but the much larger role was behind the scenes, directing all the intertwined subplots of this article. Odell was poisoned before his grade school teacher said he should be placed in special education, before his impaired mental development became more obvious in junior high school, before he was convicted of murder at the age of 16, and before he spent 41 years in prison, where he failed in

several attempts to pass the high school G.E.D. test. The cause and effect troubles of his life have been repeated in horror stories across centuries and around the world, all directed by lead poisoning.

The Atlantic later published a dissent by Hymowitz that chastised Coates for not recognizing that "black-family disruption could have some bearing on crime and incarceration".[7] She stated that 72% of black children are born to unwed mothers and "growing up in chaotic families...is itself highly correlated with the scourge of ghetto crime and incarceration". Her suggested causal relationship, however, ignores an important indicator of causation: the suspected cause must precede the

effect. She noted that black children were only slightly less likely than whites to grow up in two-parent homes before 1960, but "after 1960…the family began to unravel." If growing up in a single-parent household caused criminal behavior, then the 1960s increase in unwed birth rates should have increased crime starting in the mid-1970s, when children raised by single parents reached their teenage (juvenile offending) years. Crime and unwed birth rates increased in tandem after 1960 and have since fallen in tandem. Lead poisoning was the director of that temporal dance duet.

Coates referenced a circa-1970 memo from Daniel Patrick Moynihan acknowledging a "rather

pronounced revival - in impeccably respectable circles - of the proposition that there is a difference in genetic potential" between races. In fact, those circles included *The Atlantic*, with its 1971 publication of "I.Q." by Herrnstein, stating: "data on I.Q. and social-class differences show that we have been living with an inherited stratification of our society for some time."[8] That statement set the stage for the 1994 publication of *The Bell Curve*,[9] by Herrnstein and Murray, and the angry debate over their claims that inherited IQ has a strong causal impact on incarceration, unwed births, and high school dropout rates. *The Bell Curve* is now ridiculed in "respectable circles" but its key

findings were affirmed in a 1998 *Scientific American* article by Gottfredson,[10] who had organized the 1994 treatise "Mainstream Science on Intelligence", with 52 signatories defending *The Bell Curve*.[11] The *Scientific American* website still displays her article, but it is mathematically impossible for the behavior risks she reported for youths with low IQ to be applicable today, after two decades of steep declines in high school dropout rates, youth incarceration, and unwed teen birth rates. *The Bell Curve* drama was directed by lead poisoning, and lead poisoning prevention deserves the credit for discrediting that book.

Another publication in 1994 went

widely unnoticed during *The Bell Curve* debate over IQ and behavior. This study in the *Journal of the American Medical Association* reported that average blood lead levels for American children ages 1-5 fell by 77% from the late-1970s through the late-1980s, largely due to the USA phase out of leaded gasoline from the mid-1970s through the mid-1980s.[12]

Coates highlights the significance of 1994 for a different reason, as the year when the federal government enacted a crime bill now blamed for driving up incarceration, and he rejects claims that this bill "was a purely well-intended, logical, and nonracist response to crime". In fact, he rejects the causal relationship

between crime and incarceration trends, based on a comparison of trends from 1960 to 1974, and from 1991 to 2012, and some research suggesting that mass incarceration is partly explained by an increase in the average length of prison sentences.

Coates did not mention that black male incarceration rates fell from 2001 to 2014 by 62% for ages 18-19, 51% for ages 20-24, and 46% for ages 25-29.[13] Similarly, Hymowitz did not mention that unwed birth rates for black females fell from 1991 to 2014 by 79% for ages 15-17, 59% for ages 18-19, and 33% for ages 20-24.[14] Few people are aware of these newsworthy trends because the news media is obsessed with

the horror news genre.

During the 1994 media frenzy over *The Bell Curve*, as the 1994 crime bill was enacted, I was starting work on an Economic Analysis of lead paint hazard regulations for the Department of Housing and Urban Development (HUD). That analysis showed that the costs of lead hazard reduction were more than offset by the benefits from lead poisoning prevention affecting IQ, education, and lifetime earnings.[15] My client also mentioned that we didn't even count crime prevention benefits, suggested by recent research. In light of the strong relationship between blood lead and leaded gasoline use in the past, I wondered if there might be a relationship between crime trends

and earlier gas lead trends. What I found was a stunning visual fit with a 23-year time lag, consistent with preschool lead exposure affecting the peak age of violent offending.

In 2000, *Environmental Research* published my peer-reviewed study on "How Lead Exposure Relates to Temporal Changes in IQ, Violent Crime, and Unwed Pregnancy".[16] The same journal published my 2007 study, "Understanding international crime trends: The legacy of preschool lead exposure";[17] and my 2009 study, "Trends in Preschool Lead Exposure, Mental Retardation, and Scholastic Achievement: Association or Causation?"[18] My

2009 study also reported temporal shifts in incarceration by age and race, and showed that the lead poisoning research literature demonstrates all of the accepted indicators of causation. Lead exposure trends are not just correlated with important societal trends – lead poisoning *caused* those trends.

Coates notes that the rise and fall of crime was an "international phenomenon", and calls this pattern a "riddle". My 2007 study showed a consistent relationship between preschool lead exposure and crime trends in nine nations, explaining up to 91% of temporal variation in burglary rates, with an 18-year time lag; up to 89% of variation in robbery rates, with a

23-year time lag; and up to 95% of variation in overall "index" crime rates (violent plus nonviolent crimes), with a 19-year lag. Riddle solved.

Time lags that relate lead exposure and crime reflect analysis of a wide range of lags to identify the "best-fit" with the highest statistical significance. The best-fit lag in every crime category is consistent with lead-induced neurobehavioral damage in the first years of life and the peak age of offending for that category. Property crime arrests peak at ages 15 to 20 and fall sharply by age 30, consistent with the 18-year lag for burglary. Violent crime arrests peak at ages 15 to 24 and decline through age 50, consistent with the 23-year lag for

robbery. Nonviolent crime is much more common than violent crime, consistent with the 19-year lag for index crime.

Coates does not mention that the rise and fall of crime occurred at different times in different nations, with seemingly inexplicable contrasts in crime rates across nations over time. In 1980, the USA index crime rate was 40% higher than Australia's rate, but the 2001 USA index crime rate was 45% below Australia's rate. Canada's index crime rate was 60% higher than Britain's rate in the early-1970s, but 20% lower in 2001. In 1974, the USA burglary rate was twice the rate in Australia and 50% higher than the rate in Britain, but the 2002 USA burglary rate was

less than half of the rates in Britain and Australia. The Canadian robbery rate was five times the rate in Britain in 1962, but in 2002 the Canadian robbery rate was less than half the rate in Britain.

Crime in the USA and Canada rose earlier than in other nations because the USA and Canada accounted for the vast majority of global use of lead in gasoline before 1970, as many other nations recovered slowly from World War II. The USA and Canada phased out the use of lead in gas from the mid-1970s through the mid-1980s, but leaded gas use in many industrial nations was near its peak in the mid-1980s, delaying crime declines in those nations. Within each nation, however, crime trends have

crime arrests from 1960 to 1969.[19] The outsized youth impact was partly due to baby boomers swelling the age groups when offending peaks, but violent crime arrest *rates* for youths (arrests per 100,000 in age group) also increased over the 1960s by 83% for juveniles (under age 18) and 42% for ages 18-24. The incarceration rate didn't rise until the 1970s because first-time offenders are more likely to get probation instead of prison time, and juveniles with serious and/or multiple offenses went to juvenile detention facilities (not included in the prison data cited by Coates), and because of one other important factor affecting incarceration in the 1960s: the war in Vietnam.

There were 552,000 troops overseas in 1960, and troops overseas peaked at 1.1 million in 1968.[20] There were 205,000 men in prison in 1960, and the number of men in prison fell to a low of 182,000 in 1968.[21] Judges in this era were known to give young male defendants a choice between prison and "volunteering" for military service. Enlistment was the option chosen by many youths who expected to be drafted soon even if they served a short prison term. Troops overseas fell to 885,000 in 1970, 687,000 in 1971, and 507,000 in 1972. The draft ended in 1973, and the number of men in prison reached a new record high in 1974, and in every year for the next three decades.

The violent crime rate fell more than 50% from its 1991 peak to 2014, but Coates notes that the incarceration rate didn't peak until 2007, and fell less than 8% from 2007 through 2012. Those trends reflect a shift in arrest rates by age that is a mirror image of arrest trends over the1960s. From 1991 to 2014, violent crime arrest rates fell 65% for juveniles and 50% for ages 18-24, but increased 21% for adults ages 50-54. The same generation that caused a youth crime surge in the 1960s has caused a large increase in the over age 50 arrest rate since 1991. Moreover, surging youth crime at the start of a crime wave, and plummeting youth arrests presaging the end of a crime wave, is a pattern that has been

repeated around the world since the circa-1800 dawn of "juvenile delinquency" (trends documented in Chapter V).

Incarceration rates by age have not moved independent of arrests by age since the 1990s. From 2001 to 2014, male incarceration rates fell 62% for ages 18-19, 38% for ages 20-24, and 27% for ages 25-29, but rose 31% for ages 40-44. Data reported for older ages since 2007 show male incarceration rates rose from 2007 to 2014 by 50% for ages 50-54 and 57% for ages 55-64.

Coates faults longer sentences for mass incarceration, and Pew Trust research confirms that inmates released from state prisons in 2009 did serve longer sentences on

average than those released in 1990: 2.2 years for drug crimes, up from 1.6 years in 1990; 2.3 years for property crimes, up from 1.8 years in 1990; and 5 years for violent crimes, up from 3.7 years in 1990.[22] An increase of a half-year in average time served for nonviolent crimes can hardly explain mass incarceration. Moreover, the shift in arrests to older offenders suggests that average sentence length has increased because repeat offenders now account for a much larger share of crimes. Studies of recidivism by state prisoners released in 1983, 1994, and 2005 all report high reoffending rates, but very different age distributions for inmates released. In 1983, 36% of state prisoners released were under

25 and 9% were age 40 or older. In 2005, just 17% of prisoners released were under 25 and 32% were 40 or older.[23]

All horror stories require some suspension of disbelief, but for this story about the horrors of lead poisoning you only need to suspend judgment until you have seen all the evidence. That evidence is presented here in the context of indicators of causation delineated by Sir Austin Bradford Hill in his 1965 treatise "The Environment and Disease: Association or Causation?" The World Health Organization has recognized Hill's insights as a "public health classic" and a "mainstay of epidemiological textbooks and data

interpretation".[24] The causation indicators identified by Hill are: strength, consistency, experimental evidence, dose-response, biological plausibility, time-precedence, coherence, specificity, and analogy.

Fig. 1: 1960-2005 U.S. Troops Overseas and Men in Prison

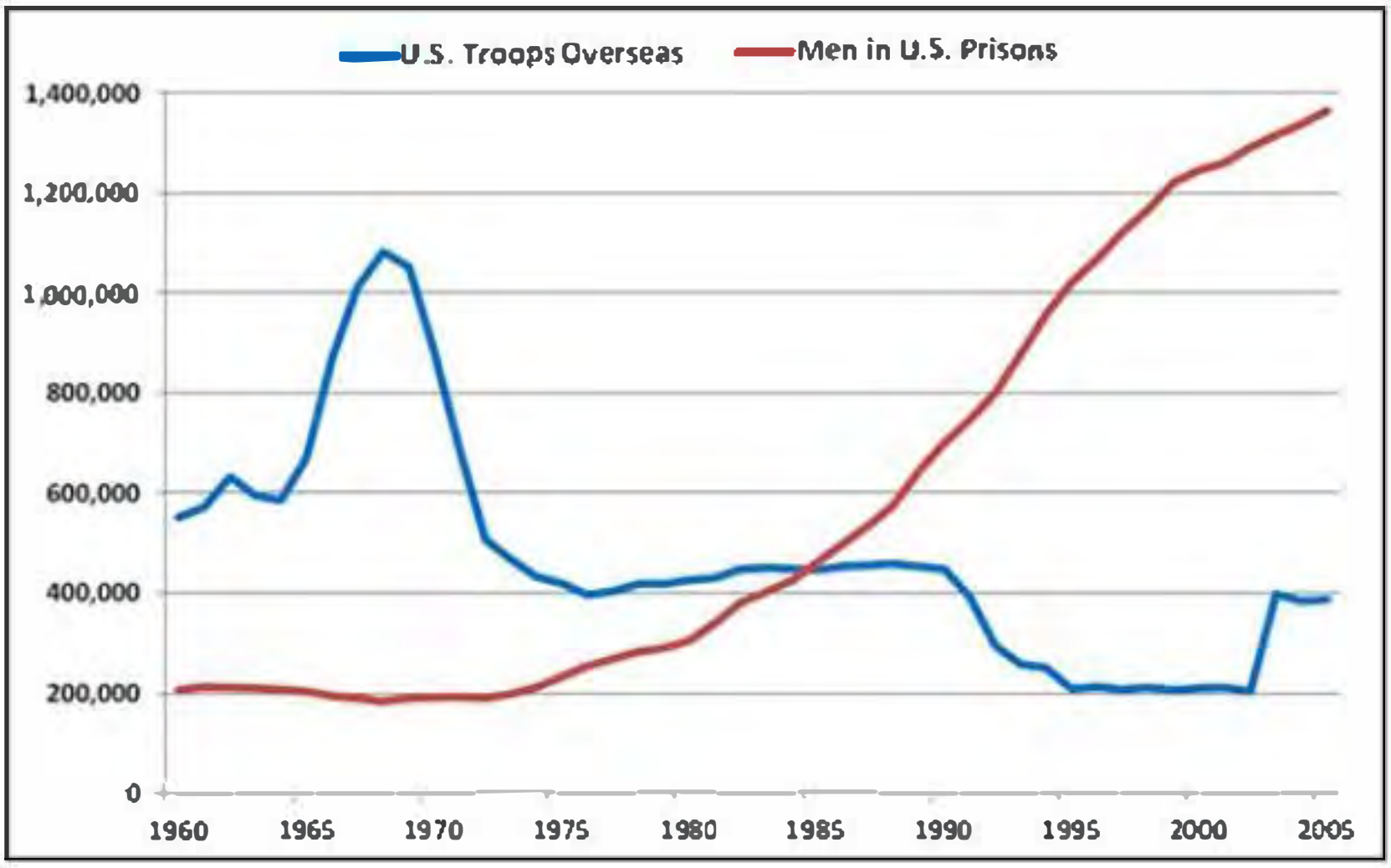
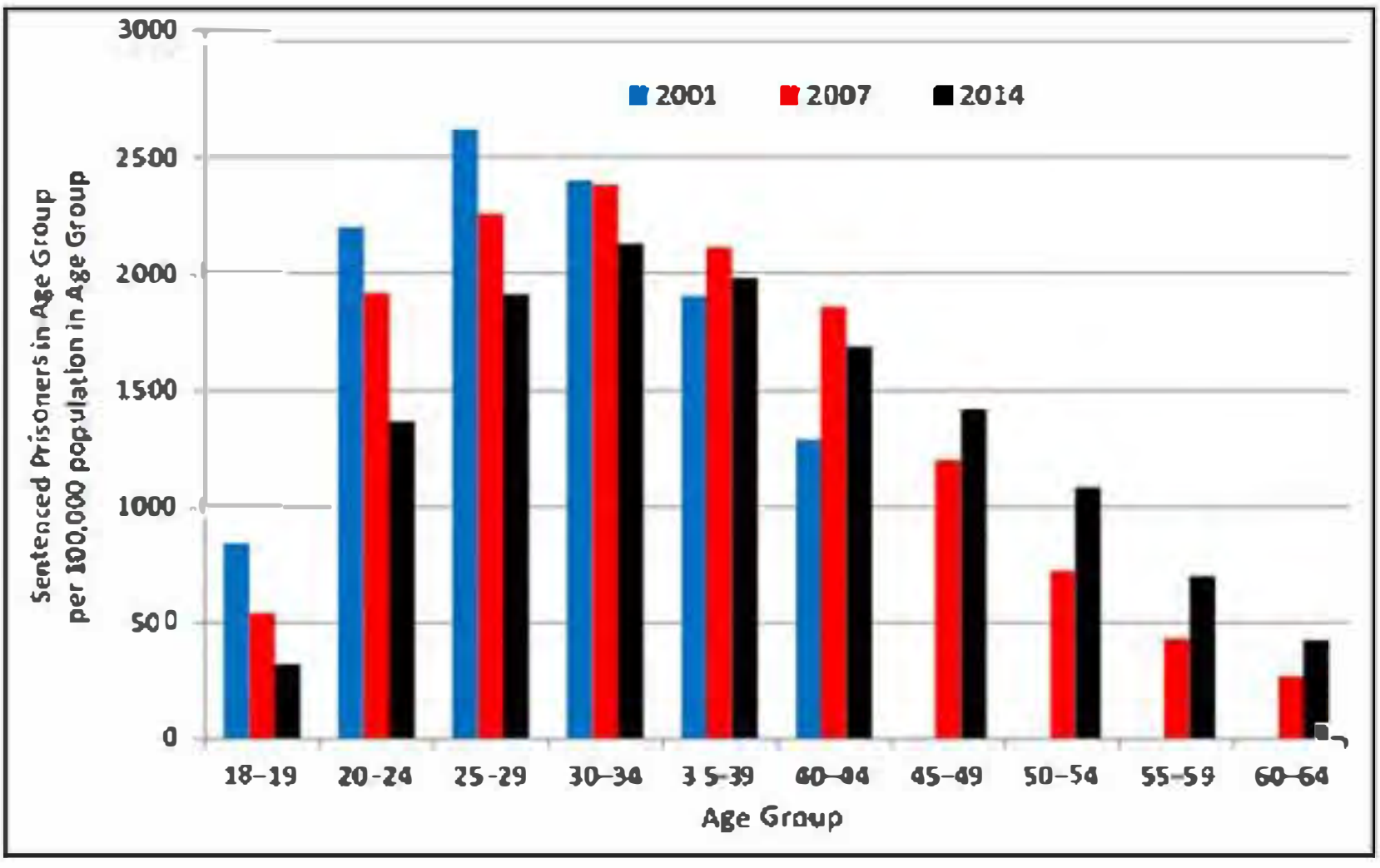


Fig. 2: 2001-2014 Shift in Male Incarceration Rates by Age



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III. Strength, Consistency, and Experimental Evidence

Hill illustrated "strength" with evidence that smokers have a lung cancer rate nine times that of nonsmokers, and a coronary death rate "no more than twice" that of nonsmokers, indicating causation in both cases, but with much stronger evidence for lung cancer. (Hill is best known for his work showing that smoking causes lung cancer, and his causation indicators rebut the "correlation does not mean causation" defense of tobacco firms in that era). The global rise and fall of crime shows that the

risk of criminal offending for those born during peak years of lead exposure was several orders of magnitude greater than the risk for those born just before and after the rise and fall of leaded gasoline use (as shown in Figures 3 through 14). In other words, this relationship is much stronger than the relationship between smoking and heart disease.

Hill recognized "consistency" in associations "repeatedly observed by different persons, in different places, circumstances and times". The association between preschool lead exposure and delinquent behavior was first discovered by Denno, based on a Philadelphia youth study reported in 1990.[25] Needleman found the same

association in Pittsburgh in studies published in 1996[26] and 2002.[27] Dietrich's 2001 study showed the same association in Cincinnati,[28] and a 2008 study by Wright, Dietrich, and colleagues confirmed an "Association of Prenatal and Childhood Blood Lead Concentrations with Criminal Arrests in Early Adulthood".[29] My 2000 study showed that lead exposure trends explained 90% of USA violent crime rate variation from the early-1960s to 1998. The association with violent crime was later confirmed by USA state[30] and city[31] crime studies. A 2001 study by Stretesky and Lynch found that USA counties with high air lead levels had murder rates four times

higher than counties with low air lead.[32] My 2007 study found that lead exposure also explained most of the violent and property crime rate variation across decades in nine nations. In seminal reporting on this issue, Kevin Drum calls this "an astonishing body of evidence. We now have studies at the international level, the national level, the state level, the city level, and even the individual level."[33]

Two studies published in 2016 further demonstrate the consistency of this relationship. Boutwell shows a clear association between census tract blood lead levels in Saint Louis Missouri and the relative risk of census tract violent and nonviolent crime.[34]

Taylor reports that Australia suburban air lead levels accounted for 30% of the local variance in assault rates 21 years later, and Australia State level analysis produced comparable results.[35]

Hill states that "the strongest support for the causation hypothesis may be revealed" when an action is taken to prevent a suspected cause of disease, and later trends show "experimental evidence" of cause and effect. "Is the frequency of the associated events affected?" In other words, does the hypothesized causal relationship have *predictive* power? Does removing the suspected cause have the expected effect in the real world?

My 2007 study reported the percent of crime rate variation through 2002 explained by trends in average preschool blood lead, measured in micrograms of lead per deciliter of blood (mcg/dl). Since 2002, another dozen years of crime data have fallen into place. The strength, consistency, and predictive power of the association between preschool blood lead and crime is evident in updated graphs (Figures 3 through 14) for burglary, robbery, and index crime trends in Canada,[36] Australia,[37] Britain,[38] the USA,[39] and New Zealand.[40]

My 2007 study found that 78% to 91% of burglary rate variation through 2002 in Canada, Britain, and Australia was explained by

earlier preschool blood lead trends. Burglary rates fell by more than 50% from 2002 to 2014 in all three nations, tracking earlier preschool blood lead trends.

My 2007 study found that preschool blood lead trends explained 71% to 89% of robbery rate trend variation through 2002 in the USA, Canada, Britain, and Australia. Robbery rates in Britain and Australia peaked in 2001 and fell about 60% from 2002 to 2014. Canada and USA robbery rates peaked 10 years earlier. The Canada robbery rate fell 50% from 1991 to 2014, including a 39% decline from 2002 to 2014. The USA robbery rate fell 63% from 1991 to 2014, including a 30% decline from 2002 to 2014.

My 2007 study found that preschool blood lead trends explained 87% to 95% of index crime trend variation through 2002 in Canada, Britain, and New Zealand. The Canada data include all offenses except traffic violations. Britain and New Zealand report similarly broad index crime data. The index crime rates in all three nations fell more than 30% from 2002 to 2014, tracking earlier preschool blood lead trends.

USA index crime includes violent crime (murder, rape, robbery, and aggravated assault) and property crime (burglary, vehicle theft, and other felony theft), but excludes petty theft, simple assault (without

serious injury or use of a deadly weapon), and other offenses included in broader index crime data available for Britain, New Zealand, and Canada. Index crime data available for Australia are comparable to USA index crime. My 2007 study found that lead exposure explained 63% and 80% of index crime rate variation through 2002 in Australia and the USA, respectively. The USA index rate peaked in 1991. Australia's rate peaked in 2001. The USA index crime rate fell 50% from 1991 to 2014, including a 28% decline from 2002 to 2014. The Australia index rate fell about 50% from 2002 to 2014.

Fig. 3: Britain Burglary and Preschool

Blood Lead Trends

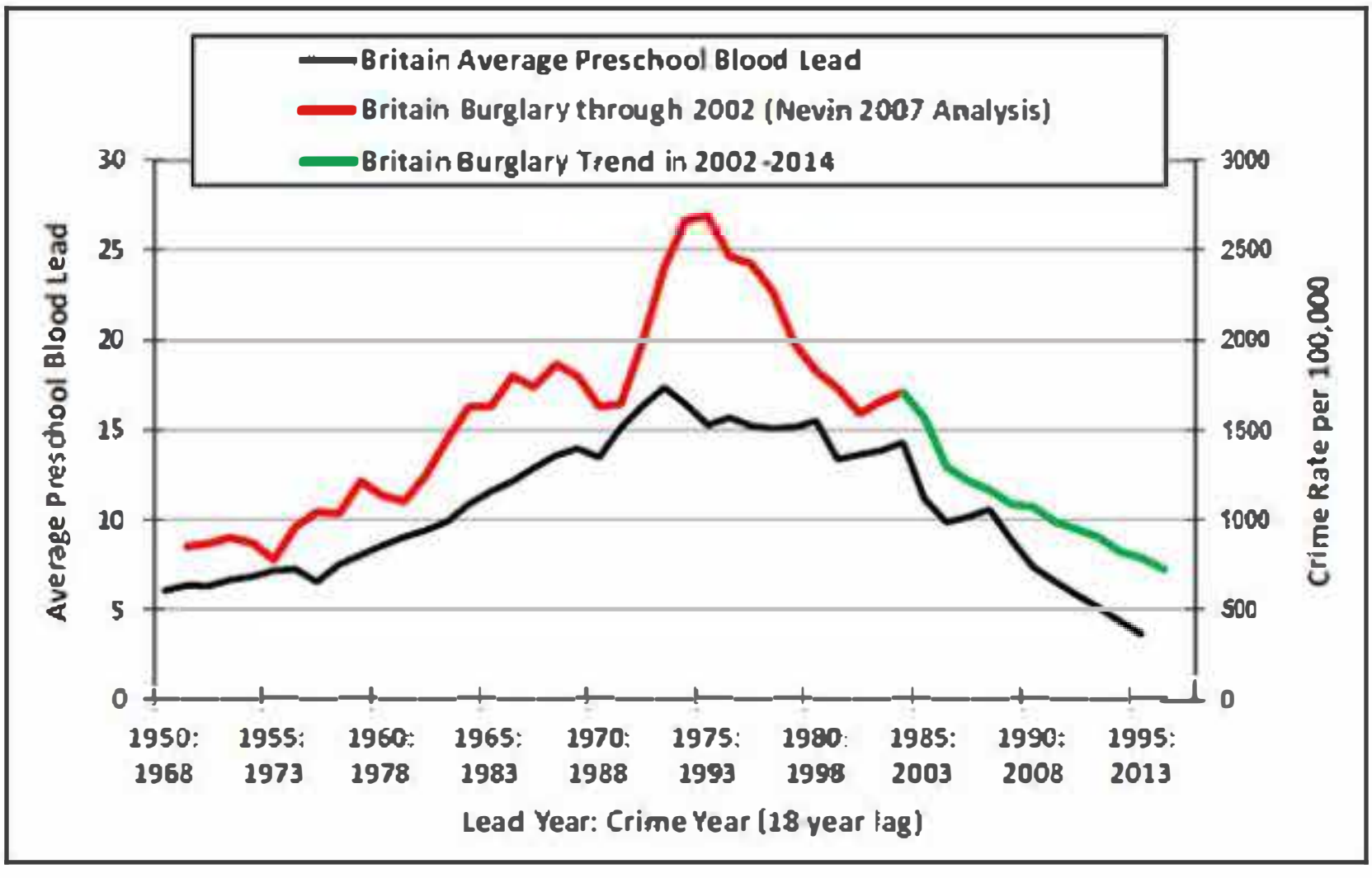


Fig. 4: Australia Burglary and Preschool Blood Lead Trends

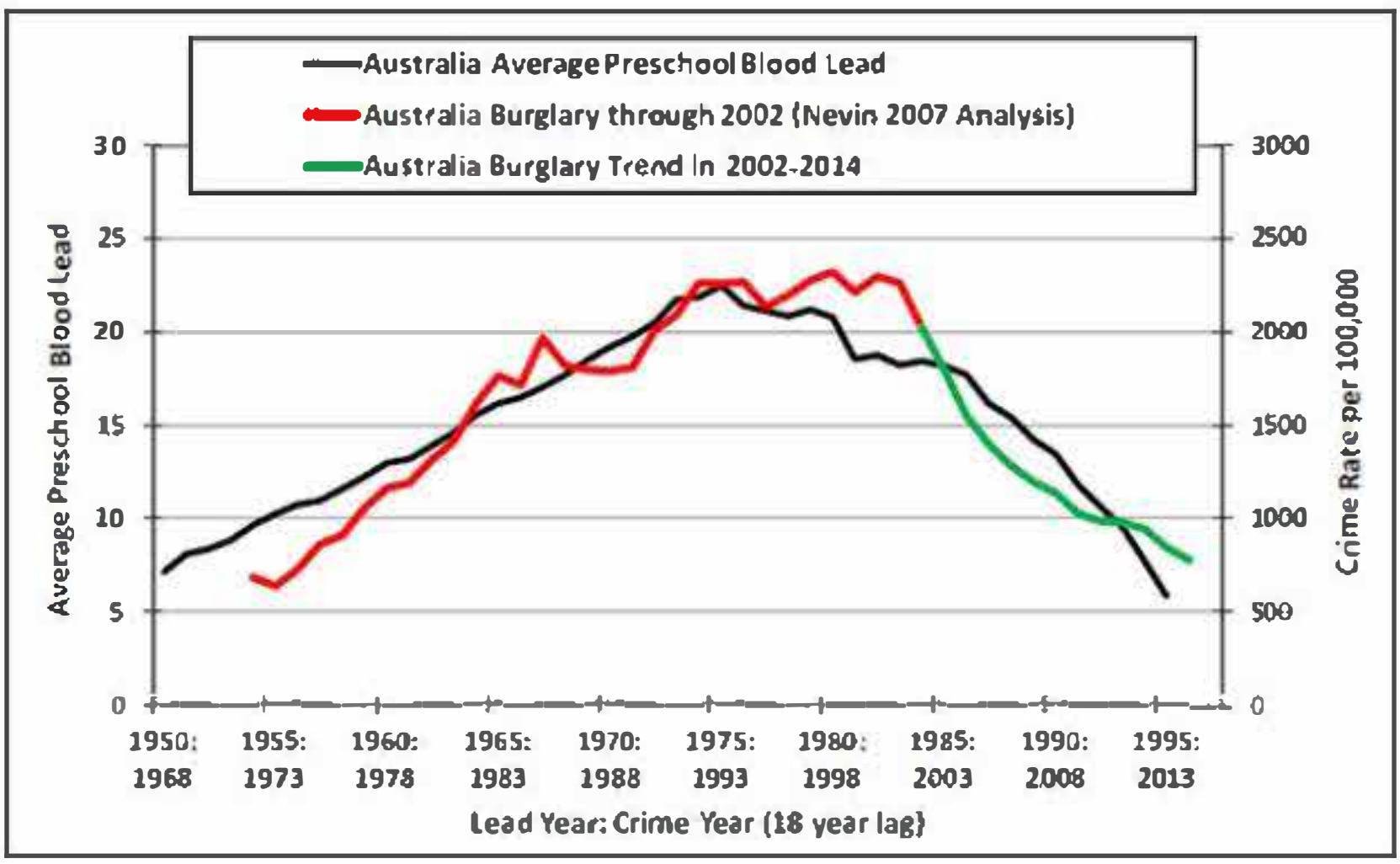


Fig. 5: Canada Burglary and Preschool

Blood Lead Trends

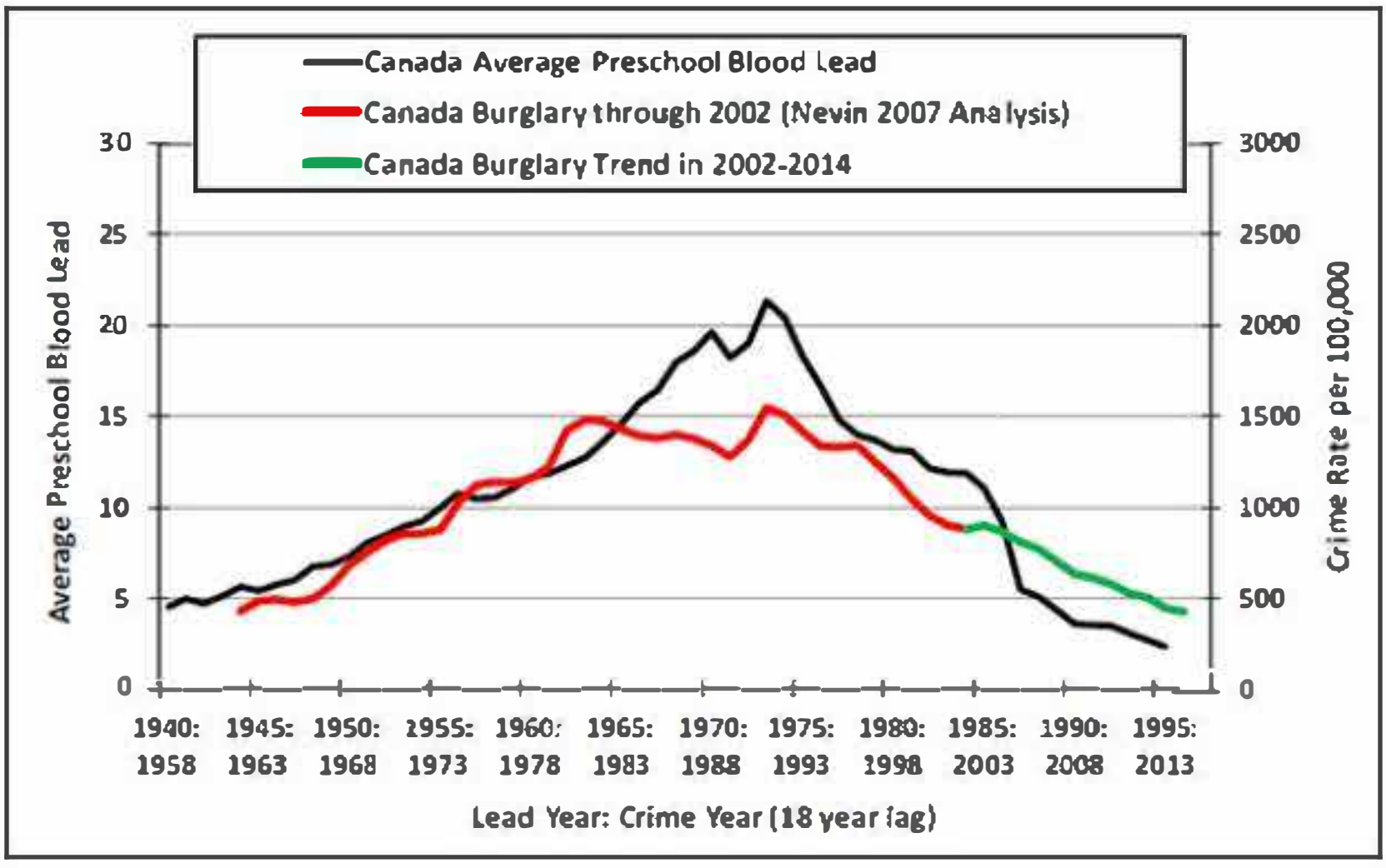


Fig. 6: Britain Robbery and Preschool

Blood Lead Trends

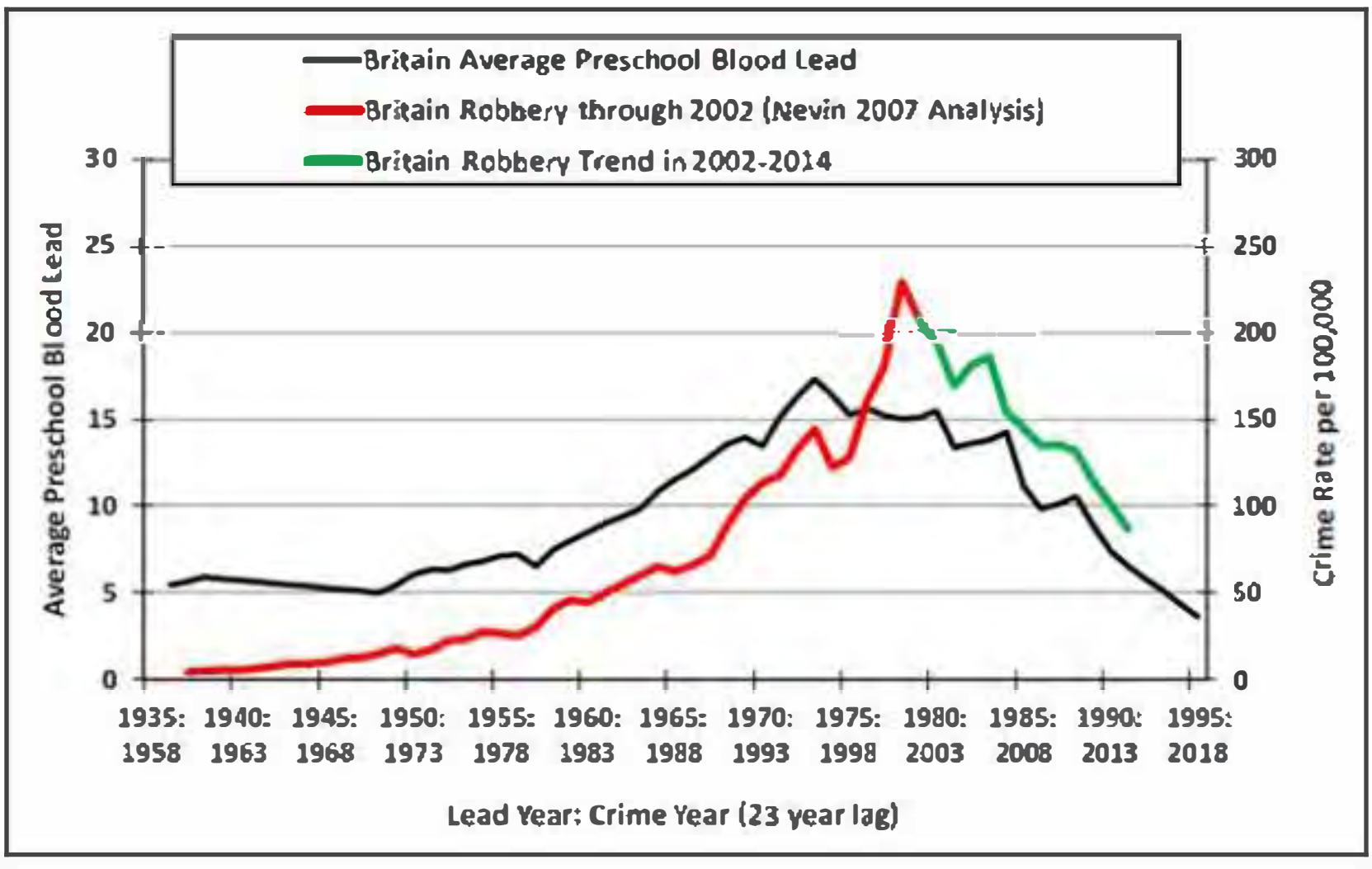


Fig. 7: Australia Robbery and Preschool Blood Lead Trends

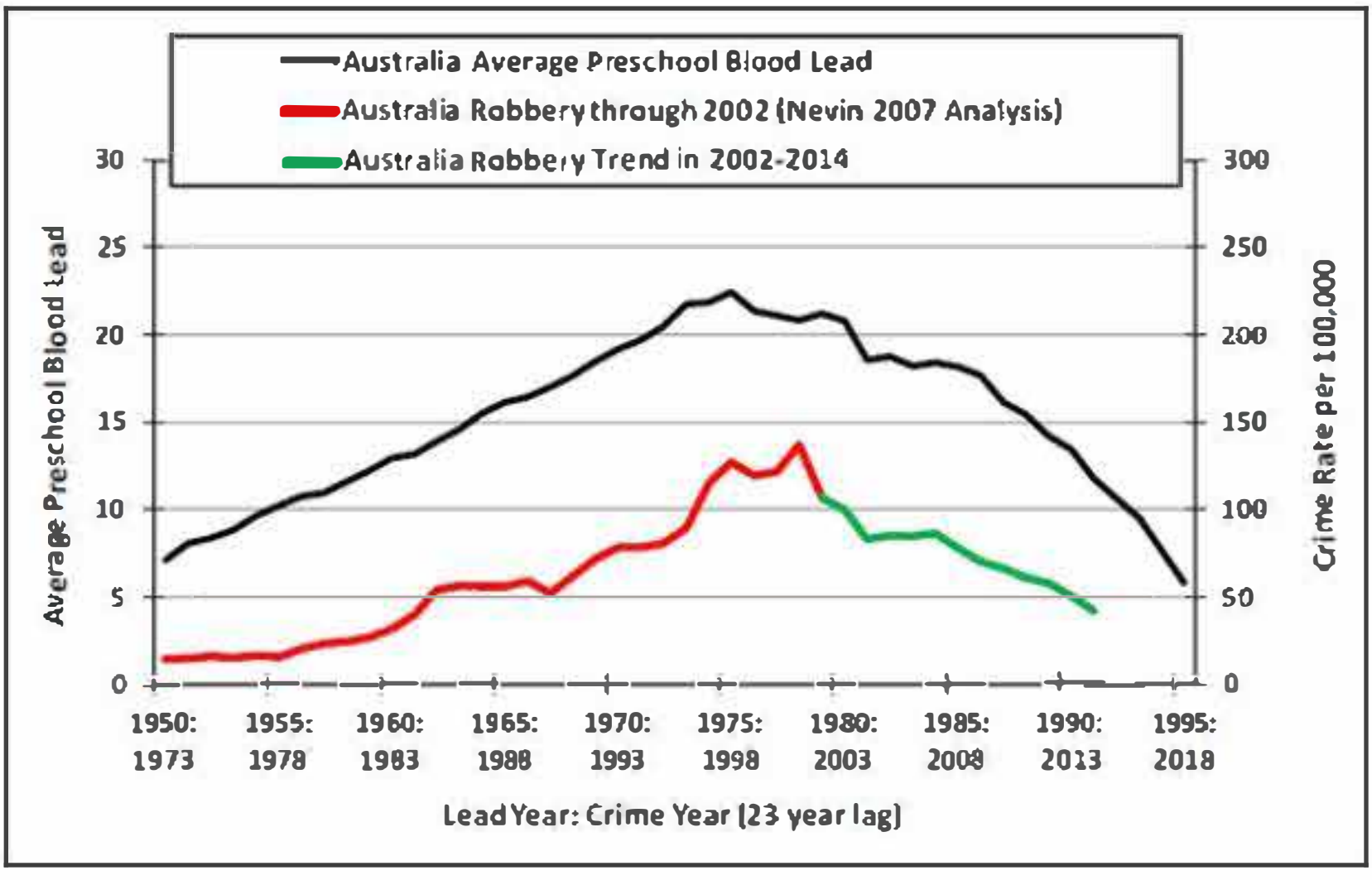


Fig. 8: Canada Robbery and Preschool Blood Lead Trends

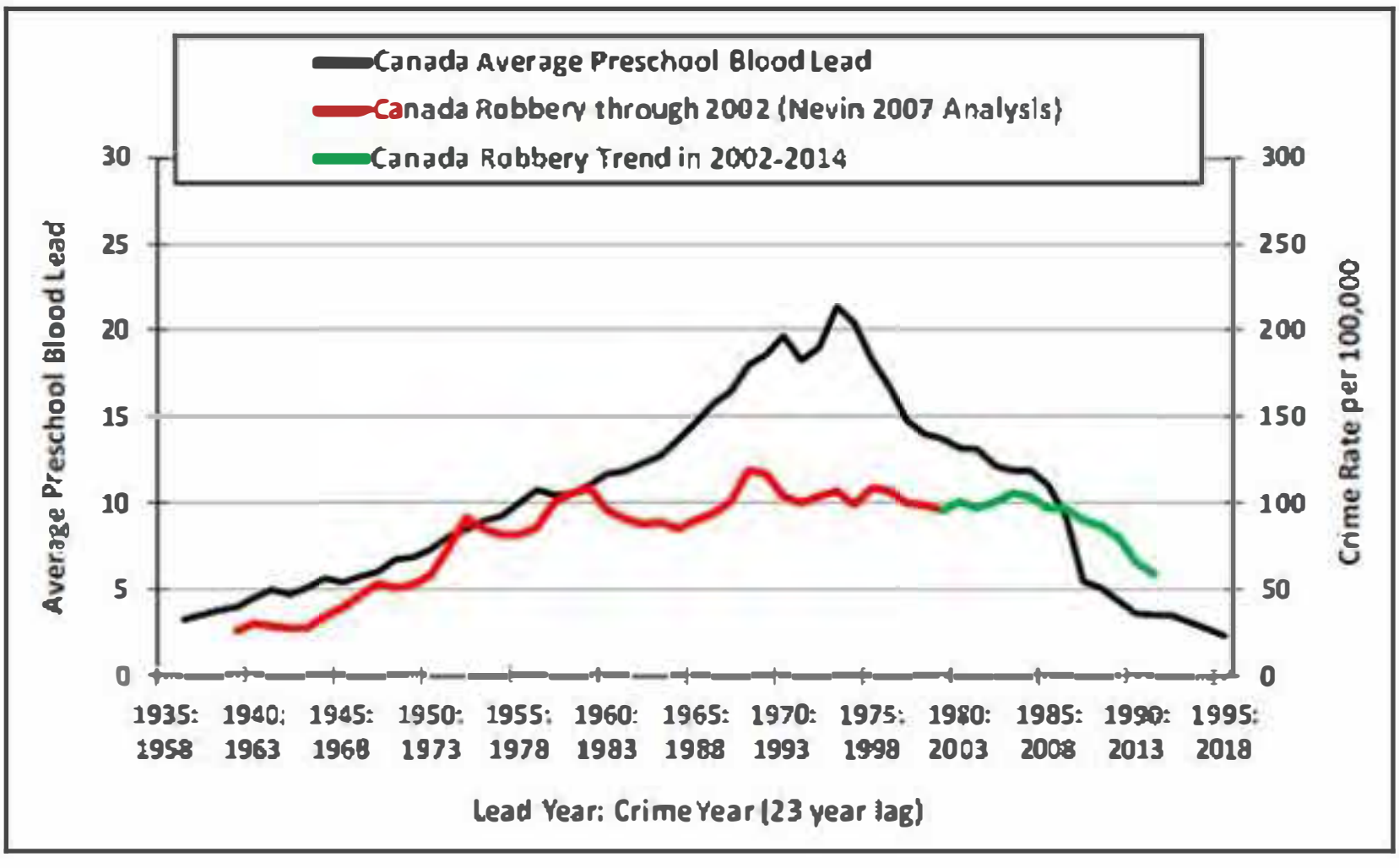


Fig. 9: USA Robbery and Preschool Blood Lead Trends

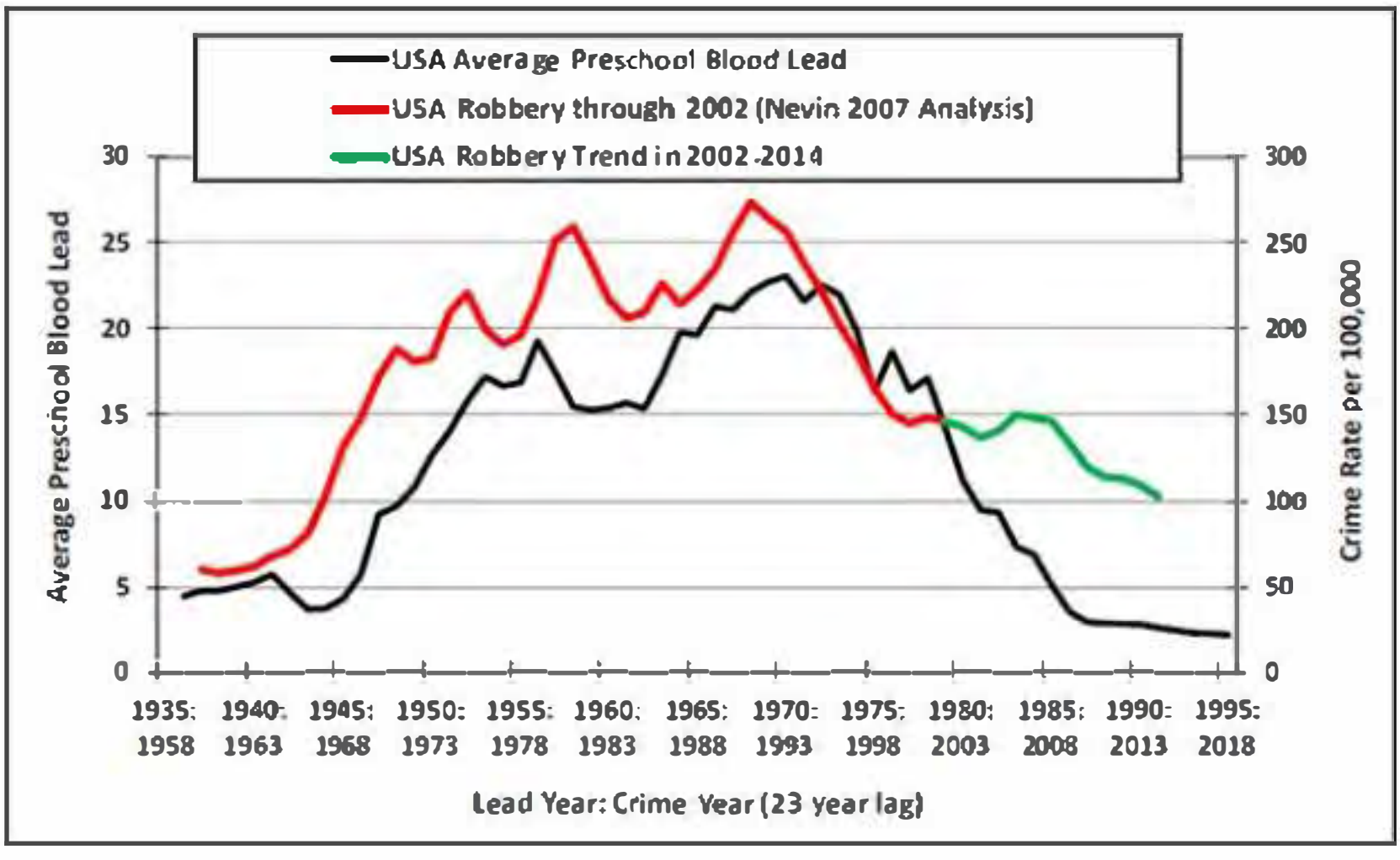


Fig. 10: Britain Index Crime and Preschool Blood Lead Trends

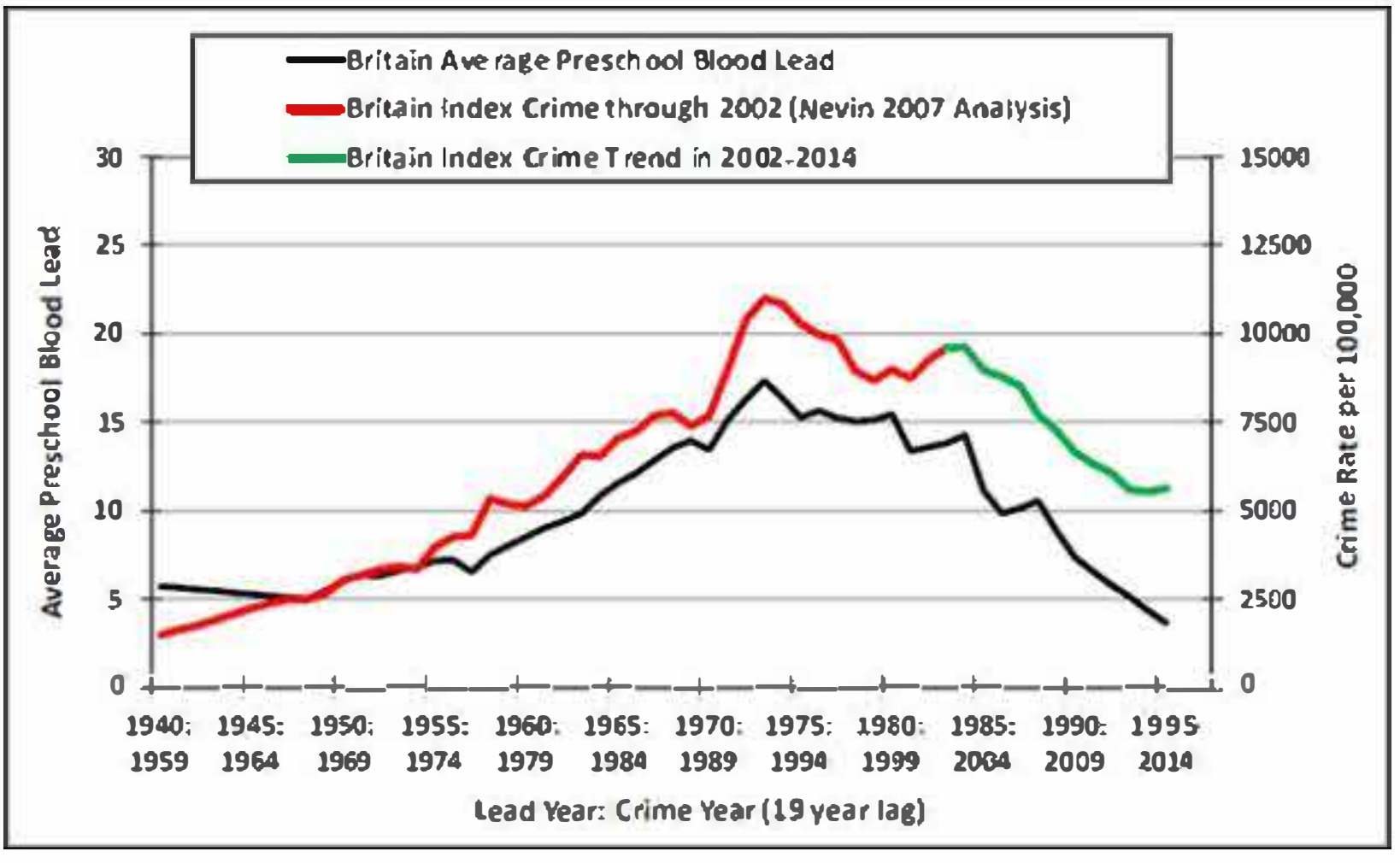


Fig. 11: Canada Index Crime and Preschool Blood Lead Trends

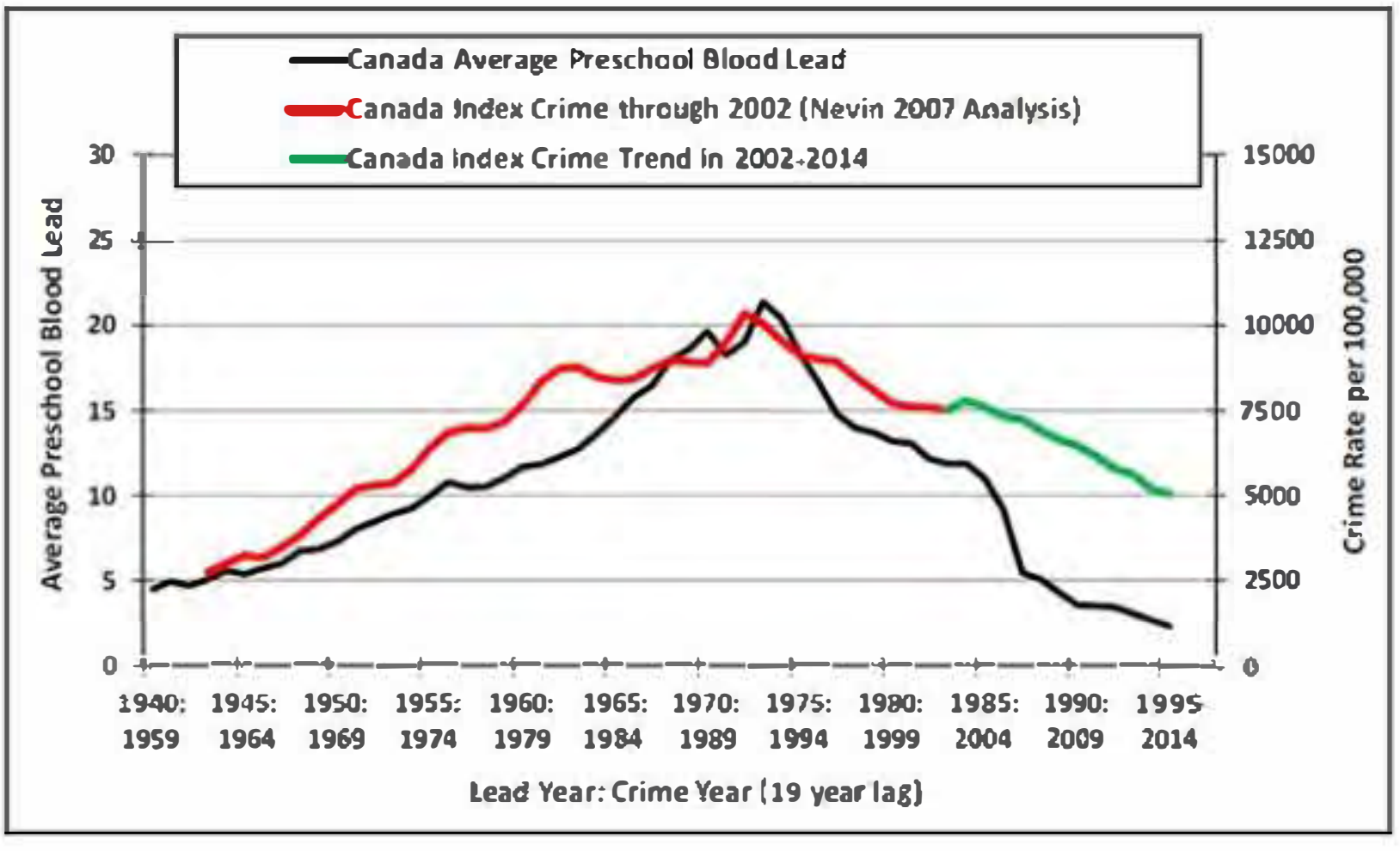


Fig. 12: New Zealand Index Crime and Preschool Blood Lead Trends

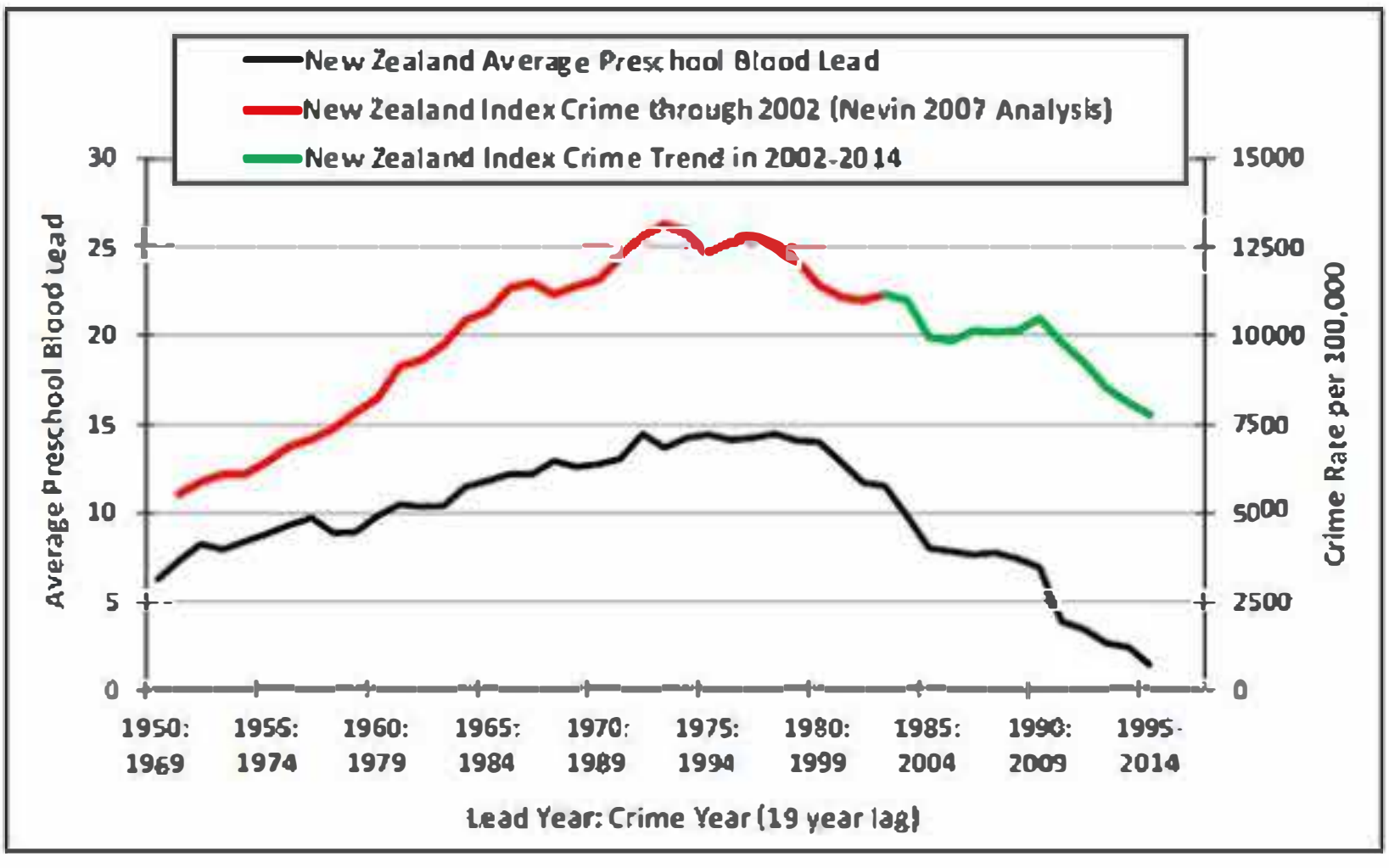


Fig. 13: Australia Index Crime and Preschool Blood Lead Trends

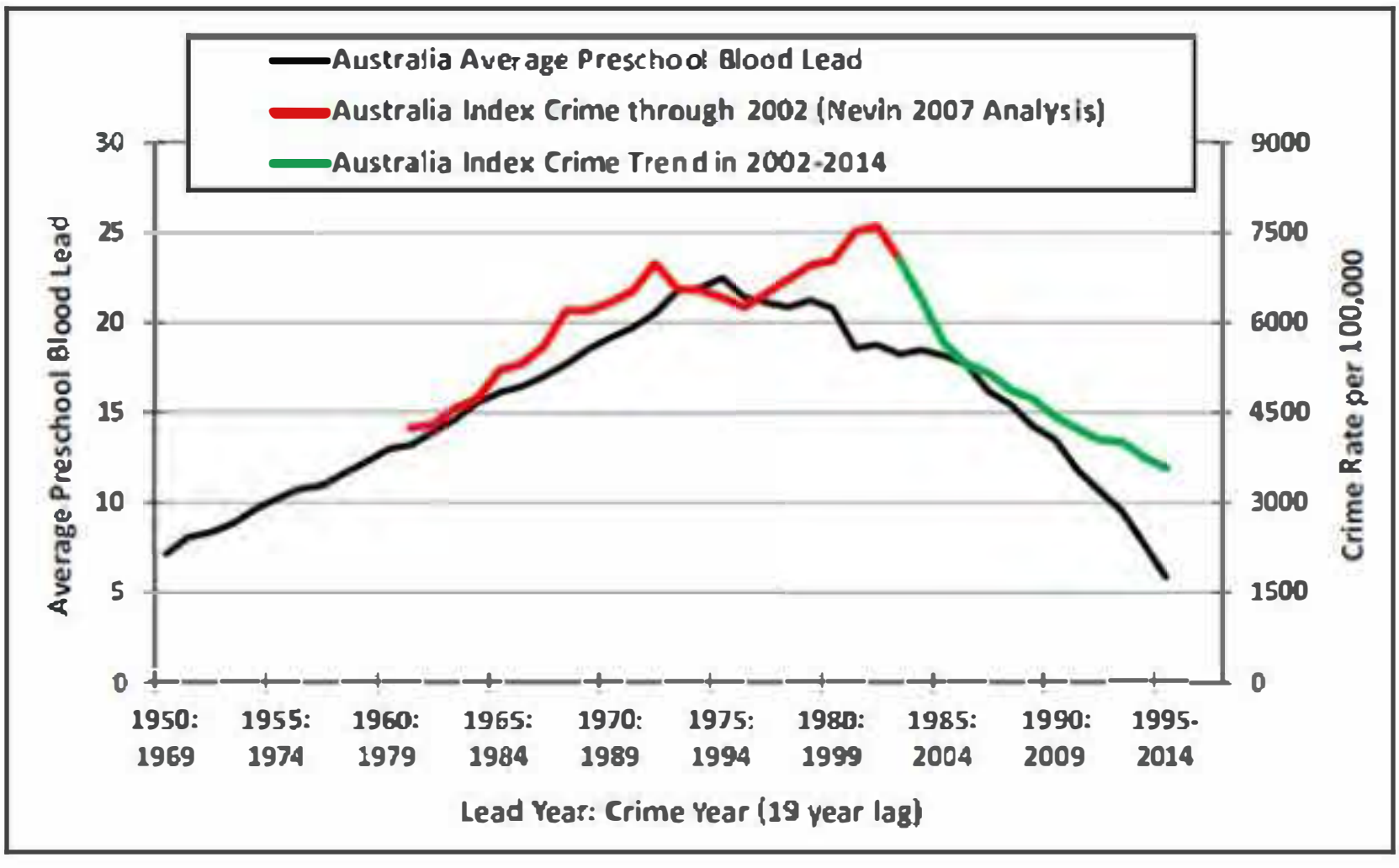
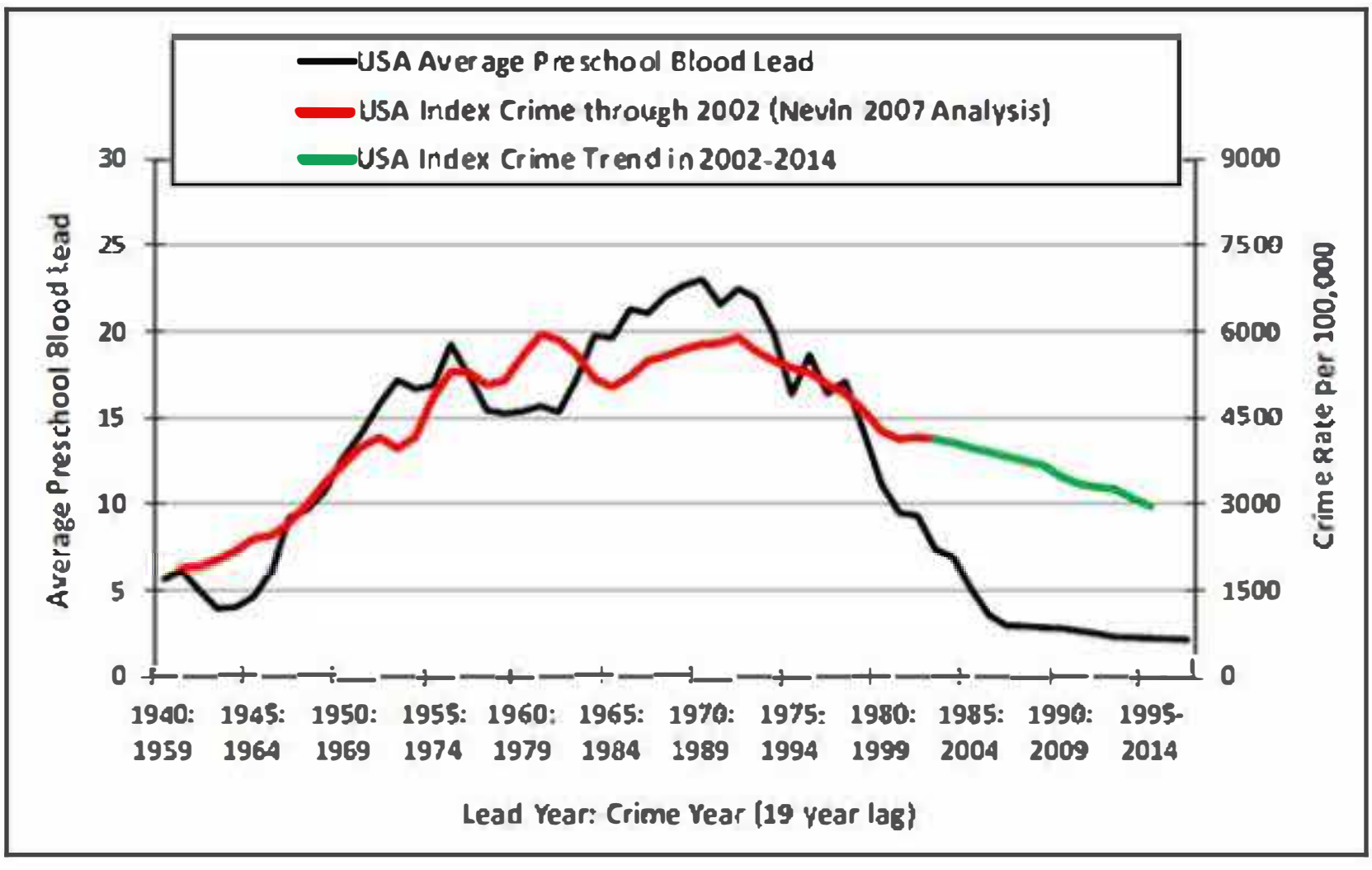


Fig. 14: USA Index Crime and Preschool Blood Lead Trends



In addition to affecting crime, my 2000 study found that lead exposure trends explained 58% of the 1972-1996 variation in pregnancy rates (births and abortions) for girls under age 15, and 90% to 96% of variation in unwed pregnancy rates from the 1950s to 1996 for ages 15-17, 18-19, and 20-24. The best-fit time lag for each age group was consistent with neurodevelopmental effects in the first year of life: A lag of 15 years for girls under the age of 15, 17 years for ages 15-17, 20 years for ages 18-19, and 24 years for ages 20-24.

My study added abortion rates by age from the Alan Guttmacher Institute (AGI) and unwed birth rates for the next year to estimate

unwed pregnancy rates by year of conception.[41] After my study was published, I contacted AGI to make them aware of my findings, and received a reply that was polite but skeptical about my cause and effect theory. The last paragraph of that letter made the following important observation about my 2000 study graphs:

"The charts show that the level of lead exposure drops off steeply... suggesting that crime and unwed pregnancy rates are about to do the same. If they do, then this will provide strong support for the proposed theory, but considering societal

> changes regarding marriage, it seems highly unlikely that unwed pregnancy rates will plummet in the next ten years." (AGI, 2000)

Hill would have applauded this emphasis on experimental evidence as the strongest indicator of causation. So what does experimental evidence tell us about lead exposure and unwed pregnancy rates? From 1996 to 2013, the pregnancy rate for girls under age 15 fell by 72%, and unwed pregnancy rates fell 66% for ages 15-17, 48% for ages 18-19, and 23% for ages 20-24.

The decline in unwed teen pregnancy reflects large declines in

abortion and unwed birth rates. For women in their early-20s the decline is mostly due to declining abortion rates. Unwed birth rates have increased since the 1990s for women over age 25 and abortion rates have increased for women over 40.[42] This is the same shift to older ages seen in arrest and incarceration rates, reflecting ongoing effects of birth years when lead exposure was still rising.

In addition to steep declines in unwed teenage births and smaller declines for ages 20-24, the CDC reports that nearly 60% of unwed births in 2006-2010 were to women in cohabiting relationships, up from 41% in 2002, and half of the births to cohabiting women in

2006-2010 were planned.[43] The CDC notes that planned births suggest more social and financial support within a cohabiting union, and that measures of father involvement are similar among married and cohabiting fathers. All of these trends reveal a steep decline in the number of children "growing up in chaotic families".

Fig. 15: USA Under-Age-15 Pregnancy Rate and Preschool Blood Lead Trends

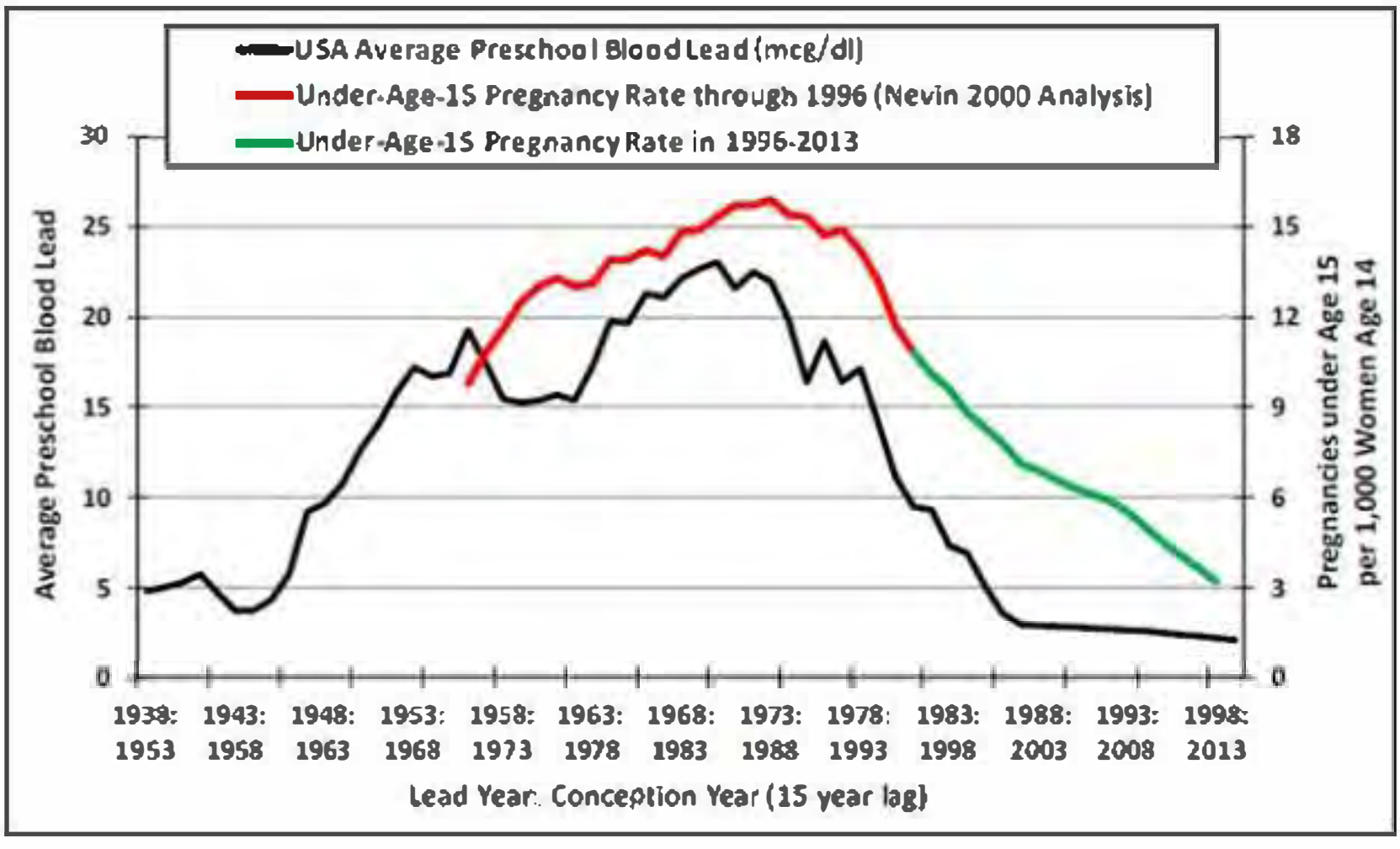


Fig. 16: USA Age 15-17 Unwed Pregnancy Rate and Preschool Blood Lead Trends

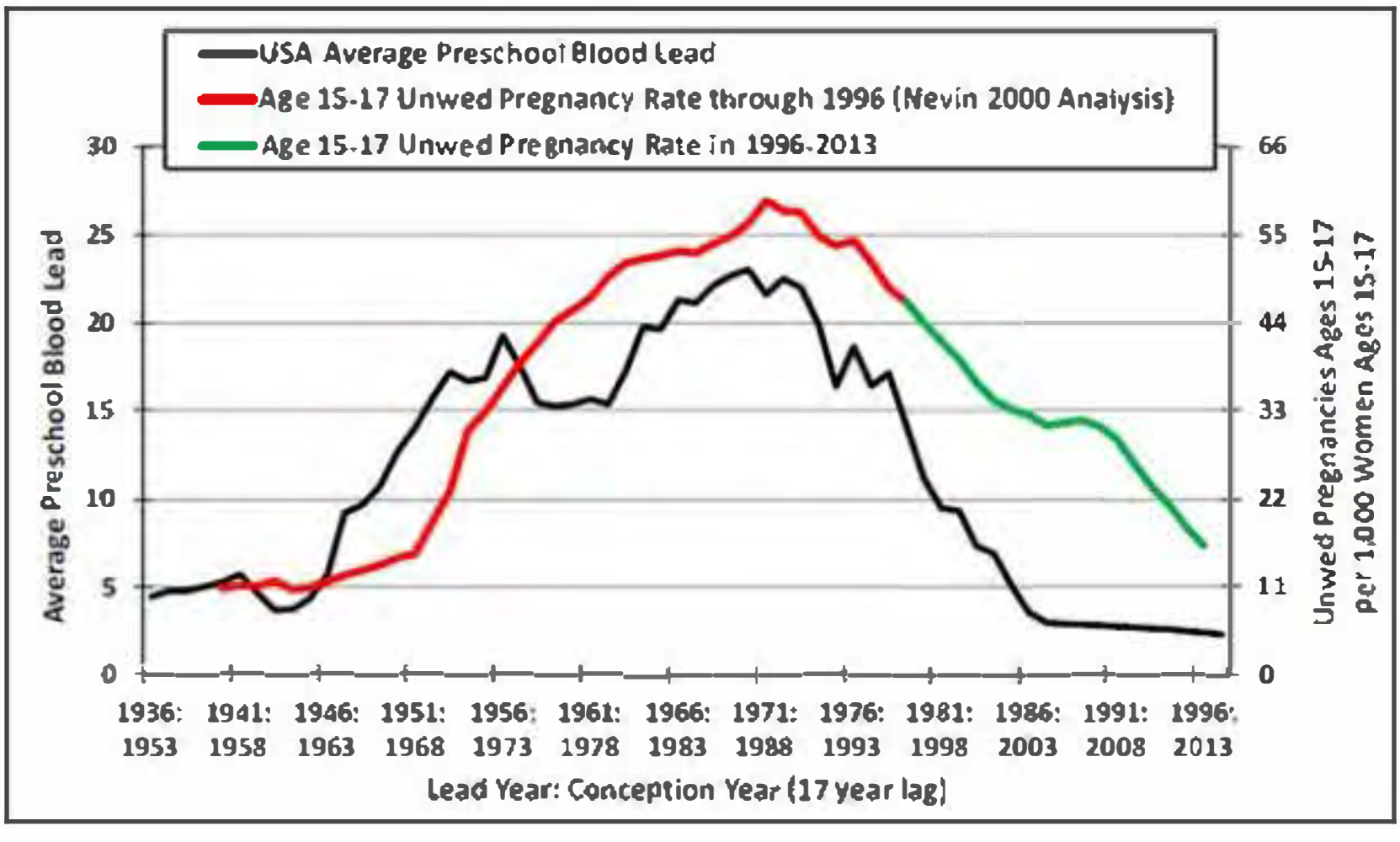


Fig. 17: USA Age 18-19 Unwed Pregnancy Rate and Preschool Blood Lead Trends

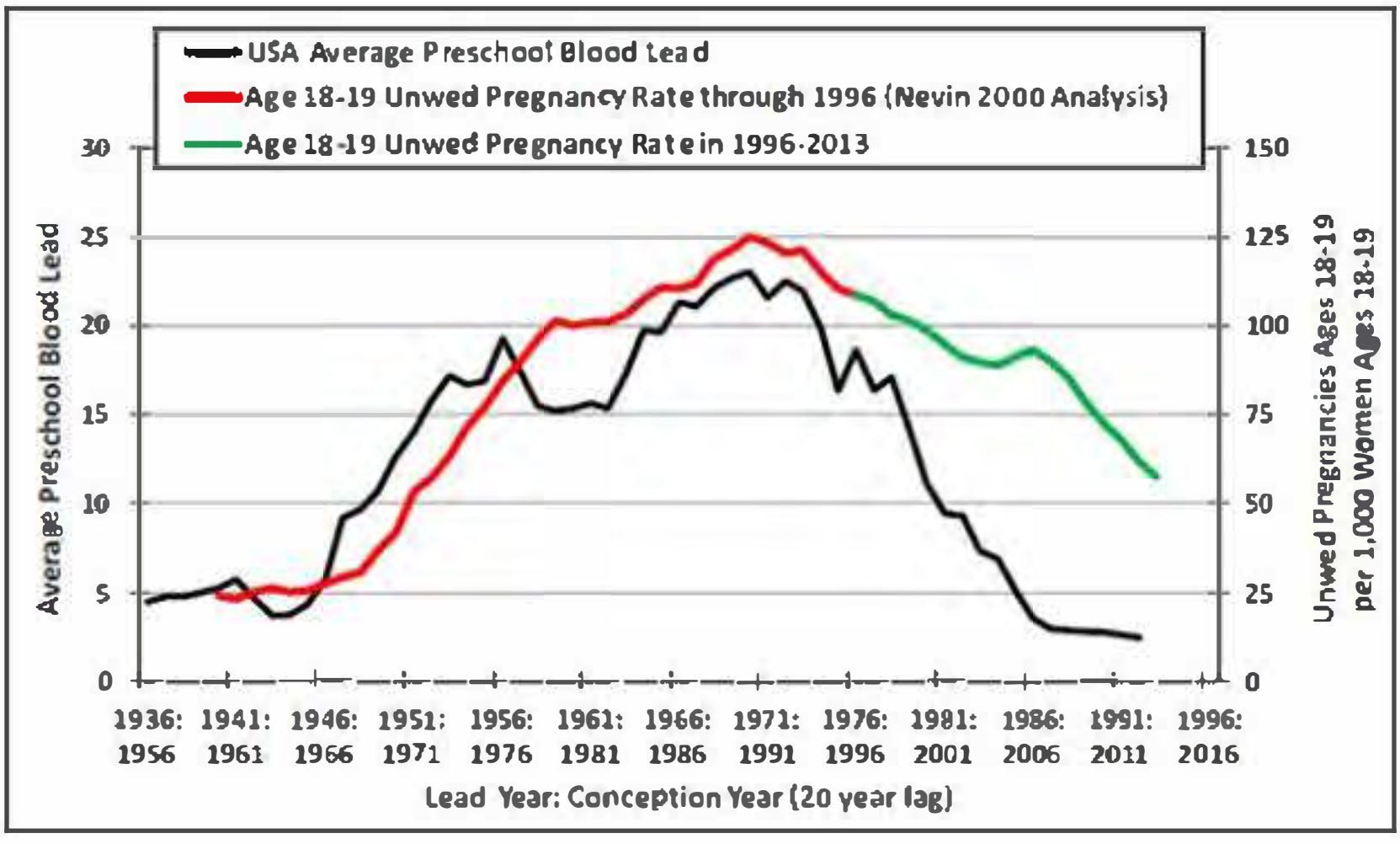


Fig. 18: USA Age 20-24 Unwed Pregnancy Rate and Preschool Blood Lead Trends

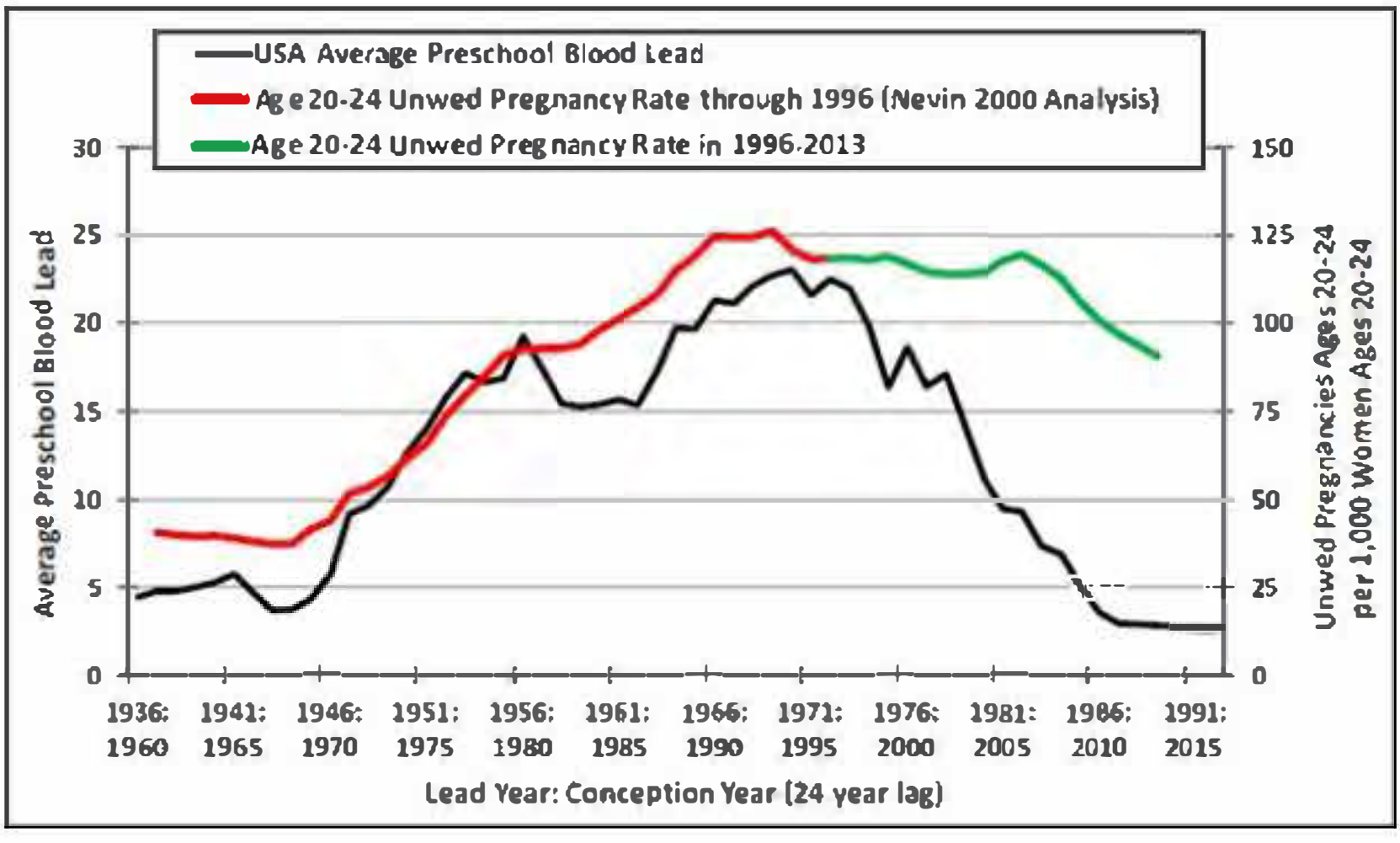
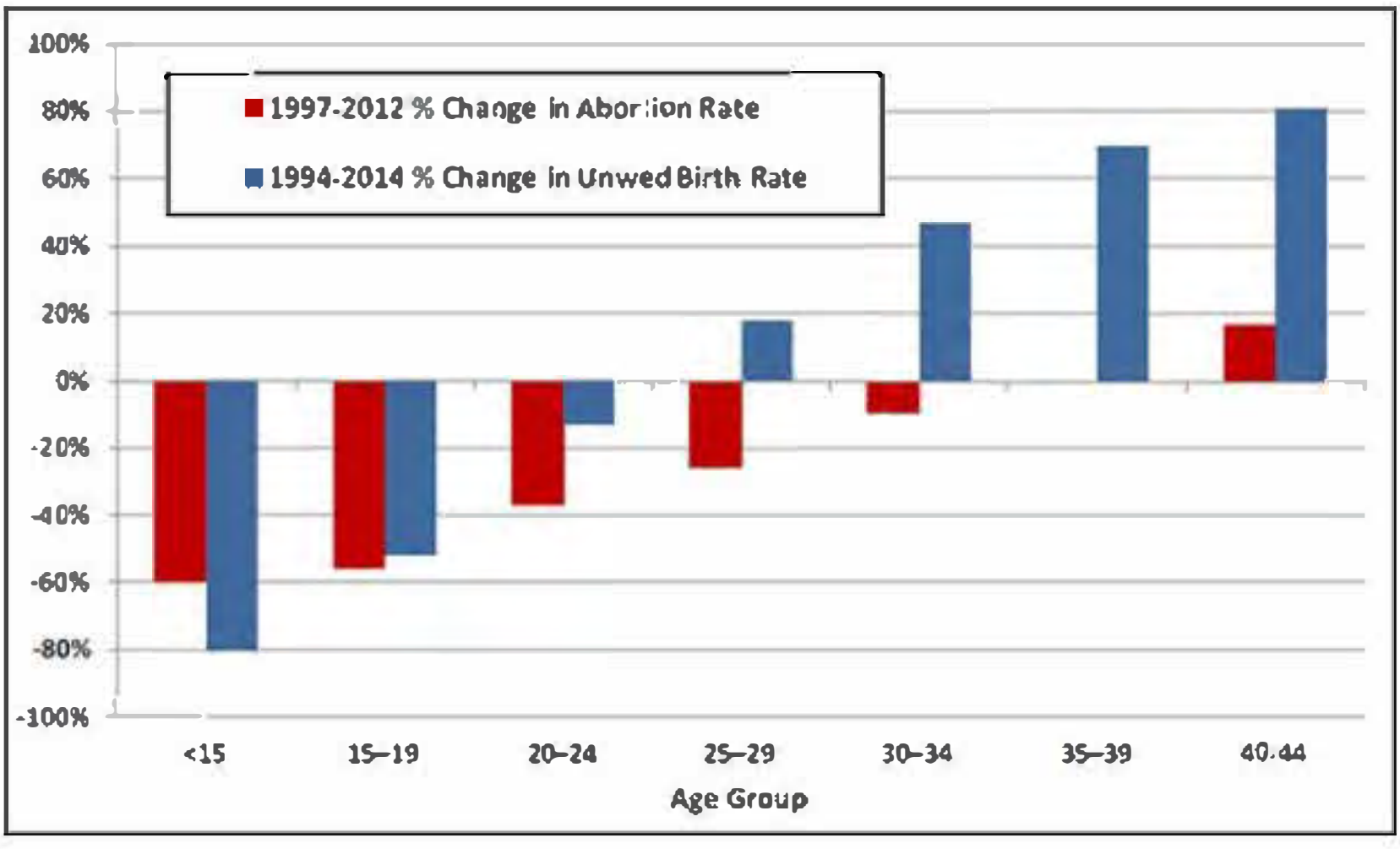


Fig. 19: Change in USA Unwed Birth

and Abortion Rates by Age



Returning to crime, what does experimental evidence tell us about my 2000 study finding that lead exposure trends explain 90% of USA violent crime rate variation from 1960 to 1998? The violent crime rate fell 36% from 1998 to 2014. That fall was not as steep as the 1975-1991 fall in lead exposure because the violent crime arrest rate for those over 50 increased 13% from 1998-2014 (as boomers also

swelled the population over 50).

Lead exposure in Latin America and related homicide trends provide more experimental evidence of the causal relationship between lead exposure and crime.[44] Columbia has not used leaded gasoline since 1990,[45] and air lead levels in Medellin Columbia fell 86% from 1980 to 1994. The Colombian homicide rate was 67 per 100,000 in 2002,[46] but fell to 26 per 100,000 in 2014,[47] down 61% from 2002-2014.

Sao Paulo did not set out to reduce lead poisoning in the 1980s, but Brazil began large-scale production of E95 (lead-free) fuel ethanol in 1979 to reduce oil imports.[48] By 1987, E95 accounted for half of all

vehicle fuel in Brazil, and the E95 share was highest in Sao Paulo state, where ethanol distilleries are concentrated. Air lead in Sao Paulo fell by about 65% from 1980-1985, and murders in Sao Paulo fell by about 65% from 1999-2008.[49]

In 1987, almost all gasoline sold in Mexico contained lead, but the maximum lead content was lowered from 0.53 to 0.28 grams per liter in 1988, and Mexico air lead fell 37% from 1988 to 1990. Mexico murders appear to have peaked in 2011, and fell by 30% from 2011-2014.[50]

El Salvador had the highest murder rate of any nation in Latin America and the Carribean in 2015,[51] but help is on the way. Unleaded

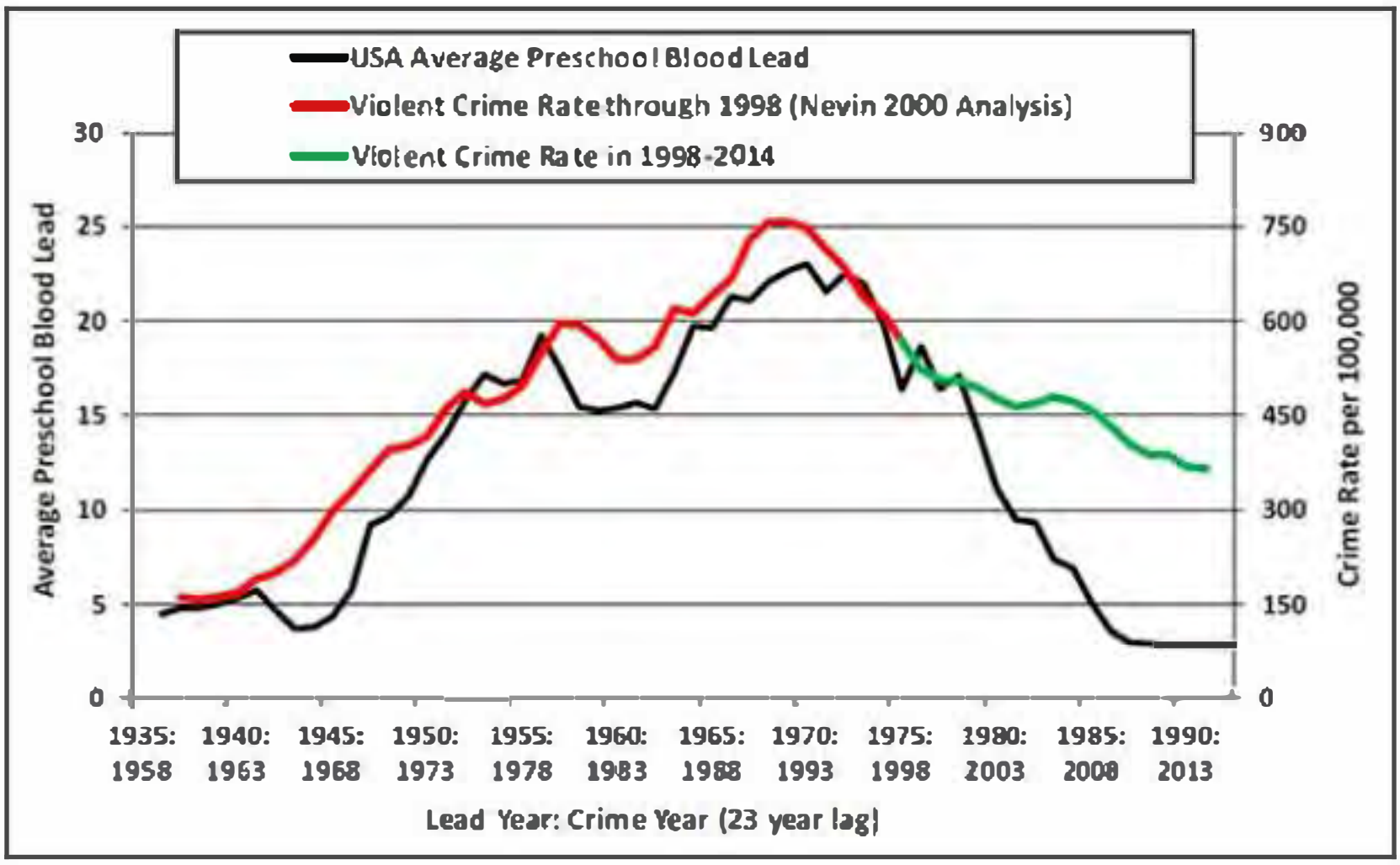
gasoline accounted for just 2% of El Salvador gasoline use in 1992, but leaded gasoline was completely eliminated in 1996.

Venezuela had the second highest murder rate in Latin America and the Carribean in 2015, and it will take a few more years for relief to arrive there. Venezuela did not begin selling unleaded gasoline until 1999, and was one of the last countries to ban leaded gasoline, in 2005.

With the notable exception of Venezuela, Latin America and Carribean nations substantially reduced leaded gas use in the first half of the 1990s. The total amount of lead used in gasoline in the region fell 62% from 1990 to 1996.

As a result, the violent crime trend in Latin America and the Carribean over the next two decades can be expected to look a lot like the USA violent crime trend over the last two decades.

Fig. 20: USA Violent Crime and Preschool Blood Lead Trends



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IV. Dose-Response, Biological Plausibility, and the Death Penalty

To illustrate dose-response, Hill states that lung cancer mortality risk that increases with number of cigarettes smoked daily "adds a very great deal to the simpler evidence that cigarette smokers have a higher death rate than non-smokers."

Extensive research shows a dose-response relationship between blood lead and IQ later in life. Preschool blood lead of 10 mcg/dl is associated with 7.4 IQ points lost relative to blood lead of one

mcg/dl.[52] Another 1.6 IQ points are lost with blood lead of 15 mcg/dl relative to 10 mcg/dl, and each mcg/dl over 15 lowers IQ by 0.23 points, on average.[53] Therefore, blood lead of 40 mcg/dl is associated with an average loss of 15 IQ points, and a 60 mcg/dl level lowers IQ by almost 20 points.

A population dose-response relationship is also evident in the graphs of preschool blood lead and burglary, robbery and index crime trends around the world. Any substantial increase in average preschool blood lead has a clear association with an increase in crime rates about two decades later, and larger increases in blood lead result in larger increases in

crime. The same population dose-response relationship is evident in graphs of preschool blood lead and USA unwed pregnancy rates.

Ongoing declines in juvenile arrests reflect declines in elevated blood lead prevalence since the 1980s, mainly due to declines in lead paint hazards (including the impact of regulations that were the subject of my 1990s analysis). The percent of children ages 1-5 with blood lead over 5 mcg/dl fell from over 31% in 1988-1991[54] to 2.6% in 2007-2010.[55] Wright found that blood lead levels above 5 mcg/dl are associated with higher violent and non-violent crime arrest rates with a lag of about 20 years, and arrest rates rose with each 5 mcg/dl increase in preschool blood lead - a dose-

response relationship.

My 2007 study also presented evidence of another type of dose-response relationship, with more severe lead poisoning especially associated with more severe violent offending. This was suggested by a cross-sectional analysis of average murder rates over the 10 years from 1985-1994 in each of 124 USA cities, compared to city size (with higher 1970 air lead in large cities) and the estimated number of preschool children in each city with 1970 blood lead over 40 mcg/dl due to severe lead paint hazards in substandard (severely deteriorated) housing. This analysis showed a very strong association between city murder rates and severe lead poisoning caused by additive

exposure to urban air lead and lead paint in deteriorated housing.

Another indication of a dose-response relationship between lead poisoning severity and offending severity was shown in racial contrasts in blood lead and juvenile arrest rates. In the late-1970s, the *average* blood lead for black children under age 3 was 50% above the average for white children, but black children were six times more likely to have blood lead of 30–39 mcg/dl and eight times more likely to be over 40 mcg/dl. Those children were juveniles in the early-1990s, when the black juvenile burglary arrest rate was 60% higher than the white rate, but the black juvenile violent crime arrest rate was five times higher,

and the black juvenile murder arrest rate was eight times higher than the white juvenile rate.

The percent of black preschool children with blood lead above 30 mcg/dl fell by 90% from the late-1970s to the late-1980s, and the black juvenile murder arrest rate then fell 83% from the early-1990s through 2004, and fell to 87% below its early-1990s peak in 2012. The "chaotic families" hypothesis provides no insight into this stunning decline in black juvenile homicide arrests over the past two decades: The percent of black children living in two-parent families was 38% in 1990 and in 2012.

An association between severe lead

poisoning and homicide would also explain why the murder rate has historically been much higher in the USA than in Canada. The USA and Canada had similar *average* preschool blood lead levels in the late-1970s, and similar burglary rates in the early-1990s, but there was a stark contrast in severe lead poisoning and murder rates. In the late-1970s, only 4% of Canadian preschool children had blood lead over 20 mcg/dl, when 18% of white children and 52% of black children in the USA had blood lead over 20 mcg/dl. In the early-1990s, the USA murder rate was four times higher than the Canadian murder rate.

The USA experienced more severe lead poisoning than other nations because lead used in paint

accounted for almost a third of USA lead output from 1900-1914, when the USA produced over 40% of world lead output. The USA also accounted for 80% of global gasoline lead use prior to 1970.[56]

My 2000 study also found that USA homicide rates from 1900-1998 tracked the rise and fall of USA per capita use of lead in paint and gasoline from 1879-1987, with preschool lead exposure affecting the homicide rate with a 21-year lag. The USA homicide rate fell 52% from 1991 to 2014, including a 26% decline from 1998-2014. This trend indicates that the USA homicide rate will soon fall to its lowest level in more than 110 years.

Fig. 21: Preschool Blood Lead and IQ

Dose-Response Relationship

Fig. 22: Decline in Percent of Children Ages 1-5 with Blood Lead > 5 mcg/dl

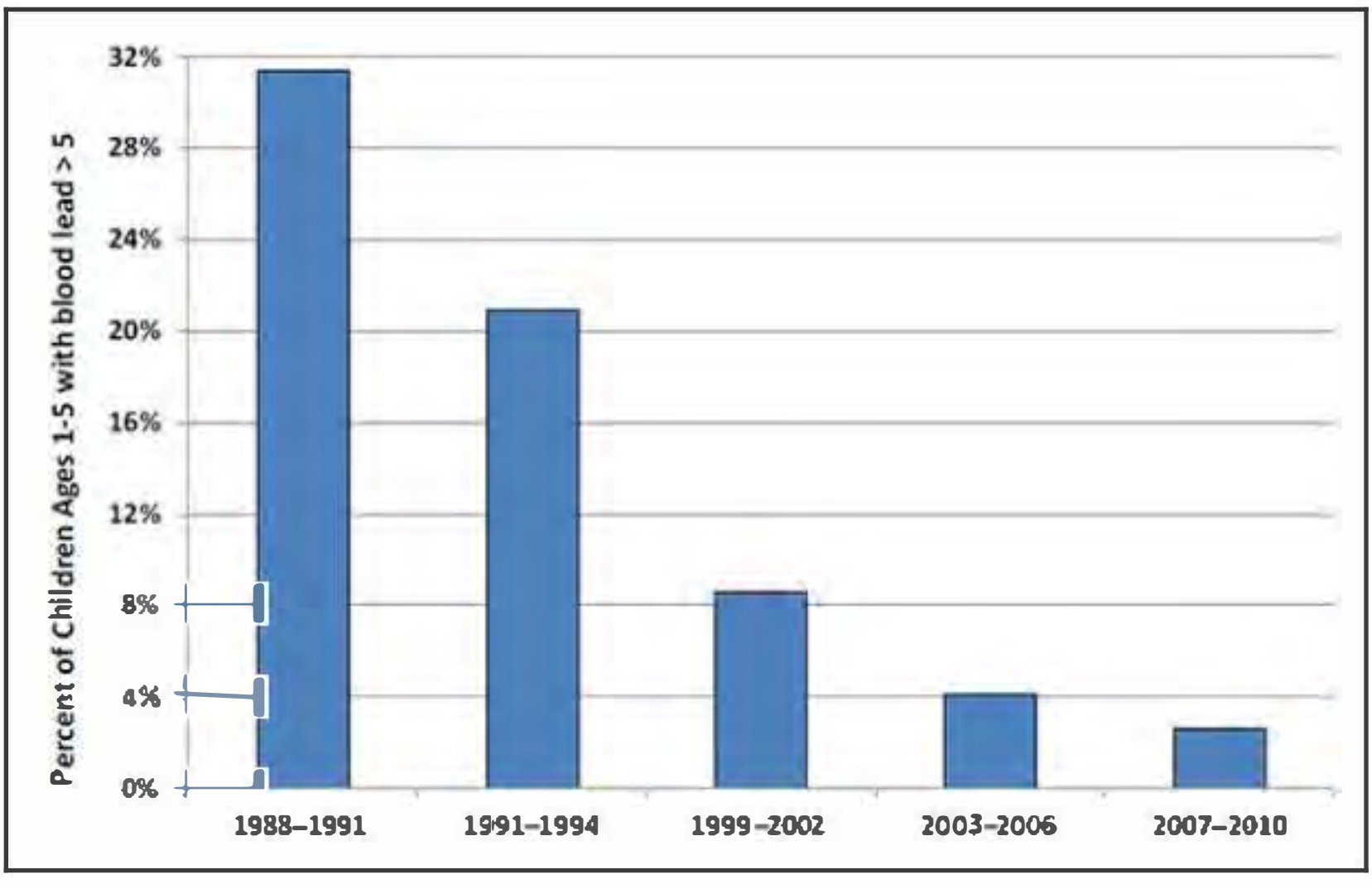
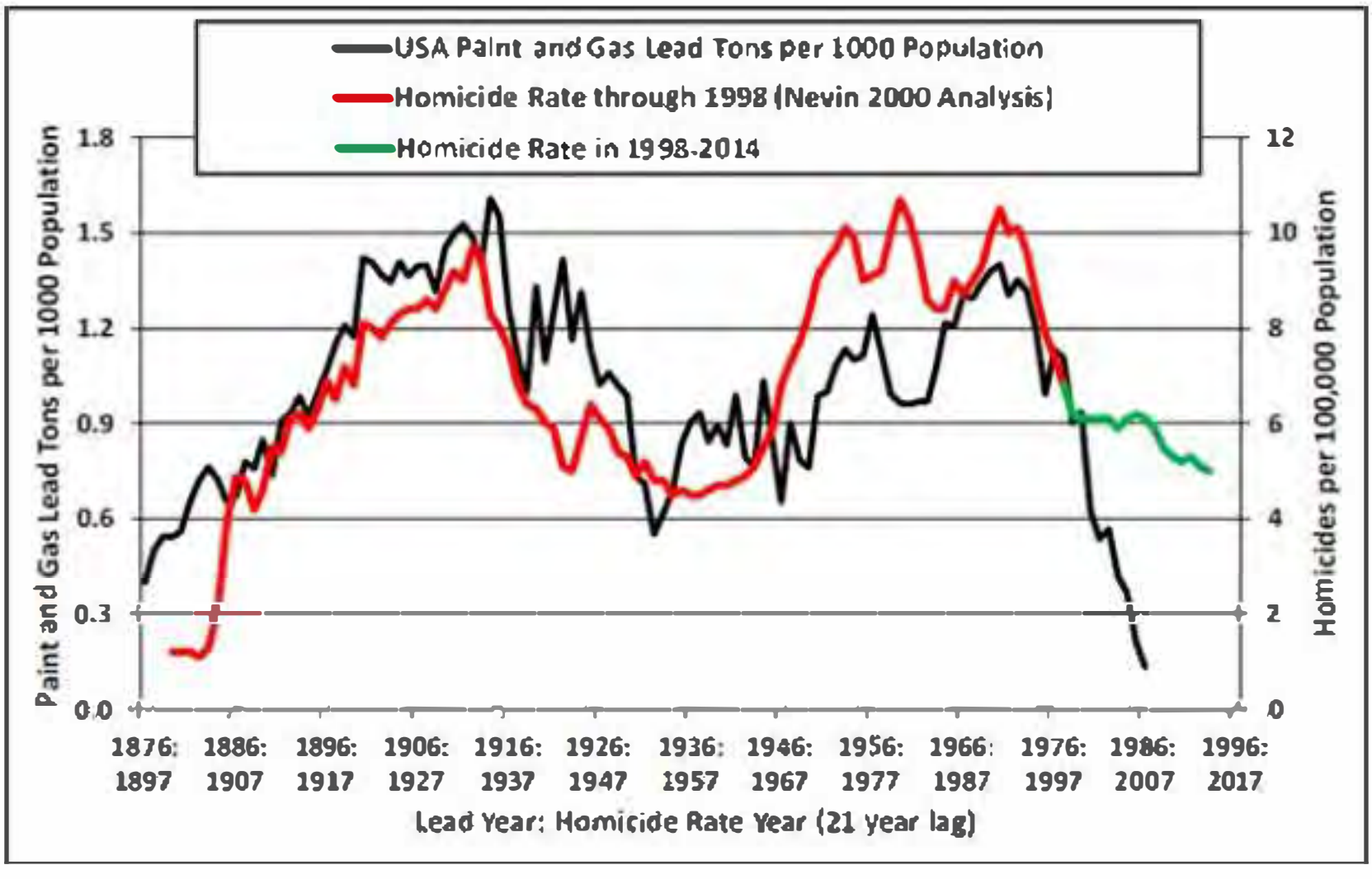


Fig. 23: USA Homicide Rate and Lead

Exposure Trends



The "biological plausibility" of lead poisoning affecting behavior is demonstrated by extensive research on exposure pathways and neurodevelopmental effects. The most common pathway affecting young children is lead in house dust, ingested via normal hand-to-mouth activity as children learn to crawl, absorbed into the bloodstream and carried to the

developing brain.[57] Heavily-leaded circa-1900 paint deteriorated by "chalking", causing severe lead dust hazards. Air lead fallout from leaded gasoline settled as lead contaminated dust. The use of lead in USA gasoline fell to near zero in 1987 and the USA banned the sale of lead paint in 1978, but lead paint in older homes affected many children throughout the leaded gasoline era and is still poisoning young children today. We now know that an amount of lead in dust that is invisible to the naked eye is enough to cause neurodevelopmental damage.

Almost all children born in the USA from 1880-1980 were exposed to lead-contaminated dust, but with widely varying degrees of lead

poisoning severity. Atmospheric emissions from gas lead affected blood lead even in rural areas, but urban traffic caused more severe city exposure because 55% of emissions settled within 12 miles of the roadway. Cities with population over a million had 1960s ambient air lead that was twice as high as cities with population of 250,000 to a million, and all large cities had higher air lead than suburbs and rural areas. The phase-out of leaded gasoline left little difference in urban and rural air lead, and the subsequent murder rate decline has been disproportionately due to large city trends. From 1992-2011, the murder rate fell 61% in cities with population over 500,000, 51% in cities with population of 100,000

to 500,000, and about 36% in smaller cities and rural areas.[58]

Urban and rural murder rates followed the opposite pattern in the early-1900s. Murder arrest data compiled by Monkkonen show no consistent 1900-1915 trend in the seven cities with 1910 population over 500,000 (New York City, Chicago, Philadelphia, Baltimore, Boston, Cleveland, and Saint Louis) even as the national homicide rate increased six-fold from 1901 to 1911.[59] The population-weighted average murder arrest rate in those seven cities was 7.1 times the national homicide rate from 1900-1904, but just 1.6 times higher from 1911-1915. Urbanization can only explain a very small part of the

1901-1911 increase in homicides, because rural areas accounted for 60% of the population in 1900, and 54% in 1910. Therefore, the six-fold increase in the national homicide rate from 1901-1911 was apparently due to a surge in rural homicides. Why?

The answer to that question begins with a seemingly unrelated question: Why are barns red?[60]

Professional painters in the 1800s prepared house paint by mixing linseed oil with white lead paste. About 90% of Americans lived in rural areas in the mid-1800s, and subsistence farmers could make linseed (flaxseed) oil, but few had access to white lead, so they mixed linseed oil with red rust to kill

fungi that increased wood decay. Red barns are a tradition in most USA farming regions, but white barns are the norm along the path of the old National Road. Why?

The National Road was the first federally funded highway, built from 1811-1834.[61] It had heavy traffic including Conestoga wagons, designed to carry freight, passing through farm areas from Baltimore to Illinois, close to major lead mines in Southeast Missouri. The reason the red barn tradition never took root along that path is likely because the National Road made white lead accessible to nearby farmers. USA lead output was a relatively stable 1000 to 2000 tons per year from 1801-1825, but output was 15,000 to 30,000 tons

per year from the mid-1830s through the mid-1860s, after completion of the National Road.[62]

USA refined lead output then surged from 26,000 tons in the early-1870s to almost 400,000 tons in 1904, and per capita use of lead in paint tripled from 1876 to 1898 due to advances in transportation and manufacturing. The first American patent for "ready-mixed" paint was filed in 1867. Railroads built almost 120,000 track miles from 1850 to 1900, and Sears Roebuck and other mail-order catalogs combined volume buying, rail transport, and rural free parcel post delivery to provide economical rural access to many products in the 1890s.

The murder arrest rate in large cities was more than seven times the national homicide rate from 1900-1904 because lead paint in the 1870s was available in large cities but unavailable in most rural areas. The early-1900s convergence in rural and urban murder rates was presaged by a late-1800s convergence in rural and urban lead paint exposure.

During the second half of the 20th Century, average preschool blood lead tracked trends in per capita emissions of lead in gasoline, as average exposure to lead paint in older homes changed very slowly. At the same time, lead paint and other sources of exposure often contributed to severe cases of

childhood lead poisoning because all lead exposure is additive.

The EPA has estimated baseline lead ingestion by two-year-olds during the leaded gasoline era, plus additive ingestion from interior lead paint, air in central cities (population over 50,000), and residence near a smelter.[63] This analysis showed that average dust lead ingestion caused by interior lead paint was similar to average dust lead ingestion caused by central city air lead exposure. These are just rough averages, with the severity of city air lead exposure affected by city size and proximity to highways, and lead paint exposure severity affected by housing age and condition. Circa-1900 housing had much higher

concentrations of lead in paint, with lead paint used on more interior surfaces. Lead in dust is also much more common in homes where deteriorated interior paint releases lead particles from old layers of lead paint. Dilapidated circa-1960 central city slum housing, built around 1900, posed extremely severe lead dust and lead paint chip ingestion risks.

The EPA analysis also showed that lead ingestion was orders of magnitude higher for children living near a lead smelter. The homicide impact of smelters and other industrial lead emissions was demonstrated by the Stretesky and Lynch analysis showing that counties with high 1990 air lead (from industrial emissions, after

the phase-out of leaded gasoline), had 1989–1991 murder rates that were four times higher than counties with low air lead, after controlling for nine air pollutants and six sociological factors. My 2007 study suggested that this finding likely reflected 1970s additive lead exposure, because the homicide rate would have fallen earlier (as air lead fell over 70% from 1975–1984) if murders were affected by contemporaneous air lead exposure. Most 1990 lead-emitting industrial facilities had been in operation for many decades, in areas with older housing and some traffic, so 1989–1991 county murder rates reflected higher 1970s preschool blood lead where children had additive

exposure to lead in paint, gasoline, and industrial emissions.

The local impact of smelters was also evident in the murder rate trend for one large city in the early-1900s. Saint Louis was the epicenter of the "Lead Belt" in the late-1800s, when St. Joseph Lead became one of the world's largest lead producers. St. Joe began operations at its first smelter in 1892 in Herculaneum, about 25 miles from St. Louis, and that smelter was still one of the largest in the world in the 1990s. USA refined lead output doubled from 1892 to 1904, and the St. Louis murder arrest rate increased 10-fold from 1910 to 1916, about 20 years after smelting operations began in Herculaneum.

Fig. 24: 1992-2011 USA Murder Rate Trends by City/Suburban/Rural Community Type

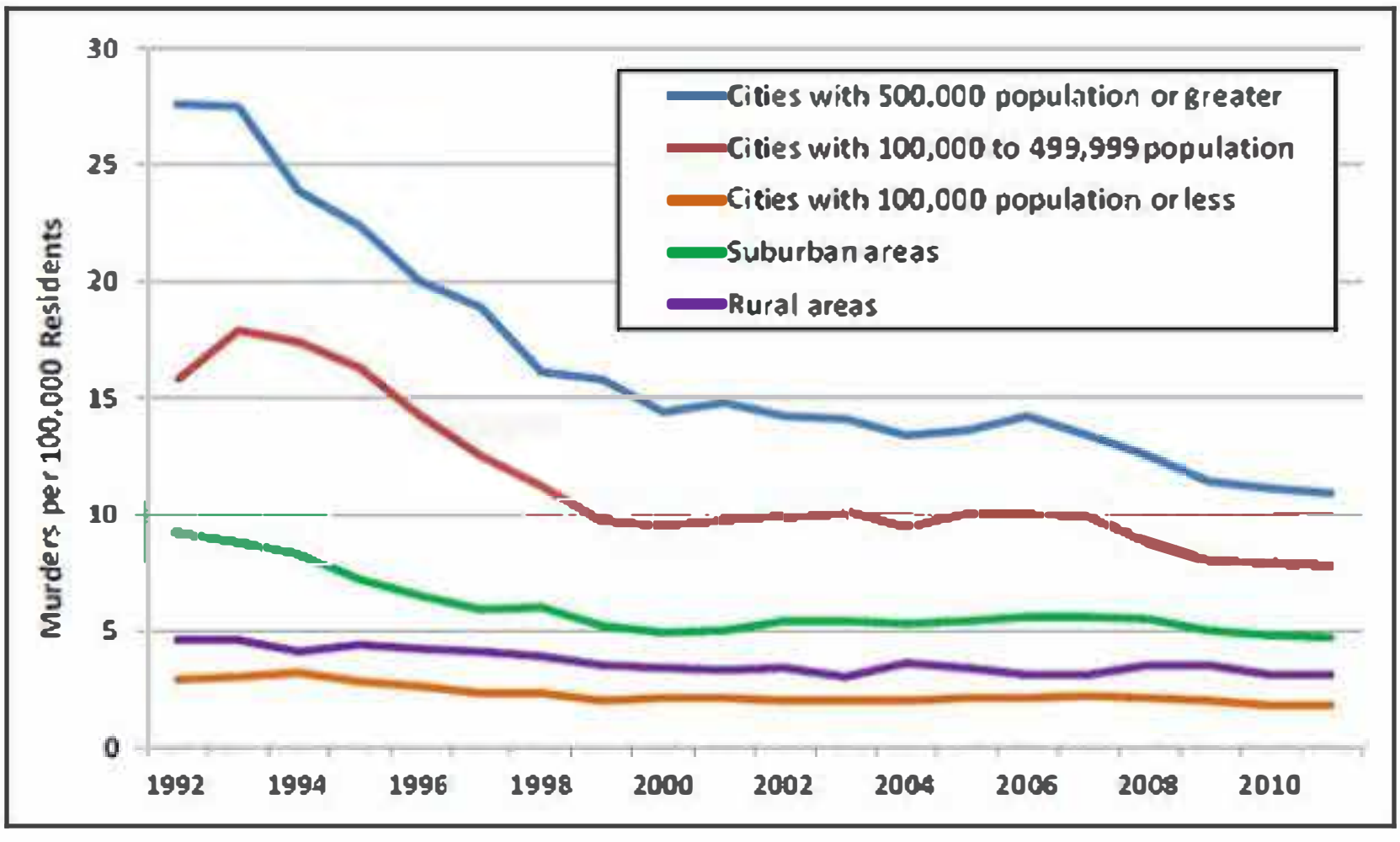


Fig. 25: Leaded Gasoline Era Average Daily Lead Ingestion by Two-Year-Olds with Baseline and Additive Lead Exposure

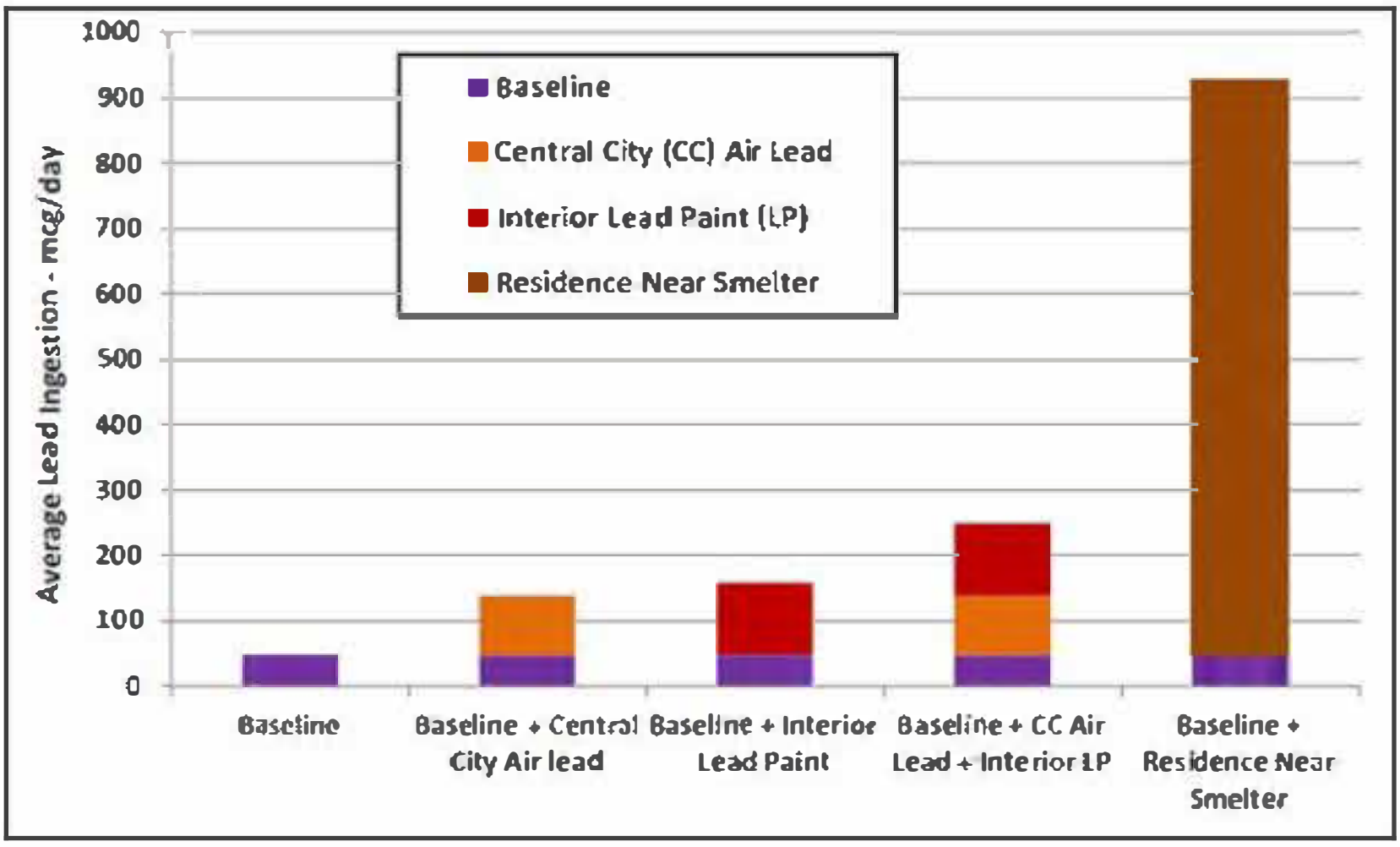
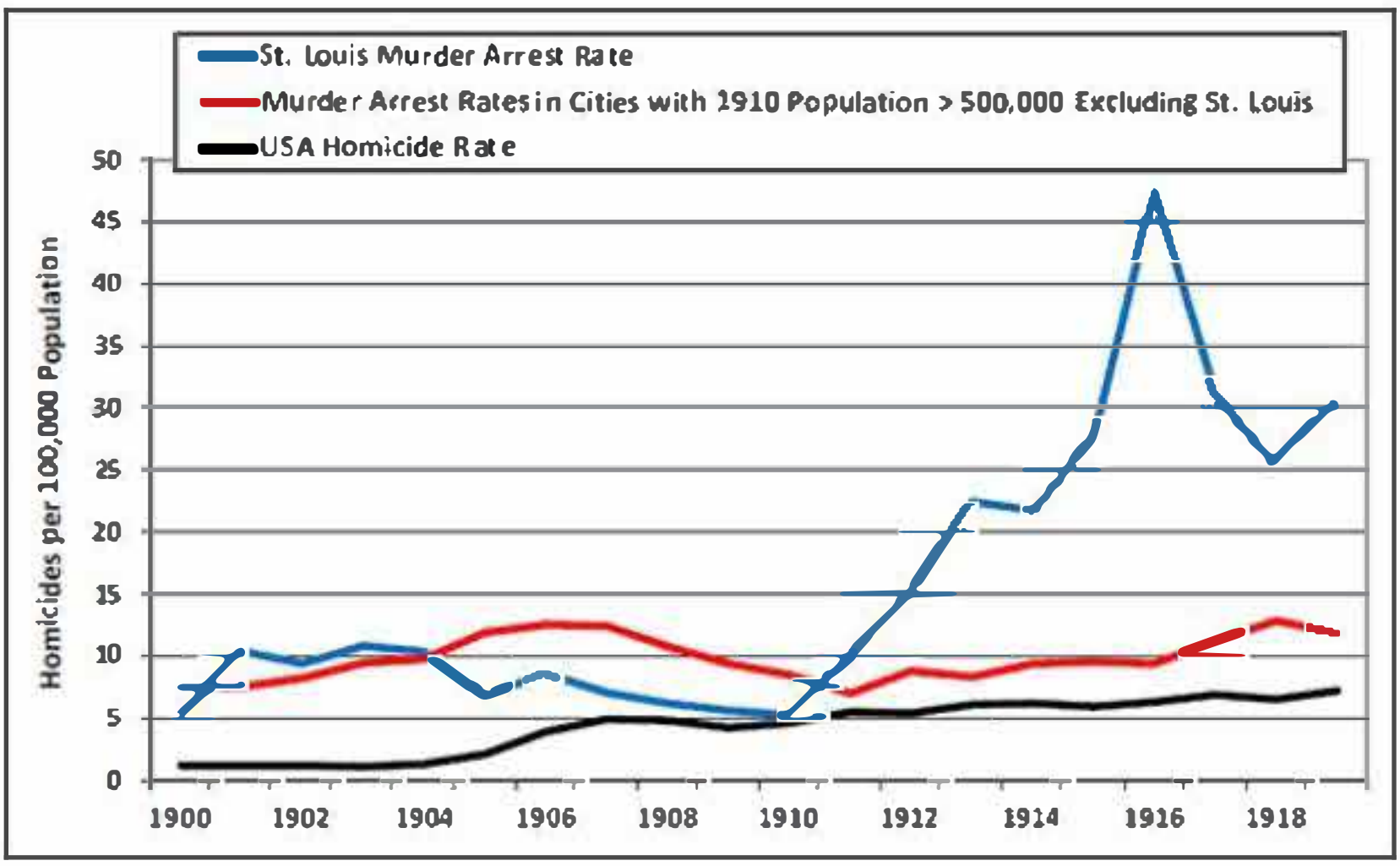


Fig. 26: 1900-1919 Trends in USA Homicide Rate and Large City Murder Arrest Rates



The “biological plausibility” of

neurodevelopmental damage caused by lead poisoning has been documented by Lidsky and Schneider in the journal *Brain*,[64] and summarized for a broader audience in a 2014 article by Wolf in *Chemical & Engineering News*.[65] Wolf observes that lead poisoning "wreaks a lot of havoc on the central nervous system. So pinpointing one - or even a few - molecular switches by which the heavy metal turns on aggression has been challenging." The impact on gray and white matter development is well known, however, with important implications for Supreme Court death penalty decisions.

In *Roper v. Simmons* (2005), the

Supreme Court held that execution is an excessive sanction for crimes committed by youths under 18, citing the "susceptibility of juveniles to immature and irresponsible behavior" indicative of "diminished culpability". An American Psychological Association (APA) *Roper* brief also cited magnetic resonance imaging (MRI) evidence of a "biological dimension to adolescent behavioral immaturity":

> "The frontal lobes, especially the prefrontal cortex, play a critical role in the executive or "CEO" functions of the brain... involved when an individual plans and

implements goal-directed behaviors.... Neurodevelopmental MRI studies indicate this executive area of the brain is one of the last parts of the brain to reach maturity.... MRI research reveals that...white matter significantly increases during adolescence [as] a substance called myelin is wrapped around brain cell axons. Myelination improves the connectivity of neural tracts by insulating the axon thereby greatly speeding up the communication between cells, allowing the brain to process

> information more efficiently and reliably."[66] (APA, 2004)

The APA *Roper* brief highlighted research by Sowell showing a gray matter growth surge around puberty, followed by white matter growth that improved connections between neurons, with scans at ages 12-16 compared to ages 23-30 showing a large difference in frontal lobe white matter.[67] The APA cites this as evidence of brain development affecting juvenile offending, but Bartzokis has also reported MRI evidence of frontal lobe white matter growth to age 50.[68] He explains: "What keeps growing is the myelin...It literally allows your brain to work in

concert; you're not as prone to impulse".[69] White matter growth to age 50 suggests that improvements in brain function could explain why arrest rates decline with age well beyond adolescence.

Moffitt has described how Adolescence-Limited offenders account for most juvenile crime and most property crimes, but some youths go on to become more violent Life-Course-Persistent offenders.[70] Even among chronic offenders, Sampson and Laub found that offense rates rose sharply after age 10; property offending peaked in adolescence and fell almost 90% by the mid-20s; and violent offending peaked in the

early-20s and fell through age 50.[71] These patterns are consistent with offending being affected by the gray matter growth surge around puberty, rapid white matter growth through the mid-20s, and ongoing white matter growth to age 50.

The APA *Roper* brief also stated that violent offense rates "build steeply to age 18 and then fall" - but that claim is less true with every passing year. In 1991, the violent crime arrest rate peaked at the age of 17, but in 2014 the arrest rate peaked in the early-20s. In 1991, the age 17 violent crime arrest rate was 1.7 times higher than the arrest rate for ages 25-29, but in 2014 the age 17 arrest rate was slightly lower than the age 25-29 arrest rate. In 1991, the violent crime arrest rate for

ages 10-14 was 2.3 times higher than the arrest rate for ages 50-54, but in 2014 the arrest rate for ages 10-14 was 35% lower than the arrest rate for ages 50-54. If brain function affects crime, then arrest rate trends by age suggest that brain function from 1991 to 2014 improved for youths but deteriorated for older adults. Why?

MRI studies by Brubaker,[72] Cecil,[73] and other University of Cincinnati researchers have shown that preschool lead exposure impairs the same types of brain development linked to "diminished culpability" in *Roper*.

> "One set of scans found that lead exposure is linked to production of the

brain's white matter - primarily a substance called myelin, which forms an insulating sheath around the connections between neurons. Lead exposure degrades both the formation and structure of myelin, and when this happens, says Kim Dietrich, one of the leaders of the imaging studies, "neurons are not communicating effectively." Put simply, the network connections within the brain become both slower and less coordinated.... A second study found that high exposure to lead during

> childhood was linked to a permanent loss of gray matter in the prefrontal cortex - a part of the brain associated with aggression control." (Drum, 2013)

These findings provide a new perspective on a 1997 study by Raine comparing brain scans for murderers and matched controls, showing that murderers had dysfunction in the prefrontal cortex and corpus callosum.[74] The corpus callosum is the largest white matter structure in the brain, facilitating communication between cerebral hemispheres. Raine's study provided "the first direct evidence supporting the long-held notion that dysfunction

in the corpus callosum may cause a predisposition to violence", and noted that a lack of hemispheric integration could "contribute to the abnormal asymmetries of function...previously observed in antisocial and violent groups". A 2009 meta-analysis of 43 imaging studies by Yang and Raine also found "significantly reduced prefrontal structure and function in antisocial individuals".[75]

In discussing his book, *The Anatomy of Violence*,[76] Raine mentions research showing that lead exposure trends explain more than 90% of violent crime variation since the 1960s. He explains that toddlers "putting their fingers in their mouths and absorbing the

lead...became the next generation of violent criminal offenders", and concludes: "to me, it's the only single cause that can both explain the precipitous rise in violence from the '70s, '80s and '90s and also the drop that we've been experiencing".[77]

Racial disparities in the criminal justice system have also tracked earlier racial disparities in lead exposure. The phase out of leaded gasoline substantially reduced lead exposure racial disparities, because black children were disproportionately concentrated in central cities, but we have not eliminated racial disparities because black children are still more likely to live in older homes with lead paint hazards. National

blood lead surveys show that the percent of young black children with blood lead over 10 mcg/dl fell from 18.6% in 1988-1991 to 11.2% in 1991-1994, as the percent of white children over 10 mcg/dl fell from 5.5% to 2.3%. The percent of black children over 5 mcg/dl then fell from 18.5% in 1999-2002 to 5.6% in 2007-2010 as the percent of white children over 5 mcg/dl fell from 7.1% to 2.4%.

National blood lead survey samples are not large enough to provide reliable estimates of the percent of children with more severely elevated preschool blood lead, but CDC surveillance data show both the progress toward eliminating severe lead poisoning, and the extent of ongoing racial disparities.

CDC surveillance recorded blood lead test results for about 7% of all children ages 1-5 in 1997, 11% in 2001, and 18% of all children ages 1-5 in 2010.[78] Despite the large increase in the percent of children tested, the total number of children with blood lead tests above 25 mcg/dl fell from 13,063 in 1997, to 7,574 in 2001, and 2,762 in 2010. The number of children with surveillance test levels of 15–24 mcg/dl also fell from 41,785 in 1997 to 7,737 in 2010. CDC surveillance data also show that black children were 5.7 times more likely than white children to have 1997 blood lead tests above 25 mcg/dl (2.06% of black children tested, versus 0.36% of white children tested), and black children were 4.5 times more likely

to have 2001 blood lead surveillance tests above 25 mcg/dl (0.85% of black children tested, versus 0.19% of white children tested).[79] In 2001, black children were also 4.3 times more likely than white children to have blood lead surveillance tests of 15–24 mcg/dl.

Racial disparities in lead poisoning were more severe in the late-1950s and 1960s, when black children were far more likely to live in substandard city housing. A Department of Commerce analysis of 1970 preschool blood lead data found that 25% of city children tested had blood lead levels over 40 mcg/dl and 95% of Census tract variation in the percent of city children over 40 mcg/dl was

explained by Census tract variation in substandard housing prevalence.[80] This suggests that severe lead poisoning was even more common in the late-1950s and early-1960s, before urban renewal projects demolished a large amount of urban slum housing.

African Americans accounted for 15% of central city households in 1960, but occupied 56% of substandard central city housing, and were disproportionately displaced by slum clearance projects in the late-1950s and 1960s. Per capita lead emissions were relatively stable in those years, as urban sprawl spread more gas lead fallout to predominantly white suburbs that had much less traffic in the early-1950s. My 2009 study

suggested that those trends likely caused severe lead poisoning for black children to peak in the early-1960s, before the early-1970s peak in lead emissions. That pattern has since been confirmed by a study in Cleveland.

Robbins found that lead concentrations in tooth enamel formed in early childhood from 1936 to1993 in Cleveland peaked in 1960-1975 at five times the levels in teeth formed in 1936–1950 and 1986–1993.[81] Tooth lead concentrations for this urban and predominantly (86%) black population were found to be highly correlated with available blood lead data for black children in cities, and supported an estimated 48 mcg/dl average blood lead among

black children in central cities from 1960-1975. The most severe lead poisoning cases were found in tooth enamel lead concentrations associated with birth years in the early-1960s.

These findings are consistent with statements from public health experts who have reported: "In the 1960s many inner city hospitals had large numbers of comatose and convulsing children with lead poisoning, with fatality rates of 5–28%".[82] Convulsions and coma are associated with preschool blood lead above 70 mcg/dl.

My 2009 study showed that an early-1960s peak in severe lead poisoning among black children, before the early-1970s peak in lead

emissions, can also explain male incarceration trends by age and race from 2000-2006.

> "Declining incarceration rates from 2000 to 2006 for males under 30 reflect declining preschool blood lead since the mid-1970s. Incarceration rates are still rising for those over age 40, born when childhood lead poisoning was epidemic. The overall age 30–39 male incarceration rate rose slightly from 2000 to 2006, but the age 30–39 rate for black males fell 12%, reflecting slum clearance birth years". (Nevin, 2009)

Those patterns are still evident in recent male incarceration trends, but the transition to rising incarceration has shifted to older age groups as the birth years of peak lead poisoning recede into the past. Declines in male incarceration rates from 2001-2014 extend through ages 35-39, and the percentage declines have been larger for black males born across birth years when black children had steeper declines in lead poisoning.

From 2007-2014, the incarceration rate only increased for men over 45. White males had a larger percent increase for ages 45-54, reflecting birth years over the 1960s when urban sprawl increased lead

fallout in suburbs. Black males had a larger percent increase for ages 55-64, reflecting birth years before 1960 when black migration to cities resulted in more black children living in city slums.

Slum demolition was criticized for displacing residents who could not find other affordable housing, but one profound benefit was the eradication of severe lead paint hazards in dilapidated housing with heavily-leaded circa-1900 paint. Ironically, another legacy of urban renewal was the effort to provide affordable apartments for displaced slum residents in high rise public housing often located beside highways built on slum clearance land, causing severe lead poisoning from the near-fallout

beside highways.

Leaded gasoline caused especially high air lead and severe lead dust hazards near highways because about 10% of emissions settled within 100 meters of the road. In the 1960s, ambient air lead in Chicago was 33% higher than in Cincinnati, but air lead measured beside Cincinnati streets with heavy traffic was about 15 times higher than Cincinnati's ambient air lead. My 2007 study used this data to illustrate the severity of air lead fallout in the long narrow Robert Taylor Homes housing project that was all within about 400 meters of Chicago's Dan Ryan expressway. The air lead beside Cincinnati streets with heavy traffic referred to air lead from

2150 cars per hour. If that traffic continued for 24 hours, then that would be 51,600 cars per day. Traffic on the Dan Ryan was 150,000 vehicles per day in 1963. Lead poisoning caused by that traffic had murderous consequences by 1980, when Robert Taylor Homes accounted for 0.5% of Chicago's population, and 11% of Chicago murders.

African-Americans now account for almost 40% of state and federal prison inmates and 42% of all prisoners on death row. That racial disparity is the causal effect of housing segregation that resulted in African-Americans occupying 56% of substandard central city housing in 1960, and accounting for 48% of all preschool children with

blood lead over 30 mcg/dl in the late-1970s.

The *Roper* decision reaffirmed: "Capital punishment must be limited to those offenders who commit a narrow category of the most serious crimes and whose extreme culpability makes them the most deserving of execution." We now know that preschool lead exposure impairs the specific types of brain development linked to "culpability" in *Roper*; Raine's research links those types of brain impairment to homicide offending; murder rate trends by race have tracked racial disparities in lead poisoning; and the USA homicide rate and murder trends by city size have tracked lead exposure trends from 1900 to 2014. Over 90% of all

death row prisoners are ages 30 to 65, with birth years spanning the rise and fall of leaded gasoline, and the peak years of severe lead poisoning in city slums. The known history of murder rate trends and lead exposure impacts clearly cast doubt on our ability to judge the extreme culpability of any specific offender.

Fig. 27: 1991-2014 Shift in USA Violent Crime Arrest Rates by Age Group

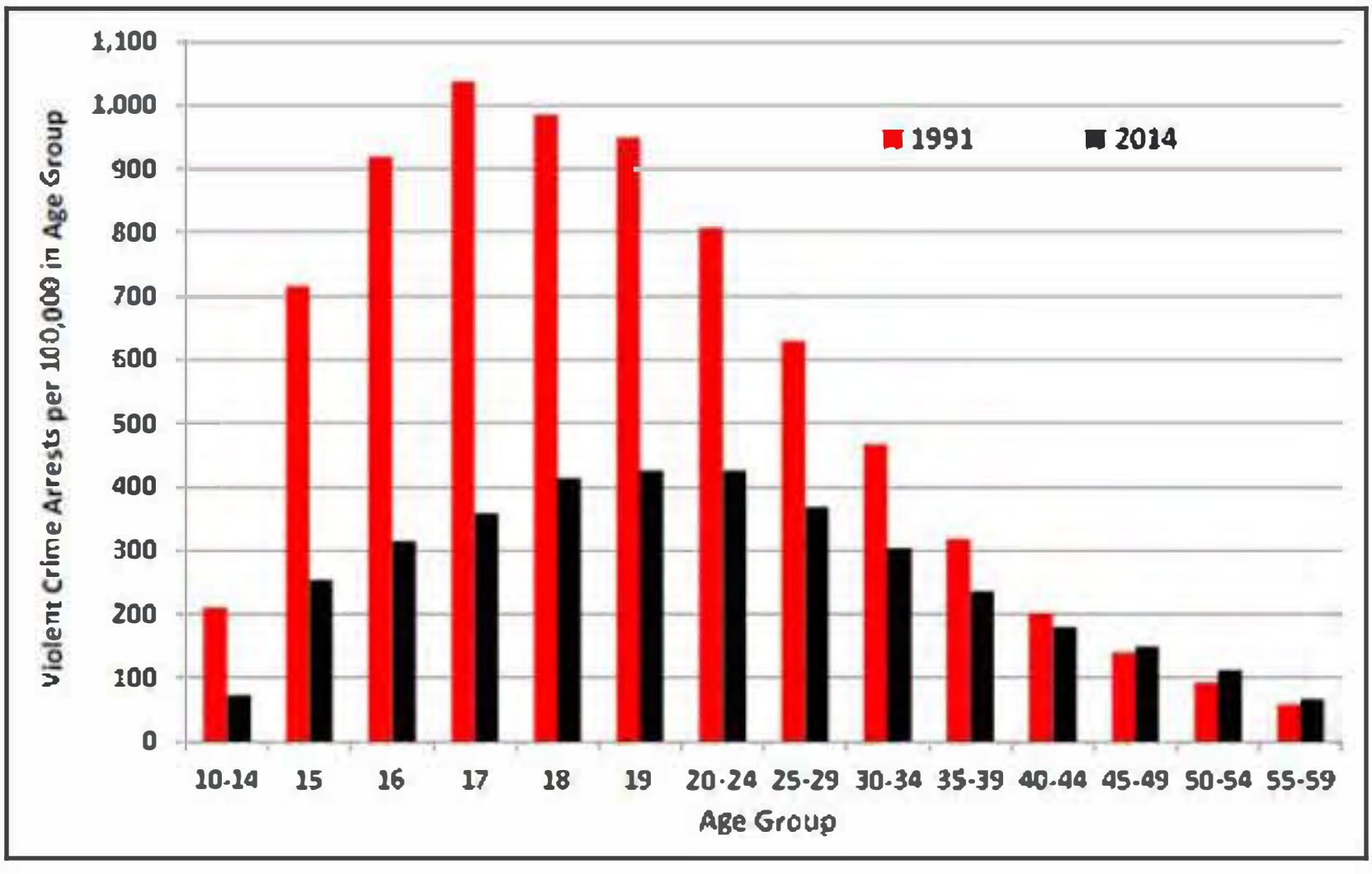


Fig. 28: 1988-2010 Black vs. White Elevated Preschool Blood Lead Prevalence

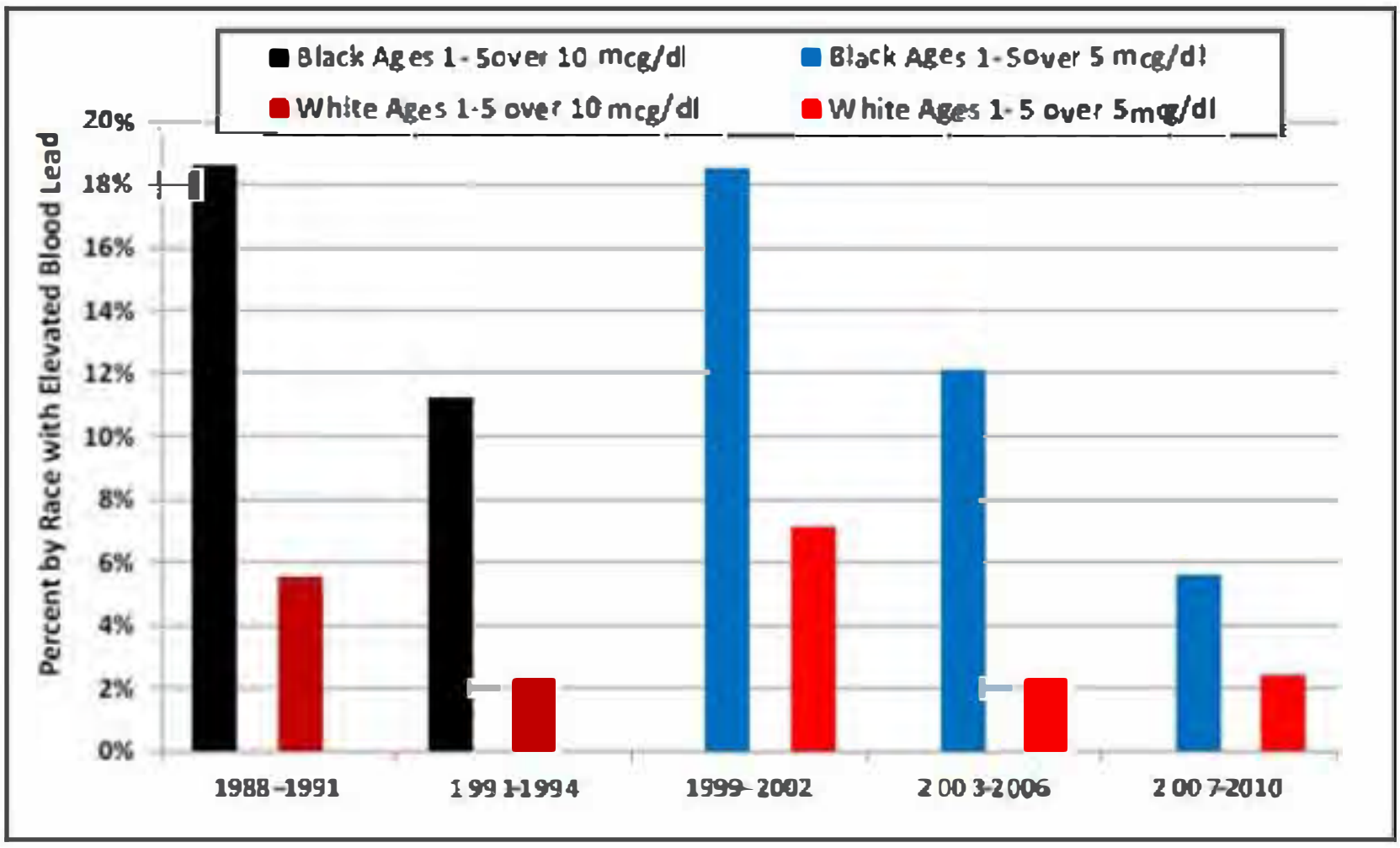


Fig. 29: 1951-1970 Trends in Urban Renewal Projects and Per Capita Use of Lead in Gasoline

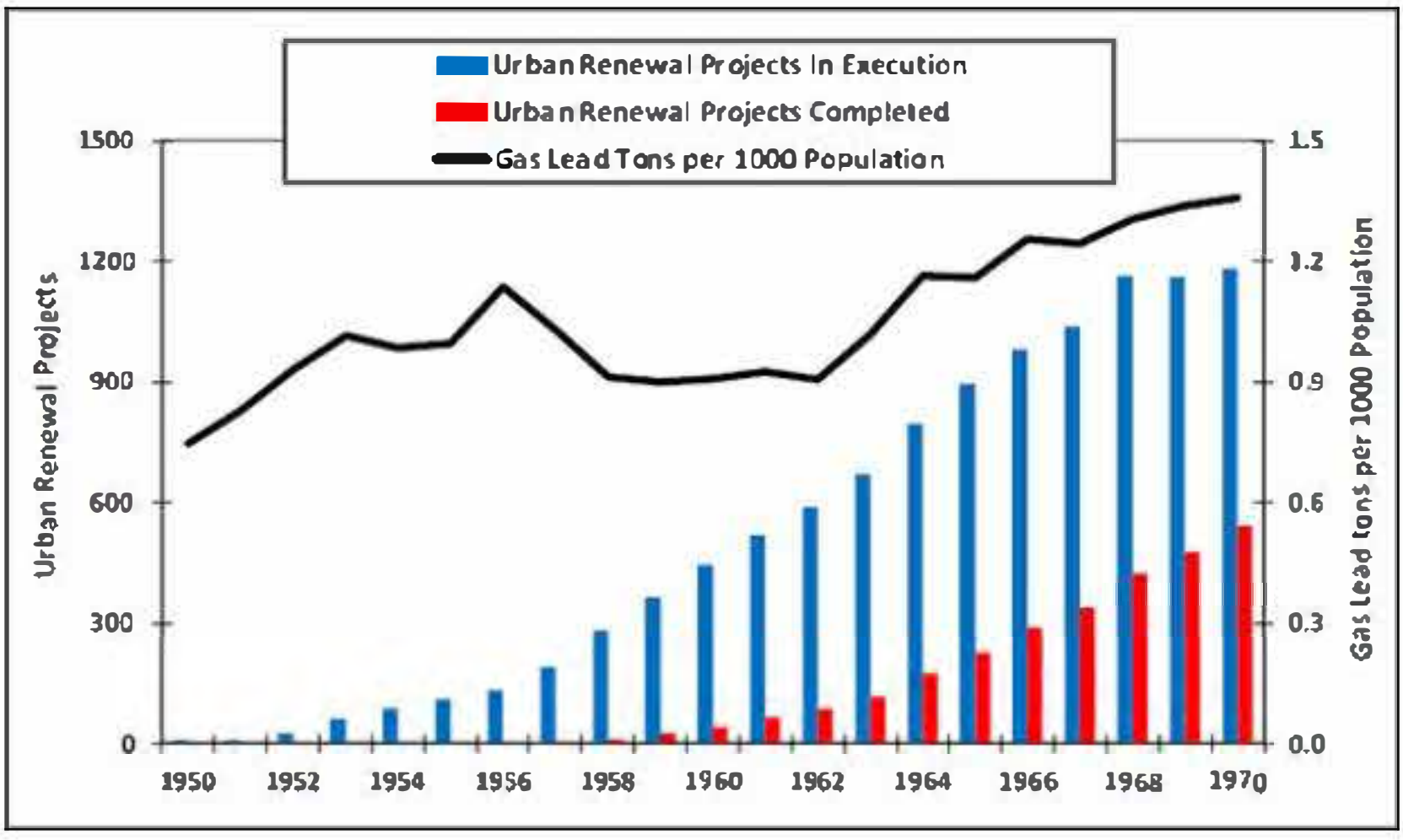


Fig. 30: 2001-2014 Percent Change in Age 18-44 Male Incarceration Rates by Age and Race

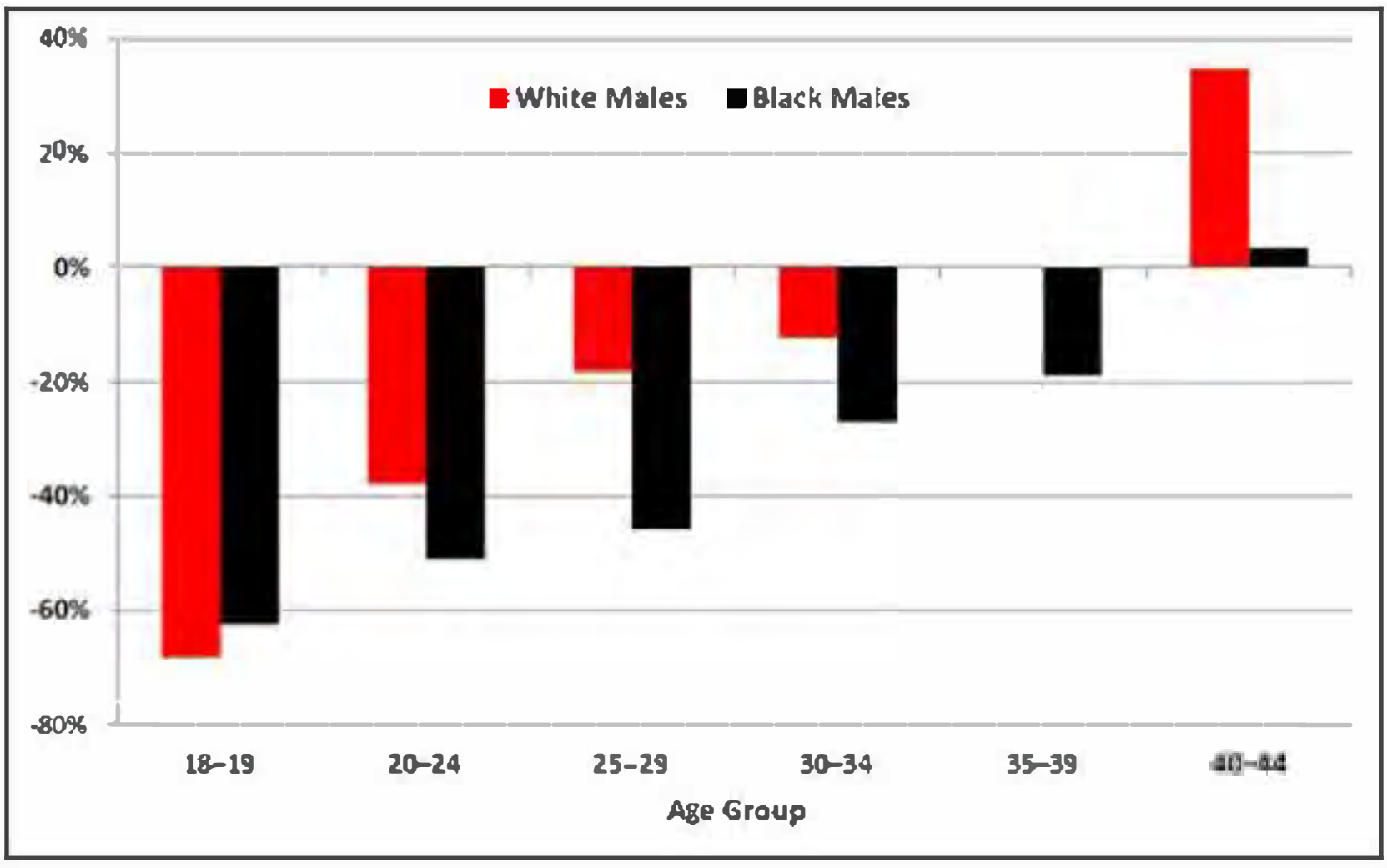
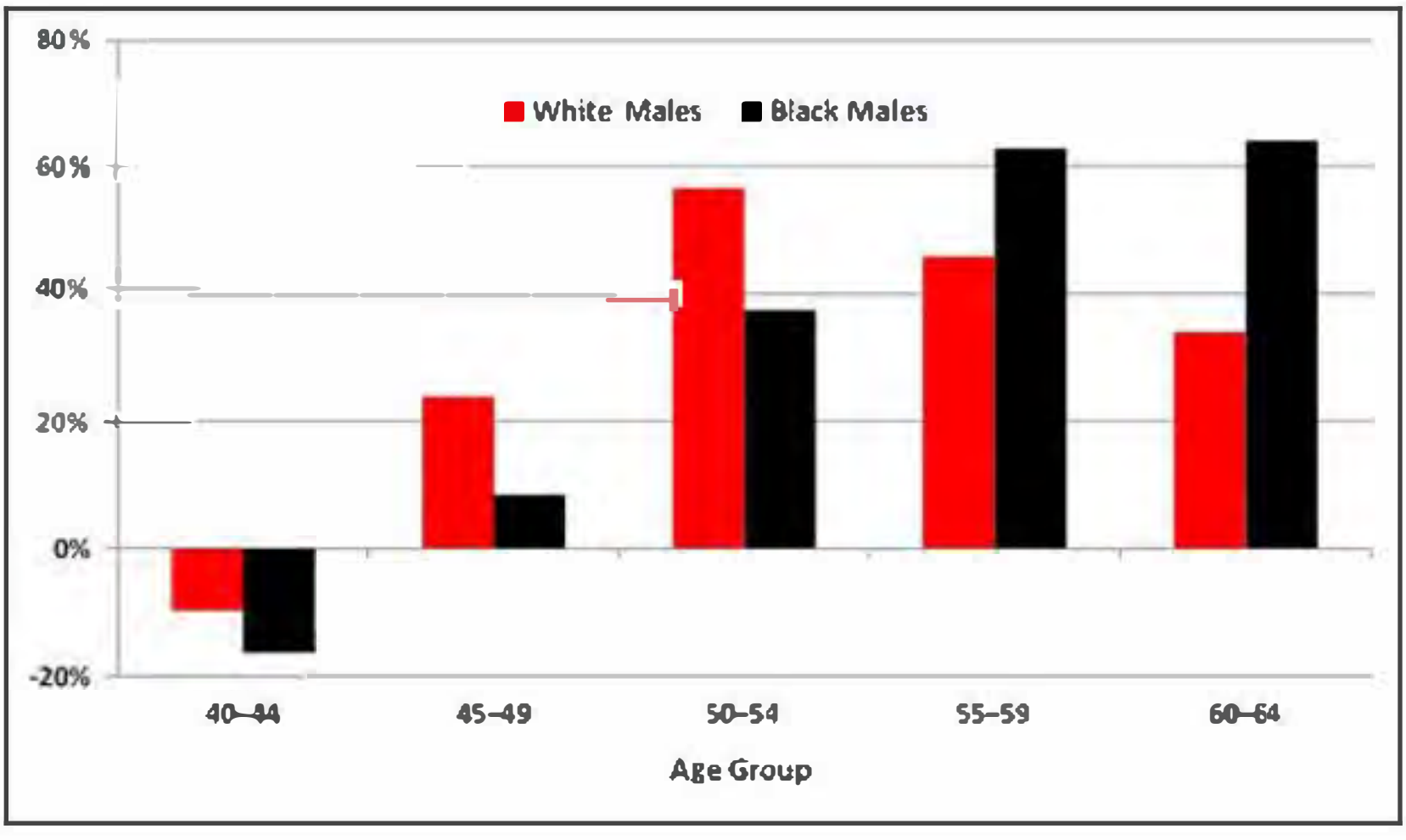


Fig. 31: 2007-2014 Percent Change in

Age 40-64 Male Incarceration Rates by Age and Race



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V. Time-Precedence and the Dawn to Dusk of Delinquency

The time-precedence indicator of causation requires, at a minimum, that the suspected cause precede the effect. The statistical best-fit time lags relating trends in lead exposure with crime and unwed pregnancy trends present an especially compelling case of time precedence because they are all consistent with neurobehavioral damage in the first years of life. The surge in youth crime over the 1960s and plummeting youth arrest rates since the early-1990s present more time-precedence evidence,

because younger age groups would be the first to show the neurobehavioral effects of both rising and falling trends in preschool lead exposure.

In response to reader questions about the October 2015 Coates article, *The Atlantic* published a follow up on whether lead poisoning contributed to mass incarceration, citing my research and a 2002 Cook and Laub study that questioned my 2000 study findings.[83] Cook and Laub focused on black male homicide *victimization* rates to show that violence fell across several age groups over the 1990s,[84] but lead exposure trends are expected to affect o*ffending* by age, not

victimization by age. There has been a historical correlation between offending and victimization by age, but the fall in lead exposure has weakened that correlation: From 1993-2013 there was a 73% decline in juvenile homicide offenders,[85] but only a 57% decline in juvenile victims,[86] as the percent of juvenile victims killed by an adult increased from 51% in the early-1990s to 66% in 2010-2013. Cook and Laub also made the following important statement about how further research could help to answer their questions:

> "Given the volatility in the rates of juvenile violence, forecasting rates is a risky

business indeed.
Effectively narrowing the range of plausible explanations for the recent ups and downs may require a long time horizon, consideration of a broader array of problem behaviors, and comparisons with trends in other countries."

(Cook/Laub, 2002)

That was a perfectly reasonable statement about plausible explanations for USA juvenile crime trends in 2002, but research since 2002 has addressed the additional perspectives suggested, and the evidence all points to lead exposure as the key determinant of

crime trends. My 2007 study provided "comparisons with trends in other countries" and found that lead exposure trends explained violent and property crime trends around the world. With respect to "consideration of a broader array of problem behaviors" the relationship between lead exposure and unwed youth pregnancy rates has now demonstrated remarkable ongoing strength and predictive power.

My 2007 study also provided the following "long time horizon" perspective on property crime arrest rates by age: "The overall USA property crime rate was about the same in 1970 and 2003, but the property crime arrest rate for youths under age 15 fell 45% from

1970–2003...and the arrest rate for adults over 24 rose 58%."

The 1970-2003 drop in the under-15 property crime arrest rate compares young juveniles in 1970, born during the mid-1950s surge in gasoline lead emissions, versus their counterparts in 2003, born after the 1980s phase out of leaded gasoline. The 1970-2003 increase in the over-24 arrest rate compares adults in 1970, born before the post-WWII rise in gas lead, versus their 2003 counterparts born before the 1980s fall in lead emissions.

In 2014, the under-15 property crime arrest rate was down 80% compared to 1970, and down 69% compared to 1960. The under-15

property crime arrest rate fell to a new record low every year from 2000 through 2014. Forecasting the juvenile crime rate has become much less risky over recent years – it just goes down.

Now, let's consider an even longer time horizon.

The "Dutch process", producing white lead in large batches, began to spread across Europe in the 1700s, and the use of white lead in paint surged in the early-1800s with the dawn of the industrial revolution. Pulsifer observed that white lead manufacture in the late-1700s "must have assumed considerable importance in Great Britain, as several patents were granted for improvements in the

old process and for its manufacture by new methods."[87] In 1810, Britain produced 12,500 tons of lead - more than the rest of Europe combined. British output then surged to 46,000 tons in 1845 and peaked at 73,000 in 1856, before falling to 54,000 in 1873.

In 1816, following the late-1700s flurry of white lead patents, the English language recorded its first known use of the term "juvenile delinquency".[88] King explains that juvenile crime was not thought to be a "particularly threatening problem" in the 1700s, but had become a "major focus of anxiety" in England by the mid-1800s.[89] Magarey called this the "invention of juvenile delinquency",[90] but

King found that this perspective was based on national data from the 1830s and 1840s, and earlier local records show that the juvenile crime surge was quite real. That crime wave began in large cities in the 1810s and 1820s, creating a large 1820s urban-rural difference in juvenile crime, but then rural juvenile crime rates surged after 1825 and had largely caught up with city juvenile crime rates in the 1840s.

In London, the percentage of property crime offenders who were juveniles nearly doubled from the 1790s to the early-1820s. Total nationwide arrests also tripled from 1805 to the early-1820s, driven by the juvenile crime surge in cities. In the 1820s, juveniles

accounted for 22% to 34% of property crime arrests in large cities but only 4% to 8% of property crime arrests in rural areas. By the mid-1840s, the percent of property crimes committed by juveniles was roughly the same in cities (30%) and rural areas (28%).

The early-1800s juvenile crime surge in Britain, like the early-1900s USA homicide surge, was presaged by a surge in white lead output. The early-1800s juvenile crime surge in Britain was concentrated in cities where white lead was widely available after 1800. The 1840s convergence of urban and rural juvenile arrest rates in Britain, like the USA 1901-1911 convergence of urban and rural murder rates, was presaged by a

transportation revolution that made white lead available in rural areas. Bogart and Majewski show that Britain enacted many improvements to roadways and canals from 1800-1830, enabling freight transport to rural areas.[91]

The amount of lead used in European paint declined over the second half of the 1800s as zinc pigments became available amid increasing concern over occupational lead poisoning risks. Orfila became a leading figure in the Paris medical community in the 1830s after publishing a book on toxicology in 1818 that included warnings about the causes of lead poisoning, a.k.a. “painter’s colic”.[92] In 1839, Tanquerel des Planches

published a study of 1,207 persons with lead colic in France, showing two-thirds were painters or lead pigment manufacturing workers.[93] Osmond states that French concern about white lead health risks and French efforts to produce safer zinc pigments date from 1780, and "production in the 1840s then facilitated a rapid and wider adoption of zinc oxide to protect the health of French pigment manufacturers and painters".[94]

That early concern about white lead presaged a large crime decline in France over the 1800s: "Violent crimes declined from 6 per 100,000 inhabitants in 1826 to 4.3 in 1913, while property crimes precipitated from about 15.9 to 3.4 per 100,000

over the same period".[95] The property crime rate decline was especially steep from the mid-1850s through the 1860s (from about 15 to 6 per 100,000), after zinc pigment output surged in the 1840s.

In Britain, an 1825 painter's manual described symptoms of severe lead poisoning, "known in some parts of England by the name of the painter's colic".[96] In 1831, Thackrah published his influential work on the effects of "Arts, Trades and Professions . . . on Health and Longevity", noting his wish to "diminish the ill health of plumbers, painters...[and] the far greater misery and mortality of the manufactures in which white lead is prepared".[97] Charles Dickens,

better known for his writing in *Oliver Twist* about delinquents in 1830s London, also described the suffering of workers in London's white lead mills in 1868: "Sure 'tis the lead-mills...and 'tis lead-pisoned she is, sur...and her brain is coming out at her ear, and it hurts her dreadful".[98] In 1897, an Illustrated London News advertisement for "Aspinall's Enamel" emphasized that this product was "NOT MADE WITH LEAD" and was "NON POISONOUS", showing broad awareness of lead paint poisoning risks in late-1800s Britain.

Himmelfarb states that births to unmarried women in Britain fell from 7% of all births in 1845 to less than 4% by 1900, and the indictable

offense rate fell by almost 50% from 1857 to 1901, which she attributes to "Victorian virtues".[99] Vickers and Ziebarth recently discovered another piece of that late-1800s crime decline puzzle: "The fraction of offenders over 40 among male offenders convicted of economic crimes increases from 18.9% in the 1860s to 35.4% in the 1890s", and the average age of men accused of simple larceny increased by seven years.[100] In other words, the steep decline in late-1800s offending in Britain was associated with even steeper declines in juvenile and young adult offending.

Himmelfarb notes that Britain maintained a low crime rate through the early-1950s, but crime

began a "dramatic rise" starting in the mid-1950s. My 2007 study highlighted 1958-1997 shifts in British caution and conviction (arrest) rates by age for indictable offenses, showing that the latest crime wave in Britain began with a new generation of *Oliver Twist* delinquents:

> "Age-14 British males had the highest caution and conviction rate for indictable offenses in 1958, but peak offending shifted to age 18 by 1997. The age-10 offense rate fell 70% from 1958–1997, as age 18–29 offending rates increased three to five-fold. Males ages 12–14 in

1958, born as gas lead exposure rose after World War II, had higher offending rates than older teens born before that rise in lead exposure. By 1997, offending declined relative to 1958 only for males under 14, born after the mid-1980s fall in British gas lead use, while offending rates rose for older teens and adults born over years of rising gasoline lead use." (Nevin, 2007)

In 1986, Britain lowered the maximum lead content of gasoline from 0.4 to 0.15 grams per liter and began the sale of unleaded

gasoline.[101] Unleaded gas grew to 19% of total gasoline sales in Britain by 1989. In 2016, Britain's Ministry of Justice reported that juvenile arrests fell 73% from 2006/2007 through 2014/2015.[102]

In Canada, the overall index crime rate fell by 18% from 1998-2008. Over those same years, the juvenile index crime arrest rate fell by 36% as the adult arrest rate fell by just 6%.[103] In the past decade, Canada introduced their "crime severity index" that takes into account both the change in the volume of crime and the relative seriousness of crimes committed. From 2007 to 2014, the Canada youth crime severity index fell by 41%.

From 2008/09 to 2014/15, Australia

burglary arrest rates fell 46% for ages 10-19 and 33% for ages 20-29, and robbery arrest rates fell 35% for ages 10-19 and 25% for ages 20-29.[104] Over those same years, Australia burglary arrest rates increased 26% for ages 45-49 and 37% for ages 50-59, and robbery arrest rates increased 18% for ages 45-49 and 27% for ages 50-59.

From 1994 to 2014, New Zealand index crime arrests fell 42% for ages 14-16, 38% for ages 17-20, and 18% for ages 21-30.[105] Over those same years, New Zealand index crime arrests increased by 28% for ages 31-50 and 96% for those over age 50.

All over the world, the sun is setting on the era of juvenile

delinquency. Remember what this era was like, because your grandchildren are going to love to hear you tell the story about how teenagers were dangerous in the olden days.

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VI. Coherence and the Bell Curve

The Bell Curve highlights the historic significance of 1904 as the year when Spearman "made a conceptual and statistical breakthrough that has shaped both the development and much of the methodological controversy about mental tests ever since." His statistical breakthrough was to use correlation coefficients to measure the extent to which individual test takers achieved similar scores on different intelligence tests. His conceptual breakthrough was to argue that the high correlation of individual scores on different tests

suggested an underlying common factor, which Spearman named g, for general intelligence.[106]

The controversy over intelligence theory was also profoundly affected by the 1904 publication of Galton's article on "Eugenics: Its Definition, Scope, and Aims".[107] He defined "eugenics" as the science that "deals with all influences that improve the inborn qualities of a race; also with those that develop them to the utmost advantage." That definition set the benign tone for his article, which sought "to secure the general intellectual acceptance of eugenics as a hopeful and most important study" and to "let its principles work into the heart of the nation, which will

gradually give practical effect to them in ways that we may not wholly foresee."

The year of 1904 was also when Binet was appointed to a commission in France, charged with developing a test to identify mentally retarded children.[108] That test was soon translated and distributed to schools in America, by psychologists who embraced Galton's theory of inherited intelligence and saw Binet's test scores as a reliable measure of that birthright.[109] While Galton encouraged "useful classes...to contribute more than their proportion to the next generation", the American eugenics movement he inspired resulted in state

sponsored forced sterilization of the mentally retarded. Nazi Germany later adopted that strategy and then extended their eugenics program to include involuntary euthanasia,[110] giving practical effect to Galton's hopeful principles in ways that he could not wholly foresee.

On the other side of the world, another article published in 1904 was the first scientific study of a childhood hazard that has also shaped the debate over mental tests ever since, unbeknown to experts in cognitive research. Gibson was the author of this *Australasian Medical Gazette* article entitled: "A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Amongst

Queensland Children".[111] His study was the first to identify chalking lead paint as the cause of life-threatening childhood lead poisoning.

Almost a century after the landmarks of 1904, the Supreme Court in *Atkins v. Virginia* (2002) held that executing the mentally retarded is an excessive sanction because their "diminished capacities" diminish their "personal culpability". The reasoning and precedent of *Atkins* was cited as the basis for the *Roper* decision. *Atkins* acknowledged "disagreement about...which offenders are in fact retarded" but noted that "clinical definitions of mental retardation require not only subaverage intellectual

functioning, but also significant limitations in adaptive skills".

Since the early-1900s, IQ tests have been used to diagnose intellectual limitations associated with mental retardation (MR). IQ tests measure individual IQ relative to the raw test scores recorded by a representative sample of test-takers, called the IQ test's "norm sample". Average IQ of 100, by definition, corresponds to the average norm sample score. A statistical method calculates individual IQ relative to norm sample test scores in a way that creates a bell curve distribution, so 5% of people have IQ over 125, 20% have IQ of 110-125, 50% have IQ of 90-110, 20% have IQ of 75-90, 5% have IQ below 75, and 2.5% have IQ

below 70.

A 1982 National Research Council (NRC) report found that IQ below 75 was the most common threshold used by States to identify students with MR, but almost half of all States used a lower threshold of IQ below 70, and some States used IQ thresholds higher than 75.[112] The way the IQ distribution is calculated means that 5% of students should have IQ below 75 and 2.5% should have IQ below 70, but the percent of public school students with MR has never been as high as 2.5% because MR also requires significant limitations in adaptive behavior. The NRC report found that special education placement for students with MR commonly begins with a referral by

teachers who observe significant academic and social behavior problems. In effect, teachers have historically identified significant limitations in adaptive behavior indicative of MR, and those students were then referred for IQ testing.

My 2009 study showed that USA blood lead trends explained 65% of the 1948-2001 variation in public school MR prevalence, which rose from 0.4% of students in 1948, peaked at 2.2% in 1976 and fell to 1.3% in 2001, tracking preschool blood lead trends with a 12-year lag. The 12-year statistical best-fit lag is consistent with neurocognitive damage in the first year of life and the average age of public school students. The

strength and predictive power of this association is evident in ongoing MR trends: Public school MR prevalence in 2013 was 0.85%, the lowest level since 1959, down 61% from its peak in 1976, including a 35% decline from 2001-2013.

MR prevalence leveled off at 1.2%-1.3% from 1991-2004 when schools switched from the 1974 WISC-R IQ test to the 1991 WISC-III, with the latter based on an IQ norm sample of children born as lead exposure fell after the mid-1970s. Kanaya has shown that students with mild MR (IQ above 55) who took both tests had WISC-III scores that were an average of 5.6 IQ points lower than their scores on the WISC-R.[113] As a result, more students have WISC-

III IQ scores below thresholds associated with MR, when many of those students would have had WISC-R scores above levels associated with MR. This test-related increase in MR prevalence dissipated after 2000, when the WISC III had been used for almost a decade, and public school MR prevalence has fallen every year since 2000.

The link between preschool lead poisoning and MR was first reported by Byers & Lord in 1943,[114] and subsequent research has shown higher risks of MR associated with blood lead above 25 mcg/dl.[115] The 1982 NRC report found that 75% to 80% of MR students in the 1970s had IQ above

55 and most of these mild cases were of unknown cause, with especially high prevalence among children living in city slums. MR prevalence peaked in 1976, about 12 years after the early-1960s birth years with the highest tooth lead concentrations reported by Robbins, before urban renewal demolished a large amount of slum housing over the 1960s.

The NRC report noted that mild MR prevalence in the 1970s was higher for ages 10-14 than for ages 5-9, as special education placement rates increased with increasing academic demands of higher grade levels. At the same time, MR prevalence for ages 10-14 was twice the prevalence for ages 15-19 because students with mild MR

were very likely to drop out and "disappear into the normal population during late adolescence, losing the label once they leave school."

My 2009 study showed that peak MR prevalence has since shifted to older ages, consistent with younger students being the first to show the effects of declines in lead poisoning. Age 14 students had the highest MR prevalence in 1993, but peak prevalence had shifted to age 16 by 2005. From 1993-2005, MR prevalence fell about 35% for ages 6-8, and 22% for ages 10-11, but declined only 4% for age 16 and rose 7% for age 17 as more MR students now stay in school until they earn a diploma or certificate. MR students who left school as

dropouts fell 23% from 1993-2005 as those earning a diploma rose 32%.

From 1976-2013, MR prevalence fell from 4.1% to 1.5% for black students and from 1.3% to 0.8% for white students. There was a steep drop in black MR prevalence from the mid-1970s to the mid-1980s, associated with 1960s slum clearance birth years. Prevalence was stable for white and black students from the late-1980s to 2000, as schools switched from the 1974 WISC-R to the 1991 WISC-III, but MR has declined for all students since 2000. This ongoing MR decline reflects declines in elevated blood lead since the Residential Lead-Based Paint Hazard Reduction Act of 1992, which authorized many lead hazard

reduction efforts, including the regulations that were the subject of my 1990s Economic Analysis.

Atkins found "no evidence" that the mentally retarded "are more likely to engage in criminal conduct than others", but that was not the early-1900s consensus, led by the influential work of Terman and Goddard. Terman, while at Stanford, published his 1916 "Stanford-Binet" with an "intelligence quotient" score later known as "IQ".[116] The first chapter of his 1916 manual stated: "the most important trait of at least 25 percent of our criminals is mental weakness" and "every study which has been made of the intelligence level of delinquents has furnished convincing testimony as to the

close relation existing between mental weakness and moral abnormality".[117] Goddard was a vocal eugenicist who translated the Binet-Simon test in 1908 and distributed 22,000 copies throughout the USA from 1908-1918. Criminology journals in the 1930s bluntly discussed the Criminal Feebleminded,[118] and Defective Delinquents,[119] citing Terman and Goddard.

Increasing use of IQ tests resulted in school MR prevalence rising from 0.06% in 1914 to 0.38% in 1935. The share of households living on farms then fell from 23% in 1935 to 13% in 1952, and one-teacher schoolhouses fell from 55% of elementary schools in 1937 to

33% in 1955. The rise in MR prevalence should have continued over those years, as urbanization increased the percent of students in larger schools where IQ tests were widely used, but MR prevalence was little changed from 1935 to 1952. Why?

The 1935-1952 plateau in MR prevalence followed the 58% decline in per capita use of lead in paint from 1914-1930, as the zinc share of USA paint pigment sales rose from 15% in 1910 to 36% in 1920 and 55% in 1930. The 1914-1930 fall in lead paint use resulted in an even steeper decline in childhood lead poisoning because lead paint was still preferred for exterior surfaces (for weather resistance) as zinc paint became the dominant

choice for interior surfaces where lead paint posed a much greater risk to young children. Zinc paint on interior surfaces reduced the risk of lead poisoning in new homes and in older homes where chalking interior lead paint was covered with zinc paint when homes were repainted.

The increase in USA zinc pigment use occurred as more than a dozen European nations signed a "White Paint Treaty" in the 1920s, limiting the lead content of interior paint to 2%. The treaty did not apply to exterior paint, and the 2% limit was far higher than current standards, but compared favorably with the 1800s, when most paint was about one-third white lead paste. The American paint industry finally

adopted a voluntary standard in 1955 limiting the lead content of interior paint to 1%, after the White Paint Treaty had been ratified by most of Europe and Latin America, Afghanistan, and Viet Nam.

Fig. 32: USA Public School Mental Retardation Prevalence and Preschool Blood Lead Trends

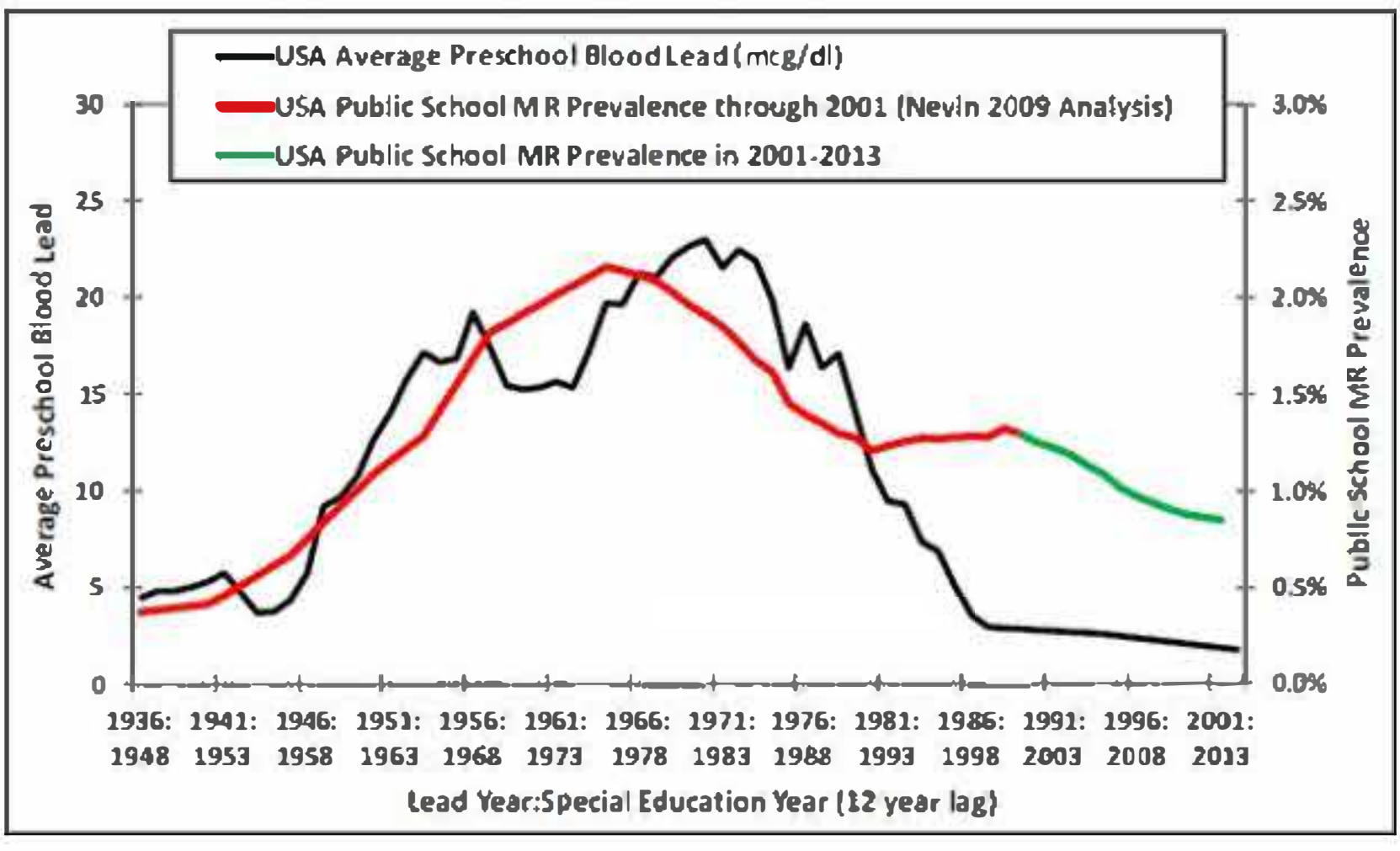


Fig. 33: USA Public School Mental Retardation Prevalence Trends by Race

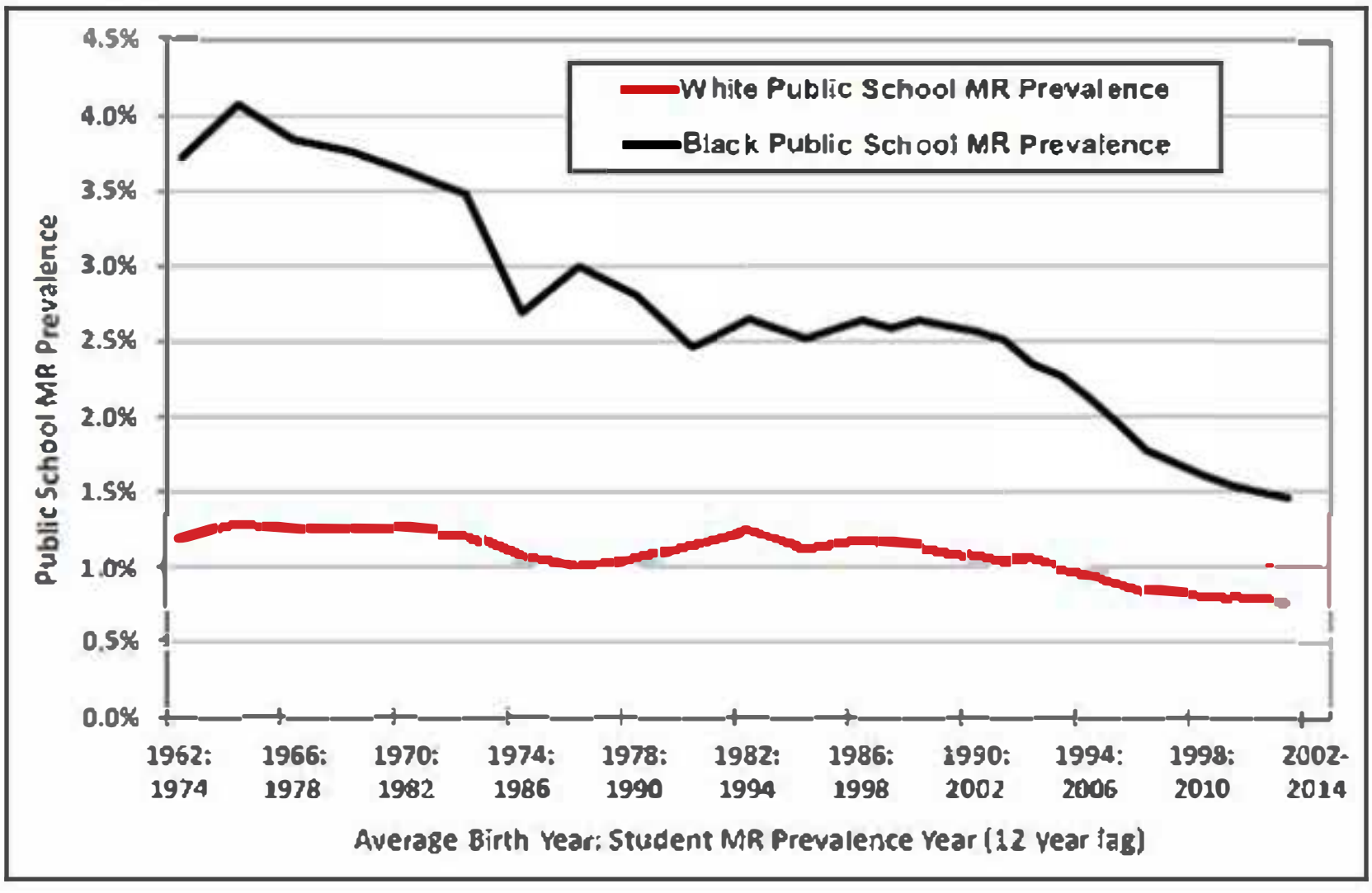
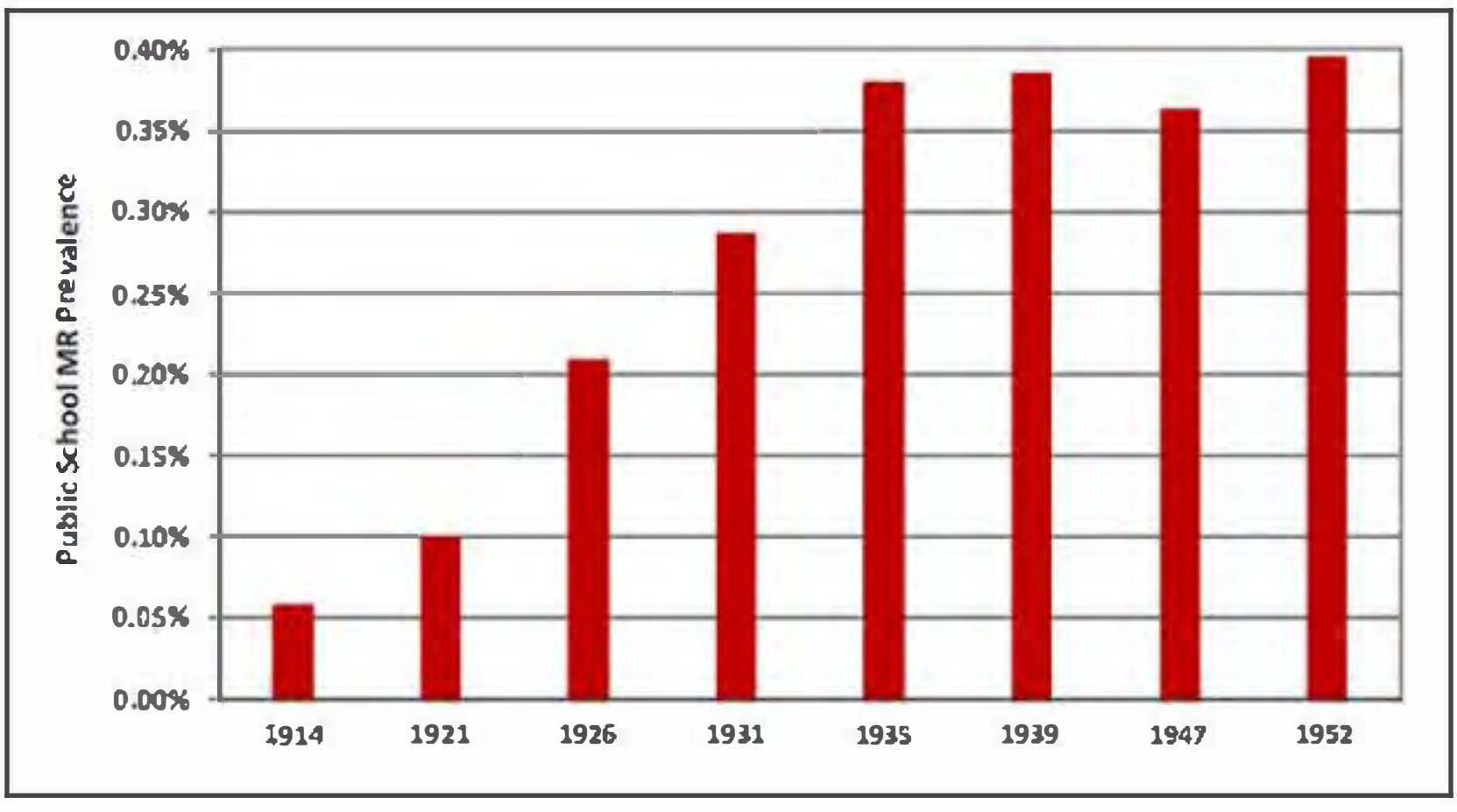


Fig. 34: 1914-1952 USA Public School Mental Retardation Prevalence



Gottfredson's *Scientific American* article claimed that "IQ of 75 is

perhaps the most important threshold in modern life" and "the odds of various kinds of achievement and social pathology change systematically across the IQ continuum". As evidence, she cited *The Bell Curve* analysis of background and annual interview data from the 1979-1990 National Longitudinal Survey of Youth (NLSY). In 1979, the NLSY participants were ages 14 to 22, and the Bureau of Labor Statistics describes this large sample as "representative of all American men and women born in the late 1950s and early 1960s".[120] NLSY participants took the Armed Forces Qualification Test (AFQT) in 1980 and their scores were converted to the IQ scale. Ironically, my 1990s

analysis of lead paint regulations cited a 1995 Salkever study that estimated education and earnings benefits associated with IQ gains from lead poisoning prevention, based on the same NLSY and AFQT data used in *The Bell Curve*.[121]

The Bell Curve found that 7% of white NLSY males with IQ below 90 reported having been incarcerated at some time before their 1980 interview, versus 3% of those with IQ of 90 to 110, and less than 1% of those with IQ over 110. Moreover, 12% of white NLSY males with IQ below 75 had at least one of their 1979-1990 interviews conducted in a correctional facility, versus 7% of those with IQ of 75 to 90, 3% of those with IQ of 90 to 110, and 1% of those with IQ over 110.

The probability of a correctional facility interview reflects both the probability of being incarcerated and the duration of time served. If time served reflects offense severity, then the high rate of correctional facility interviews for white males with IQ below 75 suggests that the lowest 5% of the IQ distribution was especially likely to engage in serious (violent) offenses.

Other research also links low IQ and criminal offending. A 2012 study in *Intelligence* includes a brief overview of three different lines of research that demonstrate: (1) Low IQ is a strong predictor of individual-level criminal behavior in the general population; (2) Known offenders score

approximately 8 IQ points lower than the general population; and (3) Macro-level IQ scores are linked to county and national crime rates.[122] The authors conclude: "Taken together, the literature suggests that IQ - at the individual and macro-level - is negatively correlated with crime and this effect remains after controlling for possible confounds such as age, race, gender, and socioeconomic status." This relationship is also evident in studies from many nations.

The Bell Curve also found that 32% of white NLSY females with IQ below 75 gave birth outside of marriage, versus 17% of those with IQ of 75 to 90, 8% of those with IQ of 90 to 110, and less than 4% of

those with IQ over 110. White NLSY youths with low IQ also had much higher risks of academic failure, with 55% of those with IQ below 75 failing to graduate from high school, versus 35% of those with IQ of 75 to 90, only 6% of those with IQ of 90 to 110, and less than 0.4% of those with IQ over 110.

In response to the controversy over *The Bell Curve*, the APA convened a task force that issued a 1996 report on "Intelligence: Knowns and Unknowns".[123] That APA report confirmed a negative correlation between IQ and juvenile crime, and a much stronger positive correlation between IQ and years of schooling. The strong correlation with education attainment is reflected in the NLSY data showing

that only 45% of white youths with IQ below 75 graduated from high school, and in the 1982 NRC finding that students with mild MR had very high dropout rates.

There are no IQ data from the late-1800s, but the strong correlation between IQ and years of schooling suggests that historic education statistics might provide some insight into what happened to IQ and MR prevalence as USA per capita use of lead in paint tripled from 1876 to 1898. School enrollment rates for age 5-19 black children rose from 2% in 1860, before the Civil War, to 34% in 1880, as age 5-19 enrollment rates for white children rose from almost 60% in 1860 to 62% in 1880. That rising enrollment trend was

reversed from 1880 to 1900, when age 5-19 enrollment rates fell from 34% to 31% for blacks and from 62% to 54% for whites.[124]

Age 5-19 enrollment rates then rose every decade from 1900-1970, reaching 89% in 1960 and 91% in 1970, but fell back to 89% in 1980, reflecting birth years when rising gas lead exposure offset declining lead paint exposure. Age 5-19 enrollment reclaimed the 91% level in 1985, and rose to 93% in 1991.

Rising education attainment has accelerated since the 1994 publication of *The Bell Curve*. The percent of the age 14-17 population enrolled in grades 9-12 rose from 91% in 1994 to 97% in 2014. The public high school average

freshman graduation rate (students who receive a regular diploma within 4 years of entering ninth grade) rose from 71% in 1995 to 82% in 2012. The percent of the age 16-24 population not in school who had not received a regular diploma or GED (status dropouts) fell from 11.4% in 1994 to 6.5% in 2014.[125]

Postsecondary education trends have followed high school completion trends since 1960. The percent of recent high school completers enrolled in college increased from 45% in 1960 to 55% in 1968, but fell below 51% in every year from 1972 to 1980. College enrollment rates for recent high school completers then rose to 60% in 1990, 63% in 2000, and 68.4% in 2014.

The theory that "an inherited stratification of our society" is dictated by inborn IQ suggests that increases in education attainment might be associated with lower achievement standards for higher education, but Scholastic Achievement Test (SAT) scores show the opposite trend. As college enrollment rates fell from 1968 to 1980, average SAT scores also fell, and as college enrollment rates increased over recent decades, average SAT scores increased. My 2009 study showed that 1936-1990 preschool blood lead trends also explained 65% of 1953-2003 variation in SAT math scores and 45% of variation in verbal scores, with a best-fit lag of 17 years. This analysis controlled for a rise in

students speaking a foreign language at home after 1990, and a surge in students taking SAT prep courses from 1977-1986.

The APA report found that the correlation of individual IQ on different IQ tests "is consistent with, but does not prove, the hypothesis that a common factor such as g underlies those correlations". The APA also noted that "there is no full agreement on what g actually means".

My contention is that Spearman did make a conceptual breakthrough when he realized that the correlation of individual IQ scores suggested an underlying common factor, but that factor was not a general intelligence gene.

Herrnstein and Murray were also correct when they argued in *The Bell Curve* that the correlation between low IQ and incarceration, unwed births, and failure to complete high school suggested a common cause, but low IQ was not the cause, it was the effect of the same underlying common factor: lead poisoning.

The APA report cited studies of adopted children showing a high correlation of IQ for genetic siblings separated early in life, and a low IQ correlation for adopted siblings who were raised in the same family, indicating that IQ is largely inherited. Identical twins separated at the age of one do not share the same socioeconomic environment, but did share the

same prenatal blood lead and exposure to lead contaminated dust in the same home as they learned to crawl. Variations in biological vulnerability to lead exposure (e.g., increasing the percent of ingested lead absorbed into the bloodstream) are also likely to affect IQ, but any inherited vulnerability would not be relevant if young children were not exposed to this neurotoxin.

In a 2010 *Physiology & Behavior* article, Carpenter and I noted: "There are undoubtedly genes that contribute to susceptibility to violent behavior, just as there are genes that determine susceptibility to development of cancer consequent to chemical exposure".[126] Raine has also

written about how crime is affected by environmental factors interacting with biological risk factors.[127] Dickens also wrote about variations in biological vulnerability to lead exposure in 1868: “lead-pisoned she is, sur, and some of them gets lead-pisoned soon, and some of them gets lead-pisoned later, and some, but not many, niver; and ‘tis all according to the constitooshun, sur, and some constitooshuns is strong, and some is weak; and her constitooshun is lead-pisoned, bad as can be, sur”.

Individual variations in biological vulnerability to preschool lead exposure, and in the timing and severity of that exposure, cause population cognitive and behavioral impacts that overlap

like Venn diagrams. Lower levels of lead exposure are associated with IQ losses, lower education attainment and achievement, and more impulsive and delinquent behavior in adolescence. More severe lead poisoning is associated with larger IQ losses, increased MR risk, more severe education and employment limitations, and greater risks of homicide and life-course-persistent violent offending.

There is no reason to assume that these impacts are mediated by some ill-defined g factor. My 2007 study noted: “Gray matter damage causing permanent IQ loss, and neurotransmission damage that affects behavior, could cause an IQ-crime correlation due to separate

lead effects." The APA states that individual IQ is relatively stable after puberty, and MRI studies show that gray matter peaks at about the same time as puberty.[128] This suggests that IQ losses could be especially associated with lead-induced gray matter losses. MRI studies show white matter growth through age 50, Silbergeld states that neurotransmission (white matter) effects of lead exposure could be reversible absent continuous exposure,[129] and arrest rates decline with age even among chronic offenders. This suggests that behavioral impacts could be especially associated with lead-induced white matter damage.

With respect to "coherence", Hill

states that a causal theory "should not seriously conflict with the generally known facts of the natural history and biology of the disease". The theory that inherited IQ caused the behavior risks reported in *The Bell Curve* clearly is not coherent with the known facts and history of behavior trends. Herrnstein and Murray explicitly conceded this point, stating that increases in crime and unwed birth rates from the 1960s to 1994 "cannot be attributed to changes in intelligence, but rather must be blamed on other factors, which may have put people of low cognitive ability at greater risk than before."

The theory that preschool lead exposure caused the behavior risks

reported in *The Bell Curve* is entirely coherent with the known facts and history of international crime trends, USA unwed pregnancy trends by age, incarceration and arrest trends by age and race, homicide trends by city size, surging youth crime at the start of crime waves, plummeting youth crime presaging the end of crime waves, MR prevalence trends from 1914 to 2013, shifts in the peak age of MR prevalence, and USA education attainment trends from 1860 to 2014.

The Bell Curve controlled for race by first examining the relationship between IQ and behavior among white NLSY participants. Herrnstein and Murray also showed that family structure and

socioeconomic status had little impact on behavior risks for white youths after controlling for IQ, and IQ still had a large impact on behavior risks after controlling for other factors. Based on these findings for white youths, the most controversial conclusion from *The Bell Curve* was that higher risks for black youths could be largely explained by differences in inherited IQ.

The Bell Curve theory that inherited IQ causes racial differences in behavior risks has failed to demonstrate any predictive power for 1994-2014 trends in unwed births, juvenile arrests, or high school dropout rates. In 1994, the age 10-14 birth rate for black girls was 5.6 times the rate for white

girls, but the 2014 rate for black girls was 25% lower than the 1994 rate for white girls. In 1994, the age 15-17 unwed birth rate for black girls was 3.1 times the rate for white girls, but the 2014 rate for black girls was 30% lower than the 1994 rate for white girls. In 1994, the age 16-24 status dropout rate for black youths was 63% higher than the rate for white youths, but the 2014 rate for black youths was 5% lower than the 1994 rate for white youths. In 1994, the black juvenile property crime arrest rate was twice the white juvenile arrest rate, but the 2014 black juvenile property crime arrest rate was 27% lower than the 1994 white juvenile arrest rate.

The theory that preschool lead

exposure caused the behavior risks reported in *The Bell Curve* is entirely coherent with 1994-2014 declines in high school dropout rates, juvenile arrests, and unwed birth rates for white and black youths, and the narrowing of racial differences in those behavior risks due to the reduction in lead poisoning racial disparities since the 1970s.

Fig. 35: Population IQ Distribution and NLSY Behavior Risks by IQ Range for White Youths

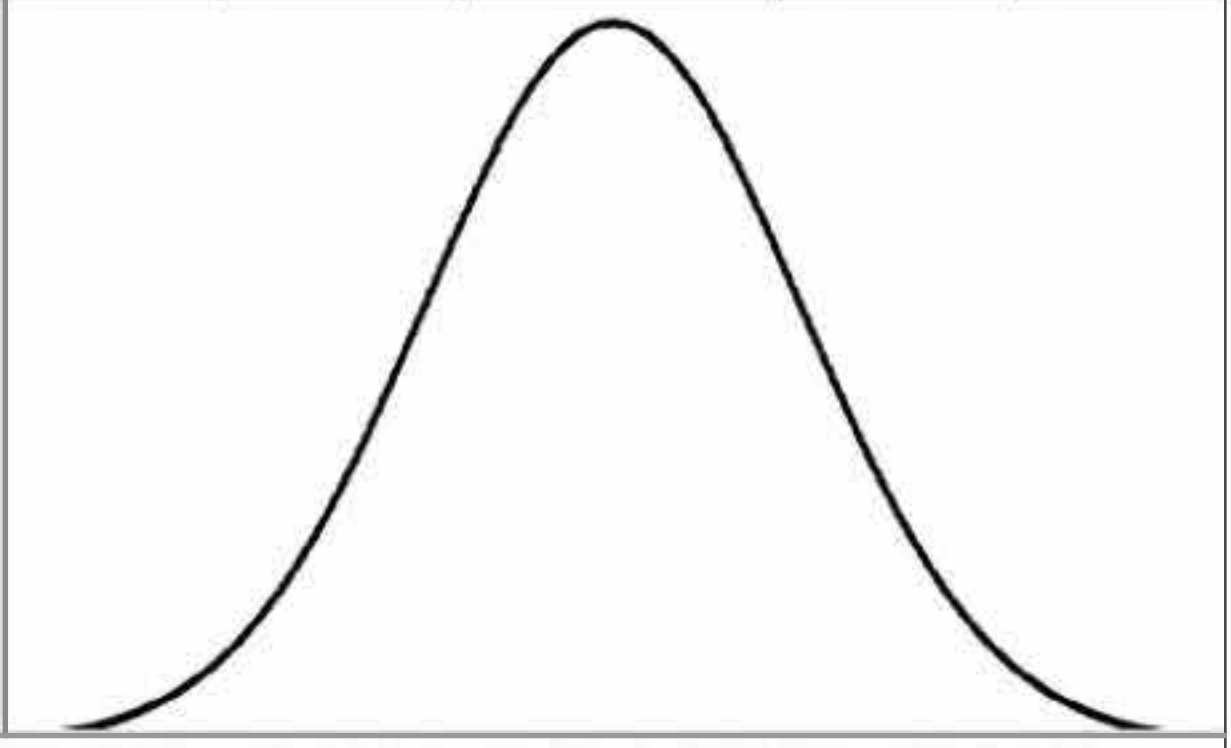
IQ Range	< 75	75-90	90-110	110-125	IQ >125
Population Distribution	5%	25%	50%	25%	5%
					
NLSY Behavior Risks by IQ Range for White Youths Born in late-1950s and Early-1960s					
IQ Range	< 75	75-90	90-110	110-125	IQ >125
1979-1990 Failure to Graduate from High School	55%	35%	6%	0.4%	0%
1979-1990 Unwed Birth (Females)	32%	17%	8%	4%	2%
Incarcerated Prior to 1980 (Males Self-Report)	7%	7%	3%	1%	0%
1979-1990 Correctional Facility Interview (Males)	12%	7%	3%	1%	1%
Source: Herrnstein and Murray (1994)					

Fig. 36: Population Impacts of Preschool Lead Exposure and Severe Lead Poisoning

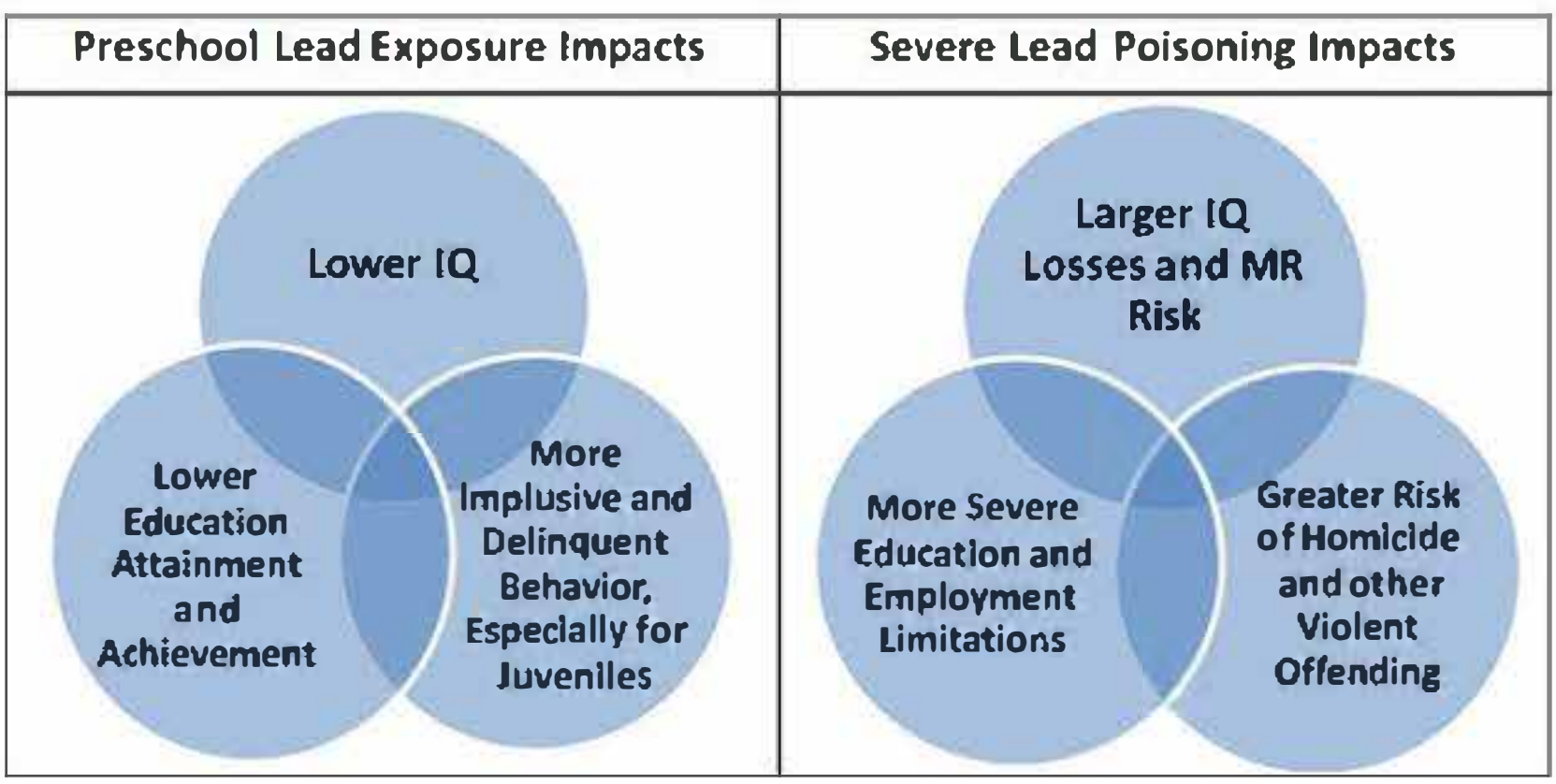


Fig. 37: 1994-2014 Change in Age 10-14 Birth Rates by Race

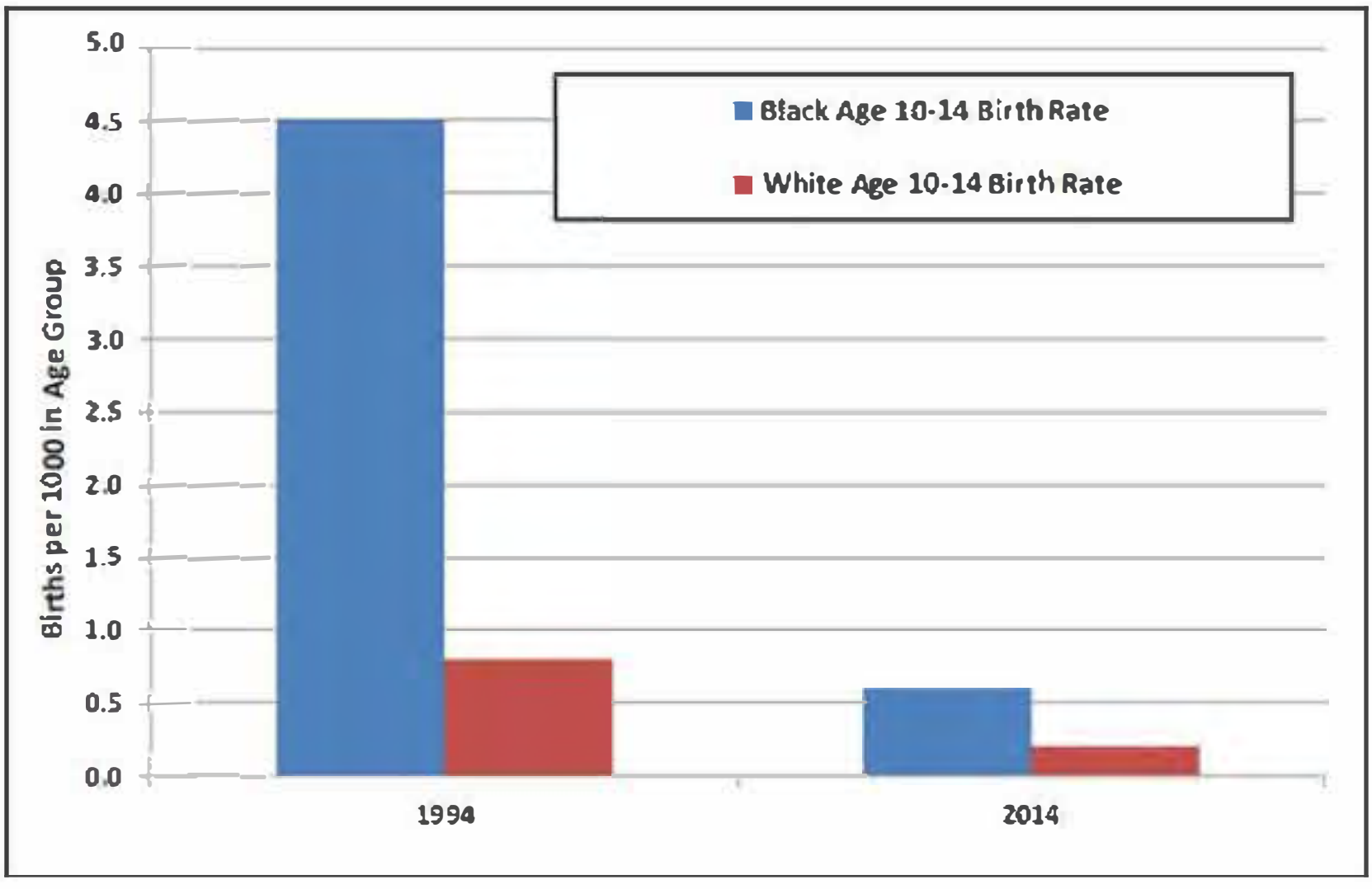


Fig. 38: 1994-2014 Change in Age 15-17 Unwed Birth Rates by Race

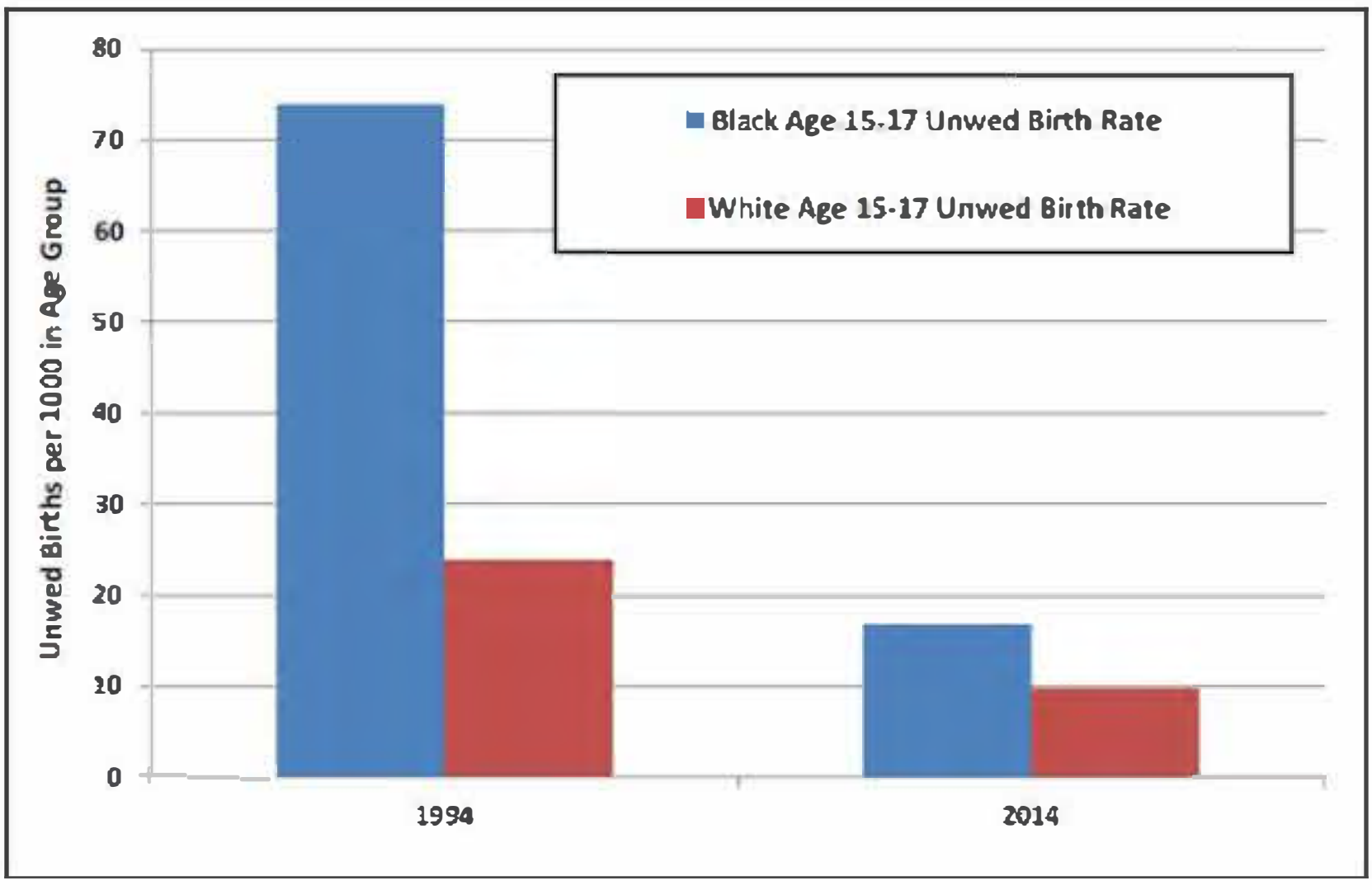


Fig. 39: 1994-2014 Change in Age 16-24 Status Dropout Rates by Race

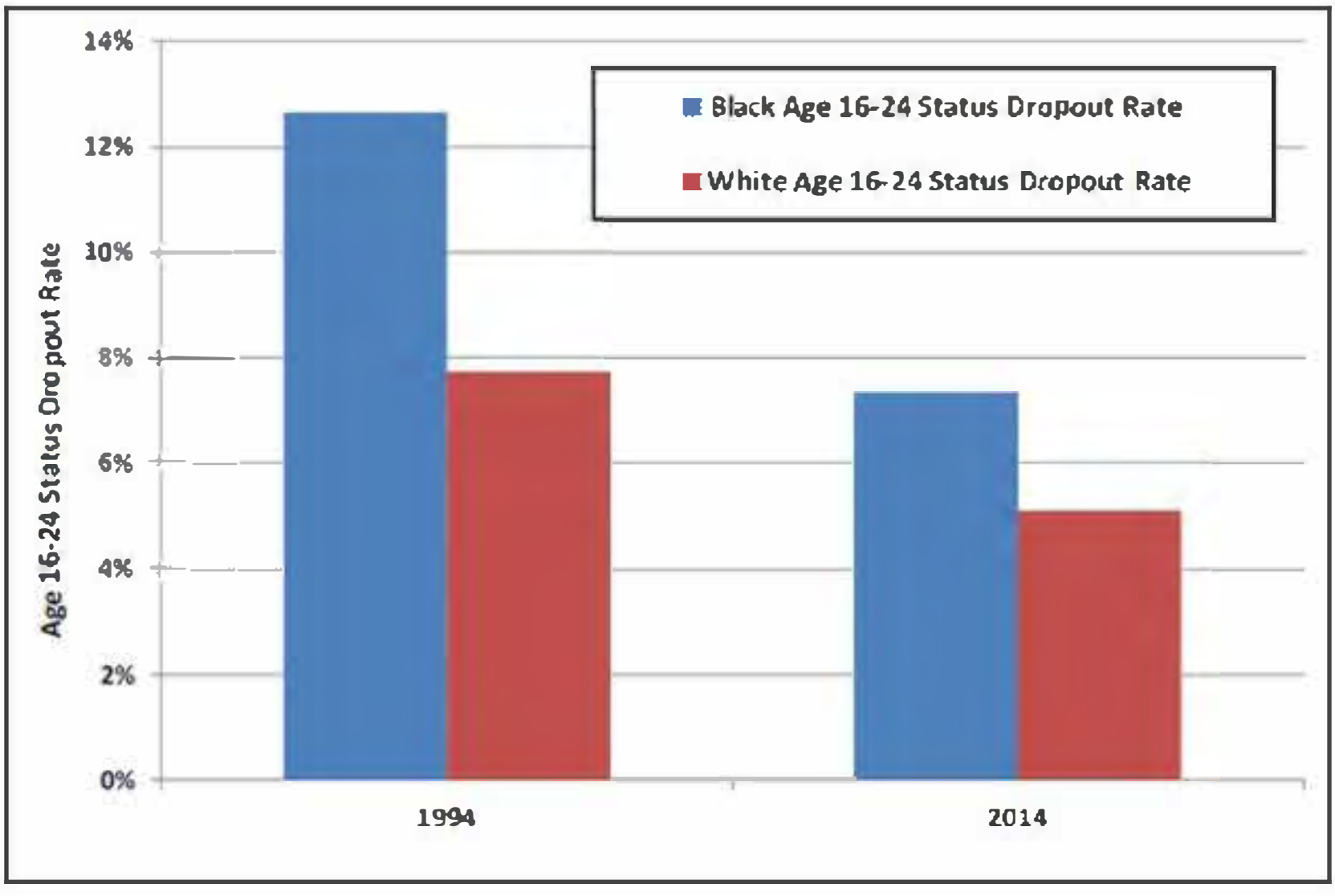
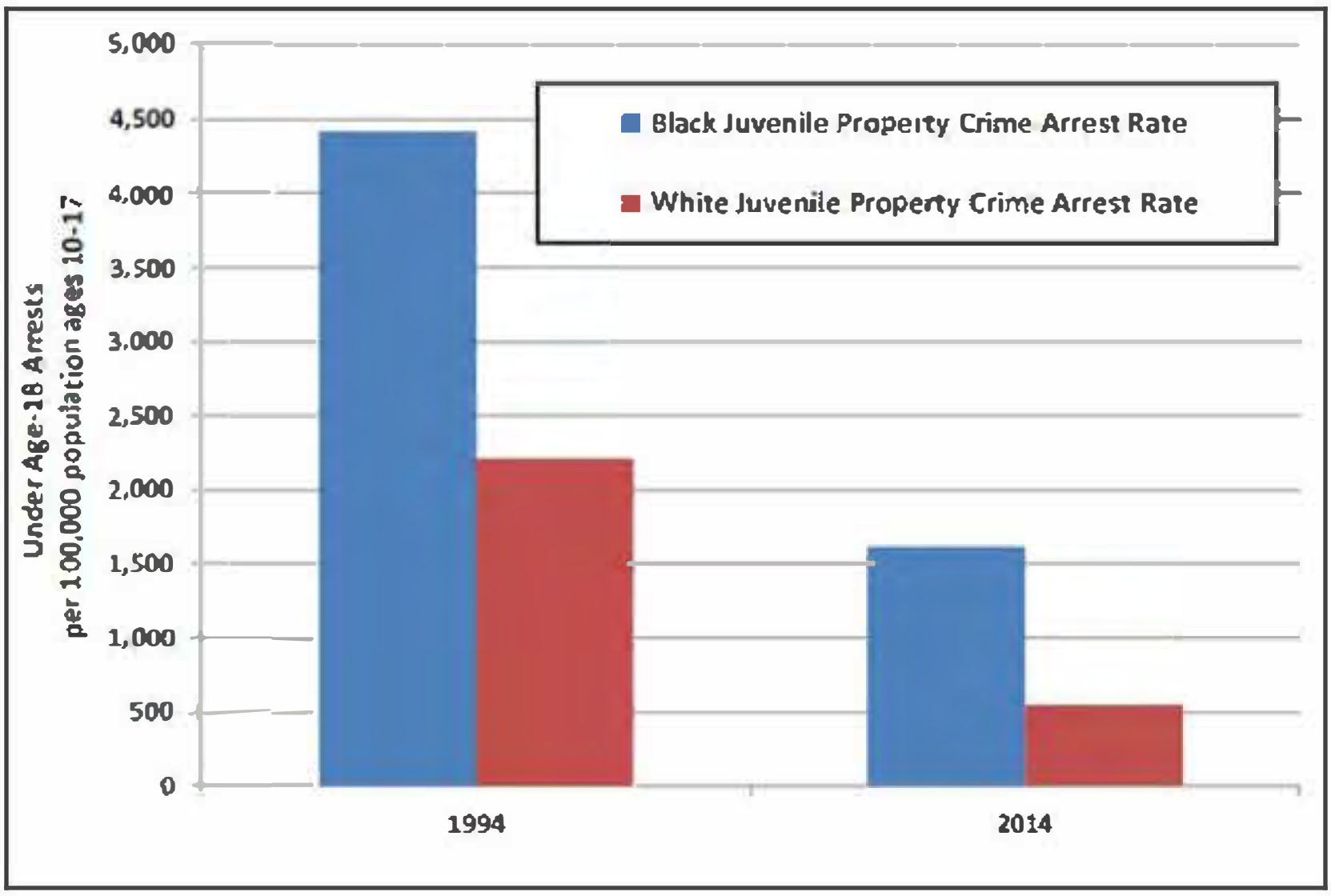


Fig. 40: 1994-2014 Change in Juvenile Property Crime Arrest Rates by Race



Lead exposure can also explain APA

findings that show population cognitive ability has changed over time in ways that are difficult to reconcile with evidence that IQ is inherited. For example, a substantial environmental impact on IQ is suggested by studies showing that USA urban IQ averaged about 6 points higher than rural IQ in the early-1900s, but the urban advantage fell to

about 2 IQ points by the late-1900s. The APA suggests that higher urban IQ could be explained by urban life complexity that somehow increases cognitive ability, and the convergence of urban and rural IQ could reflect declines in rural isolation due to mass communication and increased travel, improvements in rural schools, and other changes that increased the "complexity" of rural life.

Higher urban IQ in the early-1900s is also coherent with the older age of housing in rural areas and the related risk of lead paint exposure. The convergence of urban and rural IQ is also coherent with birth years when rural paint lead exposure fell as urban and

suburban children had more gas lead exposure than rural children. In 1940, the average farm home was 35 years old, built when heavily leaded interior paint was common, but the average non-farm home in 1940 was 21 years old, built as the zinc share of paint sales surged after 1910. By 1970, half of farm homes were built after 1935, after zinc paint had become dominant on interior surfaces, but gas lead exposure in 1970 was near its peak for urban and suburban children. My 2007 study also noted that homicide trends after 1976 were coherent with the decline in rural paint lead exposure and higher urban air lead levels in the second half of the 1900s: “The rural share of the population was 26% in

1980 and in 1990 but the rural share of USA murders fell from 14% in 1976 to 7% in 1994, and total rural murders fell 44% from 1980–1994."

In 1996, the APA task force chair and other scholars co-authored *The Rising Curve*, exploring two other APA findings suggesting large environmental impacts on population IQ.[130] First, there was a large reduction in the racial difference in USA student achievement scores after 1970. Second, there was a global rise in IQ scores throughout the 20th Century.

The Rising Curve showed that the average racial difference in National Assessment of Educational Progress (NAEP) scores

narrowed at different times for ages 9, 13, and 17, but black gains at each age were traced to the same 1962-1973 birth years. My 2009 study linked those same birth years to a narrowing of racial differences in SAT scores and public school MR prevalence, and my 2007 study linked those same birth years to a narrowing of the racial difference in juvenile burglary arrest rates. All of these trends are coherent with urban sprawl spreading more air lead to predominantly white suburbs as lead paint poisoning risks for black children were reduced by 1960s slum clearance and by housing quality standards enacted after the Housing and Urban Development Act of 1965 created HUD as a Cabinet-level

agency.

In Chapter 2 of *The Rising Curve*, Flynn presents evidence of IQ gains over the 1900s in more than 20 nations. This trend, now known as "the Flynn effect", was long obscured by the way IQ is calculated relative to norm sample scores, with average IQ "reset" to 100 with every new test. Flynn discovered the rising IQ trend by examining "norm comparisons", which validate new IQ tests by giving older IQ tests to the norm sample for the new test, to show that individual IQ scores on new and old tests are similar. Norm comparisons do show that individuals have similar scores on new and old IQ tests, but Flynn also found that almost every norm

sample had average IQ over 100 on old IQ tests. By definition, IQ of 100 was the average score for a representative norm sample for the old test, so new norm samples having average IQ above 100 on old tests means average IQ has increased. This global trend is especially remarkable since eugenicists had long fretted that higher birth rates among those with lower IQ would cause a dysgenic decline in average (inherited) IQ over time.

My 2000 study found that global IQ gains are coherent with a global 20th Century decline in lead paint exposure, and a diminishing rate of IQ gains in the late-1900s is coherent with the offsetting effect of rising gas lead exposure. Flynn's

research found that children ages 8-12 in Switzerland had gains of 0.65 IQ points per year from 1956 to 1977, but only 0.19 points per year from 1977 to 1984. Norway military tests at age 18 showed gains of 0.63 IQ points per year from 1954 to 1968, but only 0.22 IQ points per year from 1968 to 1980. In *The Rising Curve*, Flynn graphed IQ gains in Britain across birth years from 1877-1967, showing a rapid gain from 1877-1927, a slower rise for birth years from 1927-1957 (after zinc paint had become dominant on interior surfaces), and no IQ gains across birth years from 1957 to 1967.

Flynn's USA data focus on white IQ gains because early IQ norm samples included only whites.

Some of the norm samples for white IQ span a wide range of ages (e.g., ages 16-48) with many comparisons for IQ norms representing different generations. These data show steady white gains in average IQ over the 1900s, and this is coherent with generational changes in average white preschool lead exposure. EPA estimates for lead ingestion for two-year-olds during the leaded gas era (Figure 25) show that baseline ingestion (outside of central cities) was less than half of the lead ingestion from interior lead paint exposure. In the white population, steady gains in average IQ are coherent with a generational change from parents who mostly lived in homes with interior lead paint to children

mostly born in new suburbs with little or no interior lead paint and only baseline exposure to gas lead fallout.

The Bell Curve did not control for preschool blood lead, and that was a critical flaw because their NLSY sample was *only* "representative of all American men and women born in the late 1950s and early 1960s". Their data was not representative of the population *over time* because the preschool blood lead distribution has shifted over time, as shown in that other 1994 publication comparing blood lead data from 1976-1980 with 1988-1991 data. The preschool blood lead distribution resembled a bell curve in 1976-1980 and in 1988-1991, but the phase out of leaded gas shifted

the entire blood lead distribution to lower levels. In 1976-1980, 25% of preschool children had blood lead above 19 mcg/dl and 5% were above 28 mcg/dl. In 1988-1991, 25% of preschool children had blood lead above 6 mcg/dl and 5% were above 12 mcg/dl.

The dose-response relationship between IQ and blood lead means that children at the high end of the blood lead bell curve will be disproportionately concentrated at the low end of the IQ bell curve. The 1976-1980 blood lead distribution profoundly affected neurodevelopment for many of the 25% of children above 19 mcg/dl, who would be concentrated in the lowest 25% of the IQ distribution (IQ below 90) for a norm sample

representing their birth years. In absolute terms, the 1988-1991 blood lead distribution caused much less neurocognitive and neurobehavioral damage for the 25% of children above 6 mcg/dl, but those children would also be concentrated in the lowest 25% of the IQ distribution for a norm sample representing their birth years. The statistical method used to calculate IQ relative to norm sample scores obscures this impact of the blood lead bell curve, just as it obscured the rising IQ trend discovered by Flynn, because 25% of *every* IQ norm sample has IQ below 90, by definition.

The NLSY data examined in *The Bell Curve* just happened to be representative of birth years

associated with the 1976 peak in MR prevalence, the decline in education attainment and rising teen pregnancy and arrest rates through 1980, and rising incarceration rates from 2007 to 2014 for men over the age of 50. The preschool blood lead bell curve in the late-1950s and early-1960s was characterized by severe racial disparities *and* by severe disparities between white children. In 1960, black households occupied 56% of substandard central city housing, but that means that whites occupied 44% of 1960 substandard city housing. The majority of white children in 1960 lived in suburbs with little or no interior lead paint and only baseline exposure to gas lead fallout, but a large minority of

white children in 1960 lived in the same toxic environments that afflicted a disproportionate percent of black children, in substandard city housing and/or near congested highways with severe near fallout from gas lead emissions. That is why *The Bell Curve* found a very strong relationship between low IQ and behavior risks for white youths born in the late-1950s and early-1960s.

Herrnstein and Murray were perilously close to the truth when they said "other factors...may have put people of low cognitive ability at greater risk than before". The "other factor" that put people at greater risk was the severity of lead poisoning at the high end of the preschool blood lead bell curve,

and that factor affected both behavior risks *and* the absolute level of cognitive ability or "general intelligence" associated with low IQ. Declines in lead exposure have reduced neurobehavioral risks, evident in declining juvenile arrest and unwed birth rates, and reduced neurocognitive risks, evident in ongoing gains in education attainment. Gottfredson's claim that "IQ of 75 is perhaps the most important threshold in modern life" is less true with every new IQ test norm sample because the severity of preschool lead poisoning associated with IQ of 75 has declined.

In *Hall v. Florida* (2014), the Supreme Court revisited the question of "which offenders are in

fact retarded", with attorneys for Hall challenging Florida's "bright line" rule that a person is not retarded if their IQ is over 70. The *Hall* decision rejected that bright line because it did not consider "an IQ test's standard error of measurement (SEM), a statistical fact reflecting the test's inherent imprecision".

In the years separating the *Atkins* and *Hall* decisions, the half-century reign of the term "mental retardation" ended, as advocates for people with this condition embraced the term "intellectual disability" as "less stigmatizing than mental retardation".[131] I have used the "mental retardation" term, or MR, to be consistent with the terminology in the *Atkins* and

Roper decisions, my 2009 study, and the 1982 NRC report. I also believe that the "intellectual disability" term has obscured the true meaning of this condition, and done a disservice to lead poisoning victims and to an informed debate about the death penalty. An MR diagnosis has always required significant limitations in intellectual functioning *and* in adaptive behavior, but the "intellectual disability" term highlights intellectual limitations commonly associated with low IQ. Organizations that advocated for the "intellectual disability" term were then in the awkward position of submitting *Hall* briefs arguing against the Florida IQ "bright line" because adaptive behavior

limitations were actually more important to the *Atkins* rationale that "diminished capacities" of the mentally retarded diminish their "personal culpability".[132]

"Neurodevelopment disability" is a term that would be less stigmatizing than MR, and more accurate than intellectual disability, to describe a condition defined by severe neurocognitive and neurobehavioral limitations, associated with severe lead poisoning throughout the 1900s. This disability is not limited to any arbitrary IQ threshold, because variations in biological vulnerability to lead exposure cause variations in neurobehavioral damage and overlapping variations in neurocognitive damage. That is

why “the odds of various kinds of achievement and social pathology” have historically changed “systematically across the IQ continuum”.

There is a very strong correlation between the homicide rate and the condition formerly known as MR, with the USA homicide rate from 1949-2014 tracking the rise and fall of public school MR prevalence from 1940-2005 with a 9-year time lag. This correlation over 65 years does not mean that MR causes homicide offending, but this association is not a coincidence. This correlation reflects the overlapping causal impacts of lead poisoning, the underlying factor that Spearman conceptualized, and Gibson warned about, in 1904.

Fig. 41: Shift in Preschool Blood Lead Distribution from Late-1970s to Late-1980s

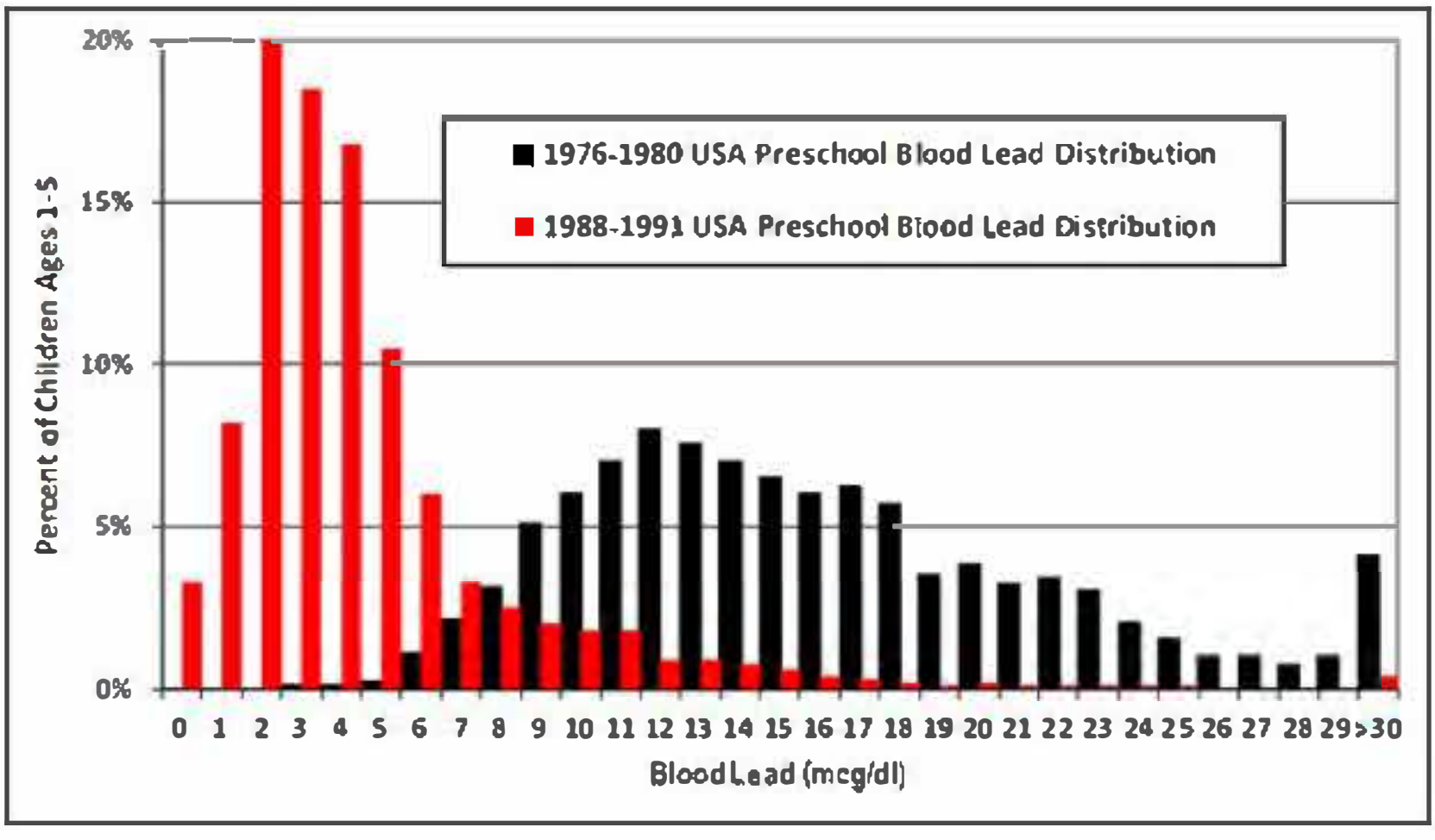
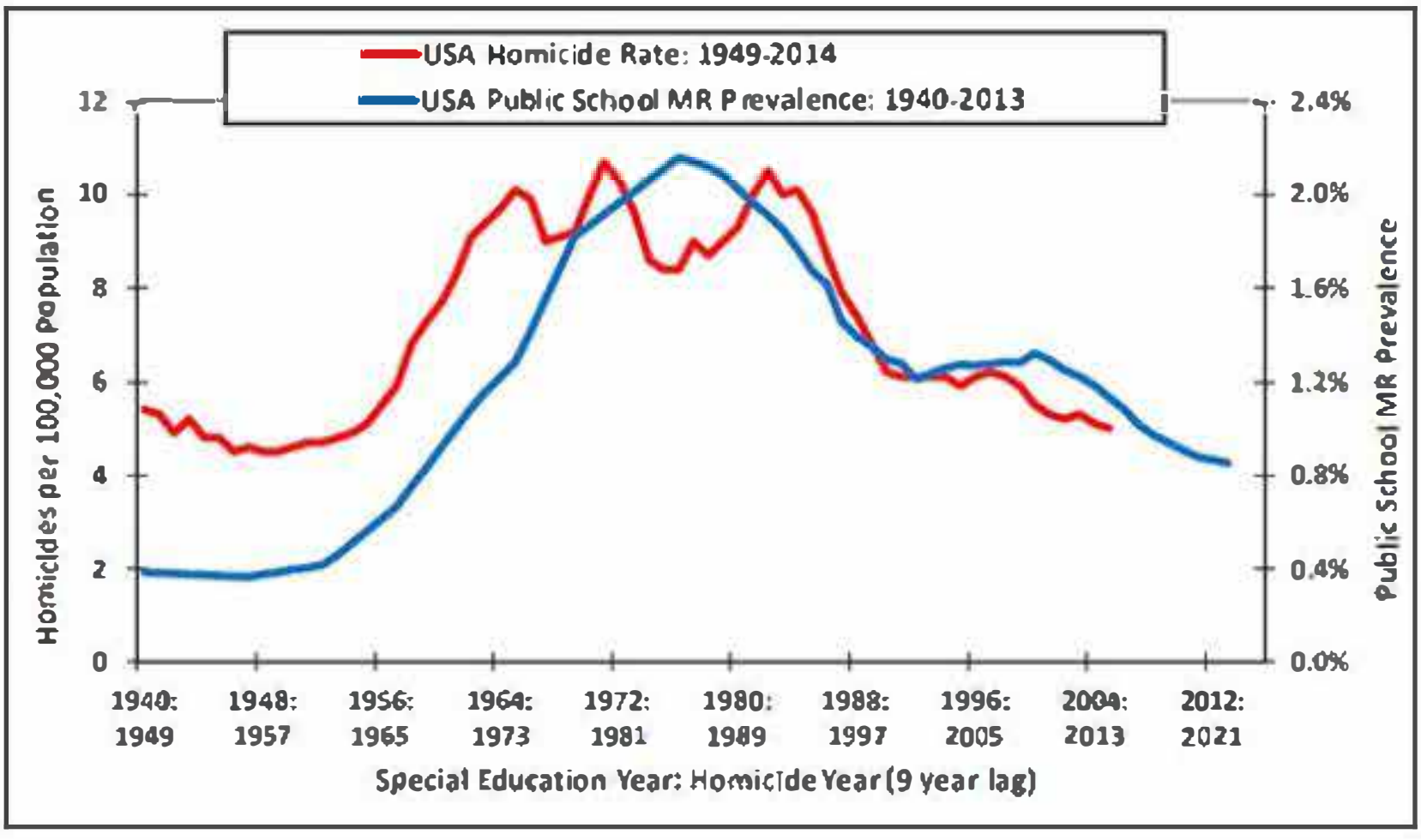


Fig. 42: USA Homicide Rate and MR prevalence Trends



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VII. Specificity and the Antebellum Puzzle

Hill states that an association "limited to specific…types of disease" is a stronger indicator of causation than associations linking an environmental exposure to diverse ailments. Still, he cautions against over-emphasizing this "specificity" indicator because many ailments can be caused by a single "underlying factor".

> "Milk as a carrier of infection and, in that sense, the cause of disease can produce such a disparate galaxy as scarlet

> fever, diphtheria, tuberculosis, undulant fever, sore throat, dysentery and typhoid fever. Before the discovery of the underlying factor, the bacterial origin of disease, harm would have been done by pushing too firmly the need for specificity as a necessary feature before convicting the dairy." (Hill, 1965)

Preschool lead exposure has caused a disparate galaxy of societal impacts through the underlying factor of neurodevelopmental damage, because lead "wreaks a lot of havoc on the central nervous system", affecting a wide variety of

"molecular switches".

There is also an association between elevated preschool blood lead and lower height.[133] One important way that lead affects the brain is by substituting for calcium during neurodevelopment, and a "biotoxic interaction of lead with calcium" could also help explain how lead exposure stunts growth.[134] One study estimated a dose-response relationship of 1.6 centimeters of height lost for each 10 mcg/dl increase in preschool blood lead.[135] This might seem unrelated to trends in IQ, education, and behavior, but it turns out that trends in height could be a big piece of the coherence puzzle.

Many coauthors of *The Rising Curve* posited that the Flynn Effect could be explained by gains in nutrition associated with economic gains, noting that IQ gains are roughly comparable to gains in height over the 20th Century. Flynn was skeptical about this theory: "That every nation has continuously enhanced nutrition by just the right amount, neither too little nor too much, for decade after decade, seems unlikely." This critique of the nutrition explanation for IQ gains could also apply to a nutrition explanation for gains in height. Gains in nutrition associated with economic gains also cannot explain trends in height from the 1700s through 1800s.

Komlos and Baten report: "Heights declined everywhere in Europe in the second half of the eighteenth century", and the average height of American Revolutionary War soldiers was "well above European standards for a very long time to come".[136] This contrast has been attributed to a late-1700s decline in real European wages, and more abundant resources in the USA, but it is also coherent with the Dutch process for white lead production spreading across Europe in the 1700s, before the first USA white-lead mill was built in 1804.

Preschool lead exposure can also help explain a decline in height in Europe and North America now known as the "Antebellum Puzzle",

because it occurred at a time of economic prosperity. Komlos highlights the global extent of this mystery, as height declined in the 1830s and 1840s in the USA, Austria, England, Ireland, Scotland, Bavaria, Netherlands, Sweden, and Russia.[137] France was an exception to the rule. This has been attributed to less urbanization, slower population growth, and wider property distribution in France,[138] but the lack of any height decline in France in the 1800s is also coherent with French concerns about white lead that date from 1780.

The height decline was especially pronounced for urban dwellers during early stages of

industrialization, but spread to rural populations as transportation improvements made urban markets more accessible to farmers. This trend has been attributed to farmers shipping more produce to urban areas, resulting in nutrition declines in rural areas, but this is the same shift from urban to rural impacts seen in crime trends, and is coherent with white lead paste being shipped to rural areas.

Komlos notes that height declines were especially severe in the early-1800s for the working class in Britain: “In fact, the “Oliver Twists” of England were shorter than any other group hitherto examined in Europe or North America”.[139] The working class in

that era had severe occupational and para-occupational exposure – lead dust brought home on work clothes – from a variety of "lead trades" including mining, smelting, ore refining, making of sheet lead and lead pipes, plumbing and soldering, white lead, paint, and ink manufacture, painting, foundry work, typesetting and printing, glazing, china and earthenware, tile and brick glazing, and glass cutting and polishing.[140] Workers in the 1800s recognized sterility, miscarriage, stillbirth, and premature delivery as being common "not only among female lead workers, but also among the wives of men who worked in the lead trades".[141] The impact on wives (and associated impact on

children) reflects the severity of lead dust hazards brought home on the clothes of workers in the lead trades. Childhood para-occupational lead exposure is still a significant risk today: A meta-analysis of data from 1987-1994 found that children of lead-exposed workers had average blood lead of 9.3 mcg/dl compared to a U.S. population average of 3.6 mcg/dl; and the children of lead exposed workers were almost 20 times more likely to have blood lead over 20 mcg/dl.[142]

Steckel reports that average height was 171.5 centimeters for native-born American men born in 1710, and rose to 173.5 centimeters for those born in 1830.[143] Average

height then fell to 170.6 centimeters for American men born in 1860, after the completion of the National Road and the 15-fold increase in USA lead output from the early-1800s to the 1850s. Pulsifer notes that the Civil War caused an 1860s surge in the price of lead that limited use of white lead in paint, and average height increased to 171.2 centimeters for American men born in 1870. Average height then fell to 169.1 centimeters for men born in 1890, as USA per capita use of lead in paint tripled from 1876 to 1898. Height was near its lows for birth years from 1880 to 1900, when education statistics show steep declines in age 5-19 enrollment rates. Height then increased over

birth years across the early-1900s, reaching 177.9 centimeters for men born in 1960, but fell to 177.4 centimeters for men born in 1970 when gasoline lead emissions peaked.

In 1984, Flynn noted that white American IQ trends from 1932-1947 showed larger gains for young children, but norm comparisons after 1947 showed no age-specific trends.[144] He concluded that "either age-specific gains occurred before 1947 and terminated abruptly at that date; or...age-specific results are artifacts of irregularities in the Stanford-Binet sample of 1932, the earliest and most primitive of our samples."

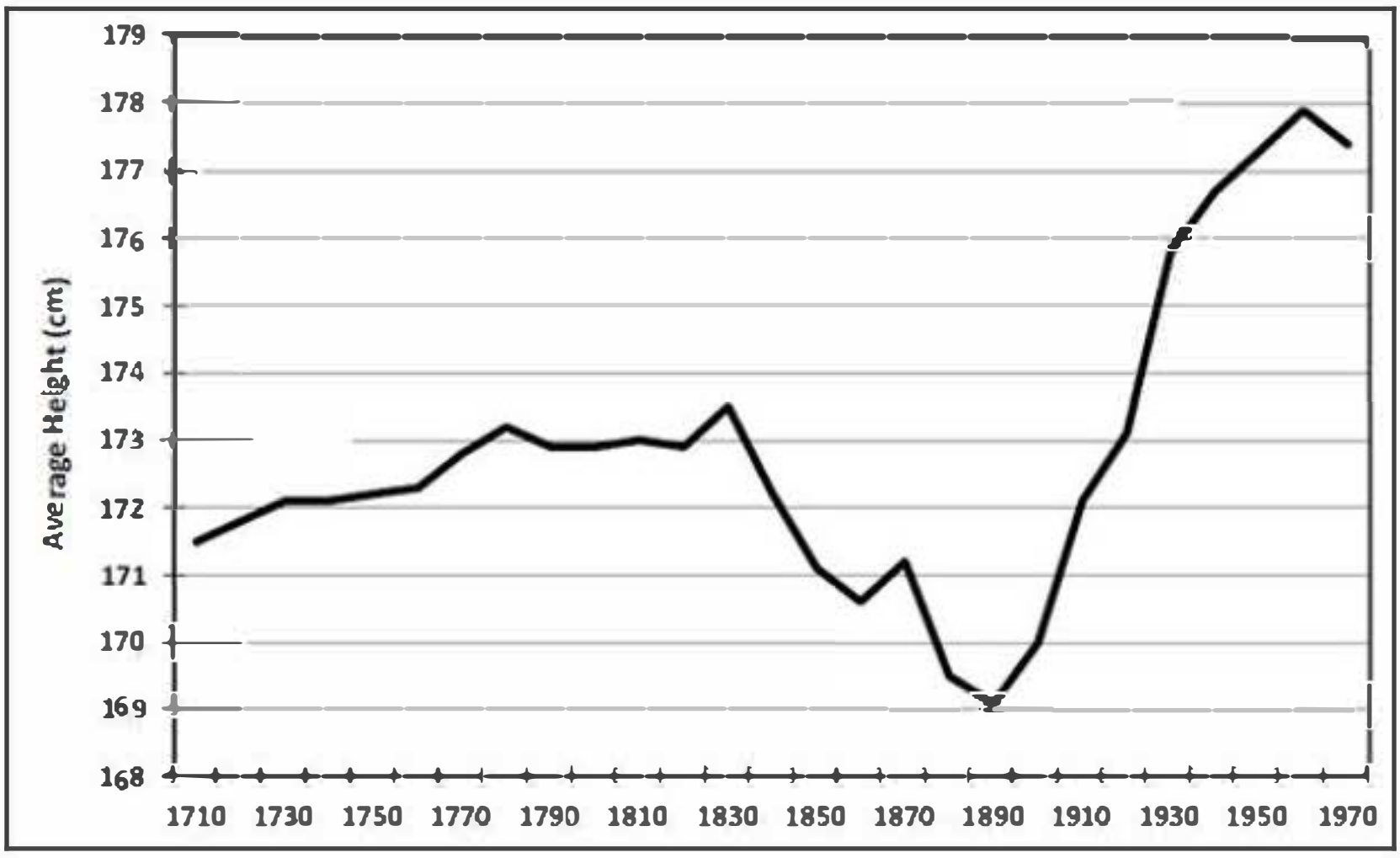
In Chapter 4 of *The Rising Curve*,

Greenfield compared IQ scores for rural Tennessee children in grades 1-8 in 1930 versus scores on the same IQ test by children in the same schools in 1940. This comparison showed a 1930-1940 IQ gain that was twice the average white American IQ gain per decade reported by Flynn, but roughly comparable to the large IQ gain Flynn reported for young children from 1932-1947.

The rapid IQ gain recorded by Tennessee children from 1930 to 1940, and the large IQ gain reported by Flynn for children in the 1932-1947 norm comparison, were associated with birth years in the 1920s and 1930s when zinc paint became dominant on interior surfaces. By contrast, adults in the

1947 norm sample were born closer to the peak use of lead in paint, and many adults in the 1932 sample were born in the late-1800s, when lead use in paint was still rising, and height, school enrollment, and IQ were falling.

Fig. 43: 1710-1970 Trend in Average Height of Native Born American Men



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VIII. Analogy and Generation Genius

With respect to “analogy”, Hill states: “With the effects of thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy.” This is the lesson we have failed to apply to lead exposure in a timely manner for decades, as public health policies failed to keep up with evidence of harm done at lower blood lead levels. The level described as “elevated” or a “level of concern” fell from 60 mcg/dl in the 1960s, to 40 mcg/dl in the early-

1970s, 30 in 1975-1985, 25 in 1985-1991, and 10 mcg/dl in 1991-2012.[145] In 2012, the CDC specified a "reference level" of 5 mcg/dl, while acknowledging evidence of neurodevelopmental damage at levels below 5 mcg/dl.[146]

While we must act on evidence of harm at lower blood lead levels, there is also reasonable concern about the way the term "lead poisoning" is used today, and the need to communicate the difference between individual risk and public health risk. The word "poisoned" is now often used to describe a child with a blood lead level above 5 mcg/dl, causing great anxiety for parents worried about how it will affect their child's life.

There is no reason to terrify individual parents this way: In 1976-1980, 99.8% of all children ages 1-5 had blood lead above 5 mcg/dl (and 88.2% had blood lead above 10 mcg/dl).

The risk that any individual child will be materially affected by a blood lead of 6 mcg/dl is remote, but more than half a million U.S. children ages 1–5 years had blood lead above 5 mcg/dl in 2010. There are many more with blood lead of 2 to 5 mcg/dl, and group differences in blood lead as low as 2 mcg/dl are associated with differences in academic test scores.[147] In fact, the CDC stopped using the "level of concern" terminology to avoid the implication that there is any "safe" level of preschool lead exposure.

The impact of blood lead below 5 mcg/dl might be insignificant for most children, but across millions of children there will be many who have some sort of biological vulnerability in their neurodevelopmental "constitooshun" that interacts with blood lead of 2 to 5 mcg/dl in a way that affects academic performance.

A large 2009 study of North Carolina school children confirmed that blood lead levels below 5 mcg/dl affect grade school test scores after controlling for parental education and economic status.[148] This study also revealed ongoing racial disparities associated with blood lead below 5 mcg/dl: Only 27% of black children in this study had blood lead below 3 mcg/dl,

versus 47% of white children. Again, it is impossible to say if blood lead above 3 mcg/dl will have any material impact on any individual child, but if white children are almost twice as likely as black children are to have blood lead below 3 mcg/dl, then that racial disparity in lead exposure will cause racial disparities in school test scores, on average, across all American children. The individual risk is extremely remote, but this is still a significant public health risk with important societal implications.

Blood lead tests should be a routine part of early childhood checkups, and the CDC should set an "action level" of 2 mcg/dl,[149] with recommended actions for children

over 2 mcg/dl that include home visits to conduct wipe tests for lead contaminated dust. Although children can be affected by many different types of lead exposure, lead in dust caused by lead paint is still the most common exposure pathway. Our failure to banish this health risk is especially tragic because we can eliminate this hazard with a simple window replacement strategy that yields many other benefits.[150]

Window replacement reduces preschool lead exposure because old windows have the highest levels of lead in paint of any building component, lead paint chips are common in old window troughs, and friction surfaces on old windows create lead dust hazards

even in homes without any deteriorated lead paint. The old windows with lead paint are single-pane windows, because double-pane windows became common in new construction about the time when lead paint was banned in 1978. Replacing single-pane windows with high-efficiency windows yields substantial energy savings and increases in home value.

In 2000, the President's Task Force Report on environmental health risks and safety risks to children used data from the early-1990s (the most recent data available at that time) to forecast lead paint hazard and elevated blood lead prevalence through 2010, based largely on housing demolition and window

replacement rates.[151] The task force model projected that housing units with interior lead paint in 1990 would follow one of three paths: some units would undergo window replacement, resulting in a very low risk of lead paint hazards; other housing units would be demolished; and other units without window replacement would have a high risk of lead paint hazards. I worked on the task force report with David Jacobs, the same client who told me years earlier about studies linking lead exposure and crime. In 2006, Jacobs and I coauthored studies in *Environmental Research*[152] and *Housing Policy Debate*[153] showing that recent lead paint hazard and elevated blood lead prevalence data

confirmed that our task force model forecasts were extremely accurate (note: experimental evidence). In 2008, we also collaborated on a study showing that "lead-safe window replacement" yields net monetary benefits from lead hazard reduction, energy savings, and increased home value.[154]

We have already made considerable progress in eliminating the risk associated with old windows. The number of pre-1950 housing units with single-pane windows fell almost 52% from 1993 to 2009.[155] Window sales in 2009 to 2015 indicate substantial additional progress since 2009.[156] We should set a national goal to replace all

single-pane windows in pre-1978 housing, targeting pre-1950 housing as the first priority. This goal can be achieved by replicating the proven success of the Illinois Comprehensive Lead Education, Reduction, and Window Replacement (Clear-Win) pilot program.[157]

Our 2008 study noted that an "increasing concentration of children with elevated blood lead in older housing is consistent with historic use of lead in paint, and a 1990s decline in lead exposure via other pathways". That trend was still evident in USA blood lead survey data from 2007-2010, showing 5.3% of all preschool children living in housing built before 1950 had blood lead levels

above 5 mcg/dl, versus 1.3% of children in 1950-1977 housing, and just 0.4% for children in housing built after 1977. The impact of lead in dust is also evident in higher risks for younger children: In 2007-2010, the percent of children ages 1-2 with blood lead over 5 mcg/dl was 35% higher than the percent of children ages 3-5 with blood lead over 5 mcg/dl (3.1% of children ages 1-2 versus 2.3% of children ages 3-5), consistent with greater risk of lead dust ingestion when very young children are crawling and engaged in frequent hand-to-mouth activity.

Lead exposure risks caused by the historic use of lead in paint were still evident in 2005-2006 housing survey data on lead levels in house

dust and soil. The average lead loading in floor dust in pre-1940 housing is more than twice the average in 1940-1959 housing, seven times the average in 1960-1978 housing, and almost 20 times the average in homes built after 1978. The average soil lead concentration around pre-1940 housing is three times the average for 1940-1959 housing, more than eight times the average around 1960-1978 housing, and 24 times the average around homes built after 1978.[158] Other housing survey data also show that lead dust hazards are especially common in homes with deteriorated interior lead paint, and soil hazards are especially common around homes with deteriorated exterior lead paint.[159]

The decline in lead exposure over the last 50 years reflects the impact of many government actions, including the EPA phase-out of leaded gasoline, Consumer Product Safety Commission limits on lead in paint, Food and Drug Administration limits on lead in food and beverage cans, HUD regulations on lead paint hazard reduction in federally-assisted housing, Occupational Safety and Health Administration actions to reduce workplace and para-occupational lead exposure, HUD grant programs for lead paint hazard reduction, EPA regulations on lead-safe housing renovation and repairs, and CDC blood lead screening and public health education efforts. There was also a

91% decline from 1992-1993 to 2000-2004 in the number of large water utilities above the EPA action level for lead in drinking water. We have come a long way, but there is still more work to do.

It is difficult now to gain public support for even the most cost-effective government actions, because many Americans are convinced that government is *always* the problem and *never* the solution. While it is easy to reach that conclusion based on anecdotes about government failures, the history of lead poisoning prevention reminds us that our government has also had some great successes. The Millennial Generation has achieved record low status dropout rates, juvenile

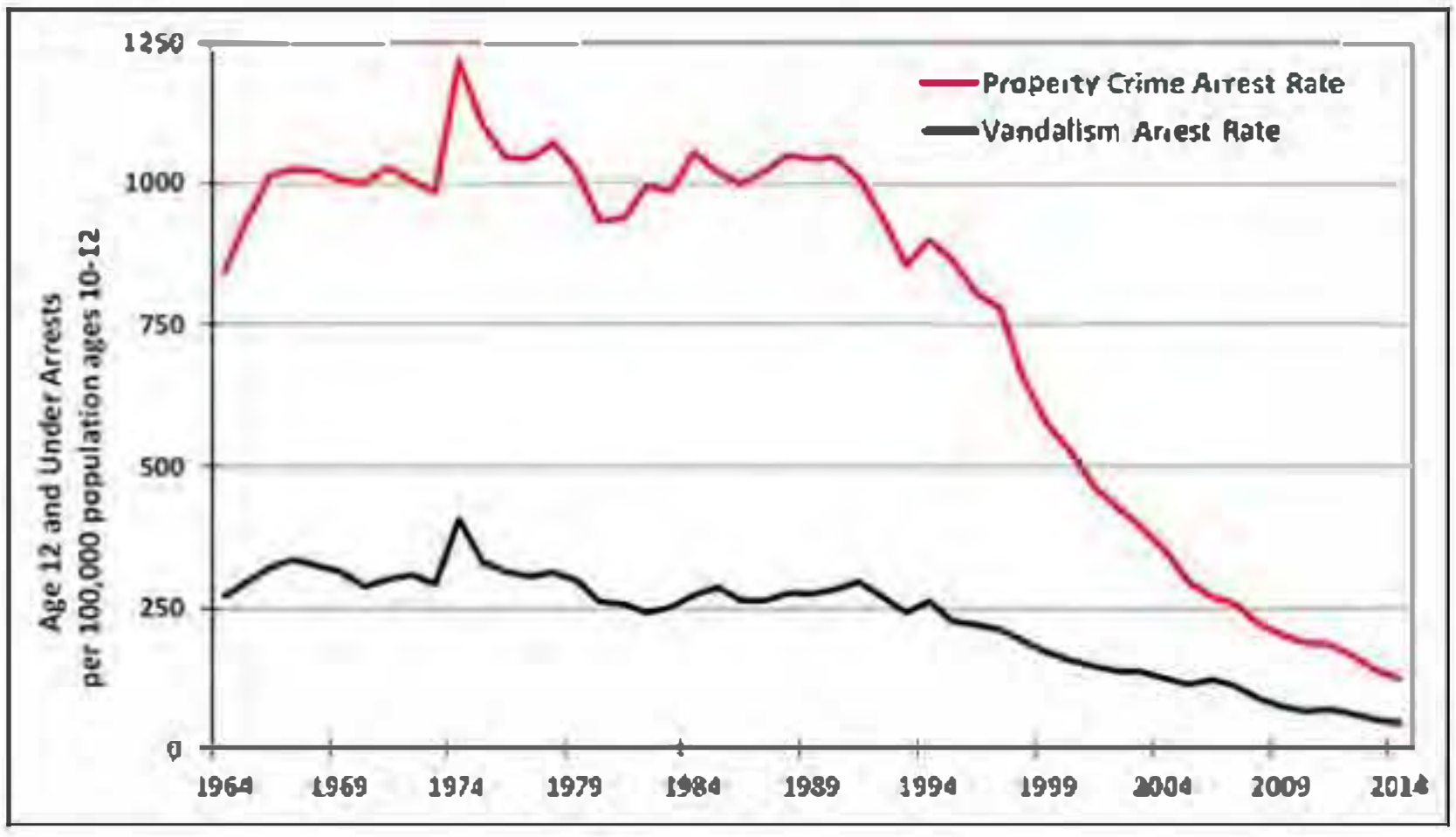
arrest rates, and unwed birth and abortion rates, and record high college enrollment rates, due to government actions taken to reduce preschool lead exposure. All of those actions were denounced as burdensome and costly when they were first proposed, but benefits have clearly exceeded costs.

The successor to the Millennial Generation has already shown their ability to set new records of their own. The largest percentage declines in unwed birth and abortion rates have been recorded by girls ages 14 and younger. The NAEP long-term trend assessment (dating back to the early-1970s) reported record high average math and reading scores for age-13 students in 2012. The FBI first

reported property crime arrest rates for ages 12 and under in 1964, and that first year of data was the record low rate for 32 years, but the age 12 and under property crime arrest rate set a new record low every year from 1996 through 2014, and the 2014 arrest rate was down 85% compared to 1964. The vandalism arrest rate has followed a similar pattern, and the 2014 age 12 and under vandalism arrest rate was down 83% compared to 1964.

We haven't yet agreed on a name for the generation that follows the Millennials. My suggestion is Generation Genius.

Fig. 44: 1964-2014 Trend in Age 12 and Under Property Crime and Vandalism Arrest Rates



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