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Environmental causes of violence

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ABSTRACT

Violent and anti-social behavior is usually attributed to social factors, including poverty, poor education, and family instability. There is also evidence that many forms of violent behavior are more frequent in individuals of lower IQ. The role of exposure to environmental contaminants has received little attention as a factor predisposing to violent behavior. However a number of environmental exposures are documented to result in a common pattern of neurobehavioral effects, including lowered IQ, shortened attention span, and increased frequency of antisocial behavior. This pattern is best described for children exposed to lead early in life, but a similar pattern is seen upon exposure to polychlorinated biphenyls and methyl mercury. Although not as extensively studied, similar decrements in IQ are seen upon exposure to arsenic and secondhand smoke (SHS) exposure. Prenatal and postnatal SHS exposure is also associated with increased rates of conduct disorder and attention deficit hyperactivity. Recent evidence suggests that temporal trends in rates of violent crime in many nations are consistent with earlier preschool blood lead trends, with a lag of about 20 years. These ecologic correlations are consistent with many controlled studies suggesting that lead-exposed children suffer irreversible brain alterations that make them more likely to commit violent crimes as young adults. If this pattern is true for lead and other contaminants, the most effective way to fight crime may be to prevent exposure to these contaminants.

1. Introduction

Violent behavior has always been a part of human existence, but the frequency of violence varies within different segments of the population and over time. Rate of crime in the US has been declining in recent years for reasons that are unclear. Homicide rates, for example, rose to high levels between 1973 and 1993, but have since declined relatively steeply [1]. Politicians and police attribute the decline in violent crime to their actions, although there is some question whether this is a justified conclusion. Certainly some extreme violent acts are a result of political or religious conflicts. However the subject of this review is violent and anti-social behavior that has its origin in personal responses to events in daily life. We present a review of literature describing associations between environmental pollutants, developmental neurotoxicity and neuro-cognitive and behavioral outcomes related to criminal behavior. The aim of this review is to establish plausible links among these previously demonstrated associations to provide policy makers with additional information which may be useful in steps to be taken to reduce crime.

Violence takes a major toll on society, and both violent crime (murder, rape, robbery, and assault) and property crime (burglary and theft) are a particular problem in teenagers and young adults, and especially in young males. Aggressive behavior of young males is characteristic of animal populations, and is almost certainly correlated with rising levels of testosterone. But all young males have testosterone, and not all young males commit violent crimes. Impulsive youth behavior has been linked to a gray matter growth surge just before puberty, followed by rapid white matter growth from ages 12–16 to 23–30, with ongoing white matter growth to age 50 that improves neural connectivity affecting behavior [2–4]. But magnetic resonance imaging (MRI) studies show that all teenagers and young adults experience these brain changes, and not all youths engage in criminal behavior. Among those who do commit crimes, there is also a documented distinction between relatively common Adolescence-Limited property crime offenders and more violent Life-Course-Persistent offenders who account for most adult offending [5].

Violence has a disproportionate effect on young adults of both genders. This is reflected in years of life lost, as shown in Fig. 1 in data from New York State. Injury constituted the largest single factor in years of life lost (23%), and intentional injury (homicide, suicide, assault) was 54% of this total. Some major fraction of the injuries listed as “unintentional” are also related to greater risk-taking, in that the major factor in this category is motor vehicle injury.

What are the factors that promote violent, risk taking and anti-social behavior? Violence is clearly coupled with poverty, and physical

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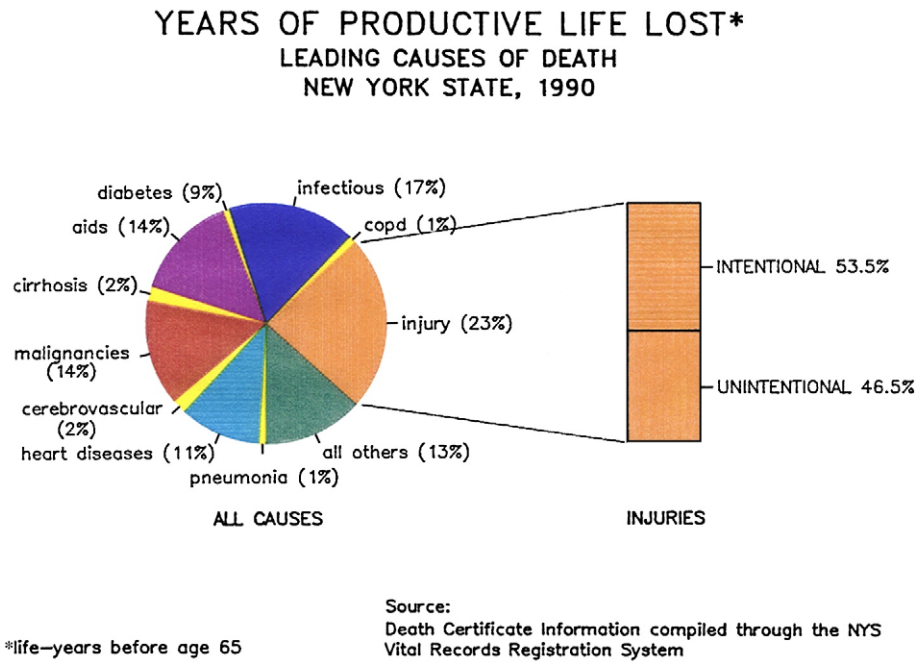

Fig. 1. Causes of mortality in New York State, 1990.

abuse of children promotes later aggression [6]. Social cohesion and neighborhood instability are risk factors for violent behavior [7]. Race is a factor closely related to violence, but it is likely that race is simply a surrogate measure of poverty and low socio-economic status. Poor academic performance, gang affiliation, and family intactness are all factors that are associated with poor and often minority populations, and are correlated with rates of violence [8]. Drugs and alcohol abuse are factors that promote violent acts, but excesses here are usually tightly associated with the above mentioned risk factors. However there is strong evidence that behavior in childhood is predictive of later criminal behavior [9]. Davidson et al. [10] have proposed that violence reflects dysfunction of the neural circuitry of emotion regulation, specifically involving serotonergic projections to the prefrontal cortex.

A variety of types of interventions have been tried as a means of reducing violence [11]. Most have involved behavioral training, psychotherapy, anger management therapy, and enriched education. While there has been some success of these programs, especially if initiated early in life, the success in preventing future violent behavior at later ages has been very limited.

2. Environmental contaminants and IQ

In 1979 Needleman et al. [12] first clearly demonstrated that children exposed to lead early in life suffered a reduced IQ. He and his colleagues measured lead in baby teeth, and found that when he comparing those with high lead to those with low lead, there was a 5–7 point decrement in IQ in those with high lead levels. Not all lead-exposed children had severely reduced IQ, but the distribution curve was shifted downward such that even the bright lead-exposed kids were not as bright as they would have been had they not been exposed. Since this initial report similar findings have been obtained in studies in many different countries. It is clear that lead exposure reduces IQ. While early life exposure appears to be particularly damaging, even exposure during adulthood has a negative impact on cognitive function [13,14]. Other metals such as arsenic also reduce IQ [15]. In a study of children in India exposed to arsenic in drinking water, Ehrenstein et al. [16] report significant negative relationships with vocabulary development, object assembly and picture completion tests. Methyl mercury also causes reduced cognitive function in children [17–19].

A variety of other chemicals have similar actions on IQ. Best studied are polychlorinated biphenyls (PCBs) and dioxins. Like lead, PCBs shift downward the IQ distribution curve [20]. As with lead this observation has been confirmed in many studies [21]. Exposure to various pesticides or mixtures of pesticides has also been shown to alter neurodevelopment [22,23].

Recent reports indicate yet other exposures that result in reduced IQ. Yolton et al. [24] reported that children exposed to secondhand smoke (SHS) (measured by urinary cotinine) showed decrements in reading and block design tests after adjustment for gender, race, poverty, parental education, ferritin and blood lead concentration. Julvez et al. [25] report that maternal smoking resulted in a significant decrease in the child's global cognitive score at age 4 years. Prenatal and postnatal SHS exposure is also associated with increased rates of conduct disorder and attention deficit hyperactivity [26]. It is interesting that there is also evidence that older adults who smoke show reduced cognitive function as compared to non-smokers [27].

3. Lead exposure and rates of mental retardation

Most lead-exposed children are not so incapacitated as to fit the definition of being retarded, but for those children at the lower end of the IQ distribution curve, exposure to lead results in a further lowering of intelligence. In the 1950s and 1960s, many children suffered severe additive exposure to gas lead emissions from urban traffic and deteriorated lead paint in city slums, while suburban children had comparatively little air lead exposure and lived in new homes with little or no lead paint. Statistical analysis of the data in Fig. 2 [28] shows that preschool blood lead trends from 1936 to 1990 appear to explain 65% of the substantial 1948–2001 variation in the percent of public school students in special education for mental retardation (MR). This relationship is characterized by a 12-year best-fit time lag (highest regression R^2 and t-value for blood lead) for students ages 6–18, consistent with lead-induced cognitive damage in the first year of life. MR prevalence peaked in 1976 at 2.16% among students born across birth years of the late-1950s to mid-1960s. The decline in MR was briefly halted when school psychologists began

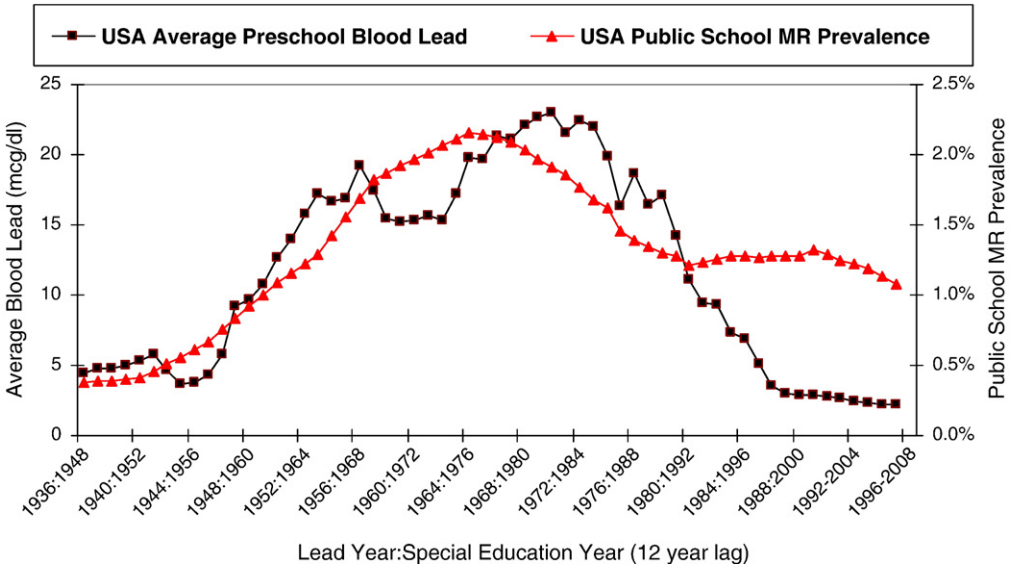

Fig. 2. USA public school MR prevalence and preschool blood lead trends (from [28] with permission). The rise and fall of preschool blood lead from 1936 to 1990 explains 65% of the substantial 1948–2001 variation in the percent of public school students in special education for mental retardation (MR). The 12-year time lag for students ages 6–18 is consistent with lead-induced cognitive damage in the first year of life.

using a new IQ test in 1992. Students with mild MR had scores on the new test more than 5 IQ points below their scores on the older test, resulting in more children having IQ below the threshold for MR diagnosis [29]. Although medical advances have reduced severe MR associated with known causes, the mildly retarded, with IQ above 55, accounted for 75% to 80% of all MR students in the 1970s [30] Most mild MR cases were of unknown cause, with especially high prevalence among "low income groups — who often live in slums".

In principle, MR is characterized by significant limitations in intellectual functioning and adaptive behavior. In practice, adaptive behavior limitations have been identified based on observed classroom learning or behavior problems, and most states defined "significant intellectual limitations" to include IQ below 75 among students referred for MR evaluation. A statistical method measures individual IQ relative to the population IQ distribution so exactly 5% of students should have IQ below 75. The fact that MR prevalence peaked at 2.16% in 1976 likely reflects a lower referral and evaluation prevalence for significant adaptive behavior limitations (i.e., many students with IQ below 75 but with no significant adaptive behavior limitations were never referred for MR evaluation).

The link between MR and lead poisoning was first reported in the 1940s [31]. In the 1960s, lead poisoning was associated only with childhood blood lead above 60 μg/dL (micrograms of lead per deciliter of blood) but subsequent research links lower blood lead to elevated MR risk [32–34]. Elevated blood lead can be due to many exposure pathways, but lead in paint and gasoline had especially pervasive effects due to contaminated dust ingested via normal hand-to-mouth activity as children crawl. The lead share of USA pigments fell from near 100% in 1900 to 35% by the mid-1930s [35], and lead paint was banned after 1977. However lead contaminated dust is still common in older homes with deteriorated paint [36]. National trends in average blood lead were highly correlated with leaded gas use and air lead trends, with median R^2 of 0.94 in 14 nations studied [37], as air lead fallout contaminated dust while lead paint exposure changed slowly with changes in the housing stock. Trends in average preschool blood lead shown in Fig. 2 were derived by using air lead and per capita gas lead trends to extrapolate from national blood lead survey data available for certain years.

The trend in average blood lead from 1936 to 1990 reflects a temporal shift in the entire preschool blood lead distribution, including a rise and fall in more severe lead poisoning associated with MR, and in marginally elevated blood lead prevalence associated with marginally lower IQ and academic achievement. Atmospheric emissions affected blood lead even in rural areas, but traffic caused more severe city exposure as 55% of emissions settled within 20 km of roadways [38]. Slum clearance in the 1960s caused a relatively rapid shift in lead paint hazard exposure because those deteriorated slum units were mostly built around 1900 with heavily leaded interior paint. Black children were disproportionately displaced by slum clearance, even as urban sprawl spread more lead emissions to predominantly white suburbs, narrowing racial disparities in preschool lead exposure over the 1960s. The decline in MR prevalence from 1976 through the 1980s was almost entirely due to a decline in MR prevalence for black students, born across the 1960s birth years when slum clearance and urban sprawl reduced the racial difference in average preschool lead exposure. These same slum clearance birth years are linked to a racial convergence in juvenile burglary arrest rates and National Assessment of Educational Progress scores and Scholastic Achievement Test scores [28,39].

4. Effects of environmental contaminants on behavior

Compounds that reduce IQ also result in behavioral changes, including a shortened attention span, promotion of hyperactivity, increase in impulsive and anti-social behavior and generally reduced overall performance. This was first demonstrated convincingly by Needleman et al. [12] as a component of his classic study of lead toxicity in Boston school children. He found that lead-exposed children were more distractible, dependent, hyperactive, impulsive, easily frustrated, less able to follow simple directions and had a low overall performance. Fig. 3 shows a plot of measures of attention in relation to lead exposure from this study, and a comparable measure of attention in children exposed to methyl mercury, data from Grandjean et al. [40].

Similar attention and behavioral deficits have been reported for children exposed to PCBs and dioxins, although such deficits have not been as systematically studied as for lead exposure. This literature has been extensively reviewed by Schantz et al. [21]. PCB-exposed children are characterized by shorter attention span and greater frequency of behavioral problems [41]. While most exposure is assumed to result from ingestion of PCBs and dioxins from foodstuffs, similar attention and emotion problems have been seen in occupational settings where the

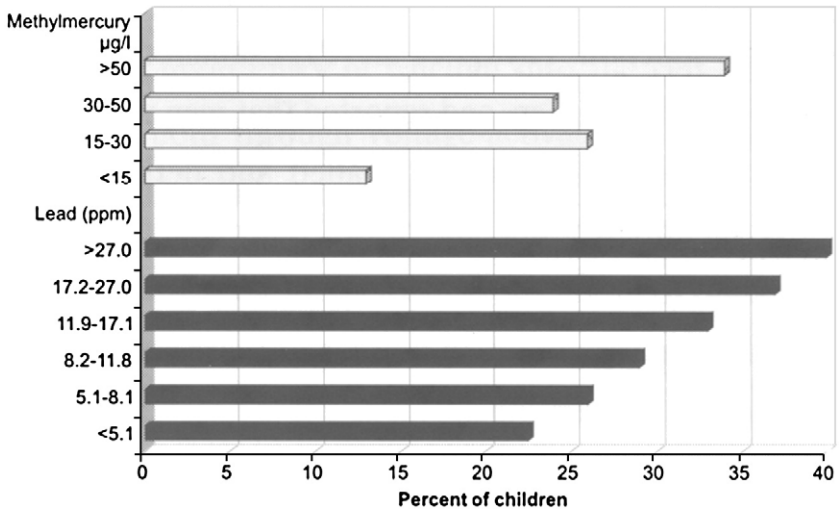

Fig. 3. Effects of methyl mercury and lead on measures of attention. Upper panel: effects of methyl mercury in blood on attention in Faroese children. Attention was measured by reaction time on the NES2 Continued Performance Test. Data derived from [40]. Lower panel: effects of dentine lead concentration on school teachers evaluation of the ease with which children were distractible. Data derived from [12]. Reproduced from Carpenter [69] with permission.

route of exposure was inhalation [42]. Stewart et al. [43] demonstrated that prenatal exposure of children to PCBs, tested at ages 4.5 years, was associated with impulsive and excessive responding on a continuous performance task.

Lee et al. [44] used data from the National Health and Nutrition Examination Survey and demonstrated that US children ages 12–15 years with elevated PCBs, dioxins, furans or persistent pesticides were significantly more likely to show learning disabilities and ADHD. Wang et al. [45] reported on 630 children ages 4–12 with ADHD and 630 controls. They found that ADHD children had a significantly higher blood lead concentration than did controls after adjustment for age and gender (OR = 6.0, 95%CI = 4.10–8.77) when comparing children with blood lead levels >10 µg/dl vs. <5 µg/dl. These are important studies because ADHD is characterized by attention problems and disruptive behavior, and they implicate PCBs, dioxins, persistent pesticides and lead in the etiology of ADHD.

Fig. 4 shows results of a study of rats exposed to PCBs [46] in which the rats must earn water by bar pressing, but where there was a 2 min delay in the delivery of the reward. After learning the task, control animals slowly increased the frequency of bar pressing as the two minute time approached. In contrast rats exposed to PCBs pressed the bar much more frequently, an indication of impatience. As shown in Fig. 5 these PCBs-exposed animals also made much more frequent response bursts, very rapid bar pressing which indicates extreme impatience. One can almost hear these animals saying "Damn it, give me that water!" In contrast, such response bursts were rare in the control animals.

Thus, at least for lead, methyl mercury and PCBs, exposure results in a syndrome of effects, including a reduction of IQ from what it would have been had there been no exposure, plus a series of behavioral effects characterized by reduced attention span and increased vulnerability to or inability to deal with frustration. The syndrome is most clearly seen following early life exposure to these chemicals. While the reduction in cognitive function is also found in exposed adults, there has at least to date not been clear demonstration of the other behavioral changes in adults. It should not be surprising that the developing brain is more vulnerable to toxicants, since this is the period of brain development and setting up of functions that will last for the rest of life.

There has not been a systematic study of behavioral alterations in children exposed to arsenic to the same degree as for lead, methyl mercury and PCBs. Such study is urgently needed. However it seems

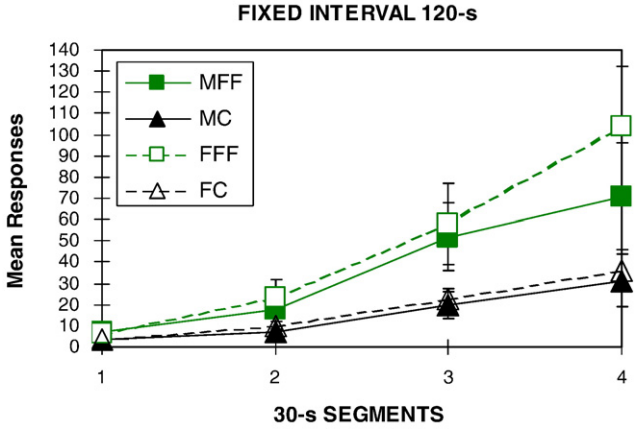

Fig. 4. Mean response in male and female rats to a bar-pressing task where the water reward was delivered only after a delay of 2 min. Control animals are compared to those exposed during gestation and through lactation to PCBs by feeding the dam contaminated fish. The fish-fed (FF) male (M) and female (F) rates pressed the bar much more frequently over the 2-min period than did the controls (C), indicating hyperactivity and impulsiveness. Error bars are +/− SEM. From [46].

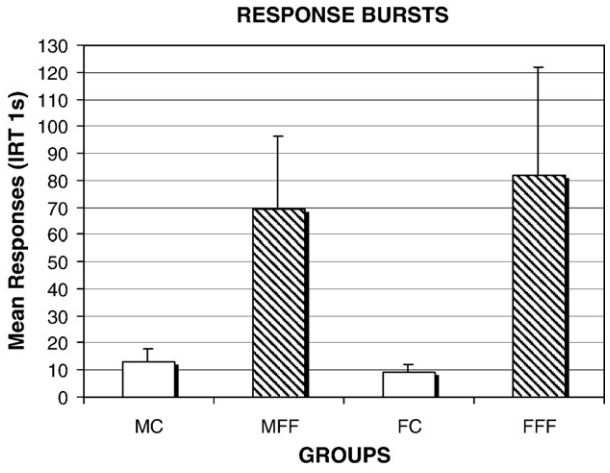

Fig. 5. Number of burst responses (responses with an inter-response interval of less than 1 s) in control and fish-fed male and female rats from the same study as in Fig. 4. These response bursts are an indication of frustration. Error bars are +/− SEM. From [46]. MC = Male Control. MFF = Male Fish Fed. FC = Female Control. FFF = Female Fish Fed.

reasonable to hypothesize that there are common mechanisms for all of these agents, even though they have very different chemical structures.

5. The possible association between IQ and violence

Lower intelligence has been correlated with a variety of risks. Increased childhood intelligence has been found to be associated with a significantly reduced risk of generalized anxiety disorder [47]. Macklin et al. [48] reported that Vietnam veterans with lower intelligence were more likely to develop posttraumatic stress disorder than those of higher intelligence. Hemmingsson et al. [49] found that cognitive ability at ages 18–20 was inversely correlated with increased mortality from cardiovascular disease, violence and alcohol-related causes over a thirty year period. Kandel et al. [50] showed that a higher IQ was protective against risk for serious criminal behavior among Danish men who were at high risk of such criminal behavior.

In their now infamous book, *The Bell Curve*, Herrnstein and Murray [51] present the data shown in Fig. 6, which relates the risk of incarceration to IQ among white males in the 1979–1990 National Longitudinal Survey of Youth (NLSY), a representative survey of American youths born in the late 1950s to mid-1960s (ages 14 to 22 in 1979). The percent of white NLSY males who acknowledged being incarcerated at some time prior to their 1980 NLSY interview shows a much higher risk among those in the lowest 25% of the IQ distribution (IQ<90). The percent who actually had one of their annual NLSY interviews from 1979 to 1990 conducted in a correctional facility shows an even higher incarceration risk for the lowest 5% of the IQ distribution (IQ<75). Herrnstein and Murray note that the probability of a correctional facility interview reflects both the risk of being incarcerated and duration of time served. If time served reflects offense severity, then these data suggest that the lowest 5% of the IQ distribution is especially likely to engage in more serious, violent offenses. The authors also showed that white male incarceration risk had a much stronger relationship with IQ than with family background or socioeconomic status.

Herrnstein and Murray interpret these data as evidence that low IQ causes criminal offending, and argue that the data for white youths suggest that racial differences in offending rates are largely explained by racial differences in inherited IQ. One serious flaw in this interpretation is that IQ is relatively stable after adolescence [52], while offending rates fall sharply with age. *The Bell Curve* also

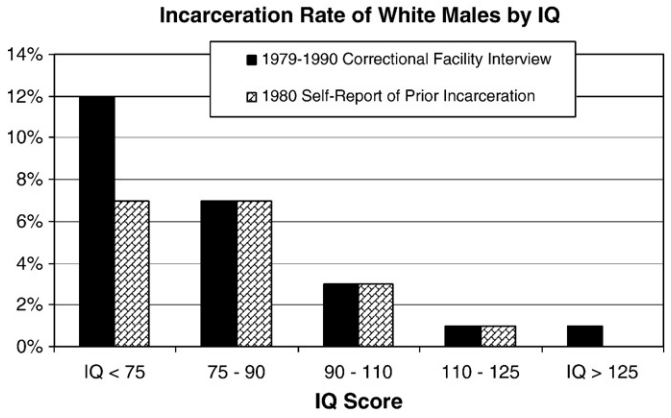

Fig. 6. The 1979–1990 relation between IQ and white male incarceration risk (data derived from [51]). Data from the 1979 to 1990 National Longitudinal Survey of Youth (NLSY), a representative sample of American youths born in the late 1950s to mid-1960s, showed that less than 1% of white males with IQ over 110 reported being incarcerated before 1980, vs. 7% of those with IQ below 90. White males with IQ below 75 had an especially high prevalence of having one or more of their 1979–1990 annual NLSY interviews actually conducted in a correctional facility, suggesting a much higher risk of more serious (violent) offending among those in the lowest 5% of IQ.

acknowledges that inherited IQ cannot explain large changes in national crime rates over time. Moreover, the steep decline in crime rates since the 1994 publication of *The Bell Curve* has been led by an even steeper and stunning decline in black juvenile offending. The black juvenile homicide offending rate fell by 83% from 1993 to 2003.

While Herrnstein and Murray interpret their data in relation to race and genetics, their results could well be explained by especially large disparities in preschool lead exposure during the NLSY birth years of the late-1950s to mid-1960s. The peak in MR prevalence was associated with these NLSY birth years and with the same IQ threshold (IQ<75) that Herrnstein and Murray [51] linked to a much higher risk of more serious (violent) offenses. Low IQ has been found to be a consistent risk factor for antisocial behavior "in both prospective and cross-sectional studies, even when other relevant risk factors are statistically controlled" [53], but genetic inferences derived from such studies overlook the research on lead poisoning and delinquency. Dietrich et al. [54] found that self-reported delinquent and antisocial behaviors were significantly associated with pre- and postnatal lead levels. Needleman et al. [55] determined bone lead levels in194 youth ages 12–18 who had been arrested and found to be delinquent in Allegheny County, PA, and 146 nondelinquent controls. They found that the mean concentration of lead in their bones was much higher than that in controls (11.0 ± 32.7 vs. 1.5 ± 32.1 ppm). Wright et al. [56] recruited pregnant women in Cincinnati, Ohio who resided in areas of the city with older and lead-containing housing. They determined prenatal maternal blood lead concentrations, and childhood blood lead concentrations up through 6.5 years of age. They found that arrest rates were greater for each 5 μg/dl increase in blood lead concentration. For prenatal blood lead the RR = 1.40 (95% CI = 1.07–1.85). For average childhood blood lead the RR = 1.07 (95%CI = 0.88–1.29). For 6-year old blood lead level the RR = 1.34 (95%CI = 1.15–1.89).

Nevin [57] has analyzed historic US rates of violent crime and related the changes in rates over time to the use of lead in gasoline. These results are shown in Fig. 7, from Nevin [39]. The upper plot shows the total tons of lead added to US gasoline per 1000 population on the left axis, and the violent crime rate per 100,000 persons on the right axis as a function of time, with the crime rate shifted by a 23 year lag. The curves overlap very well. This is consistent with the hypothesis that the amount of lead added to gasoline in the US is a surrogate measure of exposure to US children, and that early life exposure of US children resulted in an increase in risk-taking and violent behavior in later life, with the peak arrest rate occurring 23 years later. The lower plot shows arrests per 100,000 against age, demonstrating the striking increase in rates of arrest between 1980 and 1994, with a decline by 2001. In addition the peak age for arrests moved to older age over these periods, again consistent with the conclusion that children born after the early 1980s were less likely to commit crimes.

Preschool blood lead trends also appear to explain most of the substantial variation in property and violent crime rates in the USA, Canada, Britain, Australia, New Zealand, West Germany, France, Italy, and Finland across several decades [39]. Crime rates track blood lead with similar time lags within each nation, consistent with neurobehavioral damage in the first year of life: A 23-year lag for violent crime, consistent with the typical age of violent offenders, and an 18-year best-fit lag for burglary, consistent with the typical age of property crime offenders. The same time lag within each nation appears to explain divergent crime trends across nations. A shift in peak offending is also evident in Britain, where males ages 12–14 had the highest 1958 arrest rate but peak offending shifted to age 18 by 1997. The 1997 arrest rate was lower for males under age 14, born after the mid-1980s fall in British gas lead use, but 1997 arrest rates were much higher for older teens and adults born over years of rising gas lead use. The age-10 offense rate fell 70% from 1958 to 1997, as age 18–29 offending rates in Britain increased three to five fold.

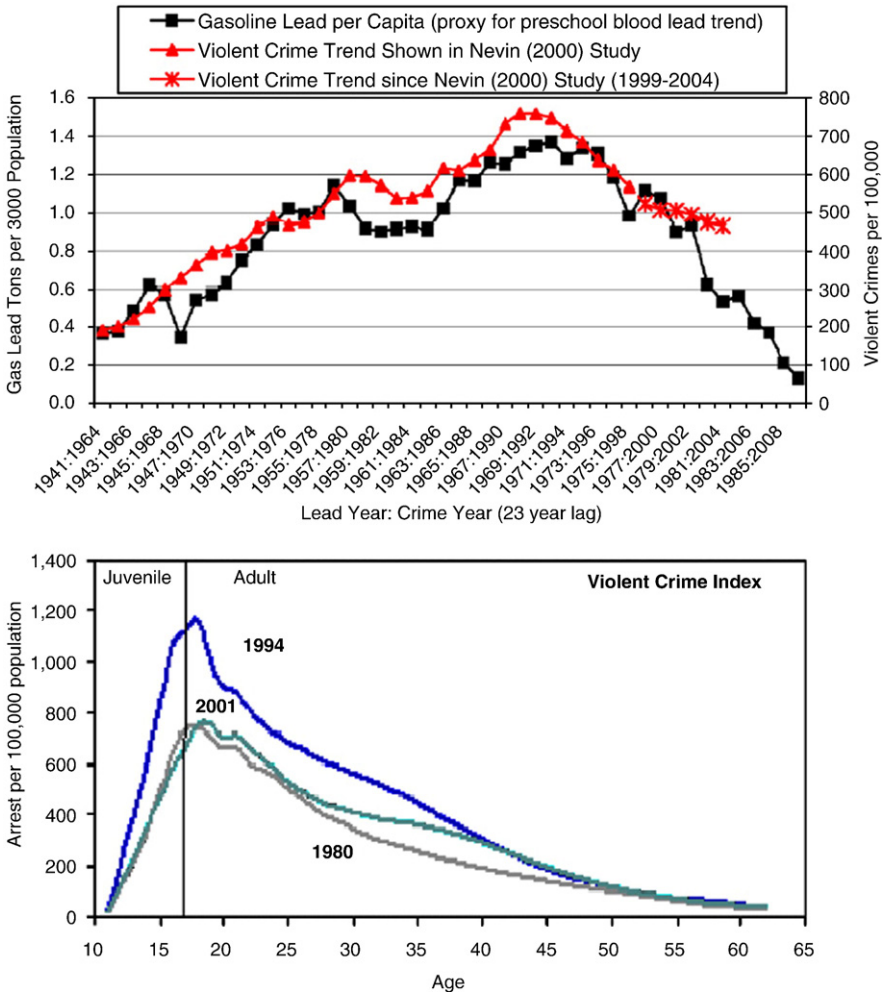

Fig. 7. USA violent crime and lead exposure trends and age-specific violent crime arrest rate shifts. In the upper plot gasoline lead per capita (left axis) and violent crime rate per 100,000 (right axis) are plotted against time, with the crime rate shifted for a 23 year lag (data from 55). The lower plot shows age-specific violent crime arrest rates (data from the Office of Juvenile Justice and Delinquency Prevention, 2004) and shows that the peak offending age shifted to older ages by 2001, as the 1990s violent crime decline was associated with an especially sharp decline among juveniles born after the early 1980s decline in gas lead levels. Reproduced from [39] with permission.

Information on contaminants and criminal behavior is currently only available for lead exposure, but for all of the reasons discussed above we hypothesize that further study will show similar effects of the other contaminants that act to shorten attention span and decrease ability to deal with frustration.

6. Correlation, causation, consistency, and coherence

Some of the evidence reviewed above comes from ecologic studies. It is well known that such studies are vulnerable to the "ecologic fallacy" of coincidental correlations that cannot by themselves demonstrate a causal relationship. Ecologic evidence is important, however, in the context of extensive animal and human research that clearly demonstrate the biological plausibility of lead-induced impacts, especially during critical brain growth before age two, and controlled studies showing lead-induced impairments in IQ, learning, and behavior [58,59]. In this context, ecologic trends can demonstrate time-precedence, consistency, and coherence that are at least indicative of causation [60]. At a minimum, time-precedence asks if the suspected cause preceded the effect, but ecologic trends reviewed here also show statistical best-fit time lags for MR and crime trends consistent with neurobehavioral damage in the first year of life. The relationship between crime trends and blood lead trends in different nations is also extremely consistent. The USA ecologic trends in MR, crime, and preschool lead exposure are also coherent with studies showing a high correlation between low IQ and criminal offending, and controlled studies showing an association between IQ, delinquency, and preschool lead exposure.

Our conclusions that environmental exposures contribute to violent behavior are not meant to discount other factors. Clearly many children exposed to contaminants during development do not go on to become criminals. 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. There is also a potential interaction between environmental exposures affecting neurodevelopment and social factors that are correlated with both criminal offending and with environmental exposures. Our point, however, is that while SES, genes, poverty, low IQ and other factors have been widely discussed in relation to violent behavior, there has been little, and totally inadequate, attention given to the role of early life environmental exposures.

One of the major limitations of ecologic data is the inability to control adequately for confounders, but highly correlated confounders also present an analytical challenge for controlled epidemiologic studies. Preschool lead exposure has been highly correlated with poverty and race because poor children are more likely to live in older housing with deteriorated lead paint and African-Americans were disproportionately concentrated in cities when leaded gas use caused severe urban air lead exposure. For this reason, it has often been asked

if the impact of lead exposure on IQ and delinquency is just a reflection of inadequate controlling for confounders. Ecologic trends suggest otherwise. Nevin [39] notes that USA juvenile offending surged in the 1960s, tracking birth year trends in lead exposure, even as the percent of children living in poverty fell sharply over the 1960s. Needleman [55] found that youths with high bone lead are twice as likely to be delinquent, after controlling for confounders, but single-parents and black race also raised delinquency risk for youths with lower bone lead. The 83% fall in the black juvenile murder arrest rate from 1993 to 2003 provides a new perspective on these findings, because the trend is obviously not explained by race, and 36% of black children lived in two-parent families in 1993 and in 2003. Separate stories in *The Washington Post* [61] and *The New York Times* [62] have noted that criminologists are mystified by ongoing large declines in USA crime rates through mid-2009, amidst a recession often described as the worst since the Depression. As economic conditions and other social confounders literally fail the test of time in predicting crime trends, it is reasonable to reverse the usual question about confounding: Has research linking criminal behavior to race, poverty, and family status adequately controlled for the confounding effect of early life environmental exposures?

7. Social and legal implications of these findings

In 1996 there was an interesting article in the New Yorker Magazine by Gladwell [63], who asked "Why is the city suddenly so much safer — could it be that crime really is an epidemic?" Nevin [39] observes that the especially large decline in the New York City crime rate was presaged by an earlier especially sharp decline in preschool lead poisoning prevalence in New York City. There is the widespread belief that criminal behavior is the result of character defects and willful voluntary actions for which society does not approve. Our observations suggest that at least one factor leading to criminal behavior is early life exposure to chemical contaminants that cause irreversible alteration in brain function and behavior, making the individual more likely to take risks and less able to deal with the frustrations of life. In this regard crime may really be an epidemic, but an epidemic caused by high exposure of a significant fraction of the population to dangerous environmental chemicals. There is without question clear evidence that criminal behavior is more common in populations that are poor, often are minority, often individuals who grew up in inner cities where housing and education are below the standards found in suburban communities. The point is that these are also the areas which are more contaminated, leading to exposure to contaminants associated with reduced IQ and behavioral changes.

Lead poisoning remains a problem in the US in spite of the major progress made over the last 40 years in removing lead from gasoline and paint, and is an even greater problem in many developing countries, some of which still have leaded gasoline. Lead is still found in many old homes in the US as a result of use of leaded paint. The soils along major roads still contain the lead that was emitted from automobiles and trucks in the past. Poor people and minorities more commonly live near hazardous waste sites, which results in exposure to a variety of hazardous chemicals. Sergeev and Carpenter [64] reported data on income and race in relation to residence in zip codes containing hazardous waste sites in upstate New York. For residence near hazardous waste sites containing persistent organic pollutants such as PCBs, families in the lowest quartile of median family income were almost twice as likely to live near a hazardous waste site as in zip codes that did not contain any hazardous waste site. While Caucasians were less likely to live in contaminated zip codes, African-Americans were 50% more likely to live near waste sites than in clean areas. This and other studies [65–69] comparing residence near to hazardous waste sites have shown that simply living near these sites results in an increased risk of a variety of disease secondary to inhalation of these semi-volatile compounds.

Exposure to lead and PCBs in the general US population has declined markedly since lead was removed from new paint and gasoline in the 1970s and 1980s and since manufacture of PCBs was stopped in 1977. However segments of the population remain more highly exposed. These are individuals living in old housing that has not been remediated, or near hazardous waste sites containing dangerous chemicals. We have new, recent standards for reducing levels of arsenic in drinking water. There has not, however, been a comparable decline in exposure to methyl mercury, for which exposure comes primarily from consumption of fish.

The evidence presented above indicates that at least some criminal and anti-social behavior is secondary to exposure to chemicals during the period of life when the brain is developing. The very possibility that early life exposure to environmental contaminants predisposes to a life of violence and other criminal offending raises difficult ethical and legal questions. An American Psychological Association [70] brief opposing juvenile executions cited MRI research showing that the adolescent brain "has not reached adult maturity", and presented an age-specific violent crime arrest rate graph, also shown in the lower half of Fig. 7, to show that violent offending declines with age. This brief did not note the shift in peak arrest rates, evident in that graph, that argues against an arbitrary demarcation at "under age 18": The violent crime arrest rate for 17-year-olds was 27% higher than the rate for 21-year-olds in 1994, but 7% lower in 2001; 17-year-olds in 2001 were born after a steep early-1980s decline in air lead, whereas 21-

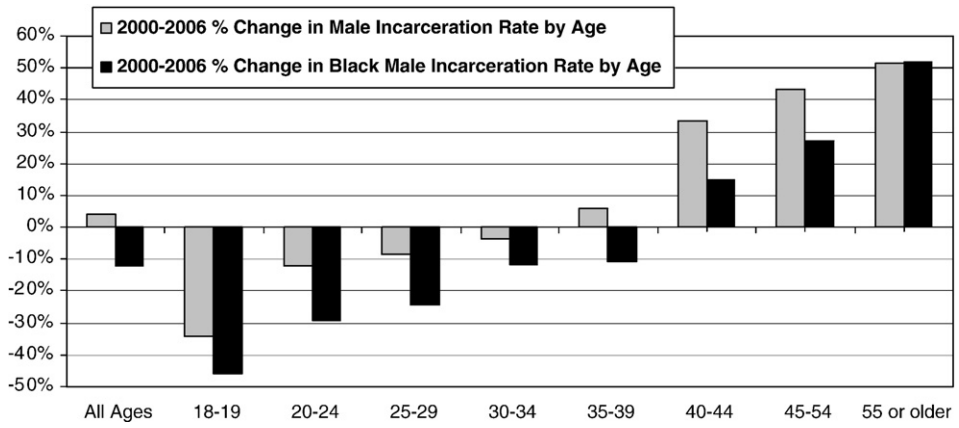

Fig. 8. 2000–2006 percent change in age-specific male incarceration rates (from [28] with permission). Declining incarceration rates from 2000 to 2006 for males under 30 reflect declining preschool blood lead since the mid-1970s. Incarceration rates are still rising for those over age 40, born when childhood lead poisoning was epidemic. The overall age 30–39 male incarceration rate rose slightly from 2000–2006, but the age 30–39 rate for black males fell 12%, reflecting slum clearance birth years also associated with a racial convergence in MR prevalence and juvenile burglary arrest rates.

year-olds in 2001 were born before that decline. The lead poisoning literature, shifts in age-specific peak offending rates, MRI brain growth research, and trends in preschool blood lead, MR prevalence, and crime rates all suggest that age, IQ, and preschool lead exposure are associated with a dose–response continuum of "diminished capacities" that could help to explain criminal offending risks, with no basis for drawing a precise demarcation at "under 18" or IQ below any specific threshold.

Recent USA incarceration rate trends are consistent with lead exposure trends, resulting in a prison population increasingly dominated by older, life-course-persistent offenders. Fig. 8 shows incarceration rates fell from 2000 to 2006 for all men under age 30, consistent with the decline in preschool blood lead since the mid-1970s, but incarceration rates are rising for men over 40, born when lead poisoning was epidemic. The overall 30–39-year-old male incarceration rate rose slightly from 2000 to 2006, but the 30–39-year-old rate for black males fell 12%, reflecting the same slum clearance birth years associated with a racial convergence in MR, SAT and NAEP scores, and juvenile burglary arrest rates.

What is society's responsibility for disproportionate exposure of certain segments of the population to dangerous chemicals? Should some criminal behavior be considered more of an illness than a voluntary act? Is our criminal justice system fair and appropriate or should criminal behavior be treated more like mental illness? And most importantly, is it possible that the most effective way to fight crime is to reduce exposure of all of the population to chemicals that alter brain development?

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