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Environmental Injustice: Childhood Lead Poisoning, Teen Pregnancy, and Tobacco

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Abstract

Purpose: This study investigates the persistent relationships between childhood lead exposure, repeat teen pregnancy, and tobacco use in a sample of teenage females in Syracuse, NY.

Methods: We analyzed the association of childhood lead poisoning with repeat pregnancy and tobacco use among 536 teens (aged 15–19 years) in Syracuse, NY, who received services at Syracuse Healthy Start between 1998 and 2002.

Results: The mothers' childhood lead exposure, controlling for race, age, and Medicaid status, was associated with repeat teen pregnancy and tobacco use.

Conclusion: Long-term negative health outcomes associated with childhood lead exposure should not be underestimated. This study helps to shore up prior research that found lead poisoning to have a long-lasting impact on children's functioning and healthy development. Policy efforts focused on neighborhood development and health education continue to be sorely needed.

Keywords: Lead poisoning; Teen pregnancy; Tobacco use; Smoking; Health disparities; Environmental injustice

Although the rates of teen pregnancy in the United States have been on the decline, many teens who give birth are likely to experience additional health problems and to have repeat pregnancies. Impoverished neighborhoods in which many pregnant teens reside contain poorly maintained housing, with lead exposure risks that help to perpetuate racial and ethnic disparities in health status throughout the U.S. Living in areas with broken windows and dilapidated housing can be indicative of neighborhoods with high crime (resulting in increased stress levels and associated effects), poverty (lack of funds to buy nutritious foods, leading to obesity, diabetes, and poor birth outcomes), and poor access to health care [1]. In addition, tobacco use is one of the most important preventable public health risks [2].

Among the environmental exposures leading to negative health outcomes is lead poisoning, which disproportionately affects impoverished children living in urban areas. Chil-

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dren of color are documented to have considerably higher levels of lead poisoning than children of white ethnicity [3]. From 2000–2001, the prevalence of elevated blood lead (EBL) in the children of Onondaga County was the second highest in New York State outside of New York City. Five zip codes in the city of Syracuse, Onondaga County's seat, accounted for 76% of the county's total childhood lead poisoning and 7.7% of the entire incidence of elevated blood lead in New York State children [4].

The greatest likelihood of exposure to lead comes from old lead-based paint in dilapidated buildings, especially around windowsills and doorframes. These windows emit powdery paint dust and chips each time they are opened or closed [5]. To a lesser, but still important extent, lead is found in the soil surrounding houses with exteriors that have peeling lead-based paint [6]. Finally, lead-tainted water, from corroded lead solder in old copper pipes, potentially also contributes to children's exposure to lead [7].

Over time, researchers have identified health consequences resulting from lead exposure at lower levels. Before 1970, a level of 60 μg/dl was considered dangerous; by 1985 the level for concern had dropped to 25 μg/dl, and in 1991 it was changed to 10 μg/dl [4]. A longitudinal study of 172 children who were followed from 6 months to 5 years of age found that IQ, as measured by the Stanford-Binet Intelligence Scale, fell 7.4 points as blood lead rose from 1 μg/dl to 10 μg/dl [8]. Lead exposure and other health disparities are estimated to account for nearly one-quarter of the racial gap in school readiness [9].

The current literature on lead's harmful effects has largely focused on lower level exposure ($<$10 μg/dl) [10]. A review conducted on these current studies by the Centers for Disease Control and Prevention (CDC) came to the following conclusions. First, lead levels $<$10 μg/dl are associated with negative cognitive effects among children, and the evidence suggests a causal relationship, but the size and shape of this relationship is not known because of methodologic limitations to studies and the confounding of socioeconomic status. Second, the evidence shows consistent associations between low levels of lead in children and other negative health outcomes, identified as other neurologic function, stature, sexual maturation, and dental problems [11].

Lead entering the bodies of very young children affects their developing brains in ways that continue to influence their behavior for many years and thus becomes a cumulative environmental injustice [5]. Neurotoxicity from lead poisoning affects not only cognitive capacity but the ability to plan, learn from prior experience, and control impulsive behavior, and creates impairments that are collectively termed deficits in "executive function" [12]. Observational studies with exposed children have found associations between lead exposure and what has been variously termed impulsive behavior, delinquency, and aggressive/anti-social behavior [13–15]. According to Bellinger, the neurotoxic effects of lead on children's development appear to be irreversible [5]. These developmental insults, Bellinger argues, need to be measured in real-world behavioral patterns, rather than on cognitive or psychological tests administered in artificial settings [16,17]. Adults who are occupationally exposed to lead also exhibit neurobehavioral deficits, including diminished cognitive ability and executive function [18–20]. Experimental animal studies have demonstrated early lead exposure to be a risk for impulse control, failure to delay gratification, and increased sensitivity to drug-seeking behavior [21,22]. A possible biological pathway for lead's promotion of maladaptive behavior has been identified in experimental trials in which lead-dosed rat pups developed disruptions in their neurochemistry [23–25].

Most of the research on the effects of lead exposure on cognitive functioning has been limited to adolescent and adult males and thus has not addressed the effects of the exposure on other health outcomes such as teenage pregnancy and tobacco use [18–20,26]. The study described in this article in part fulfills Bellinger's recommendation to investigate the lived experience of persistent effects of childhood lead exposure and the neighborhood context in which it occurs on teen pregnancy and tobacco use. The purpose of this study is to analyze the effect of childhood lead poisoning on teen pregnancy and tobacco use. We hypothesize that among females, childhood lead poisoning is associated with repeat teen pregnancy and cigarette smoking.

Methods

This research project takes a multilevel methodological approach to health problems and integrates epidemiological methods to analyze the effects of elevated blood levels on teen pregnancy and tobacco use in Syracuse, NY [27]. Syracuse is the fifth largest city in New York State, with a 2000 population of 147,306, which comprises 64.3% white, 25.3% African-American, 3.4% Asian, 1.1% Native American, and 3.4% individuals of two or more racial ancestries of whom 5.3% are Hispanic. According to the 2000 U.S. Census, nearly 43% of Syracuse children $<$5 years of age live in poverty; the poverty rate for African-American children (57%) is more than double that for white children (27%). Syracuse has New York State's third highest child poverty level, following that of Buffalo and Rochester, and the second highest Latino child poverty rate in the United States [28]. Using data from the Onondaga County Health Department Lead Program for 1992 to 1995, Griffith et al found that the mean lead level among children in Syracuse was 7.38 μg/dl for white children, 11.35 μg/dl for African-American children, and 9.83 μg/dl for Hispanic children [29]. From 2000–2003, elevated blood lead ($\geq$10 μg/dl) was identified in 10.8% of white children (425 of 3940 tested) and 22.7% of African-American children (1112 of 4899 tested) in Onondaga County (Data provided by the Onondaga County

Health Department). A similar racial disparity exists in the teen (15–19 years of age) birth rate in Syracuse (2000–2001); per year, 32.9 per 1,000 white teens gave birth, compared with 91.7 per 1,000 African-American teens (Birth data are from NYS vital records and population data are from the U.S. Census; calculations are by the authors).

This analysis used data from the Syracuse Healthy Start (SHS) participants' database [30], which monitors the receipt of preventive care of pregnant women of all ages. SHS is an infant mortality prevention project of the Onondaga County Health Department (OCHD) that has been funded by the Health Resources and Services Administration since 1997. Briefly, SHS began enrolling participants in mid-1998 and at that time began to monitor the participants' receipt of services and screening. As a part of routine screening, the SHS staff looks up the childhood lead levels in archived paper files in the OCHD. If the participant's childhood lead level is elevated, SHS staff reports this finding to the woman's obstetrician. SHS staff also complete a risk-screening questionnaire with participants that asks about tobacco use, to make a plan for intervention. This analysis is restricted to those aged 15–19 years who were enrolled in SHS from 1998 to the first quarter of 2002. The teens in this sample were born after the phase-out of leaded gasoline and after the ban on lead-based residential paint. During the period covered in this analysis, SHS enrolled over three-fourths of all mothers aged <20 years in Syracuse.

Measures

The pregnant teens in this sample had ranged from 0–24 months of age during the years 1981–1989, at which time a blood-lead level of <25 μg/dl was considered "acceptable" by the CDC [7]. In the lead records on file in the Onondaga County Health Department during these years, if a child's lead level was <20 μg/dl the staff wrote, "OK" rather than the exact number. If the child's blood lead level was ≥20 μg/dl, the staff recorded the exact number. In cases in which participants had more than one childhood lead level on file, we used the highest lead level recorded. As part of routine screening, SHS participants were asked about tobacco use early in their pregnancy and were offered assistance with reducing or quitting smoking. Participants were also asked about their previous pregnancies. If a participant had a previous pregnancy, the current pregnancy is called a "repeat teen pregnancy." Maternal age was calculated by subtracting the maternal date of birth from the expected date of her baby's birth. Maternal race/ethnicity was collected through self-report upon enrollment into SHS. Because of the small numbers of individuals of other races and ethnicities, subsequent analyses use data only for white and African-American females. All variables were coded as dichotomous: childhood blood lead level (<20/≥20 μg/dl), tobacco use (none/any), previous pregnancy (first/repeat pregnancy), maternal age (15–17/18–19 years), and maternal race (white/African-American).

Statistical analysis

Descriptive analyses were conducted on the data for teens in the SHS database with regard to childhood lead level, maternal age, Medicaid insurance, number of pregnancies, and tobacco use. Separate bivariate associations were assessed between childhood lead level (the exposure) and maternal age, Medicaid insurance, number of pregnancies, and tobacco use. Multivariate logistic regression was conducted using childhood lead level as the exposure, repeat teen pregnancy or tobacco use as the outcomes, and using maternal race, maternal age, and Medicaid use as potential confounders. Before conducting the logistic regression, we assessed effect measure modification (EMM) and confounding. To check for EMM, we used the Breslow-Day test for homogeneity, with an α level of .20 as the cutoff [31]. Potential confounding by maternal race, maternal age, or Medicaid was examined to determine whether these variables were related to either the outcomes (repeat pregnancy or tobacco use) or the exposure (elevated childhood blood lead). Once EMM and confounding were assessed, appropriate confounders were controlled for in the logistic model [31].

Results

Of the 1111 pregnant teens (aged 15–19) served by SHS, 719 (64.7%) had been tested as children and had blood lead levels on file with the county health department, of whom 26.7% were white, 48.4% African-American, 5.7% Latina, 1.7% Native American, .8% Asian, and 16.7% unknown/other. Among the 536 white and African-American pregnant teens, 71.6% had childhood lead levels <20 μg/dl, and 28.4% had levels ≥20 μg/dl (13.8% had levels 20–29 μg/dl, 9.0% had levels 30–39 μg/dl, and 5.6% had levels 40–99 μg/dl). Likewise, among this group, 64.6% (346/536) were African-American, 47.0% (252/536) were 18–19 years of age, 76.1% (408/536) were on Medicaid, 39.6% (183/462, data missing for 74) were experiencing their second or higher pregnancy, and 37.5% (201/536) were smokers.

Table 1 presents the lead levels of the pregnant teens by number of pregnancies, smoking, maternal age, Medicaid vs. private insurance, and maternal race. Repeat pregnancy was associated with elevated childhood lead and mother's age (older teens were more likely to have a repeat birth) ($p < .05$). Tobacco use was also significantly associated with maternal race (white teens smoked more than African-American teens) ($p < .05$).

We performed two separate logistic regression analyses with childhood lead level as the exposure, or risk factor, as presented in Table 2. In the first analysis repeat birth was the outcome variable (Model 1) and in the second tobacco use

Table 1
Baseline characteristics of pregnant teens by childhood blood lead levels, Syracuse, New York (1998–2002)

Characteristic	Lead level (N = 536)	
	<20 μg/dl (n = 384)	≥20 μg/dl (n = 152)
Repeat pregnancy (N = 462)		
First pregnancy	74.6% (N = 208)	25.4% (N = 71)
Second or higher-order pregnancy	65.0% (N = 119)	35.0% (N = 64)
Tobacco use during pregnancy (N = 536)		
Nonsmokers	74.9% (N = 251)	25.1% (N = 84)
Smokers	66.2% (N = 133)	33.8% (N = 68)
Maternal age, y (N = 536)		
15–17	70.8% (N = 201)	29.2% (N = 83)
18–19	72.6% (N = 183)	27.4% (N = 69)
Medical insurance (N = 536)		
Private insurance	66.4% (N = 85)	33.6% (N = 43)
Medicaid	73.3% (N = 299)	26.7% (N = 109)
Race (N = 536)		
African-American	74.7% (N = 142)	25.3% (N = 48)
White	69.9% (N = 242)	30.1% (N = 104)

Percentages are summed across the row.

was the outcome variable (Model 2). In each logistic regression, we controlled for confounders of maternal race and mother's age. Receipt of Medicaid was a confounder of Model 1 and an effect modifier of Model 2, resulting in stratified effect estimates. The analyses found childhood lead poisoning to be a risk factor for subsequent repeat birth among teens and for tobacco use. Among those not covered by Medicaid, the odds of smoking among those with an elevated childhood lead level was 4.25 (95% CI 1.89, 9.57) times higher than the odds of smoking among those with an acceptable lead level, controlling for age and race. Among those receiving Medicaid, the odds of smoking was 1.26 (95% CI 0.79, 2.03) times higher among those with an elevated childhood lead level compared to those with an acceptable lead level, controlling for age and race. Controlling for maternal age, race and Medicaid use, the odds of having a second pregnancy among those with an elevated childhood level was 1.59 (95% CI 1.04, 2.43) times the odds of having a second pregnancy among those with an acceptable lead level.

Discussion

The notion that the built environment affects health outcomes has been well documented [32]. This paper indicates a potential link between a key aspect of the dilapidated built environment—namely, lead poisoning—and subsequent health outcomes years later among young women, who are more likely to have multiple pregnancies and to smoke cigarettes than female teens without a history of elevated lead levels.

Repeat pregnancy

Most of the analyses linking prior lead exposure to deficits in executive functioning have been conducted with adolescent and adult males [18–20,26]. Among lead-poisoned adolescent males, poorer planning ability, the reduced capacity to learn from prior behavior or to delay gratification contribute to higher rates of socially deviant, and sometimes law-breaking, behavior. Teenage girls, however, are much less likely to be arrested or labeled "delinquent." For example, in Onondaga County (1995–1999) among teens aged 14–18 years, 83% of all convicted offenders were male [33]. A key area in which executive function could potentially influence the lives of teenage females is choices about their intimate partners, including when and with whom to express their sexuality, and the effective use of both contraception and protection from sexually transmitted infections. An important caveat in applying the concept of executive function to teen sexuality is that many births to younger teens are the result of coercive sex. Research has demonstrated that about half of the babies of younger teens are fathered by older male partners, who may pressure their partners into sexual relationships before they are ready and may also control condom use decisions [34]. Other variables associated with teen pregnancy include factors at the community (high unemployment and community stress levels), family (single parents), and individual (African-American race, Hispanic ethnicity, school dropout, greater number of sexual partners) levels [35].

Table 2
Logistic regression results (odds ratios and 95% confidence intervals) for models using repeat pregnancy and tobacco use as outcome variables

Predictor variables	Model 1: Repeat pregnancy (n = 462)	Model 2: Tobacco use	
		Medicaid nonusers (n = 128)	Medicaid users (n = 408)
Childhood lead level (20+ μg/dl vs. 0–19 μg/dl)	1.59 (1.04, 2.43)	4.25 (1.89, 9.57)	1.25 (.78, 2.00)
Mother's race/ethnicity (African-American vs. white)	1.46 (1.25, 1.71)	.32 (.15, .72)	.29 (.19, .45)
Mother's age, years (18–19 vs. 15–17)	1.45 (.95, 2.21)	.93 (.42, 2.05)	1.35 (.88, 2.05)
Medicaid (Medicaid vs. private insurance)	1.70 (1.06, 2.73)	—	—

Data in parentheses are 95% confidence intervals.

These factors at the community and family levels may result in residence in older housing with peeling lead paint and could be a proxy for environmental factors contributing to higher blood lead levels among children in these areas.

Developmental delay is another risk factor that has been found to be associated with teen pregnancy. A nationally representative sample of females found that those with lower scores on the Armed Services Vocational Aptitude Battery (a test of arithmetic reasoning, math knowledge, word knowledge, and paragraph comprehension) were significantly more likely to have become mothers before age 20 years, regardless of racial or ethnic background, and had nearly three times the odds of a second birth in their teen years than those with higher cognitive ability [36]. Because lead poisoning potentially impacts both cognitive ability and executive functioning, these two factors may operate synergistically in reducing the teen's ability to avoid pregnancy. Teen women with potentially weaker cognitive or judgment capacity, secondary to lead poisoning, may be less able to perceive whether potential partners sincerely care about their welfare or are manipulating them. Reduced cognitive competence and diminished ability to plan ahead and to be mindful of consequences may also play a role in the effective and consistent use of birth control.

Studies have shown that increased knowledge about pregnancy prevention does not always result in behavior change that decreases pregnancy risk. Likewise, increased access to contraception does not necessarily lead to increased condom use, subsequently reducing teen pregnancy rates [35]. This study may add more information to the overall picture of why young women are not heeding information about protective sexual behaviors.

Lead and propensity for addiction

Although smoking and race have been found to be determinants of blood lead level among women of reproductive age [37], our study is the first to investigate an association in the opposite direction—namely, between elevated childhood lead and cigarette smoking in teen years. Potentially related findings in animal studies appear to be pertinent in evaluating the potential for early lead exposure to increase sensitivity to tobacco addiction: early lead exposure has been associated with a decreased ability to delay gratification [21] and with alterations in the dopamine system and other mood-regulating neurochemicals [23–25,38]. These two factors suggest biological plausibility that early lead exposure could increase sensitivity to tobacco addiction.

Additional research has shown that small corner stores in inner-city Syracuse neighborhoods sell individual cigarettes ("loosies") to minors, thus facilitating minors' potential addiction to tobacco. The 1999 Youth Risk Behavior Survey, conducted with all 10th grade students in Syracuse, revealed that 97.5% had tried cigarette smoking at least once; 24% had smoked within the past 30 days; and 9% reported smoking more than two cigarettes per day within the past 30 days [39]. These data demonstrate that an alarming proportion of Syracuse youth risk lifetime tobacco addiction. Even though nearly all youth have tried tobacco, only about one-quarter continue to smoke. Our findings indicate that childhood lead exposure may increase the possibility of continued tobacco use.

The findings from this study, specifically the lack of a significant association between lead levels and tobacco use among Medicaid users highlight the large role that poverty plays in this relationship. Studies have found economically poorer individuals are more likely to smoke, possibly because of lower educational levels and lack of awareness of the health hazards of smoking or because of the use of smoking to manage mood and stress related to lack of resources [40].

Individual risk versus social/environmental risk

Much of the policy focused on reducing teen pregnancy and smoking has been based on the so-called individual responsibility model, in which teens are bombarded with commercial and public service messages urging them to remain abstinent and smoke free [2]. The underlying assumption of these messages and policies is that teens have complete and conscious control over their behavior. Yet the published literature and the analyses presented in this article call attention to the role of childhood lead exposure in shaping subsequent behavior. Lead poisoning likely accounts for at least a portion of the poor school performance of children and may also explain part of the racial disparity in test scores. Our data demonstrate a significant association of childhood lead poisoning with subsequent repeat pregnancy and smoking among teens, both of which are enormous public health concerns. Teen pregnancy and lead poisoning are concentrated in five zip code areas in Syracuse with high lead exposure. These neighborhoods are where people of color were segregated after urban renewal because of discrimination in rental housing and in mortgage lending. The aftershocks of urban renewal thus continue to cause harm in the form of lead-laden, dilapidated housing. Further research should integrate community-level factors into the analyses to address the confounding effect of living in these zip code areas with high degrees of health disparities.

Study limitations

There could be many proximate factors that better explain the association found between elevated childhood blood levels and poor health outcomes measured 10–15 years later. Our small sample size could mask an even larger effect estimate. Other community factors such as poverty, community stress, residence location within Syracuse, and family level factors such as single-parent families could confound the relationship between childhood lead exposure and health outcomes in teenage years. Although several of

Sir Bradford Hill's Criteria of Causality such as temporality and biological plausibility have been met, this small contribution is not definitive. Instead, this research gives support to furthering the investigation of the lifetime effects of elevated blood lead levels among young children, toward decreasing the prevalence of poor health outcomes later on in life. Further research should include neighborhood and environmental determinants, as well as individual level factors, in multilevel models, to further illuminate the impact of childhood blood lead levels on behaviors such as repeat teen pregnancy and teenage smoking.

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