Title

The Role of Maternal Adverse Childhood Experiences and Race in Intergenerational High-Risk Smoking Behaviors.

Permalink

https://escholarship.org/uc/item/0186z4gs

Journal

Nicotine & Tobacco Research, 19(5)

ISSN

1462-2203

Authors

Pear, Veronica A
Petito, Lucia C
Abrams, Barbara

Publication Date

2017-05-01

DOI

10.1093/ntr/ntw295

Peer reviewed

Original investigation

The Role of Maternal Adverse Childhood Experiences and Race in Intergenerational High-Risk Smoking Behaviors

Veronica A. Pear MPH[1], Lucia C. Petito MA[2], Barbara Abrams DrPH[1]

[1]Division of Epidemiology, School of Public Health, University of California, Berkeley, CA; [2]Division of Biostatistics, School of Public Health, University of California, Berkeley, CA

Corresponding Author: Barbara Abrams, DrPH, Division of Epidemiology, UC Berkeley School of Public Health, 103 Haviland Hall, Berkeley, CA 94720-7358, USA. Telephone: 510-642-4216; Fax: 510-643-5163; E-mail: babrams@berkeley.edu

Abstract

Introduction: A history of adversity in childhood is associated with cigarette smoking in adulthood, but there is less evidence for prenatal and next-generation offspring smoking. We investigated the association between maternal history of childhood adversity, pregnancy smoking, and early initiation of smoking in offspring, overall and by maternal race/ethnicity.

Methods: Data on maternal childhood exposure to physical abuse, household alcohol abuse, and household mental illness, prenatal smoking behaviors, and offspring age of smoking initiation were analyzed from the US National Longitudinal Survey of Youth 1979 (NLSY79, *n* = 2999 mothers) and the NLSY79 Children and Young Adults Survey (NLSYCYA, *n* = 6596 children). Adjusted risk ratios were estimated using log-linear regression models. We assessed multiplicative interaction by race/ethnicity for all associations and a three-way interaction by maternal exposure to adversity and race/ethnicity for the association between prenatal and child smoking.

Results: Maternal exposure to childhood physical abuse was significantly associated with 39% and 20% increased risks of prenatal smoking and child smoking, respectively. Household alcohol abuse was associated with significantly increased risks of 20% for prenatal smoking and 17% for child smoking. The prenatal smoking–child smoking relationship was modified by maternal exposure to household alcohol abuse and race. There were increased risks for Hispanic and white/other mothers as compared to the lowest risk group: black mothers who did not experience childhood household alcohol abuse.

Conclusions: Mothers in this national sample who experienced adversity in childhood are more likely to smoke during pregnancy and their offspring are more likely to initiate smoking before age 18. Findings varied by type of adversity and race/ethnicity.

Implications: These findings support the importance of a life-course approach to understanding prenatal and intergenerational smoking, and suggest that maternal early-life history is a potentially important risk factor that could be targeted with screening and interventions to reduce smoking in pregnant women and their children.

Introduction

Pregnancy is an ideal time for cigarette smoking cessation, as women are often highly motivated to improve their health for the sake of their fetus and antenatal care allows ongoing interaction with the health care system. In 2008, fewer than half of American women smokers quit during pregnancy,[1] suggesting the need for more effective interventions. In addition to serious risks of chronic disease, smoking during pregnancy is associated with an increased risk of miscarriage and placental abruption, cleft lip/palette, preterm birth, low birth weight, and Sudden Infant Death Syndrome.[2]

Children exposed to tobacco in utero are more likely to initiate smoking before the age of 18, even when accounting for exposure to maternal smoking after birth.[3–5] Early initiation of smoking is linked to establishing an adult smoking habit and to a higher intensity of cigarette consumption.[6] It is estimated that 90% of adult smokers tried cigarettes before the age of 18,[7] and nearly one half of high school seniors have experimented with cigarette smoking.[8] Preventing youth smoking is imperative for public health.

While the proportion of women who smoke during pregnancy has declined substantially over the past few decades, from 25.1% in 1985[9] to 10.2% in 2011,[10] it remains much higher than the Healthy People 2020 goal of 1.4%.[11] In addition to established risk factors for prenatal smoking,[1,9] three recent studies suggest that a history of Adverse Childhood Experiences (ACE), potentially traumatic events and conditions that occur before the age of 18, also increases one's risk.[12–14] Furthermore, a recent study of two large US cohorts concluded that mothers with severe childhood abuse are significantly more likely to have children who follow a high-risk smoking trajectory of early initiation leading to high consumption.[15] Given the small evidence base to date, the relationship between ACE, prenatal smoking, and child smoking deserves replication.

Unfortunately, early life adversity is quite prevalent in the United States.[16] However, the impact that childhood adversity has on health varies considerably between people. An important part of this variability is due to differences in resilience, the ability to cope with stress in a healthy way.[17] Resilience, however, is threatened by racial discrimination,[18] especially in the absence of familial social support.[17,19] Since ACE often indicates disruption in the family or a lack of social support in childhood, racial discrimination on top of ACE could be particularly damaging to resilience. Therefore, childhood adversity might impact different racial groups in different ways, yet no studies have analyzed interaction by race/ethnicity on the relationship between early life adversity and smoking behaviors.

The present study of a nationally representative sample has three major goals: (1) to analyze the relationship between three measures of ACE and prenatal smoking, and assess interaction by race/ethnicity (2) to investigate whether childhood adversity is associated with next-generation youth smoking, measuring interaction by race/ethnicity, and (3) to determine if there is effect measure modification by maternal childhood adversity and race/ethnicity on the relationship between prenatal exposure to tobacco and early initiation of smoking.

Methods

Sample

The National Longitudinal Survey of Youth 1979 (NLSY79) is an ongoing, nationally representative cohort study that enrolled 12 686 individuals between the ages of 14 and 21 years in 1979. Participants were selected using a multistage stratified sampling procedure, which has been described in detail previously.[20] The NLSY79 cohort was interviewed annually from 1979 to 1994, after which the survey was conducted biennially. With sample weights, this cohort is representative of young adults residing in the United States in 1979.

All children of women in the NLSY79 cohort were invited to participate in the biennial NLSY79 Children and Young Adults survey (NLSYCYA), which began in 1986. This cohort represents over 95% of the biological children of NLSY79 women.[21] As of 2012, there were 11 504 participants.

For our analyses, we used all mother–child dyads with complete information on maternal ACE exposure, smoking status during pregnancy, offspring age at smoking initiation, and race/ethnicity. The eligible sample consisted of 3191 women who were interviewed in 2012, the year in which childhood adversity questions were added to the survey, along with their 7886 children. The final sample included 2999 women and 6596 children. Supplementary Figure 1 provides stepwise details on the exclusion process.

The UC Berkeley Committee for Protection of Human Subjects approved this study.

Measures

Adverse Childhood Experiences

Three questions from the ACE questionnaire[22] measuring childhood physical abuse, household alcohol abuse, and household mental illness were added to the NLSY79 survey in 2012; over 99% of women interviewed that year answered all three questions about childhood adversity. Childhood exposure to physical abuse was defined as being "hit, beat[en], kick[ed] or physically harm[ed]" more than once by a parent before the age of 18. Household alcohol abuse was defined as living with a "problem drinker or alcoholic" before the age of 18, and household mental illness was defined as living with someone who was "depressed, mentally ill, or suicidal" before the age of 18 (see Supplementary Table 1 for full question text).

Pregnancy Smoking Behaviors

Since 1983, at each wave of data collection, NLSY79 women have been asked about their smoking habits during the 12 months before a child was born, and since 2000, they have also been asked about the number of cigarettes they smoked during a given pregnancy. These data were collected retrospectively within 2 years of a pregnancy. We used reported smoking to define two categories of pregnancy smokers: nonsmokers (no regular smoking during pregnancy) and smokers (any regular smoking during pregnancy, such that the average number of cigarettes smoked per week was reported to be greater than or equal to one). Women who quit at the start of pregnancy were classified as nonsmokers, and women who quit during pregnancy were classified as smokers.

Youth Smoking Behaviors

Among children 10 years of age and older, age of smoking initiation in the NLSYCYA survey has been measured with a self-administered questionnaire since 1988. Children are asked whether they ever smoked and at what age they first smoked (Supplementary Table 1). Impossible answers, such as smoking initiation ages that were greater than the child's age at the time of the interview, were discarded (*n* = 28). For both questions, reported initiation ages were averaged across all available survey years for each child. We found that these reported initiation ages were very consistent (*r* = 0.91). Due to the high correlation between answers, each child's mean self-reported age at first cigarette was used as their initiation age, unless this age was equal to zero. Children with mean self-reported smoking initiation ages of zero who did not report ever smoking were treated as nonsmokers. If the mean age was zero but they reported having ever smoked, the age at which they first provided a "yes" response to the question "has child ever smoked?" was used as their smoking initiation age. Children were categorized as youth smokers if their initiation age was less than 18 years.

Covariates

All potential confounders were identified a priori using theory-based causal diagrams (Supplementary Figure 2).[23] Confounders of the relationships between ACE and smoking during pregnancy and ACE and next-generation youth smoking included the women's mothers' education,[24,25] in years of completed schooling; growing up in the south[26]; growing up in an urban area[27,28]; being born outside of the United States[29–31]; and birth decade.[9,32–34] All covariates were self-reported in 1979.

Potential confounders of the relationship between smoking during pregnancy and early initiation of smoking included all potential confounders listed above, the women's age at childbirth[9]; decade of childbirth[9]; education at child's birth year,[35,36] in years completed; marital status at child's birth year[37,38]; employment status at child's birth year[39,40]; and income at child's birth year,[35,36] equivalized to account for family size[41]; as well as the child's birth order.[42,43]

Race/ethnicity was considered a potential effect measure modifier in all relationships and was categorized as white/other, black, and Hispanic. Approximately 88% of the white/other group was white.[44]

Statistical Analysis

We first assessed differences between included participants and excluded, eligible participants. Then, we described the distribution of demographic and study characteristics across racial/ethnic groups. We also assessed the crude associations between each measure of childhood adversity and smoking during pregnancy, between childhood adversity and next-generation youth smoking, and between smoking during pregnancy and youth smoking. Survey-adjusted Rao-Scott chi-squared tests[45] were used to measure significance for categorical variables and generalized linear regressions were used for continuous variables.

Subsequently, we estimated adjusted risk ratios (*aRR*) to assess the associations between maternal childhood adversity and (1) any smoking during pregnancy and (2) next-generation youth smoking using weighted multiple Poisson regression models accounting for the complex survey design. Multiplicative interaction terms were included to assess whether these associations differed by race/ethnicity. Because black women had the lowest prevalence of childhood adversity and smoking outcomes, we chose to use them as the reference group in all race/ethnicity related analyses.

Our last model tested the association between maternal smoking in pregnancy and offspring early smoking initiation. We first measured multiplicative two-way interactions between prenatal smoking and race/ethnicity, and prenatal smoking and each measure of childhood adversity. We then assessed multiplicative three-way interactions between race/ethnicity, maternal ACE, and prenatal smoking using separate models for each ACE exposure. The significance levels of all interactions were determined with a survey-adjusted Wald test.

As the excluded participants had significantly different sociodemographic characteristics than the included participants, missing data on confounders were addressed with multiple imputation by chained equations (missingness ranged from 0.4% to 20.4%).[46] The multiple imputation by chained equations procedure used a logistic regression, multivariate logistic regression, or truncated linear regression as appropriate for the variable type, and iterated 100 times to achieve stability in the imputed values. Fifty datasets were imputed, using all outcomes, exposures, and covariates with complete information, as well as quintiles of the sampling weights, to leverage auxiliary information to improve precision of the imputed values.[46,47]

To make the sample nationally representative, all analyses were weighted using the NLSYCYA sample weights. Standard errors were adjusted for the complex survey design,[48] which also accounted for potential correlation between pregnancies in the same woman. Statistical significance was determined at α < 0.05 except for effect modification, which was significant at α < 0.20.[49] Univariate analyses were done in RStudio. Bivariate and multivariate analyses were done in Stata 14.0.

Results

Characteristics of Study Population

Table 1 displays the prevalence of reported exposure to childhood adversity, sociodemographic, and pregnancy characteristics, including smoking prevalence overall and by race/ethnicity. After weighting to be representative of the United States in 1979, three-quarters of the sample was white/other, close to 20% was black and about 8% Hispanic. Thirty-six percent reported exposure to at least one of the adversities measured, the most common of which was household alcohol abuse (23%); 17% reported childhood physical abuse and 11% reported exposure to household mental illness. Overall, 27% reported smoking in pregnancy and 11% of the prepregnancy smokers quit during pregnancy. The majority of children were born between 1980 and 1999. More than 40% of children reported smoking before age 18.

All adversity exposures and smoking behaviors in both mothers and children were differentially distributed by race/ethnicity. Hispanic women were the least likely to smoke during pregnancy, but their children were more likely to initiate smoking than children of black or white mothers. Black mothers reported less exposure to the three ACEs studied, and black children were the least likely to experiment with smoking before the age of 18.

Bivariate analyses revealed that physical abuse and household alcohol abuse were differentially distributed by prenatal smoking status, both being more common in smokers than nonsmokers or quitters; these maternal adversity exposures were also significantly associated with offspring smoking before the age of 18 (Supplementary Table 2). Early smoking initiation was, additionally, more common in children who were exposed to tobacco prenatally (61%), and the least common in children of nonsmokers (41%).

Association Between Maternal Childhood Adversity and Smoking Behaviors

Table 2 presents associations between the maternal childhood adversity exposures and smoking during pregnancy. After adjusting for covariates, history of physical abuse and alcohol in the home were significantly associated with a 39% and 20% increased risk of prenatal smoking, respectively. Exposure to household mental illness was not related to prenatal smoking overall. Although there was evidence of interaction by race/ethnicity on the relationship between childhood household mental illness and prenatal smoking (*p* = .03), subgroup analyses showed that these relationships were not statistically significant in any racial/ethnic group

Table 3 shows that maternal history of childhood physical abuse and household alcohol abuse were associated with increased risks of offspring youth smoking by 20% and 17%, respectively; however household mental illness was not a risk factor. There was no evidence that these findings differed by race–ethnicity.

Table 1. Descriptive Statistics by Maternal Race/Ethnicity (Weighted Proportions)

Mothers' characteristics	Total N = 2999[a]	White/other N = 1561 (73.6%)	Black N = 929 (18.9%)	Hispanic N = 509 (7.6%)	p
ACE, %					
Physical abuse	17.2	18.0	12.5	20.7	<.00
Household alcohol abuse	22.8	24.5	16.5	20.4	<.00
Household mental illness	11.2	12.9	5.7	6.7	<.00
Mother's education, mean ± SD (N = 2824[b])	11.4 ± 2.8	11.9 ± 2.4	10.7 ± 2.5	7.4 ± 3.8	<.00
Southern childhood, % (N = 2976[b])	34.3	29.4	58.2	25.2	<.00
Urban childhood, % (N = 2988[b])	78.5	76.8	81.7	88.1	.02
Foreign born, %	4.1	2.7	2.0	24.5	<.00
Year of birth, %					.66
1957–1959	26.9	26.5	27.7	29.1	
1960–1965	73.1	73.5	72.3	70.9	
Pregnancies' characteristics	N = 6596	N = 3218	N = 2144	N = 1234	
Age at childbirth, mean ± SD	26.1 ± 5.8	26.9 ± 5.6	23.6 ± 5.7	24.7 ± 5.7	<.00
Smoking in pregnancy, %					<.00
Nonsmoker	73.2	72.4	71.6	85.2	
Smoker	26.8	27.6	28.4	14.8	
Birth year, by decade, %					<.00
1970–1979	10.6	7.7	20.2	14.1	
1980–1989	51.5	49.3	57.0	59.3	
1990–1999	35.9	40.6	21.7	24.9	
2000–2009	2.1	2.4	1.1	1.7	
Education, mean ± SD (N = 5806[b])	12.9 ± 2.4	13.3 ± 2.4	12.3 ± 1.9	11.2 ± 2.8	<.00
Marital status, % married (N = 5816[b])	74.5	84.0	35.3	69.0	<.00
Employment, % employed (N = 5834[b])	65.8	70.2	52.3	52.2	<.00
Equivalized income, mean ± SD (N = 5250[b])	9.8 ± 1.2	10.1 ± 1.0	8.9 ± 1.3	9.3 ± 1.2	<.00
Prepregnancy BMI, mean ± SD (N = 6431[b])	23.5 ± 5.0	23.4 ± 5.1	23.6 ± 5.0	23.8 ± 4.3	.07
Children's characteristics	N = 6596	N = 3218	N = 2144	N = 1234	
Smoking before age 18, %	46.4	46.6	43.6	51.7	.02
Birth order, %					<.00
1	43.0	44.2	40.3	38.4	
2	33.4	34.4	30.8	29.9	
≥3	23.6	21.4	28.8	31.7	
Birth weight, mean ± SD (N = 6360[b])	3355.2 ± 604.5	3416.6 ± 584.1	3134.1 ± 634.5	3311.9 ± 589.7	<.00

BMI = body mass index.

[a]All Ns are unweighted.

[b]Number of women with complete information.

Association Between Prenatal Exposure to Tobacco and Youth Smoking

Smoking during pregnancy was associated with a significant 18% increase in offspring early smoking initiation (95% confidence interval [CI]: 1.11, 1.26). When we tested whether this relationship differed by maternal exposure to childhood adversity by testing all two- and three-way interactions, we found evidence of a difference in experiences with household alcohol abuse (p = .14). Compared to children of nonsmoking mothers without a history of childhood household alcohol abuse, the risk of youth smoking was increased for children of prenatal smokers (without maternal childhood household alcoholism: aRR 1.21 [95% CI: 1.13, 1.31]; with maternal childhood household alcoholism: aRR 1.25 [95% CI: 1.13, 1.38]). However, there was also an increased risk of offspring smoking in nonsmoking mothers who reported exposure to household alcohol abuse (aRR 1.13; 95% CI: 1.03, 1.24). No other tested interactions between maternal ACE and smoking during pregnancy were significant.

Finally, we explored possible differences by race/ethnicity in the associations between maternal childhood adversity, prenatal smoking, and child smoking. Table 4 shows that compared to the reference group of black women without a history of childhood household alcohol abuse who did not smoke during pregnancy, prenatal smoking was associated with 69%–72% increased risk of youth smoking in children of white/other women, and did not vary by maternal exposure to household alcoholism. Black children were generally not at increased risk of youth smoking, but they were at 17% increased risk (p < .05) when their mothers smoked during pregnancy but did not have a history of childhood household alcoholism. Among Hispanics, maternal childhood household alcoholism increased risk of early initiation of offspring smoking (by 67%) only when the mother smoked during pregnancy.

Discussion

Results from this nationally representative sample confirm links between maternal childhood adversity and smoking behaviors in pregnant women and their offspring. These associations varied according to type of abuse and race/ethnicity. Generally, maternal smoking during pregnancy was associated with a higher risk of initiating smoking among children, but these results also varied by race/

Table 2. Associations Between Childhood Adversities and Smoking in Pregnancy, Modified by Race/Ethnicity

	Nonsmokers vs. pregnancy smokers (N = 6596)	
	aRR[a] (95% CI)	Wald *p* value for interaction by race/ethnicity
Physical abuse		.39
Overall	1.39 (1.19, 1.62)***	
Household alcohol abuse		.52
Overall	1.20 (1.03, 1.38)*	
Household mental illness		.03
White/other[b]	0.97 (0.92, 1.01)	
Black[b]	1.04 (0.98, 1.10)	
Hispanic[b]	0.86 (0.67, 1.11)	

aRR = adjusted risk ratio; CI = confidence interval.
[a]Log-linear regressions adjusted for mothers' education, growing up in the south, growing up in an urban area, being born outside of the United States, and birth decade; all but mental illness adjusted for race/ethnicity.
[b]Reference group is black women who did not experience childhood household mental illness.
*$p < .05$, ***$p < .001$.

Table 3. Associations Between Maternal History of Childhood Adversity and Next-Generation Youth Smoking

	Youth smokers vs. nonsmokers and adult initiators (N = 6596)	
	aRR[a] (95% CI)	Wald *p* value for interaction by race/ethnicity
Physical abuse	1.20 (1.10, 1.32)***	.22
Household alcohol abuse	1.17 (1.08, 1.27)***	.70
Household mental illness	1.01 (0.89, 1.15)	.30

aRR = adjusted risk ratio; CI = confidence interval.
***$p < .001$.
[a]Log-linear regressions adjusted for mothers' education, growing up in the south, growing up in an urban area, being born outside the United States, birth decade, and race/ethnicity.

ethnicity and maternal ACE exposure. Overall, our findings suggest that maternal childhood adversity and race/ethnicity are potential predictors of high-risk smoking behavior in many pregnant women and their children.

In our sample, 17.2% of women experienced physical abuse, which is very similar to the 15.4% estimate based on data from five states using the Behavioral Risk Factor Surveillance System (BRFSS) questionnaire in 2009.[22] This study's 22.8% prevalence of household alcohol abuse was lower than the 30.6% of women who reported household substance abuse in the BRFSS survey,[22] but this is to be expected, as alcoholism is only one kind of substance abuse. The NLSY79 prevalence of household mental illness, however, was much lower than reported in BRFSS (11.2% vs. 22.0%)[22] for reasons unknown.

Our first study goal was to analyze the relationship between childhood adversity and smoking during pregnancy, and to test for interaction by race/ethnicity. In this sample, maternal exposure to childhood physical abuse was a risk factor for smoking during pregnancy, regardless of race/ethnicity. This is consistent with a previous

finding that physical abuse is associated with smoking during pregnancy in a primarily white sample of German women.[13]

We were also able to confirm Chung and colleagues' findings that household substance abuse is a risk factor for prenatal smoking.[12] In our sample, household alcohol abuse was associated with smoking during pregnancy. We also extended Chung et al.'s findings to a more general population, as their sample of 1476 women in Philadelphia largely consists of young, black, low-income single women with little education, and ours is weighted to be nationally representative.

Our second study goal was to analyze the relationship between maternal ACE and next-generation youth smoking. We found that maternal history of childhood physical abuse and of household alcohol abuse were both associated with offspring smoking, with no difference by race/ethnicity. We are aware of only one previous study that assessed the association between maternal childhood adversity and next-generation child smoking: using linked data from the Nurses Health Study II and the Growing Up Today Study, Roberts et al. found that mothers with severe childhood physical, emotional, or sexual abuse (measured with questions from the Childhood Trauma Questionnaire), were more likely to have offspring who followed the most dangerous smoking trajectories of early initiation and high consumption.[15] In our nationally representative sample, we found a slightly stronger association between physical abuse and offspring early smoking initiation (*aRR* = 1.20 for the NLSY cohort, compared to adjusted odds ratio [*aOR*] = 1.16 reported by Roberts et al.). Using mediation analysis, Roberts et al. found that a 34% reduction in effect size was explained by maternal smoking alone, and a 61% reduction was explained by maternal smoking together with the offspring's experience of child abuse.

The final objective of our study was to explore whether the relationship between prenatal smoking and youth smoking differed by race/ethnicity and maternal adversity in childhood. We found evidence of interaction only by race/ethnicity and exposure to alcohol abuse in the home. Compared to children of nonsmoking black mothers with no childhood exposure to household alcohol abuse, black children were at significantly increased risk of youth smoking only when their mothers smoked during pregnancy but did not have childhood exposure to household alcohol abuse. Children of Hispanic women with exposure to childhood household alcoholism were at increased risk of smoking before the age of 18 only if their mothers also smoked during pregnancy. White/other children were always at higher risk of early smoking, but those born to smoking mothers with a history of household alcohol problems had the highest risk.

Our finding that household alcohol abuse, race/ethnicity, and smoking during pregnancy had a significant three-way interaction underscores the importance of incorporating intersectionality[50] into the way we study and understand health, especially in marginalized populations.[51] Intersectionality posits that individuals live at the intersection of multiple social categories (eg, race, gender, and sexual orientation), which reflects overlapping systems of discrimination and privilege. Systemic oppression can, therefore, only be understood by attending to the complex, multilayered nature of identity. Our original hypothesis was that people of color would be more vulnerable to smoking in the presence of ACE because of the additional damage inflicted on their resiliency as a result of racism. What we found, however, suggests that resiliency varies significantly between racial/ethnic groups of color. Future studies using an intersectional approach to understand these differences are needed.

The relative new and rapidly expanding field of epigenetics suggests mechanisms by which childhood adversity can impact high-risk

Table 4. Association Between Smoking in Pregnancy and Youth Smoking, Modified by Maternal Childhood Household Alcohol Abuse and Race/Ethnicity[a]

| | | Youth smokers vs. nonsmokers and adult initiators (N = 6596) | | |
| | | White/other | Black | Hispanic |
Household alcohol abuse	Smoking in pregnancy	aRR[b] (95% CI)	aRR[b] (95% CI)	aRR[b] (95% CI)
Yes	Yes	1.72 (1.53, 1.94)***	1.16 (0.89, 1.50)	1.67 (1.41, 1.99)***
	No	1.59 (1.41, 1.78)***	1.11 (0.93, 1.32)	1.17 (0.93, 1.48)
No	Yes	1.69 (1.53, 1.86)***	1.17 (1.02, 1.33)*	1.40 (1.16, 1.69)***
	No	1.35 (1.22, 1.51)***	1.00 (Reference)	1.30 (1.16, 1.46)***

aRR = adjusted risk ratio; CI = confidence interval.

[a]Wald test *p* value for interaction = .05.

[b]Log-linear regressions adjusted for mothers' age at childbirth, decade of childbirth, education at birth year, marital status at birth year, employment status at birth year, income at birth year, and children's birth order.

*$p \leq .05$, ***$p \leq .001$.

smoking behaviors. Specifically, ACE is thought to have lifelong repercussions due to epigenetic changes that result from toxic stress in childhood.[52] Both human and animal studies have found childhood maltreatment to be associated with DNA methylation changes that regulate stress-responsivity.[53,54] Chronic stress in childhood is also associated with having a small prefrontal cortex, which impairs self-regulatory behavior.[52] Moreover, recent studies have shown that these epigenetic changes due to stress can be heritable,[55–58] meaning that one's own stress-response can be affected by one's parents' exposure to childhood adversity. Children of women with a history of ACE may therefore be predisposed to maladaptive stress responses, such as smoking. Our understanding of epigenetic inheritance is in its infancy, but it provides a very promising mechanism for how stress can get under the skin and be transmitted across generations.

Intergenerational smoking patterns are also likely influenced by the transmission of familial dysfunction from one generation to the next. Children of mothers with ACE are much more likely to experience ACE themselves,[59,60] for example, which is a direct risk factor for smoking during childhood.[61] Furthermore, childhood trauma is associated with depressed socioeconomic status in adulthood,[62] so children of mothers with ACE are more likely to be exposed to stressors stemming from growing up with a lack of capital. Chronic stress in childhood, as was previously mentioned, is a known risk for smoking.

This study was subject to several limitations. Self-reported pregnancy and youth smoking questions surely introduced social desirability bias,[63] although recent validation studies have found self-reported smoking to be highly correlated with cotinine levels in adolescents and pregnant women.[64,65] Since this would underestimate the true number of smokers, however, the findings are likely biased toward the null.[66] Another limitation was that, while this is a representative sample, it is representative of youths in the United States in 1979, which was quite different demographically than the country today. An additional limitation was the restriction of race/ethnicity to three groups, where whites were grouped with Native Americans, Asians, and all other people identified as non-black, non-Hispanic.

We would have liked to control for the women's mothers' smoking status in the analysis of childhood adversity and smoking during pregnancy, as this is a likely confounder,[67–69] but this information was not collected in the survey. Similarly, the NLSY79 only asked questions about three ACE exposures, so other important adversities were not included in this analysis. In addition, the ACE questions were only included in 2012, limiting our study sample to women who were still participating in NLSY79 33 years after the study began. It is possible that the women who dropped out of the study before 2012 are different with regard to ACE and/or smoking than women who remained, which would bias our findings. Furthermore, as is the case in most studies of child adversity, information was collected retrospectively, when participants were in middle age; however, answers to ACE questions were previously found to be reliable in a sample of older adults.[70] If there is misclassification of ACE, it is likely to be nondifferential with regard to the outcome,[71] so we would expect our results to be biased toward the null.

This study also had several important strengths. The prospective NLSY cohort is large, diverse, and nationally representative. We were able to adjust for important sociodemographic covariables. We also had smoking data on two generations, allowing us to study the intergenerational impact of childhood adversity. Additionally, self-administered questionnaires, rather than interviews, were used to gather all NLSYCYA smoking information, which reduced the probability of information bias in this measure.[72]

Future studies should examine the role of additional ACE exposures in high-risk smoking behaviors among pregnant women and their offspring. Additionally, the association between maternal ACE, smoking in pregnancy, and youth smoking in offspring could be further elucidated by a mediation analysis, measuring how much additional risk is attributable to ACE. Findings from the current study suggest that such an analysis ought to include interaction by race/ethnicity.

Despite evidence for ACE underlying the adoption of a variety of health risk behaviors and the subsequent acquisition of disease,[73] most physicians do not screen their patients for childhood adversities.[74] However, health care providers are ideally positioned to screen women before and during pregnancy. Pregnant women with a history of childhood adversity should be offered a targeted smoking intervention that would aim to replace smoking with a healthier coping mechanism. Additionally, targeting racial/ethnic groups who are more likely to engage in high-risk smoking behaviors could help narrow existing health disparities. Ideally, we should prevent childhood adversities from occurring in the first place, but while ACE persists, we must work on preventing its pernicious health repercussions throughout the lifespan and across generations; specialized training and awareness campaigns could help screening for childhood adversities become routine practice.[74]

Supplementary Material

Supplementary Figures 1 and 2, and Tables 1 and 2 can be found online at http://www.ntr.oxfordjournals.org

Declaration of Interests

None declared.

References

1. PRAMS and Smoking. Centers Dis Control Prev Web site. 2012. www.cdc.gov/prams/tobaccoandprams.htm. Accessed February 7, 2016.
2. Tobacco Use and Pregnancy. Centers Dis Control Prev Web site. 2016. www.cdc.gov/reproductivehealth/maternalinfanthealth/tobaccousepregnancy/index.htm. Accessed February 7, 2016.
3. Cornelius MD, Leech SL, Goldschmidt L, Day NL. Is prenatal tobacco exposure a risk factor for early adolescent smoking? A follow-up study. *Neurotoxicol Teratol*. 2005;27(4):667–676. doi:10.1016/j.ntt.2005.05.006.
4. Al Mamun A, O'Callaghan F V, Alati R, et al. Does maternal smoking during pregnancy predict the smoking patterns of young adult offspring? A birth cohort study. *Tob Control*. 2006;15(6):452–457. doi:10.1136/tc.2006.016790.
5. Rydell M, Cnattingius S, Granath F, Magnusson C, Galanti MR. Prenatal exposure to tobacco and future nicotine dependence: population-based cohort study. *Br J Psychiatry*. 2012;200(3):202–209. doi:10.1192/bjp.bp.111.100123.
6. Klein H, Sterk CE, Elifson KW. Initial smoking experiences and current smoking behaviors and perceptions among current smokers. *J Addict*. 2013;2013:1–9. doi:http://dx.doi.org/10.1155/2013/491797.
7. Smoking and Tobacco Use: Fact Sheet. CDC's Off Smok Heal Web site. 2016. www.cdc.gov/tobacco/data_statistics/fact_sheets/youth_data/tobacco_use/. Accessed February 7, 2016.
8. Kann L, Kinchen S, Shanklin SL, et al. Youth risk behavior surveillance--United States, 2013. *MMWR Surveill Summ*. 2014;63(4)(suppl 4):1–168. doi:ss6304a1 [pii].
9. Office on Smoking and Health (US). Chapter 2. Patterns of Tobacco Use Among Women and Girls. 2001. www.ncbi.nlm.nih.gov/books/NBK44311/. Accessed January 14, 2016.
10. PRAMStat. Centers Dis Control Prev Web site. 2016. www.cdc.gov/prams/pramstat/. Accessed February 7, 2016.
11. Maternal, Infant, and Child Health. Healthy People 2020 Web site. 2016. www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives. Accessed February 7, 2016.
12. Chung EK, Nurmohamed L, Mathew L, Elo IT, Coyne JC, Culhane JF. Risky health behaviors among mothers-to-be: the impact of adverse childhood experiences. *Acad Pediatr*. 2010;10(4):245–251. doi:10.1016/j.acap.2010.04.003.
13. Leeners B, Rath W, Block E, Gorres G, Tschudin S. Risk factors for unfavorable pregnancy outcome in women with adverse childhood experiences. *J Perinat Med*. 2014;42(2):171–178. doi:10.1515/jpm-2013-0003.
14. Roberts AL, Lyall K, Rich-Edwards JW, Ascherio A, Weisskopf MG. Maternal exposure to childhood abuse is associated with elevated risk of autism. *JAMA psychiatry*. 2013;70(5):508–515. doi:10.1001/jamapsychiatry.2013.447.
15. Roberts AL, Galea S, Austin SB, Corliss HL, Williams MA, Koenen KC. Women's experience of abuse in childhood and their children's smoking and overweight. *Am J Prev Med*. 2014;46(3):249–258. doi:10.1016/j.amepre.2013.11.012.
16. About Behavioral Risk Factor Surveillance System ACE Data. Centers Dis Control Prev Web site. 2015. www.cdc.gov/violenceprevention/acestudy/ace_brfss.html. Accessed October 28, 2016.
17. Logan-Greene P, Green S, Nurius PS, Longhi D. Distinct contributions of adverse childhood experiences and resilience resources: a cohort analysis of adult physical and mental health. *Soc Work Health Care*. 2014;53(8):776–797. doi:10.1080/00981389.2014.944251.
18. Brown DL, Tylka TL. Racial discrimination and resilience in African American young adults: examining racial socialization as a moderator. *J Black Psychol*. 2011;37(3):259–285. doi:10.1177/0095798410390689.
19. Rutter M. Annual research review: resilience--clinical implications. *J Child Psychol Psychiatry*. 2013;54(4):474–487. doi:10.1111/j.1469-7610.2012.02615.x.
20. National Longitudinal Surveys: Sample Design & Screening Process. US Bur Labor Stat Web site. 2012. www.nlsinfo.org/content/cohorts/nlsy79/intro-to-the-sample/sample-design-screening-process. Accessed February 7, 2016.
21. The NLSY79 Child/Young Adult Sample: An Introduction. US Bur Labor Stat Web site. 2012. www.nlsinfo.org/content/cohorts/nlsy79-children/intro-to-the-sample/nlsy79-childyoung-adult-sample-introduction. Accessed February 7, 2016.
22. Bynum L, Griffin T, Ridings DL, et al. Adverse Childhood Experiences Reported by Adults --- Five States. 2009. *MMWR*. 2010;59(49):1609–1613. www.cdc.gov/mmwr/preview/mmwrhtml/mm5949a1.htm. Accessed December 31, 2015.
23. Pearl J. Causal diagrams for empirical research. *Biometrika*. 1995;82(4):669–688. doi:10.1093/biomet/82.4.669.
24. Runyan D, Wattam C, Ikeda R, Hassan F, Ramiro L. Child abuse and neglect by parents and other caregivers. In: Krug EG, Dahlberg LL, Mercy JA, Zwi AB, Lozano R, eds. World Report on Violence and Health. Geneva, Switzerland: World Health Organization; 2002: 57–86.
25. Gilman SE, Breslau J, Subramanian SV, Hitsman B, Koenen KC. Social factors, psychopathology, and maternal smoking during pregnancy. *Am J Public Health*. 2008;98(3):448–453. doi:10.2105/AJPH.2006.102772.
26. Shopland DR, Hartman AM, Gibson JT, Mueller MD, Kessler LG, Lynn WR. Cigarette smoking among U.S. adults by state and region: estimates from the current population survey. *J Natl Cancer Inst*. 1996;88(23):1748–1758. www.ncbi.nlm.nih.gov/pubmed/8944005. Accessed February 11, 2016.
27. U.S. Department of Health and Human Services, Health Resources and Services Administration, Maternal and Child Health Bureau. *The Health and Well-Being of Children in Rural Areas: A Portrait of the Nation, 2011-2012*. Rockville, MD: U.S. Department of Health and Human Services; 2015.
28. Jacobson LT, Dong F, Scheuermann TS, Redmond ML, Collins TC. Smoking behaviors among urban and rural pregnant women enrolled in the Kansas WIC program. *J Community Health*. 2015;40(5):1037–1046. doi:10.1007/s10900-015-0029-x.
29. Vaughn MG, Salas-Wright CP, Huang J, Qian Z, Terzis LD, Helton JJ. Adverse childhood experiences among immigrants to the United States [published online ahead of print June 24, 2015]. *J Interpers Violence*. doi:10.1177/0886260515589568.
30. National Research Council (US) and Institute of Medicine (US) Committee on the Health and Adjustment of Immigrant Children and Families; Hernandez DJ. Immigration and Infant Health: Birth Outcomes of Immigrant and Native-Born Women. 1999. www.ncbi.nlm.nih.gov/books/NBK224443/. Accessed January 30, 2016.
31. Singh GK, Yu SM. Adverse pregnancy outcomes: differences between US- and foreign-born women in major US racial and ethnic groups. *Am J Public Health*. 1996;86(6):837–843. www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1380404&tool=pmcentrez&rendertype=abstract. Accessed January 30, 2016.
32. Felitti VJ, Anda RF. The relationship of adverse childhood experiences to adult medical disease, psychiatric disorders, and sexual behavior: Implications for health care. In: Lanius RA, Vermetten E, Pain C, eds. *The Impact of Early Life Trauma on Health and Disease: The Hidden Epidemic*. New York, NY: Cambridge UP; 2010: 77–87.

33. Vandewater EA, Park SE, Carey FR, Wilkinson AV. Intergenerational transfer of smoking across three generations and forty-five years. *Nicotine Tob Res*. 2014;16(1):11–17. doi:10.1093/ntr/ntt112.

34. Acevedo-Garcia D, Pan J, Jun H-J, Osypuk TL, Emmons KM. The effect of immigrant generation on smoking. *Soc Sci Med*. 2005;61(6):1223–1242. doi:10.1016/j.socscimed.2005.01.027.

35. Tong V, Dietz PM, Morrow B, et al. *Trends in Smoking Before, During, and After Pregnancy — Pregnancy Risk Assessment Monitoring System, United States, 40 Sites, 2000–2010.* 2013. www.cdc.gov/mmwr/preview/mmwrhtml/ss6206a1.htm. Accessed January 30, 2016.

36. Soteriades ES, DiFranza JR. Parent's socioeconomic status, adolescents' disposable income, and adolescents' smoking status in Massachusetts. *Am J Public Health*. 2003;93(7):1155–1160. www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1447926&tool=pmcentrez&rendertype=abstract. Accessed January 30, 2016.

37. Kiernan K, Pickett KE. Marital status disparities in maternal smoking during pregnancy, breastfeeding and maternal depression. *Soc Sci Med*. 2006;63(2):335–346. doi:10.1016/j.socscimed.2006.01.006.

38. Powell LM, Chaloupka F. Parents, public policy and youth smoking. *J Policy Anal Manag*. 2004;24(1):93–112.

39. Lu Y, Tong S, Oldenburg B. Determinants of smoking and cessation during and after pregnancy. *Health Promot Int*. 2001;16(4):355–365. doi:10.1093/heapro/16.4.355.

40. Ruhm CJ. Maternal employment and adolescent development. *Labour Econ*. 2008;15(5):958–983. doi:10.1016/j.labeco.2007.07.008.

41. Rehkopf DH, Krieger N, Coull B, Berkman LF. Biologic risk markers for coronary heart disease: nonlinear associations with income. *Epidemiology*. 2010;21(1):38–46. doi:10.1097/EDE.0b013e3181c30b89.

42. Bai J, Wong FWS, Bauman A, Mohsin M. Parity and pregnancy outcomes. *Am J Obstet Gynecol*. 2002;186(2):274–278. doi:10.1067/mob.2002.119639.

43. Avenevoli S, Merikangas KR. Familial influences on adolescent smoking. *Addiction*. 2003;98(suppl 1):1–20. doi:10.1046/j.1360-0443.98.s1.2.x.

44. National Longitudinal Surveys: Race, Ethnicity & Immigration Data. US Bur Labor Stat Web site. 2012. www.nlsinfo.org/content/cohorts/nlsy79/topical-guide/household/race-ethnicity-immigration-data. Accessed February 8, 2016.

45. Rao J, Scott A. On chi-squared tests for multiway tables with cell proportions estimated from survey data. *Ann Stat*. 1984;12(1):46–60.

46. Rubin D. *Multiple Imputation for Nonresponse in Surveys*. New York, NY: Wiley; 1987.

47. Sterne JAC, White IR, Carlin JB, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. *BMJ*. 2009;338:b2393. www.ncbi.nlm.nih.gov/pubmed/19564179.Accessed August 1, 2016.

48. StataCorp L. *Stata Survey Data Reference Manual, Release 11*. College Station, TX: Stata Press Publication; 2009.

49. Selvin S. *Statistical Analysis of Epidemiologic Data*. Vol. 3. New York, NY: Oxford UP; 2004.

50. Crenshaw K. Mapping the margins: intersectionality, identity politics, and violence against women of color. *Stanford Law Rev*. 1991;43(6):1241–1299.

51. Bowleg L. The problem with the phrase women and minorities: intersectionality-an important theoretical framework for public health. *Am J Public Health*. 2012;102(7):1267–1273. doi:10.2105/AJPH.2012.300750.

52. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav*. 2012;106(1):29–39. doi:10.1016/j.physbeh.2011.08.019.

53. Lutz P-E, Turecki G. DNA methylation and childhood maltreatment: from animal models to human studies. *Neuroscience*. 2014;264:142–156. doi:10.1016/j.neuroscience.2013.07.069.

54. Weaver ICG, Cervoni N, Champagne FA, et al. Epigenetic programming by maternal behavior. *Nat Neurosci*. 2004;7(8):847–854. doi:10.1038/nn1276.

55. Metz GA, Ng JWY, Kovalchuk I, Olson DM. Ancestral experience as a game changer in stress vulnerability and disease outcomes. *BioEssays*. 2015;37(6):602–611. doi:10.1002/bies.201400217.

56. Franklin TB, Mansuy IM. Epigenetic inheritance in mammals: evidence for the impact of adverse environmental effects. *Neurobiol Dis*. 2010;39(1):61–65. doi:10.1016/j.nbd.2009.11.012.

57. Francis D, Diorio J, Liu D, Meaney MJ. Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science*. 1999;286(5442):1155–1158. www.ncbi.nlm.nih.gov/pubmed/10550053. Accessed August 1, 2016.

58. Bowers ME, Yehuda R. Intergenerational transmission of stress in humans. *Neuropsychopharmacol*. 2016;41(1):232–244. doi:10.1038/npp.2015.247.

59. Narayan AJ, Kalstabakken AW, Labella MH, Nerenberg LS, Monn AR, Masten AS. Intergenerational continuity of adverse childhood experiences in homeless families: unpacking exposure to maltreatment and family dysfunction [published online ahead of print January 11, 2016]. *Am J Orthopsychiatry*. 2016. doi:10.1037/ort0000133.

60. Ertem IO, Leventhal JM, Dobbs S. Intergenerational continuity of child physical abuse: how good is the evidence? *Lancet*. 2000;356(9232):814–819. doi:10.1016/S0140-6736(00)02656-8.

61. Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA*. 1999;282(17):1652–1658. doi:10.1001/jama.282.17.1652.

62. Zielinski DS. Child maltreatment and adult socioeconomic well-being. *Child Abuse Negl*. 2009;33(10):666–678. doi:10.1016/j.chiabu.2009.09.001.

63. Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res*. 2009;11(1):12–24. doi:10.1093/ntr/ntn010.

64. Mattsson K, Källén K, Rignell-Hydbom A, et al. Cotinine validation of self-reported smoking during pregnancy in the swedish medical birth register. *Nicotine Tob Res*. 2016;18(1):79–83. doi:10.1093/ntr/ntv087.

65. Valladolid-López M del C, Barrientos-Gutiérrez T, Reynales-Shigematsu LM, et al. Evaluating the validity of self-reported smoking in Mexican adolescents. *BMJ Open*. 2015;5(10):e007485. doi:10.1136/bmjopen-2014-007485.

66. Rothman KJ, Greenland S, Lash TL. *Modern Epidemiology*. Vol. 3. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.

67. John U. Strength of the relationship between tobacco smoking, nicotine dependence and the severity of alcohol dependence syndrome criteria in a population-based sample. *Alcohol Alcohol*. 2003;38(6):606–612. doi:10.1093/alcalc/agg122.

68. Smith PH, Mazure CM, McKee SA. Smoking and mental illness in the US population. *Tob Control*. 2014;23(e2):e147–e153. doi:10.1136/tobaccocontrol-2013–051466.

69. Gilman SE, Rende R, Boergers J, et al. Parental smoking and adolescent smoking initiation: an intergenerational perspective on tobacco control. *Pediatrics*. 2009;123(2):e274–e281. doi:10.1542/peds.2008–2251.

70. Dube SR, Williamson DF, Thompson T, Felitti VJ, Anda RF. Assessing the reliability of retrospective reports of adverse childhood experiences among adult HMO members attending a primary care clinic. *Child Abuse Negl*. 2004;28(7):729–737. doi:10.1016/j.chiabu.2003.08.009.

71. Rehkopf DH, Headen I, Hubbard A, et al. Adverse childhood experiences and later life adult obesity and smoking in the United States. *Ann Epidemiol*. 2016;26(7):488–492.e5. doi:10.1016/j.annepidem.2016.06.003.

72. Bowling A. Mode of questionnaire administration can have serious effects on data quality. *J Public Health (Bangkok)*. 2005;27(3):281–291. doi:10.1093/pubmed/fdi031.

73. About the CDC-Kaiser ACE Study. Centers Dis Control Prev Web site. www.cdc.gov/violenceprevention/acestudy/about.html. Last updated 2016. Accessed October 28, 2016.

74. Weinreb L, Savageau JA, Candib LM, Reed GW, Fletcher KE, Hargraves JL. Screening for childhood trauma in adult primary care patients: a cross-sectional survey. *Prim Care Companion J Clin Psychiatry*. 2010;12(6):1–18. doi:10.4088/PCC.10m00950blu.