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Resilience and Biomarkers of Health Risk in Black Smokers and Nonsmokers

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Abstract

Objectives—Blacks are disproportionately affected by tobacco-related illnesses as well as traumatic events that are associated with psychiatric conditions and smoking. We examined the potential protective nature of resilience within this context, hypothesizing that resilience differentially moderates the associations of traumatic experiences to depressive symptoms as well as to biomarkers of health risk among Black ever versus never smokers.

Methods—Measures of resilience, traumatic experiences, depressive symptoms, and biomarkers (interleukin-6 [IL-6], C-reactive protein [CRP], allostatic load) were obtained among 852 Blacks recruited from Grady Memorial Hospital in Atlanta to participate in a study of trauma.

Results—Ever smokers experienced more trauma (p’s<.001) and depressive symptoms (p=.01). Structural equation modeling indicated that, in ever smokers, childhood trauma was positively associated with depressive symptoms (p<.001); resilience was negatively associated with depressive symptoms (p=.01). Depressive symptoms were positively associated with IL-6 (p=.03), which was positively associated with allostatic load (p=.01). Adulthood trauma was associated with higher CRP levels (p=.03). In never smokers, childhood (p<.001) and adulthood trauma (p=.01) were associated with more depressive symptoms. Adulthood trauma was also associated with
higher CRP levels (p<.001), which was positively associated with allostatic load (p<.001). Never smokers with higher resilience had a negative association between childhood trauma and depressive symptoms, whereas those with lower resilience had a positive association between childhood trauma and depressive symptoms. Resilience was negatively associated with CRP levels (p<.001).

Conclusions—Interventions targeting resilience may prevent smoking and adverse health outcomes as well as provide a basis for understanding mechanisms of intervention impact.

Keywords
Tobacco use; Health disparities; Trauma; Depression; Resilience

INTRODUCTION
Health is impacted by social environment, health behaviors, and endogenous biological responses that occur across the lifespan, a complement that is increasingly referred to as the exposome (Miller & Jones, 2014). The exposome comprises overlapping domains that include: 1) general external environments (e.g., urbanicity, social capital, interpersonal relationships); 2) specific external environments (e.g., dietary habits, tobacco use); and 3) internal biological environments (e.g., metabolic factors, inflammation, oxidative stress; Miller & Jones, 2014). These environmental domains interact with each other to effect health outcomes (Miller & Jones, 2014; Wild, 2012). The public health exposome model conceptualizes these pathways to include environmental exposures, personal characteristics, and moderating factors, that in turn, are associated with health disparities among communities at greatest risk (Juarez, 2013). One such community is Blacks in the US. Indeed, it is this chronicity of lifetime exposures that Geronimus and colleagues posited in their “weathering hypothesis”: Blacks show higher cumulative risk measurements than Whites, and these differences are not explained by poverty alone (Geronimus, Bound, Waidmann, Hillemeier, & Burns, 1996; Geronimus, Hicken, Keene, & Bound, 2006). This hypothesis provides a framework for examining the impact of exposure factors with greater prevalence among Blacks that may together influence health outcomes.

An important exposure associated with multiple negative health outcomes is the experience of traumatic events (e.g., experiencing, witnessing, or being confronted with death, serious injury, or physical threat to self or others (American Psychological Association [APA], 1994)), particularly those that occur in childhood. Such trauma has been associated with the development of substance abuse and dependence, including cigarette smoking (Hovdestad, Campeau, Potter, & Tonmyr, 2015; Simpson & Miller, 2002), as well as mental health problems (e.g., depression, anxiety, suicidality; Enns et al., 2006; Gilbert et al., 2009; Kessler, Davis, & Kendler, 1997; MacMillan et al., 2001; Scott, Smith, & Ellis, 2010), interpersonal difficulties (Jaffee & Gallop, 2007; Wolfe, Scott, Wekerle, & Pittman, 2001), compromised academic and occupational functioning (Cicchetti & Rogosch, 1997; Gilbert et al., 2009), aggression, violence, and criminal behaviors (Casiano, Mota, Afifi, Enns, & Sareen, 2009; Gilbert et al., 2009; Ou & Reynolds, 2010). Cumulatively, the impact of traumatic experiences on health are significant and multidimensional. Blacks are disproportionately exposed to traumatic events of various types (Breslau, Davis, & Andreski,
1995; Green, Grace, Lindy, & Leonard, 1990). Compared to Whites, Blacks are more likely to report higher rates of childhood trauma, including parental death and severe alcoholism, as well as extreme economic deprivation (Breslau et al., 1995; Green et al., 1990). Blacks experience more traumatic events in adulthood, such as being attacked or raped, threatened with a weapon, or witnessing an injury or murder (Breslau et al., 1995). Blacks also have higher lifetime and current rates of posttraumatic stress disorder (Breslau et al., 1995; Green et al., 1990).

Blacks are disproportionately affected by tobacco-related illnesses. Each year, approximately 45,000 Blacks die from smoking-related disease (American Cancer Society [ACS], 2011; Centers for Disease Control and Prevention [CDC], 1998). Smoking-related illnesses are the number one cause of death in the Black community, surpassing all other causes of death, including AIDS, homicide, diabetes, and accidents (ACS, 2011). Blacks suffer disproportionate tobacco-related morbidity and mortality despite the fact that they tend to smoke fewer cigarettes per day and begin smoking later in life (Alexander et al., 2016). This phenomenon remains unexplained but has been attributed to a variety of factors including stress associated with living in conditions of socio-environmental disadvantage. Additionally, Blacks are less likely to quit smoking than Whites, resulting in sustained exposure to tobacco smoke for a longer period of their life (Holford, Levy, & Meza, 2016).

The disproportionate burden of smoking-related diseases among Blacks is further compounded by the co-occurrence of smoking and depression that can result from trauma. Studies have shown an increased risk of nicotine dependence among individuals who report exposure to trauma or adverse events (Al Mamun et al., 2007; Breslau, Davis, & Schultz, 2003; Hapke et al., 2005), an association that may be particularly unique to smoking. For example, Breslau and colleagues (Breslau et al., 2003) found an association between trauma exposure and 10-year cumulative incidence of nicotine dependence but no association between trauma exposure and alcohol use. Depressive symptoms also commonly co-occur among those who have been exposed to a traumatic life event (Fergusson, Horwood, & Lynskey, 1996; O'Donnell, Creamer, & Pattison, 2004). Depressive symptoms are strongly associated with nicotine dependence (Breslau et al., 1998; Fergusson et al., 1996; M. Windle & Windle, 2001).

Resilience is a personal characteristic that may account for differential health outcomes among people with histories of trauma. Definitions of resilience have included: positive outcomes despite serious threats to adaptation and development (Masten, 2001); the absence of a particular negative outcome in at-risk populations (e.g., absence of psychiatric disorder or suicidality) (Collishaw et al., 2007); or a composite score based on indices of functioning reflecting both positive outcomes (e.g., educational attainment, employment history) and a lack of negative outcomes (e.g., homelessness, substance use) (McGloin & Widom, 2001). The Connor-Davidson Resilience Scale (CD-RISC) is a comprehensive assessment of personal strengths that contribute to positive outcomes among individuals faced with adversity. The CD-RISC is one of few multi-dimensional measures available for use with adults and includes: personal competence, perseverance, tolerance of negative affect, acceptance of change, sense of social support, trust in one's instincts, spiritual faith, and an action-oriented approach to solving problems (Campbell-Sills & Stein, 2007; Connor &
Davidson, 2003). Prior research has found that this measure of resilience may buffer the effects of traumatic experiences on mental health and cigarette smoking (Campbell-Sills & Stein, 2007; Goldstein, Faulkner, & Wekerle, 2013). However, no previous research has examined the differential role of resilience on depressive symptoms and smoking among Blacks, which is particularly relevant given the range of sociodemographic and psychosocial differences between smokers and nonsmokers (e.g., educational attainment, income levels, age) (Agaku, King, Dube, CDC, 2014; Agaku, King, Husten, et al., 2014). Additionally, while resilience has frequently been examined in terms of mental health outcomes, it has been infrequently examined in relation to the internal environmental component of the exposome, such as physiological biomarkers associated with illness or disease (e.g., cancer risk). Such biomarkers might include interleukin-6 (IL-6), C-reactive protein (CRP), and allostatic load. IL-6 is an interleukin that acts as both a pro-inflammatory and anti-inflammatory cytokine (Ferguson-Smith et al., 1988). It is secreted by T cells and macrophages to stimulate immune response (e.g. during infection and after trauma) and has been found to be associated with several types of chronic diseases including cardiovascular disease and cancer as well as increased mortality (Volpato et al., 2001). CRP is a sensitive marker of systemic low-grade inflammation and is currently recommended as the principal inflammatory marker in research and clinical practice (Pearson et al., 2003). Elevated plasma levels of CRP have been associated with an increased risk of coronary heart disease (Koenig et al., 1999; Ridker, Rifai, Rose, Buring, & Cook, 2002), ischemic stroke (Rost et al., 2001), peripheral artery disease (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1998), hypertension (Sesso et al., 2003), and any cardiovascular disease (Ridker, Buring, Cook, & Rifai, 2002; Ridker et al., 2002) in individuals who have no prior cardiovascular disease. CRP has also found to predict the risk of recurrent myocardial infarction (Ridker, 1998) and mortality (Lindahl, Toss, Siegbahn, Venge, & Wallentin, 2000) in patients with coronary heart disease and is also associated with cancer risk (Chaturvedi et al., 2010; Trichopoulos, Psaltopoulou, Orfanos, Trichopoulos, & Boffetta, 2006). Moreover, elevated plasma CRP levels have been associated with obesity, insulin resistance, the metabolic syndrome, endothelial dysfunction, and an increased risk of developing Type 2 diabetes (Festa et al., 2000; Freeman et al., 2002; Ridker et al., 2003; Yudkin, Stehouwer, Emeis, & Coppack, 1999). Allostatic load is one biomarker that captures the “weathering” the body experiences as it strives to achieve stability in disruptive environments, and elevated IL-6 and CRP levels may be precursors to increased allostatic load (McEwen, 1998). Allostatic load is commonly conceptualized as an aggregate measure of disease risk markers, such as blood pressure, body mass index (BMI), waist circumference, and blood glucose (Barboza Solis et al., 2015; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). Having a history of smoking has been associated with IL-6 (Bermudez, Rifai, Buring, Manson, & Ridker, 2002), CRP (Ohsawa et al., 2005), and allostatic load (McEwen, 1998), and thus, those with a history of smoking should be examined separately from those without such a history in terms of other environmental exposures related to these outcomes. Consistent with the weathering framework, we examined whether the association of exposure to traumatic experiences’ with depressive symptoms, and in turn, allostatic load would be moderated by resilience in a heterogeneous cohort of black adults (see Figure 1). Additionally, we hypothesized that these associations may differ for smokers and

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nonsmokers. Our assumptions in testing the models were that childhood traumatic experiences pre-date tobacco initiation and ongoing use as well as depressive symptoms. However, the availability of additional measures of adult exposure to traumatic experiences provides a lifetime exposome indicator that has not been available in other studies. Given consistent evidence of positive associations between smoking and trauma exposure as well as depression, we hypothesized that their joint influences on weathering might differ for smokers and nonsmokers. We tested structural equation models to capture the complexity of the potential associations among these factors. Such approaches are essential for sorting out the complex and interdependent factors that are likely to contribute to health disparities. Characterizing groups for whom the link between depression and resilience is most pronounced could inform future interventions for tobacco users.

METHODS

Participants & Procedures

This analysis was part of a larger study investigating genetic and trauma-related risk factors for post-traumatic stress disorder and depression in a population of urban, low-income, highly traumatized, predominantly Black men and women (Binder et al., 2008; Bradley et al., 2008). Inclusion criteria included ages 18 to 75 years, understanding English, and ability to give informed consent. The study was approved by the Emory University Institutional Review Board.

Members of the research team approached adult patients waiting for their outpatient appointments at the primary medical care or obstetrical-gynecological clinics of Grady Memorial Hospital in Atlanta, Georgia, to solicit for study participation (Binder et al., 2008; Bradley et al., 2008). If the first participant that the staff approached in the waiting room did not agree to do the study, the staff approached at least two additional possible participants. Of those approached first by staff, 58% agreed, and a cumulative percentage of 84% and 91% of the second and third participants approached agreed if the initial participant approached did not participate. Participants gave informed consent and completed a battery of self-report measures. Due to variation between participants with respect to literacy, all self-report measures were obtained by interview. Blood pressure, weight (kg), height (cm), and waist circumference (cm) were obtained by trained nursing staff at the Grady Hospital Clinical Research Center. Current analyses focused on all Black participants with data available regarding smoking history, traumatic experiences, depression, resilience, and measures of IL-6, CRP, and physiological measures used to calculate allostatic load (n=852).

Measures

Lifetime history of smoking was assessed using responses from the Kreek-McHugh-Schluger-Kellogg (KMSK) scale (Kellogg et al., 2003). Participants were grouped into “never smokers” and “ever smokers” for structural equation modeling.

Covariates & Predictors—Sociodemographics included age, sex, education, income, employment, and disability.
**Childhood Trauma Questionnaire (CTQ):** Childhood abuse was assessed retrospectively using the 28-item Childhood Trauma Questionnaire (CTQ) (D. Bernstein & Fink, 1998; D. P. Bernstein et al., 2003). This scale uses a five-point Likert scale, which range from “never true” (scored 1) to “very often true” (scored 5). Reliability for the CTQ is good with high internal consistency scores. Sexual Abuse, Emotional Neglect, Emotional Abuse, and Physical Abuse have reported coefficients of .93–.95, .88–.92, .84–.89, and .81–.86, respectively. Over a three-month period, the test-retest coefficient was 0.80. Factor analysis on the five-factor CTQ model showed structural invariance, demonstrating good validity (D. Bernstein & Fink, 1998; D. P. Bernstein et al., 2003).

**Traumatic Events Inventory (TEI):** Adulthood trauma was assessed using the Traumatic Events Inventory (Gillespie et al., 2009; Schwartz, Bradley, Sexton, Sherry, & Ressler, 2005). This instrument screens for lifetime exposure to different categories of trauma, including natural disaster, serious accident or injury, sudden life-threatening illness, military combat, being attacked with a weapon, witnessing a family member or friend being attacked with a weapon, being attacked without a weapon, witnessing a family member or friend being attacked without a weapon, witnessing the murder of a friend or family member, or being sexually assaulted under duress. For each category of the instrument, having had the exposure was scored as 1 and no exposure as 0. Score ranges from 0 to 15 for adulthood trauma, with higher scores reflecting exposure to more types of trauma. The childhood trauma items in this inventory were excluded to avoid overlap with the information collected with the CTQ.

**Beck Depression Inventory (BDI):** Depression was measured with the 21-item Beck Depression Inventory (BDI), which has a high degree of reliability and validity (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Items were rated on a four-point Likert scale of 0 to 3; total score ranges from 0 to 63, with higher scores reflecting higher levels of depression. Levels of depression severity are suggested by the following score ranges: ≤9 reflects no depressive symptoms, 10 to 18 mild symptoms, and ≥19 moderate to severe depressive symptoms (Beck et al., 1961). This variable was operationalized as a continuous variable.

**Moderator**

**Connor-Davidson Resilience Scale (CD-RISC):** Resilience was assessed with the ten-item Connor-Davidson Resilience Scale (CD-RISC) (Campbell-Sills & Stein, 2007; Connor & Davidson, 2003). Example items include: “can deal with whatever comes” and “think of self as strong person.” The full CD-RISC was found to have an unstable structure across two demographically equivalent samples and was thus modified into a ten-item scale with good internal consistency, construct validity, and excellent psychometric properties for efficient measurement of resilience (Campbell-Sills & Stein, 2007; Connor & Davidson, 2003) (e.g., Cronbach’s alpha=.91 (Gonzalez, Sierra, Martinez, Martinez-Molina, & Ponce, 2015); item separation reliability=.98 (Gonzalez et al., 2015); high content validity (G. Windle, Bennett, & Noyes, 2011)). Items were rated on a five-point Likert scale, ranging from “not true at all” (scored 0) to “true nearly all the time” (scored 4). Scores range from 0 to 40, with higher score reflecting greater resilience.
**Biomarker Outcomes**

**Interleukin-6 (IL-6) and C-Reactive Protein (CRP):** Whole blood was collected under fasting conditions between 8:00 am and 9:00 am and processed by centrifugation to separate the plasma from the buffy coat. Plasma samples were used to assess IL-6 and CRP, as well as cortisol (used in the measure of allostatic load below).

**Allostatic Load:** Allostatic load was operationalized as an aggregate measure of biomarkers of disease risk. Individual biomarker scores were standardized and summed, creating a cumulative allostatic load score. We adapted our measure from Seeman et al.’s (Seeman et al., 1997) seminal measure of allostatic load to include a culmination of biomarkers known to be linked with various health outcomes. This is a standard way to measure allostatic load in the literature, as sums across physiological systems are better indicators of cumulative dysregulation than individual biomarker scores (Geronimus, Hicken, Keene, & Bound, 2006; Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002; Seeman, McEwen, Rowe, & Singer, 2001; Seeman et al., 1997). This approach has been studied in population-based survey research and indicated that higher allostatic load scores were associated with higher morbidity and mortality (Seeman et al., 2001; Seeman et al., 1997). Moreover, lower allostatic load scores were associated with higher educational levels and better psychosocial factor scores (Seeman et al., 2004; Seeman, Singer, Ryff, & Levy-Storms, 2002).

The five factors comprising the allostatic load measure were: cortisol, systolic blood pressure, diastolic blood pressure, BMI, and waist-to-hip circumference. We selected these biomarkers from available data based on their roles as indicators of disease risk and use in prior allostatic load research (Doamekpor & Dinwiddie, 2015; Mauss, Li, Schmidt, Angerer, & Jaraczok, 2015; Seeman et al., 1997; Steptoe et al., 2014; Upchurch et al., 2015). For each factor, the following dichotomizations were made: 1) cortisol ≥16.6 mg/dL, which represents the highest quartile of this cohort; 2) systolic blood pressure ≥140; 3) diastolic blood pressure ≥90 mmHg; 4) BMI ≥30; and 5) abdominal obesity based on a waist circumference of >102 cm for men or >88 cm for women. A point was assigned for each criterion, yielding scores of 0 to 5, with 5 indicating highest risk.

**Data Analysis**

Descriptive univariate analyses were conducted to assess distributions and the extent of missing data. Bivariate analyses were used to examine the differences between ever smokers and never smokers. These were followed by analyses into the patterns of missing data and multivariable regression analyses (including residual analyses) for each of the endogenous constructs (e.g., depression, IL-6, allostatic load). Joint normality was assessed. Structural equation modeling was then conducted to examine the mediating role of depressive symptoms as well as the moderating role of resilience comparing relationships between ever and never smokers. Age and sex were included as covariates for IL-6, CRP, and allostatic load. Estimation for the two-group model used maximum likelihood with missing values. No auxiliary variables were included, as missingness in the data was due to administration of different modules at different times independent of specific participant characteristics and were thus assumed to be missing completely at random. Indices used to assess model fit were chi-squared, the Root Mean Square Error of Approximation (RMSEA), and the
Comparative Fit Index (CFI). Post SEM, residuals were inspected. Data were cleaned and
descriptive, univariate, bivariate, and multivariable analyses were conducted using SAS 9.4.
Structural equation modeling analyses were conducting using Stata 14. Based on results
from the bivariate analyses, age and sex were included as covariates in the structural
equation model.

RESULTS

Participant Characteristics

Ever smokers and never smokers differed across all sociodemographic dimensions, with ever
smokers being older, more likely to be male, less educated, lower income earners (p’s<.001),
more likely to be unemployed (p=.02), and more likely to receive disability (p=.04; Table 1).
As the literature would suggest, ever smokers also reported experiencing more childhood
and adulthood trauma (p’s<.001) and depressive symptoms (p=.01).

Structural Equation Model Results

The final structural equation model presented had good model fit: \( \chi^2(30)=46.501, p=.03 \)
overall, \( \chi^2(15)=8.156, p=.92 \) for ever smokers, \( \chi^2(15)=38.345, p=.001 \) for never smokers,
RMSEA = 0.036, CFI = 0.950. The overall coefficient of determinant (CD) was 0.532, with
CD=0.426 and 0.683 for ever and never smokers, respectively.

Table 2 and Figure 2 display the results of the structural equation models examining the
impact of childhood and adulthood trauma (per the CTQ and TEI, respectively), resilience
(per the CD-RISC), and depressive symptoms (per the BDI) on biomarkers of health risk
(i.e., IL-6, CRP, and allostatic load) among ever and never smokers, respectively. In ever
smokers, childhood trauma was positively associated with depressive symptoms (p<.001);
resilience was negatively associated with depressive symptoms (p=.01). Depressive
symptoms were positively associated with IL-6 (p=.03), which was positively associated
with allostatic load (p=.01). Adulthood trauma was positively associated with CRP levels
(p=.03). Older age was also associated with higher IL-6, and being female was associated
with higher allostatic load.

In never smokers, childhood and adulthood trauma were positively associated with
depressive symptoms (p<.001, p=.01). Adulthood trauma was also associated with higher
CRP levels (p<.001), which was positively associated with allostatic load (p<.001). Never
smokers with higher resilience showed a negative association between childhood trauma and
depressive symptoms, whereas those with lower resilience showed a positive association
between childhood trauma and depressive symptoms (see Figure 3). Resilience was
negatively associated with CRP levels (p<.001). Older age and being female was also
associated with higher CRP.

In comparing coefficients across groups, differences were found regarding the interaction of
childhood trauma and resilience on depressive symptoms (p=.03), adulthood trauma on CRP
levels (p<.001), the interaction between adulthood trauma and resilience on CRP (p<.001),
and age on CRP levels (p<.001).
DISCUSSION

The current study examined the role of resilience in moderating the impact of trauma exposure, depression, and resilience on biomarkers of “weathering” among Black ever versus never smokers. Examining these groups separately is critical, as our results and the prior literature (Agaku, King, Dube, et al., 2014; Agaku, King, Husten, et al., 2014) have demonstrated marked differences in the range of exposures across the exposome (e.g., sociodemographic, sociocultural, psychological characteristics), with smokers experiencing more risk exposures across these dimensions (Al Mamun et al., 2007; APA, 1994; Breslau et al., 2003; Hapke et al., 2005). As anticipated, resilience served as a buffer between specific upstream risk factors (i.e., traumatic experiences) and downstream risk factors (i.e., depressive symptoms, health risk biomarkers). In addition, our hypothesis that resilience would differentially moderate the associations between traumatic experiences, depressive symptoms, and health risk biomarkers among ever versus never smokers was also supported.

First, these findings highlight the need for early intervention among Blacks experiencing childhood trauma. Our results show that, among ever smokers, childhood trauma – but not adulthood trauma – was associated with higher depressive symptoms. This finding points to biological embedding (Hertzman, 1999, 2012). Biological embedding suggests that long-term and stable differences in experience, under different nurturant conditions, lead to differences in learning and behavior over the life course. These differences can impact the weathering process and ultimately health outcomes (Essex et al., 2013). Our data indicates that childhood trauma leads to higher depressive symptoms. These higher depressive symptoms put one at risk for smoking initiation, which, in turn, can increase depressive symptoms (M. Windle & Windle, 2001) and, consequently, reduce the odds of successful cessation (Brody, Hamer, & Haaga, 2005; Catley, Ahluwalia, Resnicow, & Nazir, 2003; Chen, White, & Pandina, 2001; Covey, Glassman, & Stetner, 1990; Husky, Mazure, Paliwal, & McKee, 2008). This line of downstream consequences potentially prompted by early childhood environmental factors – specifically trauma – has long-term and potentially difficult-to-modify consequences, both in terms of depressive symptoms and health risk biomarkers, specifically IL-6 and allostatic load. However, resilience was associated with decreased depressive symptoms in ever smokers. This suggests the potential importance of promoting resilience as a construct among young people who have experienced traumatic events in order to prevent biological embedding that may directly lead to depression and indirectly to smoking initiation and progression (Brody et al., 2005; Catley et al., 2003; Chen et al., 2001; Covey et al., 1990; Husky et al., 2008).

Among never smokers, both childhood and adulthood trauma was associated with higher depressive symptoms. Whereas resilience was not significantly associated with depressive symptoms (as with ever smokers), it did serve as a moderator between childhood trauma and depressive symptoms in never smokers. Specifically, those with greater resilience demonstrated a negative association between levels of childhood trauma and depressive symptoms, whereas those with less resilience demonstrated a positive association between levels of childhood trauma and depressive symptoms. Interestingly, resilience was negatively
associated with CRP levels among never smokers, indicating that resilience may particularly serve as a buffer for disease risk among Blacks who have never smoked.

The distinct roles of resilience among ever versus never smokers are difficult to interpret. One explanation is that relative to nonsmokers, smokers have a complement of interdependent vulnerabilities (e.g., less educated, lower income, unemployed) (Al Mamun et al., 2007; APA, 1994; Breslau et al., 2003; Hapke et al., 2005) along with their experiences of depression and trauma history. As such, the earlier childhood experiences of this group may have shaped their subsequent behaviors (e.g., substance use, academic performance). Those with higher levels of childhood trauma may have engaged in these damaging behaviors, which resulted in poorer quality of life. Yet, a higher resilience within this high-risk group may contribute to lower levels of depressive symptoms despite these vulnerabilities. On the other hand, individuals who never engaged in cigarette smoking, and who were less vulnerable in terms of sociodemographics and psychosocial experiences, were differentially impacted by childhood trauma. However, our cross-sectional data did not allow us to test this assumption. The impact of childhood traumatic events on depressive symptoms may have been mitigated by higher resilience.

Another finding that is interesting to note is that the two groups showed different associations between experiencing traumatic events in adulthood and CRP levels, such that experiencing greater adulthood traumatic events was associated with higher CRP levels in smokers while lower adulthood trauma was associated with higher CRP levels in never smokers. These findings warrant replication to ensure that these were not spurious findings and, if replicated, additional research to understand the differential mechanisms impacting the relationship between adulthood trauma and CRP levels in ever versus never smokers.

These findings collectively suggest that increasing resilience may buffer the impact of environmental risk factors. Further prospective study designs will be needed to confirm this. This research could take the form of randomized intervention trials that vary resilience-building activities in groups at high risk of accelerated weathering. Limited research has examined the utility of interventions targeting resilience in order to prevent or treat substance use, specifically smoking or nicotine dependence. While there is some controversy regarding whether resilience is a personality trait or a characteristic amenable to intervention (Yilmaiz, Unal, Gencer, Aydemir, & Selcuk, 2015), some research indicates the promise of intervening on resilience. For example, some intervention research has shown that attempts to increase resilience in young adults have been effective in not only increasing resilience but also increasing coping skills and protective factors (e.g., positive affect, self-esteem) and decreasing symptoms of depression, stress, and negative affect (Steinhardt & Dolbier, 2008). Other research using a family-based resilience intervention has documented reductions in alcohol use among adolescents (Toumbourou, Gregg, Shortt, Hutchinson, & Slaviero, 2013). Indeed, there has been recent calls for social policies that promote resilience in order to decrease substance use and related costs (Luthar, Cicchetti, & Becker, 2000) and for interventions to increase resilience among youth that are multi-level, coordinated, continuous over time, and shown to be effective in order to meet the long-term needs of youth facing the cumulative disadvantages of family, community, school, and individual challenges (Ungar, Liebenberg, Landry, & Ikeda, 2012).
Limitations

Several study limitations are worth noting. First, the cross-sectional nature of this study and the use of retrospective self-reports prohibits us from making causal attributions and is vulnerable to bias. Prospective, longitudinal studies are required to examine the temporality of trauma in relation to smoking, depressive symptoms, and resulting health outcomes as well as the impact of resilience on these outcomes. Another limitation is that our sample was disproportionately female and low income, and we do not yet have the data to demonstrate if these findings would apply in other segments of the Black population. However, this weakness is counterbalanced by the public health importance of studying these variables in an often under-researched and under-served population with disproportionately high rates of trauma exposure as well as mental and physical health problems. Additionally, this is a study exclusively of Blacks. Thus, it is not possible to infer from the current analyses whether the documented associations would also be demonstrated within other racial or ethnic groups. Finally, it is important to note that several of our hypothesized associations were not significant. The strongest associations were found in the model representing never smokers, particularly the moderating effect of resilience on the relationship between childhood trauma experiences and depressive symptoms. The model representing ever smokers showed less robust associations, with the strongest associations being those between adulthood traumatic events and CRP levels as well as between resilience and depressive symptoms. As such, these analyses warrant replication in similar samples and in other more representative samples to establish which relationships are most robust, both in this population and in others.

Conclusions

The current findings point to the distinct role of resilience in buffering the impact of environmental stressors in Black ever versus never smokers. While resilience did not moderate the association between trauma and depressive symptoms in ever smokers, it was directly related to lower levels of depressive symptoms. In contrast, resilience was not directly related to depressive symptoms in never smokers but rather buffered the impact of traumatic experiences on depressive symptoms. In addition, resilience directly impacted specific health risk biomarkers in never smokers but only indirectly impacted specific health risk biomarkers in smokers via depressive symptoms. Ultimately, this study highlights the importance of resilience in preventing adverse health outcomes and provides a basis for understanding the mechanisms of change that might result from interventions targeting resilience in Black ever versus never smokers.

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Figure 1.
Conceptual framework examining exposures across the exposome and impact on health risk biomarkers
Figure 2.
Structural equation models examining the impact of trauma, resilience, and depressive symptoms on biological outcomes among ever smokers and never smokers, respectively.

Note: Solid black lines indicate significant associations, whereas gray lines indicate insignificant associations.
Figure 3.
Resilience moderating the relationship between childhood trauma and depression among never smokers

Note: No relationship among ever smokers (figure not shown).
### Table 1

Participant characteristics and bivariate analyses examining differences between ever smokers and never smokers

<table>
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<tr>
<th></th>
<th>Total</th>
<th>Ever smoker</th>
<th>Never smoker</th>
<th>p</th>
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<tbody>
<tr>
<td></td>
<td>M/N SD/%</td>
<td>M/N SD/%</td>
<td>M/N SD/%</td>
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<tr>
<td>Sample size</td>
<td>852 100</td>
<td>608 71.36</td>
<td>244 28.64</td>
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<td>Sociodemographics</td>
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<td>Age</td>
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<td>44.32 11.62</td>
<td>38.14 13.39</td>
<td>&lt;.0001</td>
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<td>Gender</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>302 35.53</td>
<td>246 40.53</td>
<td>56 23.05</td>
<td>&lt;.0001</td>
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<td>548 64.47</td>
<td>361 59.47</td>
<td>187 76.95</td>
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<td></td>
<td></td>
<td>&lt;.0001</td>
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<td>172 28.67</td>
<td>35 14.58</td>
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<tr>
<td>12th grade or high school</td>
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<td>193 32.17</td>
<td>101 42.08</td>
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<td>45 7.50</td>
<td>9 3.75</td>
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<td>124 20.67</td>
<td>62 25.83</td>
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<td>Tech school graduate</td>
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<td>24 4.00</td>
<td>8 3.33</td>
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<td>34 5.67</td>
<td>23 9.58</td>
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<td>274 33.29</td>
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<td>25 10.73</td>
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<tr>
<td>$500–$999</td>
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<td>163 27.63</td>
<td>72 30.90</td>
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<tr>
<td>$1000–$1999</td>
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<td>108 18.31</td>
<td>55 23.61</td>
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<tr>
<td>$2000 or more</td>
<td>64 7.78</td>
<td>41 6.95</td>
<td>23 9.87</td>
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<tr>
<td>Employment status (no reported)</td>
<td>668 79.43</td>
<td>490 81.53</td>
<td>178 74.17</td>
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<td>Received disability (yes reported)</td>
<td>210 25.06</td>
<td>162 27.00</td>
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<td>Primary Predictors</td>
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<td>Childhood Trauma Questionnaire (CTQ)</td>
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<td>45.20 19.32</td>
<td>39.72 14.65</td>
<td>&lt;.0001</td>
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<td>3.95 2.55</td>
<td>2.64 2.14</td>
<td>&lt;.0001</td>
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<td>Connor-Davidson Resilience Scale (CDR)</td>
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<td>30.87 8.15</td>
<td>30.90 8.78</td>
<td>.98</td>
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<td>Beck Depression Inventory (BDI)</td>
<td>15.93 12.54</td>
<td>16.69 12.65</td>
<td>13.97 12.06</td>
<td>.01</td>
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<td>Outcomes</td>
<td>Total</td>
<td>Ever smoker</td>
<td>Never smoker</td>
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<td>-------------</td>
<td>--------------</td>
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<tr>
<td></td>
<td>M/N</td>
<td>SD/%</td>
<td>M/N</td>
<td>SD/%</td>
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<td>Interleukin-6 (IL-6)</td>
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<td>1.42</td>
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<td>C-Reactive Protein (CRP)</td>
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<td>5.70</td>
<td>8.44</td>
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<td>Allostatic Load (AL)</td>
<td>2.08</td>
<td>1.34</td>
<td>2.04</td>
<td>1.36</td>
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M: mean; N: sample size; SD: standard deviation
Table 2

Equation estimates for structural equation models for ever smokers and never smokers, respectively, and comparisons of coefficients across groups

<table>
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<tr>
<th></th>
<th>Ever smokers</th>
<th></th>
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<th>95% CI</th>
<th>Ever smokers</th>
<th></th>
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<th>95% CI</th>
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<td></td>
<td>Unstand. Coefficient</td>
<td>SE</td>
<td>z</td>
<td>p</td>
<td>Stand. Coefficient</td>
<td>SE</td>
<td>z</td>
<td>p</td>
</tr>
<tr>
<td>CTQ→BDI</td>
<td>0.17</td>
<td>0.03</td>
<td>5.66</td>
<td>.001</td>
<td>0.11</td>
<td>0.23</td>
<td>0.35</td>
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<tr>
<td>TEI→BDI</td>
<td>0.33</td>
<td>0.22</td>
<td>1.49</td>
<td>.14</td>
<td>-0.10</td>
<td>0.77</td>
<td>0.15</td>
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<tr>
<td>CDR→BDI</td>
<td>-0.44</td>
<td>0.18</td>
<td>-2.46</td>
<td>.01</td>
<td>-0.79</td>
<td>-0.09</td>
<td>-0.06</td>
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<td>CTQ*CDR→BDI</td>
<td>0.06</td>
<td>0.41</td>
<td>0.15</td>
<td>.88</td>
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<td>0.87</td>
<td>0.33</td>
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<td>TEI*CDR→BDI</td>
<td>-5.96</td>
<td>3.68</td>
<td>-1.62</td>
<td>.11</td>
<td>-13.18</td>
<td>1.25</td>
<td>0.04</td>
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<tr>
<td>BDI→CRP</td>
<td>-0.10</td>
<td>0.05</td>
<td>-1.91</td>
<td>.06</td>
<td>-0.20</td>
<td>0.00</td>
<td>0.00</td>
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<td>TEI→CRP</td>
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<td>0.03</td>
<td>0.85</td>
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<td>CDR→CRP</td>
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<td>0.14</td>
<td>-0.38</td>
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<td>-0.33</td>
<td>0.23</td>
<td>0.17</td>
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<tr>
<td>TEI*CDR→CRP</td>
<td>-1.54</td>
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<td>Age→CRP</td>
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<td>0.04</td>
<td>0.94</td>
<td>.35</td>
<td>-0.04</td>
<td>0.12</td>
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<td>Female→BDI</td>
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<td>BDI→IL-6</td>
<td>0.01</td>
<td>0.01</td>
<td>2.16</td>
<td>.03</td>
<td>0.00</td>
<td>0.03</td>
<td>0.27</td>
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</tr>
<tr>
<td>Age→IL-6</td>
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<td>.02</td>
<td>0.00</td>
<td>0.03</td>
<td>0.25</td>
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<tr>
<td>Female→IL-6</td>
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<td>BDI→Allostatic load</td>
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<td>0.00</td>
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<tr>
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<td>0.02</td>
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<td>1.62</td>
<td>.11</td>
<td>0.00</td>
<td>0.04</td>
<td>0.22</td>
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<tr>
<td>IL-6→Allostatic load</td>
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<td>0.06</td>
<td>2.68</td>
<td>.01</td>
<td>0.04</td>
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<td>Age→Allostatic load</td>
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<td>0.89</td>
<td>.37</td>
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<td>0.13</td>
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<tr>
<td>Female→Allostatic load</td>
<td>0.55</td>
<td>0.12</td>
<td>4.48</td>
<td>.001</td>
<td>0.31</td>
<td>0.79</td>
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Equation-level goodness of fit

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<th></th>
<th>r-squared</th>
<th>mc</th>
<th>mc²</th>
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<tbody>
<tr>
<td>BDI</td>
<td>0.366</td>
<td>0.605</td>
<td>0.366</td>
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<tr>
<td>CRP</td>
<td>0.035</td>
<td>0.188</td>
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<tr>
<td>IL-6</td>
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<td>0.205</td>
<td>0.042</td>
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<tr>
<td>Allostatic load</td>
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<td>0.286</td>
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</table>

<table>
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<th>r-squared</th>
<th>mc</th>
<th>mc²</th>
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<td>BDI</td>
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<tr>
<td>CRP</td>
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<tr>
<td>IL-6</td>
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<td>0.288</td>
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<td>Allostatic load</td>
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<td>0.110</td>
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<tr>
<td>Ever smokers</td>
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<td>Stand.</td>
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<td>Overall</td>
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<tr>
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<tr>
<td>Stand.</td>
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*Comparing coefficients across groups.*

mc: correlation between the dependent variable and its prediction
mc²: Bentler Raykov squared multiple correlation coefficient