

## Childhood Neighborhood Disadvantage, Parenting, and Adult Health



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**Introduction:** Growing up in disadvantaged neighborhoods is associated with poor adult health indicators. Consistent and supportive parenting plays a key role in life-long health, but it is not known whether positive parenting can mitigate the relationship between neighborhood adversity and poor health. This study examines parenting as a moderator of the links between childhood neighborhood characteristics and adult health indicators.

**Methods:** A sample of 305 individuals (61% female; 82% African American, 18% Caucasian) were assessed in childhood (T1; age 11 years; 2003–2004) and adulthood (T2; age 27 years; 2018–2021). At T1, neighborhood poverty was derived from census data; neighborhood disorder was reported by parents. Children reported on parental harsh discipline, inconsistent discipline, and parental nurturance. At T2, health outcomes included BMI, serum cortisol and C-reactive protein (CRP), and salivary DNA methylation index related to CRP. Regression models predicted T2 health outcomes from T1 neighborhood and parenting variables and their interactions, adjusting for clustering and confounders. Data were analyzed in 2021.

**Results:** Neighborhood poverty was associated with lower cortisol, whereas neighborhood disorder was linked with CRP-related DNA methylation. Multiple interactions between neighborhood and parenting variables emerged, indicating that adverse neighborhood conditions were only related to poor adult health when combined with inconsistent discipline and low parental nurturance. By contrast, warm and supportive parenting, consistent discipline, and to a lesser extent harsh discipline buffered children from poor health outcomes associated with neighborhood disadvantage.

**Conclusions:** Interventions enhancing consistent and nurturing parenting may help to reduce the long-term associations of neighborhood disadvantage with poor health.

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### INTRODUCTION

Adverse childhood experiences show strong associations with adult health through obesity, inflammation, and endocrine disruptions.<sup>1–3</sup> Living in poor urban neighborhoods exposes children to numerous chronic stressors, including violence and social and physical disorder.<sup>4–6</sup> Even after adjusting for family SES and other confounders, growing up in disadvantaged neighborhoods is associated with obesity, inflammation, and atypical cortisol levels that persist into adulthood<sup>7–10</sup> and contribute to cardiovascular disease, diabetes, cancer, and other chronic disorders.<sup>11–13</sup> These relationships are thought to reflect multisystem dysregulation of the stress

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response and metabolic and inflammatory systems induced by exposure to chronic stress during development.<sup>14-16</sup> This dysregulation also involves epigenetic changes in gene expression through DNA methylation,<sup>17-19</sup> which then contributes to cardiovascular disease, diabetes, cancers, and other age-related diseases.<sup>20-22</sup>

Adverse neighborhoods disproportionately affect African American youth, who are more likely to grow up in racially segregated urban neighborhoods characterized by poverty, crime, drug use, and violence.<sup>23-25</sup> These racial inequalities in exposure to neighborhood stressors contribute to profound racial disparities in chronic diseases.<sup>26-28</sup> By young adulthood, African Americans already show higher levels of poor health indicators, including elevated BMI and inflammation markers, than non-Hispanic Whites.<sup>29,30</sup> Over time, these health biomarkers translate into chronic diseases and premature mortality.<sup>31-33</sup>

Although policies and interventions designed to reduce neighborhood poverty and disorder are needed to improve health and reduce disparities,<sup>34</sup> it is also important to identify the protective factors that may mitigate the negative associations between neighborhood disadvantage and health. Multiple prospective studies show the importance of consistent and supportive parenting for better adult health, including lower BMI and inflammation.<sup>35-37</sup> Some of these relationships appear mediated by differential DNA methylation in inflammation-related genes.<sup>36</sup> Consistent and supportive parenting also buffer the links between adverse neighborhoods and poor mental health, delinquency, brain development, and academic outcomes.<sup>38-40</sup> However, few studies have investigated parenting as a moderator of the associations between childhood neighborhood adversity and adult health.

This study examines whether children's perceptions of parenting moderate the prospective relationship between childhood neighborhood adversity and adult health indicators. The sample represents mostly urban African Americans who carry a disproportionate burden of neighborhood adversity and poor health.<sup>25,28</sup> To identify the most relevant dimensions that can guide the development of targeted interventions, we examine 2 aspects of neighborhood disadvantage (concentrated poverty and neighborhood disorder) and 3 dimensions of parenting (harsh discipline, inconsistent discipline, and nurturance). Because chronic stress dysregulates multiple physiologic systems, the study includes health indicators associated with the stress response (cortisol), metabolism (BMI), and inflammation (C-reactive protein [CRP]). To elucidate the underlying biological mechanisms, a DNA methylation index related to higher CRP levels<sup>41</sup> is also included. We hypothesize that childhood neighborhood poverty and disorder will be associated with lower cortisol, higher BMI, and higher CRP and CRP-related DNA

methylation in adulthood but that these associations will be attenuated by parental nurturance and lower harsh and inconsistent discipline experienced in childhood.

## METHODS

### Study Sample

This study includes 305 participants from the Birmingham Youth Violence Study who were assessed in childhood (T1) and adulthood (T2). Children and their caregivers were recruited from 5th-grade classrooms in 17 public schools in Birmingham, Alabama and completed individual interviews at T1 in 2003–2004. Approximately 16 years later (in 2018–2021), the children (now adults) participated in a T2 interview where they provided blood and saliva samples. Height and weight were measured at both T1 and T2. All interviews were conducted by trained interviewers utilizing computer-assisted interview procedures. Informed consent was obtained from adults and caregivers; children provided informed assent. Participants received financial compensation. All procedures were approved by the University of Alabama at Birmingham IRB.

### Measures

Neighborhood poverty was measured with Census data tied to participants' geocoded addresses at T1. Consistent with previous research,<sup>42-44</sup> 4 indicators of poverty were extracted from the 2000 U.S. Census for each of the 28 represented census tracts: percentage of unemployed adult males, percentage of households below poverty level, percentage of single-parent households, and percentage of renter-occupied households. These variables were standardized and averaged (Cronbach's  $\alpha=0.88$ ).

Neighborhood disorder was measured with caregiver report at T1 using 11 items from the Neighborhood/Block Conditions scale.<sup>45,46</sup> This scale evaluates the degree of social (e.g., drug dealing, organized gangs, physical assaults, fighting, gunshots, feeling unsafe) and physical (e.g., property damage, inadequate city services) disorder in the neighborhood. For each item, caregivers stated whether it is *No problem* (0), a *Minor problem* (1), or a *Serious problem* (2) in their neighborhood. The responses were averaged ( $\alpha=0.92$ ).

Harsh discipline was assessed at T1. Children responded to 3 questions about the frequency of their parents yelling, spanking, and hitting them on a 5-point scale ranging from *Never* (0) to *Always* (4).<sup>47,48</sup> The items were averaged ( $\alpha=0.67$ ).

Inconsistent discipline was assessed at T1 with child report; 4 questions asked how often punishment depends on caregiver's mood, how often punishment is inconsistent from one time to another, or how often requests and punishments are not followed through.<sup>47,48</sup> The items were rated on a 5-point scale from *Never* (0) to *Always* (4) and averaged ( $\alpha=0.67$ ).

Parental nurturance was assessed at T1 by child report. Children were asked 5 questions about their parents' nurturing behavior and close parent-child relationship (parents praising and encouraging child; giving physical affection, advice, and guidance; doing enjoyable things together; child discussing problems with parents).<sup>48,49</sup> The items were rated on a 3-point scale from *Almost never* (0) to *Almost always* (2) and averaged ( $\alpha=0.57$ ).

Serum CRP and cortisol levels came from blood samples drawn at T2 from the forearm vein in glass gold-topped vacuum tubes without anticoagulants. Time of sample acquisition was recorded and used as a covariate for cortisol analyses. Samples were allowed to clot for 30

minutes and then were centrifuged (1,100g–1,300g for 10 minutes). Serum was aliquoted and stored at  $-80^{\circ}\text{C}$ . CRP was measured on a Stanbio Sirius (Boerne, TX) analyzer using Pointe Scientific (Canton, MI) turbidometric reagent following the manufacturer's instructions. The interassay coefficient of variation was 5.33%; the intra-assay coefficient of variation was 7.49%. Minimum detectable serum concentrations were 0.5 mg/L. Cortisol was measured on a Tosoh Bioscience AIA900 (South San Francisco, CA) using immunofluorescence following the manufacturer's instructions. The interassay coefficient of variation was 1.66%; the intra-assay coefficient of variation was 5.19%. Minimum detectable serum concentrations were 5.52 nmol/L. CRP levels reflect systemic inflammation,<sup>50</sup> whereas cortisol levels reflect acute stress response.<sup>51</sup>

DNA methylation was derived from saliva samples collected at T2 with Oragene DNA OG-500 kits. DNA was extracted using the PureGene extraction method (Qiagen, Hilden, Germany) following the manufacturer's specifications. All samples yielded more than 2.1  $\mu\text{g}$  of high-quality DNA. Methylation analysis of DNA was performed with the Illumina Infinium MethylationEPIC BeadChip. Normalization and quality control (QC) were conducted in the R package minfi<sup>52</sup> and included probe level QC, sample level QC, background correction, within-array normalization, Types I and II chemistry correction, and batch/plate/chip adjustment. Total methylation levels were quantified as *Beta-values*, defined as the ratio of methylated fluorescent intensity and overall intensity.<sup>53</sup> The reference-based deconvolution method<sup>54</sup> was utilized to correct for differences in cell composition.

The CRP methylations index was derived from candidate CpG associations, with CRP levels reported in a recent meta-analysis<sup>41</sup> and validated in an independent sample.<sup>55</sup> A total of 6 CpG sites spanning 7 genes that showed the strongest evidence of a functional association with CRP levels were used. To enhance robustness, weights from *z*-statistics in the meta-analysis and the validation study were combined using the METASOFT program<sup>56</sup> and used to weigh methylation values at each CpG. The index was standardized and inverted so that higher scores are associated with higher CRP levels.<sup>41,55</sup> These scores reflect an epigenetic profile associated with inflammation.

Covariates included parent reports of the child's sex, ethnicity, and date of birth; their own highest education completed; marital status; and annual household income at T1. BMI was calculated for children and caregivers at T1 and for adults at T2 from the average of 2 measurements. For children, BMI percentiles were derived on the basis of age and sex. Because tobacco smoking is associated with cortisol levels<sup>57</sup> and DNA methylation,<sup>58</sup> we included current smoking for cortisol and history of smoking (at T2 and age 18 years) for DNA methylation. The estimated cell proportions of cell types found in the saliva samples were measured.

### Statistical Analysis

Descriptive statistics and bivariate correlations were examined. Main analyses were conducted in Mplus 8.6 using hierarchical regression models predicting BMI, cortisol, CRP, and CRP methylation index at T2 from neighborhood and parenting variables at T1. Step 1 included neighborhood poverty, neighborhood disorder, harsh discipline, inconsistent discipline, parental nurturance, and covariates. Step 2 added interactions between neighborhood poverty and each parenting variable (Step 2a) or between neighborhood disorder and each parenting variable (Step 2b). Significant interactions were followed by visualization<sup>59</sup> and tests of

simple slopes for neighborhood effects at average, low, and high levels of parenting (1 SD below and above the mean).<sup>60</sup>

Covariates used in all the models were child sex, race, parental education, household income; single-parent household; child BMI percentile and parent BMI at T1; and adult age at T2. Models for cortisol, CRP, and CRP methylation index also controlled for adult BMI at T2. Cortisol models controlled for time of day and current smoking. CRP methylation models controlled for history of smoking and saliva cell types<sup>61</sup> (granulocytes,  $\text{CD4}^+\text{T}$ ,  $\text{CD8}^+\text{T}$ , B cells, and monocytes; natural killer cells were not present). In all models, parameter estimates and SEs were adjusted for clustering of participants within neighborhoods at T1. Because some variables were not normally distributed, the maximum likelihood estimator with robust SEs was used. Missing data (3.45% of data points) were handled with full information maximum likelihood, which reduces bias and preserves the overall sample size.<sup>62</sup> Thus, all available data from the full sample of 305 participants were analyzed.

## RESULTS

The T1 sample included 704 children, of which 305 (43%) were retained at T2. The retained participants did not differ from those lost to attrition at T1 age, household income, BMI percentile, neighborhood poverty or disorder, and any of the parenting variables. Retained participants had higher parental education ( $M=4.29$  vs 4.01,  $p=0.031$ ) and were more likely to be female (61% vs 37%,  $p<0.001$ ) and African American (82% vs 75%,  $p=0.014$ ).

Descriptive statistics are shown in Table 1. The sample included 61% females, 82% African Americans, and 18% Caucasians. Average ages were 11.71 years ( $SD=0.77$ ) at T1 and 27.37 years ( $SD=1.19$ ) at T2. Although the sample overrepresented low-income urban African American families, household annual income ranged from  $<\$5,000$  to  $>\$90,000$  (median= $\$25,000$ – $\$30,000$ ), and caregiver education ranged from  $<9^{\text{th}}$  grade to graduate degree (median=some college); 53% of caregivers were single.

Bivariate correlations revealed that T1 neighborhood poverty was associated with greater neighborhood disorder ( $r=0.37$ ,  $p<0.001$ ) and lower T2 cortisol ( $r=-0.20$ ,  $p<0.001$ ). T1 harsh discipline, inconsistent discipline, and low parental nurturance were intercorrelated ( $r=-0.15$  to 0.40,  $p<0.05$ ). T2 BMI was related to higher CRP ( $r=0.39$ ,  $p<0.001$ ) and CRP methylation ( $r=0.22$ ,  $p<0.001$ ). CRP was modestly associated with CRP methylation ( $r=0.27$ ,  $p<0.001$ ).

Standardized coefficients from the hierarchical regression models are shown in Table 2. After adjusting for covariates and clustering, neighborhood poverty at T1 was uniquely associated with lower cortisol levels at T2, whereas neighborhood disorder predicted higher CRP methylation. In Step 2, all the 3 T1 parenting variables interacted with neighborhood poverty in predicting CRP. Neighborhood poverty also interacted with inconsistent discipline in predicting cortisol and with parental

**Table 1.** Descriptive Statistics

Variables	Mean (SD)
Female, n (%)	183 (61)
African American, n (%)	249 (82)
Single parent household, n (%)	153 (53)
Age, T1	11.71 (0.77)
Age, T2	27.37 (1.19)
Parental education <sup>a</sup> , T1	4.28 (1.70)
Household income <sup>b</sup> , T1	6.49 (3.78)
Neighborhood disorder, T1	0.32 (0.45)
Neighborhood % males unemployed, T1	4.78 (3.46)
Neighborhood % renter occupied, T1	34.25 (17.17)
Neighborhood % poverty, T1	18.43 (11.97)
Neighborhood % single parent, T1	24.64 (12.07)
Harsh discipline, T1	0.91 (0.80)
Inconsistent discipline, T1	0.69 (0.72)
Parental nurturance, T1	1.58 (0.39)
Parent BMI, T1	33.62 (8.41)
Child BMI %, T1	74.43 (27.34)
Adult BMI, T2	31.53 (11.46)
C-reactive protein (mg/L), T2	4.75 (8.14)
Cortisol (μg/dL), T2	10.93 (4.44)

<sup>a</sup>Scores ranged from 1 (less than high school) to 8 (graduate or professional degree).

<sup>b</sup>Scores ranged from 1 (<\$5,000 a year) to 13 (>\$90,000 a year).

nurturance in predicting CRP methylation. Finally, inconsistent discipline interacted with neighborhood disorder for BMI, CRP, and CRP methylation.

Examples of the significant interactions are visualized in [Figures 1](#) and [2](#). Simple slope analyses indicated that

neighborhood poverty was related to lower cortisol at low and average inconsistent discipline ( $\beta_{low} = -0.29, p < 0.001$ ;  $\beta_{mean} = -0.20, p < 0.001$ ) but only marginally related at high inconsistent discipline ( $\beta = -0.12, p = 0.091$ ). Neighborhood poverty was associated with greater CRP levels at low harsh parenting ( $\beta = 0.22, p = 0.027$ ), high inconsistent parenting ( $\beta = 0.30, p = 0.030$ ), and low nurturance ( $\beta = 0.26, p = 0.001$ ); marginally related at average levels of all types of parenting ( $\beta = 0.12, p = 0.062$ ); and unrelated at high harsh parenting ( $\beta = 0.03, p = 0.614$ ), low inconsistent parenting ( $\beta = 0.05, p = 0.459$ ), and high nurturance ( $\beta = -0.01, p = 0.847$ ). Neighborhood poverty was associated with lower CRP methylation at high parental nurturance ( $\beta = -0.12, p = 0.042$ ) but unrelated at average ( $\beta = -0.04, p = 0.464$ ) or low ( $\beta = 0.05, p = 0.437$ ) parental nurturance. Neighborhood poverty became related to higher methylation at more extreme levels of low parental nurturance (e.g., at 2.5 SD below the mean  $\beta = 0.18, p = 0.048$ ).

Neighborhood disorder predicted higher CRP methylation at high and average inconsistent discipline ( $\beta_{high} = 0.18, p < 0.001$ ;  $\beta_{mean} = 0.10, p = 0.019$ ) but not at low inconsistent discipline ( $\beta = 0.03, p = 0.599$ ). Neighborhood disorder was related to lower CRP at low inconsistent discipline ( $\beta = -0.26, p = 0.002$ ) but unrelated at average or high inconsistent discipline ( $\beta_{mean} = -0.05, p = 0.333$ ;  $\beta_{high} = 0.11, p = 0.244$ ). Finally, neighborhood disorder was unrelated to BMI at low, average, or high inconsistent discipline ( $\beta_{low} = -0.07, p = 0.495$ ;  $\beta_{mean} = 0.00, p = 1.000$ ;  $\beta_{high} = 0.07, p = 0.359$ ). Only at more extreme levels of inconsistent discipline did the impacts of neighborhood disorder on BMI

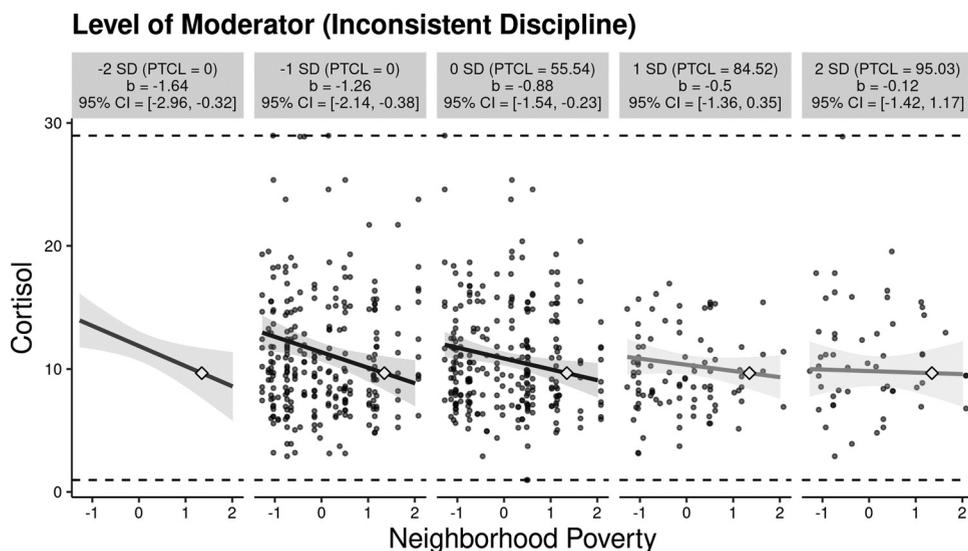
**Table 2.** Regression Models Predicting Adult Outcomes From Childhood Neighborhood Characteristics and Parenting

Variables	Cortisol $\beta$	BMI $\beta$	CRP $\beta$	CRP methylation index $\beta$
<b>Step 1</b>				
Neighborhood poverty	<b>-0.19***</b>	-0.08	0.10	-0.04
Neighborhood disorder	0.02	0.00	-0.06	<b>0.11**</b>
Harsh discipline	-0.05	0.02	0.01	-0.03
Inconsistent discipline	-0.05	0.02	0.03	0.02
Parental nurturance	-0.01	0.05	0.03	-0.02
<b>Step 2a</b>				
N poverty X harsh	-0.02	0.07	<b>-0.10*</b>	0.01
N poverty X inconsistent	<b>0.10*</b>	0.05	<b>0.09*</b>	0.00
N poverty X nurturance	0.04	-0.07	<b>-0.14***</b>	<b>-0.09*</b>
<b>Step 2b</b>				
N disorder X harsh	0.04	-0.03	-0.02	-0.01
N disorder X inconsistent	0.03	<b>0.07*</b>	<b>0.17**</b>	<b>0.08*</b>
N disorder X nurturance	0.06	-0.02	-0.05	0.00

Note: Boldface indicates statistical significance (\* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ ).

N=305. All models adjusted for sex, race, parental education, family income, single-parent household, child BMI percentile, and parent BMI in childhood as well as age and BMI in adulthood. Cortisol models also adjust for current smoking and time of day; methylation models adjust for history of smoking and cell types in saliva samples.

CRP, C-Reactive Protein.



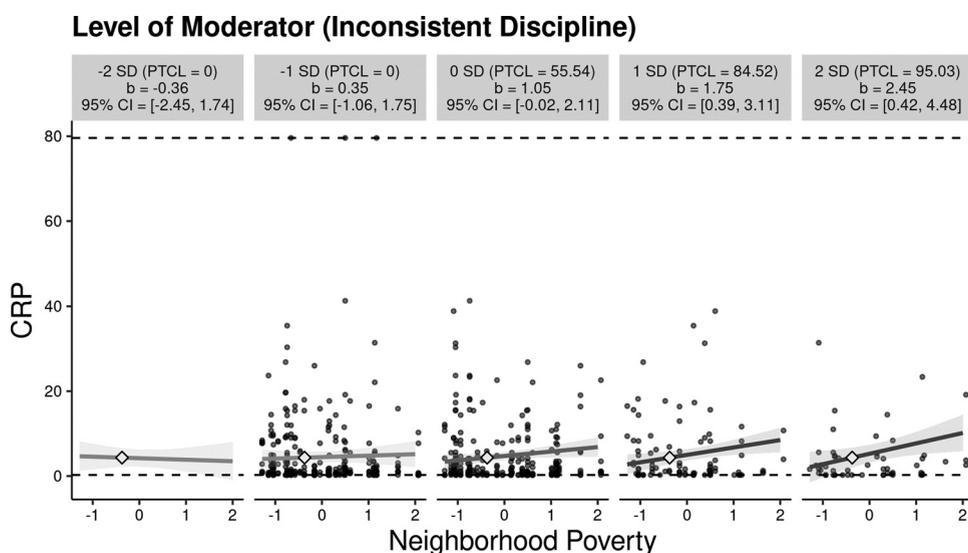
**Figure 1.** Parental inconsistent discipline moderates the relationship between neighborhood poverty and cortisol. PTCL, percentile.

approach significance (e.g., at 3 SD above the mean  $\beta=0.20$ ,  $p=0.078$ ).

**DISCUSSION**

This is the first investigation that has examined childhood parenting as a moderator of the associations between childhood neighborhood disadvantage and adult health indicators. Using a cohort of mostly urban African American youth followed over 16 years, the results showed that objectively assessed neighborhood

poverty in childhood was associated with lower cortisol levels in adulthood. In addition, greater parent-reported neighborhood social and physical disorder in childhood was related to a proinflammatory DNA methylation profile in adulthood. Importantly, the relationships between childhood neighborhood characteristics and adult health varied by parental discipline and nurturance in childhood, so the relationships between adverse neighborhood conditions and poor adult health were exacerbated by inconsistent discipline and low parental nurturance. By contrast, supportive parenting, consistent discipline,



**Figure 2.** Parental inconsistent discipline moderates the relationship between neighborhood poverty and CRP levels. CRP, C-Reactive Protein; PTCL, percentile.

and, to a lesser extent, harsh discipline buffered children from the long-term associations between neighborhood poverty and disorder and poor adult health.

Childhood neighborhood poverty was related to lower cortisol in adulthood, consistent with lower cortisol levels among adults residing in more disadvantaged and disordered neighborhoods.<sup>63</sup> Studies with young children linked community disadvantage with higher cortisol<sup>64</sup> but also blunted diurnal cortisol patterns,<sup>65,66</sup> suggesting that neighborhood disadvantage leads to blunted cortisol responses to acute stress.<sup>17</sup> The pattern of blunted cortisol responses is consistent with the lower cortisol levels among adults exposed to neighborhood poverty in childhood observed in this study. The stronger relationship between neighborhood poverty and lower cortisol at low levels of inconsistent discipline suggests that cortisol blunting may be an adaptive response that is supported by consistent parenting.

Contrary to the findings of some previous research,<sup>7</sup> this study did not find links between neighborhood disadvantage and adult BMI. It is possible that this association is more consistently present in national samples that cover a broader range of neighborhood conditions<sup>7</sup> because neighborhood factors do not seem consistently related to obesity among disadvantaged populations.<sup>67</sup> In this study, parental inconsistent discipline moderated the link between neighborhood disorder and BMI so that neighborhood disorder marginally predicted higher BMI only at very high levels of inconsistent discipline. One other study tested the interaction of neighborhood and parenting characteristics on obesity, finding that positive parenting mitigated the association between neighborhood crime and higher BMI in children aged 5 years.<sup>68</sup>

CRP was positively associated with BMI ( $r=0.39$ ,  $p<0.001$ ), consistent with the finding of previous research ( $r=0.36$  in a meta-analysis of 51 studies).<sup>69</sup> This study extends previous work linking elevated CRP levels with both neighborhood disadvantage<sup>8,70,71</sup> and nurturing parenting<sup>72-75</sup> by showing that the relationship between neighborhood disadvantage in childhood and systemic inflammation in adulthood is buffered by consistent and supportive parenting and exacerbated by inconsistent and unsupportive parenting. These findings are consistent with research showing that positive parenting mitigates the long-term associations between childhood adversities and poor adult health.<sup>76-78</sup>

Moreover, at low inconsistent discipline, neighborhood disorder was related to lower instead of higher adult CRP, suggesting a compensatory effect. Harsh discipline also emerged as a protective factor against the relationship between childhood neighborhood poverty and adult inflammation. Although seemingly counterintuitive, this result is consistent with research showing

that harsh discipline is adaptive in disadvantaged, high-crime neighborhoods.<sup>79-81</sup> Indeed, high parental control was associated with better immune functioning in adults exposed to high neighborhood risk as children.<sup>82</sup>

Finally, this study showed that inconsistent discipline exacerbates the relationship between neighborhood disorder and inflammation-related DNA methylation. This finding extends previous research linking neighborhood disadvantage and less supportive and consistent parenting with inflammation-related DNA methylation<sup>18,83,84</sup> and supports DNA methylation as a biological mechanism through which adverse neighborhoods and parenting affect adult inflammation. The results suggest that parental interventions targeting consistent and nurturing parenting in disadvantaged communities may improve children's health into adulthood. Indeed, parenting interventions reduced children's inflammation in adulthood<sup>37</sup> and buffered the impact of childhood adversities on adult health.<sup>78,85,86</sup>

### Limitations

The study limitations include attrition, with only 43% of T1 participants taking part in T2. Females and African Americans were more likely to be retained, so the results may be less generalizable to other groups. Health outcomes were only measured in adulthood, so their previous levels could not be controlled, and causal inferences cannot be made. Children's reports of parenting had low internal consistency, similar to previous studies using the same measures,<sup>48,87</sup> which may have contributed to the results not being consistent across all dimensions of parenting. Replication with more reliable measures is needed. Finally, DNA methylation was derived from salivary DNA, so replication in other tissues would be informative.<sup>88</sup>

### CONCLUSIONS

In conclusion, this was one of the first studies to investigate whether parenting moderates long-term associations between childhood neighborhood disadvantage and adult health. The study used a prospective design that followed a community sample of mostly African American children for more than 16 years, utilizing multisource data to assess neighborhood characteristics, parenting, and health outcomes. The results provide strong evidence that consistent and supportive parenting mitigates the associations between growing up in disadvantaged neighborhoods and multisystem markers of chronic disease in young adulthood—whereas inconsistent and unsupportive parenting exacerbates them—and that these relationships may be explained by inflammation-related DNA methylation. Interventions enhancing consistent and nurturing parenting may help to reduce

the long-term links between neighborhood disadvantage and health and reduce health disparities.

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## SUPPLEMENT NOTE

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