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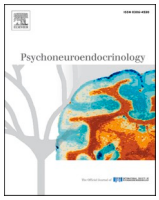

The association between levels of GPS-tracked activity space violent crime and the relationship between cortisol and a biomarker of inflammation amongst Black and White adolescents

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ABSTRACT

Exposure to areas high in violent crime is a potent stressor that influences health outcomes by chronically undermining safety and upregulating biological stress responses. We tested the hypothesis that the association between cortisol, as measured in head hair, and inflammation, as measured by C-Reactive Protein (CRP) in capillary blood, is dependent on the degree of violent crime within adolescents' everyday activity spaces. Because structural inequities cause Black adolescents to spend more time in areas with higher rates of violent crime, we tested this hypothesis in Black and White youth separately. 137 adolescents (M_{age} = 15.55, 57 % female, 52 % Black, 48 % White) participated in the study. We obtained continuous GPS-tracked data for one week to assess the average violent crime rate across the areas where participants spent time; biosamples were collected at the end of the week. Among Black adolescents, there was an interaction such that higher GPS-tracked activity space violent crime levels were associated with a positive and significant association between CRP and cortisol, consistent with models suggesting that stress can dysregulate immune-endocrine functioning. Conversely, for Black adolescents with low rates of exposure, cortisol had a negative association with CRP, consistent with a normative effect of glucocorticoid inhibition of inflammation. For White adolescents, cortisol and violence levels were significantly lower than for Black adolescents, and in this context, there was a weak main effect of violence exposure on CRP but no significant interaction. Results suggest the association between cortisol and inflammation varies across violent crime levels within the areas adolescents spend time and emphasize the importance of studying how an adolescent's environment shapes biological responses to chronic stressors.

1. Introduction

Community violence is a pervasive stressor that harms mental and physical health (Boynton-Jarrett et al., 2008; Fowler et al., 2009) even if it is not experienced or seen directly (Sharkey, 2018). Indeed, the psychological and biological effects of violence in the community can extend well beyond those who experience or witness an act of violence. Spending time at or near locations that have a high probability of violent crime can undermine perceived safety (Browning et al., 2024) and lead to hypervigilance (Smith et al., 2019). With respect to mental and physical health outcomes, living in neighborhoods with higher violent crime rates is associated with higher rates of perceived distress and depression (Baranyi et al., 2021), substance use (Reboussin et al., 2015), elevated blood pressure (Tung et al., 2019), and heightened mortality (Wilkinson et al., 1998) relative to neighborhoods with lower violent crime. Violence is far-reaching, and higher exposure to violent areas is a stressor that warrants closer investigation, particularly with respect to the biological mechanisms through which exposure to such violence impacts health.

Inflammation has been suggested as a potential mechanism through

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which violence exposure during adolescence influences long-term health outcomes (Finegood and Miller, 2021). In youth and adult samples, rates of violent crime based on home address have been directly associated with a biomarker of inflammation, C-Reactive Protein (CRP) (Browning et al., 2012; Broyles et al., 2012), as well as an index of inflammatory markers that included CRP (Finegood et al., 2020). Neighborhood-level violent crime rates remained a robust predictor of inflammatory activation after controlling for experiencing or witnessing violence (Miller et al., 2022), supporting the importance of studying area-level violent crime as a distinct stressor beyond experiences of violence.

Another biological pathway theorized to be a critical mediator of stress effects on health is the hypothalamic-pituitary-adrenal axis (HPA), which elicits hormonal responses to stressors. While HPA activation is adaptive in the short-term, chronic HPA axis activation can lead to negative health outcomes (McEwen, 1998a). One measure of cumulative output of the HPA axis over several months is the level of cortisol in head hair (Short et al., 2016; Singh Solorzano et al., 2023; Sugaya et al., 2020). Using this methodology, higher levels of exposure to stressors have been associated with higher cortisol in both children (Li et al., 2023) and adults (Stalder et al., 2017). Accordingly, when participants reported momentary perceptions of safety during their day-to-day routines, higher perceived unsafety was associated with higher levels of cortisol in Black adolescents (Browning et al., 2023). Similarly, Latine youth who scored higher on a questionnaire assessing perceptions of lack of safety in their neighborhood had higher hair cortisol levels (Hollenbach et al., 2019). In a longitudinal sample of youth in Quebec, greater perceived neighborhood dangerousness as rated by the mother at eight time points between age 5 months and 15 years was associated with higher levels of hair cortisol concentration relative to those who grew up in neighborhoods with moderate levels of dangerousness (Ouellet-Morin et al., 2021). Because higher head hair cortisol is a risk factor for future health outcomes like cardiovascular disease (Iob and Steptoe, 2019), HPA axis overactivation as suggested by elevated hair cortisol is important to consider as a mechanism through which exposure to areas high in violence increases risk for poor health outcomes. Because inflammation and cortisol are both elevated by violent crime exposure, we sought to extend prior work by examining their association as a function of activity space violent crime exposure.

In classical work from the 1940's that led to widespread clinical use of glucocorticoids, cortisol had inhibitory effects on inflammation (Cain and Cidlowski, 2017). Conversely, cytokines that are released during an inflammatory response can activate the HPA axis and increase cortisol levels (Besedovsky et al., 1986). This crosstalk between the HPA axis and the immune system appears to be dysregulated in the context of living in a location with high levels of violent crime. In one study over a two-year period, adolescents residing in higher violent crime neighborhoods showed a decrease in the expression of genes within circulating peripheral immune cells (monocytes) that had response elements for the glucocorticoid receptor (Miller et al., 2022). Because the glucocorticoid receptor transduces the downstream effects of cortisol (Cain and Cidlowski, 2017), including the anti-inflammatory effects, this suggests that living in a high violence location may be associated with a less-responsive glucocorticoid signaling pathway. The association between the glucocorticoid and inflammatory signaling pathways was not directly assessed in this prior work. Here we investigate the impact of violence exposure on this immune-endocrine relation by examining the association between levels of cortisol and levels of CRP, a biomarker of inflammation, across different levels of violence exposure.

Stress and persistent elevated activity of the HPA axis can alter the association that cortisol has with inflammation. Although the association between glucocorticoids and inflammation is nuanced and likely bidirectional (Amasi-Hartoonian et al., 2022; Pariante et al., 1999), persistent activation of the HPA axis is thought to precede disruptions in immune-endocrine functioning that can lead to a positive feedback loop between cortisol and inflammation (Silverman and Sternberg, 2012b). Black youths and adolescents exhibit evidence of chronic HPA activation as indicated by elevated head hair cortisol in earlier waves of the study presented here (Ford et al., 2021). Thus, in examining the effects of activity space violent crime on the association between cortisol and CRP, we focus on CRP as the dependent measure.

Understanding the risk factors that might influence the association between cortisol and inflammation is critical because of the associated health risks posed by dysregulation of these systems. Black adolescents are particularly at risk due to disparities in exposure to violent crime and access to resources within the areas where they live and spend time, which is rooted in structural inequity and can result in health disparities even at an early age (Yusuf et al., 2022). Recommendations have been made to examine within-group differences, rather than solely relying on between-group differences, based on the varying environments and life experiences of different groups (Whitfield et al., 2008). This is particularly relevant to the present work given that Black adolescents with "low" violent area exposure relative to other Black adolescents still have higher exposure levels relative to White adolescents, and Black adolescents with high exposure to violent areas may experience a threshold of exposure that chronically undermines safety (Browning et al., 2024; Pinchak et al., 2022). In addition to experiencing higher levels of violence exposure, Black adolescents in prior waves of this sample also exhibit higher hair cortisol levels (Ford et al., 2021). Thus, it is possible that both the magnitude of stress exposure and the magnitude of biological stress responses alter the association between inflammation and cortisol. Yet, no prior work has investigated how the impact of violence exposure on the interplay between the immune and endocrine systems might be different across groups with starkly different levels of exposure and chronic activation of biological stress pathways.

Furthermore, the relatively small number of studies examining the effects of community violence on inflammatory activation and cortisol have generally measured violent crime rates of participants' residential neighborhood based on their home address. Yet, adolescents' *activity spaces*, or the comprehensive network of areas where they spend time, have been found to be heterogeneous, with adolescents spending more than 30 % of their time on average outside of the home neighborhood (Browning et al., 2021). To fully understand the effects of violent crime on biological and psychological stress outcomes, calls have been made for studies obtaining continuous, GPS-tracked data to obtain incidences of violent crime comprehensively within the full spectrum of the areas where individuals spend time (Sharkey, 2018), which we utilize in the present research.

The aims of the present research are thus a) to investigate the impact of activity space violent crime levels on immune-endocrine functioning by examining the moderating effect of activity space violence on the association between head hair cortisol and CRP b) to address this aim through a structural lens by examining this interaction separately for Black and White adolescents due to the fundamentally different nature of the environments they grow up in and c) to extend past work assessing violence exposure through self-report measures or crime rates measured at the home, school, or neighborhood-level to determine if *activity space* violent crime, a chronic and consistently safety-threatening stressor, influences the association between inflammation and cortisol.

2. Material and method

2.1. Procedure

This study draws from one time point of data (Wave 3) from The Adolescent Health and Development in Context (AHDC) study, a longitudinal cohort study investigating the role of social and spatial environments in shaping health outcomes of youth aged 12–20 in Columbus, OH and collected during 2018–2020. For more information on the AHDC study design and procedures, see Browning et al., (2021). In brief, the AHDC study was designed to improve understanding of how the contexts of adolescent development – including schools, residential

areas, activity spaces, and social network ties- contribute to risk behavior and mental health. Recruitment information was mailed to households within the Columbus, OH Interstate 270 beltway that were likely to have an adolescent based on vendor provided lists as well as data from public school districts. This yielded 1405 adolescents in the first wave of the study, which was conducted between the spring of 2014 and the summer of 2016. The sample matched the income and racial demographics of the city of Columbus with the exception of a slightly higher proportion of Black adolescents in the original wave of the sample (Boettner et al., 2019). Wave 2 was fielded between January and December 2016. The sample was restricted to respondents who were under age 18 and due to funding constraints was limited to those for whom Wave 1 participation had occurred at least 9 months prior. Wave 3 commenced in July of 2018 and ended with the COVID-related research shutdown in March of 2020. In addition to participants recruited from the original sample, 117 youth in Wave 3 were newly recruited using the same methods from low-income census tracts as well as tabling at schools in these tracts. Wave 3 was the first wave of this study in which samples were collected to assess C-reactive protein due to additional grant funding and research questions that were added to the study related to inflammation. Ethics approval was obtained by the Social and Behavioral Sciences and the Biomedical Sciences Institutional Review Boards at Ohio State University. All participants gave informed consent or assent; a guardian provided permission for participants under 18 in accordance with the guidelines set by the Office of Responsible Research Practices at The Ohio State University.

The AHDC study design included an entrance survey where we obtained demographic, socioeconomic, and self-reported health data. Then, youth participants completed a seven-day smartphone-based geographically explicit ecological momentary assessment period. Participants were provided with phones by the project and were instructed to carry the phones with them. During this time, we obtained continuous, GPS-tracked location from the participants' phone. After the seven-day smartphone period, participants took part in a laboratory session at the Ohio State University. During this session, if participants consented to providing biological samples, we collected hair and finger prick blood samples for cortisol and C-reactive protein, respectively.

2.2. Participants

The present analyses consist of 137 youths and adolescents from the greater area of Columbus, OH ($M_{age} = 15.55$, $SD_{age} = 2.09$, 57 % female, 52 % Black/ African American, 48 % White/ Caucasian, see Table 1 for more detailed sample characteristics). 59 participants in this analysis were participating in this study for the first time through the recruitment methods outlined above, while 78 participants had participated in prior waves. The analyses presented here are limited to Black and White participants due to both the nature of our research question and an insufficient sample size of other racial/ethnic groups to examine our variables of interest (13 Hispanic/Latin American, 2 Asian/ Pacific Islander, 20 Multiracial, and 1 First Nations/ Native American youth). Participants were included in the analyses if they identified as Black/ African American or White/Caucasian and if they had complete data for cortisol, C-Reactive Protein (CRP), and GPS-tracked, activity space violent crime levels.

2.3. Measures

2.3.1. C-reactive protein

CRP was assayed in duplicate from dried blood spots following an established protocol from prior work (McDade, 2014). Finger-prick blood samples were collected on a 903 Protein Saver Card (Whatman) and kept for 24 h to dry at room temperature. Next, we punched the samples with a 3-mm punch and stored them in microcentrifuge tubes at –80 °C. To assay for CRP, we thawed a single 3-mm punch and added 200 μL of buffer (phosphate buffered saline with 0.1 % Tween 20; ThermoFisher Scientific) overnight incubation at 4 °C while shaking at 60 rpm. We diluted this eluate 1:10 and assayed CRP the following morning using Vplex Plus kits (K151STG; Meso Scale Delivery). All samples were successfully measured (i.e., within the linear range of the standard curve). The intraassay coefficient of variation was 2.8 %, while the interassay coefficient of variation was 11.8 %.

Table 1
Characteristics of the sample.

Variable	*Mean(SD) or N(%)*	
	White Participants (N = 66)	*Black Participants (N = 71)*
C-Reactive Protein (mg/L)	1.20 (2.06)	.60 (.99)
Mean Activity Space Violent Crime Rate (2014–2016)	4.50 (4.45)	10.93 (7.72)
Hair Cortisol Concentration (pg/mg)	28.52 (101.04)	38.42 (93.69)
Sex= Female	39 (59.1 %)	39 (54.9 %)
Age	16.20 (2.14)	14.96 (1.86)
Household Income Under 30,000	6 (9.1 %)	37 (52.1 %)
Household Income 30,000–60,000	21 (31.8 %)	18 (25.4 %)
Household Income 60,000–150,000	24 (36.4 %)	12 (16.9 %)
Household income Over 150,000	10 (15.2 %)	0 (0 %)
Caregiver Education Level: Less than High School	2 (3.0 %)	9 (12.7 %)
Caregiver Education Level: High School or GED	13 (19.7 %)	15 (21.1 %)
Caregiver Education Level: Some College	14 (21.2 %)	34 (47.9 %)
Caregiver Education Level: Bachelor's Degree	22 (33.3 %)	8 (11.3 %)
Caregiver Education Level: Graduate Degree	13 (19.7 %)	3 (4.2 %)
Using Birth Control	13 (19.7 %)	8 (11.3 %)
BMI Percentile	.75 (.25)	.80 (.25)
Using Steroids	7 (10.6 %)	9 (12.7 %)
Cigarette Use in Past 30 Days	3 (4.5 %)	2 (2.8 %)
Alcohol Use in Past 30 Days	16 (24.2 %)	5 (7.0 %)
Exercised Day of Lab Session	4 (6.1 %)	4 (5.6 %)
Hair Weight	39.79 (19.54)	28.84 (15.05)
Hair Length	12.33 (12.01)	7.78 (5.78)

2.3.2. Hair cortisol concentration

Research staff were instructed to obtain a hair sample if youth had at least 1 cm of hair (or enough hair to cut with thinning shears). We included all samples that exceeded 0.5 cm in the analysis as those with shorter hair lengths were disproportionately Black. As recommended, longer hair samples were cut in the laboratory to 3 cm from the root (Meyer et al., 2014) capturing the accumulation of cortisol for a roughly three-month period prior to measurement (Stalder et al., 2016).

The hair samples were prepared using an adapted protocol (Meyer et al., 2014) that has been described in previous research (Ford et al., 2019, 2021). We used the Salimetrics® high sensitivity enzyme immunoassay cortisol kit to assay the hair samples (in duplicate). Inter- and intra-assay coefficients of variation were 9.1 % and 6.7 %, respectively. We followed Salimetrics® protocol and used the My Assay® analytic software program to calculate cortisol concentrations in μg/dL. The limit of detection for Salimetrics Cortisol ELISA is 0.012 μg/dL. A total of 17 youth had non-detectable values and they were excluded from analysis. We used the formula provided by Meyer et al. (2014) to convert the hair cortisol concentration to pg/mg.

2.3.3. Activity space violent crime levels

Activity space violent crime is based on reported violent crime incidents in the Ohio Incident-Based Reporting System between 2014 and 2016. Incidents are geocoded to Census block groups, which are geographic units with roughly 1000 residents on average that are used for providing summary statistical data. The violent crime rate for each block group is calculated as the count of homicide, robbery, aggravated assault, and rape incidents per 1000 residents. Participants' GPS-derived locations are linked to the block groups and mean exposure to violent

crime rates during the 7-day GPS data collection is weighted by the proportion of time a participant spent in each block group during waking hours. For greater detail regarding the collection and cleaning of continuous GPS data, as well as the calculation of violent crime rates per block group, see (Browning et al., 2017).

2.3.4. Demographic and health-related covariates

We adjusted for a number of covariates that could influence our variables of interest. Age and sex were self-reported, and we coded sex as 0 =female (reference group) and 1 =male. Caregivers reported their household income and level of education. Household income was a four-category measure: $30,000 or less, $30,001–60,000, $60,001–150,000, and over $150,000. Caregiver education was a five-category measure: less than a high school degree, high school degree, some college, college degree, or a graduate/professional degree. Hair weight (mg) and hair length (cm) were provided at the time the hair samples were assessed for cortisol concentration. Youth participants self-reported birth control use, steroid use, alcohol and cigarette use over the prior 30 days, and whether they had exercised that day, all of which were dichotomous measures where 0 =no and 1 =yes. Participants were weighed and their height was measured during the home visit and Body Mass Index (BMI) scores were calculated as weight (kg) / height $(m)^2$ using the Centers for Disease Control and Prevention's recommendations (Cole et al., 2000; Kuczmarski, 2002). Given that our sample consists of youth and adolescents, we adjusted for BMI percentile scores to account for the fact that participants are still growing, thus BMI is expressed as relative to others of their same age and sex. To calculate BMI percentiles, we used the Analyses of Growth Data (AGD) package (van Buuren, 2018).

2.4. Analytic strategy

We conducted a series of multiple regression analyses with C-reactive protein as the outcome variable. These analyses were done for each racial group separately according to Whitfield (2008) and prior work in this sample (e.g. Browning et al., 2021). Our analyses consisted of four models which sequentially controlled for an increasing number of covariates (e.g. Model 4 contains hair sample covariates in addition to all the covariates in Models 1–3): Model 1 (no covariates), Model 2 (sociodemographic covariates), Model 3 (health covariates), and Model 4 (hair sample covariates). We also report models in the supplementary material adjusting for a variety of measures assessing more direct and acute violence exposures, demonstrating our results are driven by area-level and not direct violence exposure. After establishing that the interaction term between cortisol and activity space violent crime remained a significant predictor of C-reactive protein (CRP) above and beyond any potentially confounding variables, we conducted moderation analyses using Model 1 of the PROCESS program (Hayes, 2017) to examine simple slopes of the association of cortisol on CRP at low (-1*SD*), mean, and high (+1 *SD*) levels of activity space violent crime. CRP and cortisol were natural log transformed, and we standardized our key variables (cortisol, activity space violent crime, and CRP). While one Black participant had a CRP value above 4 SD from the mean, this case was not influential on the results of the interaction, thus we did not exclude any participants with complete values from the analyses. For our regression tables, we calculated the interaction term as the product of cortisol and activity space violent crime after standardizing these variables. Given the starkly higher levels of violent area exposure and hair cortisol concentration amongst Black participants, we standardized our variables separately for Black and White participants to interpret the interactions in the context of these different mean levels. Thus, when we examine and interpret "low" and "high" values of activity space violence exposure, we interpret these slopes separately based on the mean for Black participants and the mean for White participants. To illustrate why within-group standardization is most appropriate for this sample, a Black participant with "low" exposure relative to other Black participants have exposure that is about sixty times higher than a White participant with "low" exposure relative to other White participants. Pooled standardization would not account for these environmental differences. Thus, we standardize separately in line with prior work in this sample (e.g. Browning et al., 2021) and with past work arguing the importance of considering within-group heterogeneity as opposed to solely relying on simple between-group comparisons (Whitfield, 2008).

3. Results

3.1. Descriptives

Replicating findings from past work in prior waves of this sample (Ford et al., 2021), Black participants had significantly higher hair cortisol concentration ($M_{\text{Black Participants}}$ = 38.42, $M_{\text{White Participants}}$ = 28.52, $t(135)$ = -3.28, $p < .001$) and significantly higher exposure to violent areas ($M_{\text{Black Participants}}$ = 10.93, $M_{\text{White Participants}}$ = 4.50, $t(135)$ = -6.03, $p < .001$), as was seen in prior waves (Pinchak et al., 2022). Black participants exhibited lower C-reactive protein ($M_{\text{Black Participants}}$ =.60, $M_{\text{White Participants}}$ = 1.20, $t(135)$ = 2.13, p = .035) than White participantssteroid. For a list of correlations amongst all study variables, please see Tables 1–2 in the supplementary material.

3.2. Do activity space violent crime levels moderate the association between hair cortisol and C-reactive protein?

For Black participants, there was a significant interaction between cortisol and violent crime rates within participants' GPS-tracked activity spaces on C-reactive protein (β=.50, p = .002**, 95 %CI [.158,.861; Fig. 1]. This interaction held after adjusting for a variety of covariates (Table 2). BMI was the only covariate significantly associated with C-reactive protein across models, thus we adjusted for BMI in final analyses (N = 70) to assess simple slopes at low (+1 *SD*), mean, and high (+1 *SD*) levels of violent crime rates within activity spaces. At low violent crime rates, higher cortisol was significantly associated with lower inflammation (β = −.34 p = .023, 95 %CI [-.63, −.05]). At mean violent crime rates, cortisol was not significantly associated with C-reactive protein (β =.05, p = .66, 95 % CI [-.16,.26]). At high violent crime rates, higher cortisol was significantly associated with higher C-reactive protein (β =.43, p = .017, 95 %CI [.08,.79]).

For White participants, there was no significant interaction between cortisol and violent crime rates of participants' GPS-tracked activity spaces on C-reactive protein (β= −.16, p = .27, 95 % CI [-.45,.13]; Fig. 1) across models (Table 3). Thus, the violent crime rate of participants' activity spaces did not appear to influence the relationship between cortisol and inflammation. However, for White participants, higher violent crime rate within their activity spaces did significantly predict higher inflammation in Model 1 (β =.27, p = .029, 95 %CI [.03,.05] and Model 4 (β =.33, p = .049, 95 %CI [.002,.64], but was only marginal in Model 2 and was not significant in Model 3.

4. Discussion

Among Black adolescents, we found that a measure of recent, cumulative cortisol exposure showed different associations with a biomarker of inflammation (CRP), depending on the level of violent crime within youths' activity spaces. Additional analyses confirm that these effects held after adjusting for direct measures of both experiencing and witnessing violence (see supplementary material), underscoring the importance of studying indirect and continuous measures of violence exposure (Sharkey et al., 2018). Objective, GPS-tracked activity space violent crime exposure appears to have a distinct and robust association with immune-endocrine functioning, as indicated by the association between cortisol and inflammation.

Structural racism leads to predominantly Black communities and areas having higher rates of violent crime, higher levels of poverty, and fewer resources. This inequity can generate the experience of chronic

Table 2
Coefficients from linear models predicting C-reactive protein for black participants.

	Model 1 (No Covariates)		Model 2 (Demographic Covariates)		Model 3 (Health Covariates)		Model 4 (Hair Covariates)	
	Black Participants (N = 71)		*Black Participants (N = 65)*		*Black Participants (N = 55)*		*Black Participants (N = 55)*	
Predictor	**b(p)**	**95 % CI**	**b(p)**	**95 % CI**	**b(p)**	**95 % CI**	**b(p)**	**95 % CI**
Cortisol	.18 (.130)	[-.05,.41]	.21 (.094)	[-.04,.46]	.21 (.146)	[-.08,.50]	.25 (.098)	[-.05,.55]
Area-Level Violence	.12 (.424)	[-.18,.42]	.13 (.476)	[-.23,.48]	.21 (.355)	[-.24,.66]	.22 (.335)	[-.24,.68]
Interaction Term	.51 (.005**)	[.16,.86]	.50 (.013*)	[.11,.89]	.49 (.018*)	[.09,.89]	.47 (.024*)	[.07,.88]
Sex			.02 (.853)	[-.48,.58]	-.01 (.941)	[-.67,.62]	.06 (.734)	[-.60,.84]
Age			.12 (.369)	[-.08,.21]	.07 (.645)	[-.13,.21]	.03(.851)	[-.16,.19]
Household Income			-.13 (.333)	[-.51,.17]	-.03 (.801)	[-.41,.32]	-.06 (.671)	[-.45,.29]
Caregiver Education			.14 (.306)	[-.13,.40]	.15 (.272)	[-.13,.44]	.16 (.234)	[-.12,.47]
Birth Control					-.01 (.977)	[-.95,.92]	.05 (.756)	[-.83, 1.14]
BMI Percentile					.35 (.016*)	[.27, 2.54]	.33 (.028*)	[.15, 2.47]
Steroid Use					.25 (.052)	[-.01, 1.59]	.27 (.042*)	[.034, 1.66]
Cigarette Use					-.05 (.752)	[2.90, 2.11]	-.01 (.969)	[-2.66, 2.56]
Alcohol Use					.09 (.608)	[-1.05, 1.78]	.05 (.809)	[-1.31, 1.67]
Exercise					.13 (.322)	[-.55, 1.62]	.15 (.272)	[-.494, 1.71]
Hair Weight							.11 (.471)	[-.01,.03]
Hair Length							.10 (.563)	[-.04,.08]

stress rooted in the physical spaces where Black adolescents spend time, which the present work demonstrates can manifest into disrupted stress biology, even at a young age. Adolescence is a sensitive period during which stressors, such as exposure to violent areas, can influence long-term health and stress trajectories (Eiland and Romeo, 2013; Ford et al., 2021; Heinze et al., 2017). The HPA-axis and immune system have both been suggested to be key pathways through which stress can lead to disease (Cohen et al., 2016; McEwen, 1998b), underscoring the importance of studying the interplay between these systems in the context of chronic stressors.

For Black adolescents with low violence exposure, cortisol had a significant negative association with inflammation as would be expected under normative conditions. It is important to note that Black adolescents with low levels of violence exposure relative to other Black participants still experienced levels of exposure that were about *sixty* times higher compared to White adolescents with low exposure relative to other White adolescents. Thus, low exposure for Black and White participants represents drastically different levels of exposure. Relatively low exposure for a Black adolescent may still represent a level of exposure high enough to be a stressor. The negative association between cortisol and inflammation for Black adolescents with relatively low exposure may be consistent with "suppressive" effects, where stressor exposure and cortisol levels become high enough to inhibit inflammatory activation but are not indicative of endocrine-immune dysregulation (Sapolsky, 2015). In line with the framework of suppressive effects, Black participants with low violence exposure but high cortisol exhibited the lowest average levels of inflammation observed in the sample (see Fig. 1).

High violence exposure was associated with a significant *positive* association between cortisol and inflammation for Black adolescents. This association is consistent with multiple mechanistic accounts. For example, in the glucocorticoid resistance model, persistent exposure to glucocorticoids can lead to multiple downstream adaptations in glucocorticoid signaling pathways that culminate in reduced inhibition of inflammatory synthesis pathways (Avitsur et al., 2001; Miller et al., 2002). Other models suggest that cortisol does not fail to inhibit inflammation under chronic stress but rather upregulates inflammation to prime the immune system for future stressors. For example, the pro-inflammatory cortisol model suggests cortisol may be triggering the increases in inflammation (do Prado et al., 2017; Horowitz et al., 2020; Nikkheslat et al., 2020; Raison and Miller, 2003). Similarly, cortisol has been theorized to have "stimulating" effects on other biological systems, including the immune system, in response to chronic and unpredictable stressors (Sapolsky, 2015). Regardless of the actual mechanism(s) underlying this association, a positive association between cortisol and inflammation may indicate risks to mental and physical health for these adolescents.

Over time, HPA axis overactivation and a lack of inhibitory association between glucocorticoids and inflammation are thought to contribute to the pattern of hypercortisolism and elevated inflammation observed in illnesses related to chronic stress (Perrin et al., 2019; Silverman and Sternberg, 2012; Amasi-Hartoonian et al., 2022; Miller and Raison, 2016; Raison and Miller, 2003). In addition to observing a significant and positive association between cortisol and inflammation for Black adolescents with high violence exposure, the highest average levels of inflammation observed in the sample occurred for Black adolescents with high violence exposure and high cortisol (Fig. 1). Thus, over time, a positive association between cortisol and inflammation could lead to chronic hypercortisolism and elevated inflammation.

With respect to White adolescents, there was a different pattern of associations between exposure to neighborhood violence and the biomarkers. Notably, there was no interaction between measures for White adolescents. Thus, we did not find evidence that differences in rates of activity space violent crime levels were associated with differential relationships between cortisol and CRP. However, consistent with past work suggesting that community violence is associated with elevated inflammation (Browning et al., 2012; Broyles et al., 2012; Finegood et al., 2020), White adolescents did exhibit a positive association between violence exposure and inflammation, although the effect was not consistently significant across all models. This main effect should be interpreted in the context of White adolescents' activity spaces being approximately one third lower in violent crime on average than the activity spaces of Black adolescents, and that mean levels of cortisol were also significantly lower amongst White adolescents than Black adolescents.

The stress of exposure to violence amongst White adolescents, albeit comparatively lower than that of the Black adolescents, would appear to be consistent with "permissive" effects that are theorized to occur at basal levels of glucocorticoids (Munck and Náray-Fejes-Tóth, 1992; Sapolsky et al., 2000). At low levels of glucocorticoids, inflammation triggered by other pathways such as the sympathetic nervous system (Way and Uchino, 2025) is not inhibited. Within this framework, permissive effects are consistent with the weak main effect of violence exposure on elevated inflammation that we observed for White participants as well as White participants having lower cortisol levels on average. Although violence exposure is a stressor for White participants, they experienced significantly lower levels of exposure and exhibited lower levels of cortisol. Relatively lower stressor exposure may not generate a cortisol response sufficient to inhibit inflammation, permitting low levels of elevated inflammation. This framework may also contextualize why higher average CRP levels were observed for White participants and why cortisol did not have a significant negative

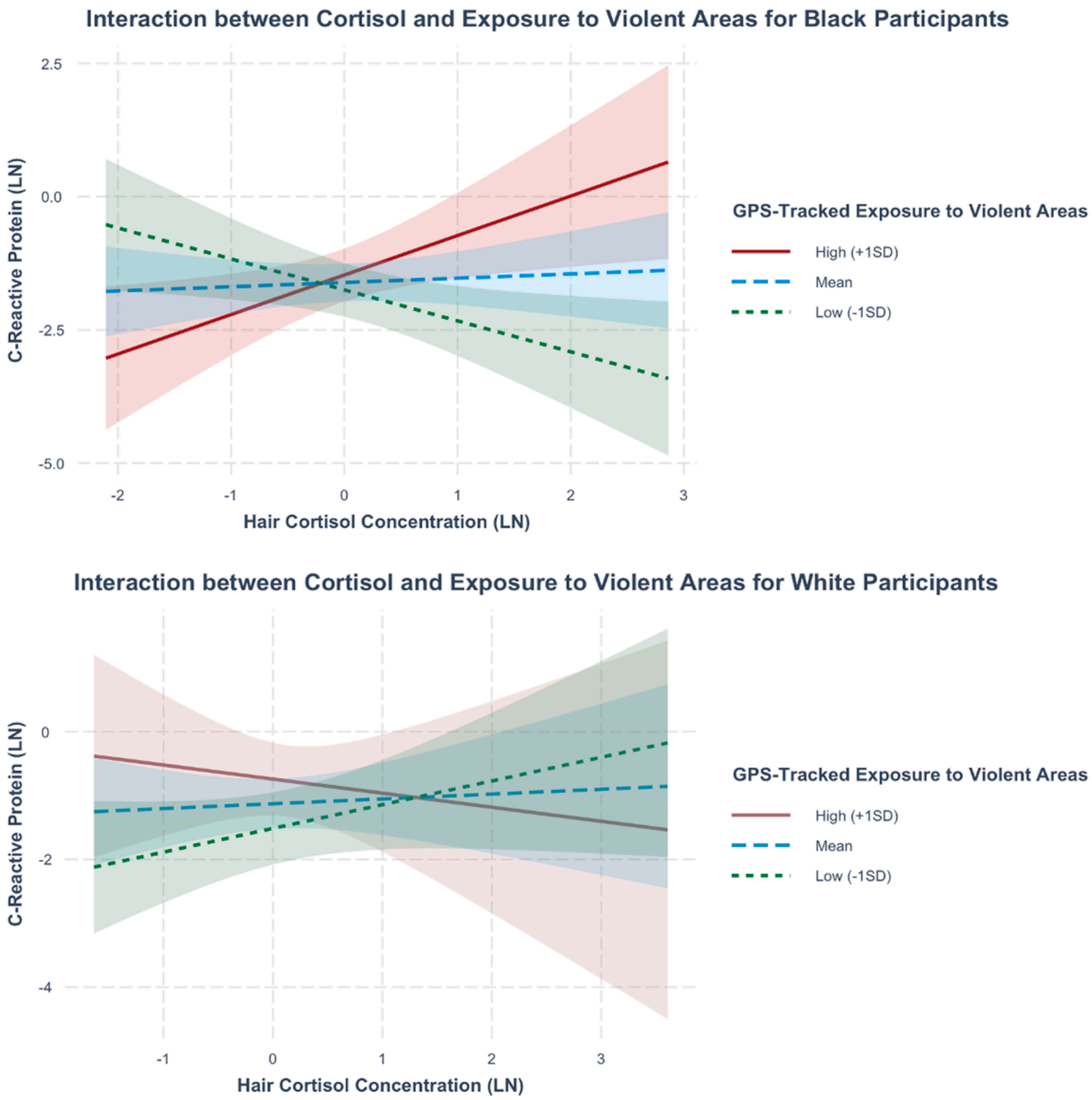

Fig. 1. Predicted C-reactive protein (CRP) values as a function of hair cortisol concentration (HCC) and GPS-tracked, activity space violent crime levels for Black and White participants.

Table 3
Coefficients from linear models predicting C-reactive protein for White participants.

	White Participants (N = 66)		*White Participants (N = 59)*		*White Participants (N = 53)*		*White Participants(N = 52)*	
Predictor	**b(p)**	**95 % CI**	**b(p)**	**95 % CI**	**b(p)**	**95 % CI**	**b(p)**	**95 % CI**
Cortisol	.07 (.604)	[-.19,.32]	-.01 (.927)	[-.29,.27]	.031 (.034*)	[-.30,.36]	.09 (.594)	[-.24,.41]
Area-Level Violence	.27 (.029*)	[.03,.05]	.27 (.083)	[-.04,.57]	.27 (.113)	[-.07,.60]	.33 (.049*)	[.002,.64)
Interaction Term	-.16 (.304)	[-.47,.15]	-.04 (.782)	[-.35,.26]	.016 (.939)	[-.40,.44]	-.03 (.873)	[-.44,.37]
Sex			-.19 (.145)	[-.88,.13]	-.21 (.193)	[-1.03,.21]	-.23 (.171)	[-1.08,.20]
Age			.24 (.070)	[-.01,.23]	.25 (.155)	[-.04,.26]	.29 (.092)	[-.02,.27]
Household Income			-.34 (.083)	[-.81,.05]	-.25(.246)	[-.75,.20]	-.18 (.416)	[-.68,.29]
Caregiver Education			.44 (.007**)	[.11,.66]	.36 (.041*)	[.01,.60]	.40 (.019*)	[.06,.62]
Birth Control					.20 (.233)	[-.30, 1.17]	.18 (.250)	[-.31, 1.16]
BMI Percentile					.30 (.035*)	[.08, 2.16]	.33 (.019*)	[.06,.62]
Steroid Use					-.04 (.770)	[-.99,.74]	-.08 (.564)	[-1.07,.60]
Cigarette Use					-.16 (.258)	[-2.16,.60]	-.08 (.545)	[-1.76,.94]
Alcohol Use					.07 (.610)	[-.46,.77]	-.002 (.987)	[-.61,.60]
Exercise					-.09 (.540)	[-1.34,.71]	-.07 (.629)	[-1.25,.76]
Hair Weight							.39 (.034*)	[.001,.04]
Hair Length							-.38 (.038*)	[-.06, −.002]

association with CRP for White participants across any regression models. If conditions for most White participants were consistent with permissive effects (i.e. lower cortisol and relatively lower violence exposure), we would expect to see a lack of negative association between cortisol and inflammation, and in turn, slightly elevated inflammation on average.

The results highlight that understanding how the magnitude of stressor exposure influences the association between cortisol and CRP requires consideration of the environments where adolescents spend time. Here, "low" and "high" exposure was shown to be starkly different for Black and White adolescents, and the effects of low and high exposure on the association between cortisol and CRP were best understood within the context of the differing environments where Black and White adolescents spend time.

4.1. Limitations and future directions

One limitation of the current study is the cross-sectional nature of the data. We cannot establish causality between activity space violent crime levels and the association between cortisol and inflammation. Thus, it cannot be established whether cortisol is inhibiting or escalating inflammation (or vice versa). Clarifying directionality would require a longitudinal study with multiple measurements of violence exposure and levels of cortisol and inflammation, as well as a more direct measurement of immune-endocrine functioning such as ex vivo blood stimulation. Such a design would clarify whether violence exposure and elevated cortisol levels precede disruptions to immune-endocrine functioning and variation in inflammatory activation. Implementing a multiwave design in a young sample with a broader age range of both children and adolescents could also provide insight into the age or developmental stage at which violence has more pronounced effects on cortisol, inflammation, and their interactive influences.

Second, there were differing time frames amongst our study measures and potential confounding measures not adjusted for in the present study. The GPS tracking week was designed to capture an average week in the participants' lives. Thus, if participants' GPS-tracked data suggested high violence exposure, it is likely, though not certain, that this is a stressor they encounter chronically and not only characteristic of the week when data was collected. Similarly, our hair cortisol measure assessed cortisol concentration over about a three-month period, suggesting a more chronic measure of biological stress (Russell et al., 2012) but we are still limited by only one time point of cortisol levels in the present study. CRP typically changes in the order of days to weeks (Mehta et al., 2010; Paine et al., 2013), though there are reports of stability over longer periods (Hardikar et al., 2014; Navarro et al., 2012). Our measurement of cortisol and GPS-tracked activity space violent crime levels suggest that these measures have some temporal precedence to our outcome measure, CRP. However, we cannot definitively establish temporal precedence amongst study measures. The conclusions are also limited by potentially confounding variables not adjusted for in the present study. Due to an administrative error, we did not collect information about hair products or hair treatments from all participants. Additionally, although we adjusted for steroid use, we did not adjust for all medications used by participants.

Finally, although a strength of using GPS-tracked exposure is that it is objective, it is solely a measure of stressor exposure and does not capture appraisals or psychological responses to stressors associated with biological changes. Future research can seek to understand the psychological factors (e.g. predictability, controllability, upregulation of vigilance) that make this such a powerful stressor.

4.2. Conclusions and implications

Our results suggest that environmental exposures can lead to different patterns of associations between outputs of the HPA axis and the immune system. Furthermore, results suggest a novel biological link through which heightened exposure to activity space violent crime levels, rooted in structural inequity, might contribute to poorer health outcomes. If replicated, the results suggest the association between glucocorticoids and inflammation may be an important avenue for intervention in ameliorating the effects of heightened exposure to violence on mental and physical health outcomes.

Observing these effects in a relatively young and healthy sample underscores the need for interventions aimed at reducing area-level inequity and chronic stressor exposure, especially during key developmental periods where chronic stress can create lasting harm on mental and physical health into adulthood. The most impactful way to prevent these harmful outcomes is to address the root of inequity, which stems from longstanding policies and practices that marginalize and divert resources away from predominantly Black communities. Poverty, poor housing conditions, and a lack of resources to care for community spaces have been associated with higher rates of violent crime (For a review, see MacDonald et al., 2024), thus investing in communities to improve these factors may in turn reduce the inequity driving higher exposure to chronic, area-level stressors in adolescence.

At the individual level, future work should examine the features of communities and areas that positively influence adolescent health and might buffer against the effects observed in this study. Establishing safe social networks, as well as financial and residential stability, is critical in the face of chronic stress due to inequity. Prior work highlights the factors at various levels (e.g. family, neighborhood, policies and laws) that can help an individual establish feelings of security, safety, and connection (Diamond and Alley, 2022; Hurd et al., 2013).

CRediT authorship contribution statement

Christopher R. Browning: Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Conceptualization. **Bethany L. Boettner:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization. **Wilson Kendra:** Writing – original draft, Visualization, Project administration, Investigation, Formal analysis, Data curation, Conceptualization. **Baldwin M. Way:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization. **Jodi L. Ford:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Conceptualization.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Baldwin Way, Christopher Browning reports financial support was provided by National Institute on Drug Abuse. Baldwin Way reports financial support was provided by John Templeton Foundation. Catherine Calder, Christopher Browning reports financial support was provided by National Institute of Child Health and Human Development. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2025.107722.

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