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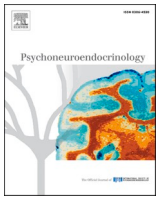

Interpersonal violence exposure and inflammation during adolescence and young adulthood

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ABSTRACT

Exposure to violence increases young peoples' risk of developing mental and physical health problems. Chronic stress-related upregulation of innate immune system activity and the development of low-grade inflammation may partially underlie this health risk. However, much of the previous research has been limited to cross-sectional studies utilizing between-person analytic designs, susceptible to confounding by unmeasured factors. In this six-wave panel study of N=157 female adolescents and young adults, we tested within-person associations between interpersonal violence exposure and multiple measures of inflammatory activity. Ex vivo culture studies suggested that participants' immune cells were more reactive to microbial stimulation and less sensitive to inhibition by glucocorticoids after violence. Numbers of circulating monocyte cells increased after violence, but serum levels of interleukin-6 and c-reactive protein did not. Findings from this within-person analysis suggest that violence exposure up-regulates innate immune system activity during adolescence and young adulthood in ways that may increase mental and physical health risk.

1. Introduction

Interpersonal violence or "the intentional use of physical force or power, threatened or actual, by a person or a small group of people against another person or small group that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation" (World Health Organization, 2014, p. 82), is a significant cause of injury and mortality among youth (Centers for Disease Control and Prevention, 2021; World Health Organization, 2014). Interpersonal violence can take many forms and is prevalent. Over 1 in 5 adolescents aged 14–17 have been injured by an assault, over 1 in 3 (38%) have experienced maltreatment, and 1 in 4 have witnessed interparental violence (Finkelhor et al., 2015). In addition to the risk of physical injury, violence increases young people's risk of developing psychiatric problems including posttraumatic stress disorder (PTSD) and depression (Foster and Brooks-Gunn, 2009; Fowler et al., 2009; McLaughlin et al., 2013; Slopen et al., 2012) as well as physical health problems including asthma, chronic pain (McLaughlin et al., 2016), and high blood pressure (Wright et al., 2017).

Relatively little is known about the mechanisms that explain how violence exposure in early life could translate into later life health problems, although, increasingly, studies suggest that a psychobiological mechanism involving stress-induced upregulation of innate immune system activity and inflammation may play a role (Finegood and Miller, 2021; Miller et al., 2011; Suglia et al., 2015; Tawakol et al., 2019). The body's inflammatory response is a critical defense against infections and injuries and is essential to survival. However, when inflammation is sustained chronically at a low-grade level it plays a role in psychiatric problems linked to violence exposure including PTSD and depression (Miller and Raison, 2016), and is a major player in health problems like diabetes, some cancers, and CVD (Hotamisligil, 2006; Nahrendorf, 2018). A nascent body of research suggests that violence exposure in

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childhood and adolescence, for example, exposure to maltreatment (Ehrlich et al., 2020; Jonker et al., 2017; Serbulent et al., 2017) or community violence (Finegood et al., 2020) is associated with higher inflammatory activity in youth, and there is some evidence that this relation may persist across life. For example, children who are exposed to violence evidence excess inflammation in early adulthood (Baldwin et al., 2018; Danese et al., 2007; Rasmussen et al., 2020). Collectively, this literature suggests that violence in early life gives rise to a pro-inflammatory phenotype that increases risk for a range of stress-related mental and physical health problems in adulthood.

Still, this literature is subject to three important limitations. First, much of the existing evidence has come from cross-sectional studies of children or from studies of adults retrospectively reporting on their early life experience; neither design providing a strong account of how violence-related health effects might unfold across development. To address this, the current study utilized panel data in young people to explore whether individual changes in violence exposure were associated with changes in inflammatory activity. Second, nearly all studies in this area assess inflammation using circulating biomarkers such as C-reactive protein (CRP) or cytokines such as interleukin-6 (IL-6). Although these biomarkers predict adverse health outcomes, they are coarse indicators of inflammation, and difficult to interpret. They are released by multiple body tissues, not just cells of the immune system (Furman et al., 2019; Medzhitov, 2021). To get deeper mechanistic insights, the current study supplemented these biomarkers with counts of monocytes, immune cells that play a key role in sensing threats and mobilizing the inflammatory response. We also conducted functional studies of subjects' monocytes, stimulating them in vitro with a bacterial product, lipopolysaccharide (LPS), and measuring their production of the cytokine IL-6. The stimulated cells were also treated with glucocorticoids, a steroid hormone that inhibits inflammation, to model how effectively their inflammatory response is down-regulated. Based on studies of other early-life stressors (Chiang et al., 2022), we predicted that violence would be associated with a more aggressive monocyte response to bacterial stimulation and less sensitivity to downregulation by glucocorticoids.

A third limitation is that studies in this area often utilize correlational between-person analytic designs, which are limited (to varying degrees) in terms of internal validity. For example, selection and omitted variable bias are often of concern given that childhood violence exposure can co-occur with other health-relevant psychosocial and environmental stressors (e.g., economic hardship, harsh caregiving, neighborhood disadvantage). One analytic strategy that can reduce these concerns is to test within-person associations between violence and inflammatory activity; comparing individuals to themselves at times when they have vs. have not experienced violence, which controls for the influence of variables that are invariant at the person level (Allison, 2009). With this approach, any observed within-person association between violence and inflammation would control for aspects like individuals' family and socioeconomic context, to the extent that these aspects are invariant across the study, as well as all other person-level factors including heritable genetic makeup.

To advance understanding in this area, we applied a within-person design to questions concerning violence exposure and inflammatory activity. In a six-wave study of adolescents and young adults, we hypothesized that violence exposure would be positively associated with pro-inflammatory activity on a within-person basis. Specifically, we expected that participants would evidence increased inflammatory activity (both in terms of circulating biomarkers, like CRP and IL6, and indicators of monocyte response to stimulation) at times when they had recently been exposed to violence compared to times when they had not been recently exposed to violence.

2. Material and methods

2.1. Participants and procedure

The study took place from years 2004–2007. Study participants were 147 female adolescents and young adults from Vancouver, Canada. Details regarding study recruitment and eligibility criteria have been described elsewhere (Miller and Cole, 2012) and are summarized here. Participants had to be 15–19 years of age, fluent in English, free of acute and chronic medical illnesses, without a history of major psychiatric disorders, and not currently taking medications other than birth control. Subjects had to be at high risk for having an episode of depression over the study follow-up, operationalized as having a first-degree relative with a history of depression or having elevated cognitive vulnerability to depression (scoring in the top quartile on the Dysfunctional Attitudes Scale (Beck et al., 1991) or the Adolescent Cognitive Style Questionnaire (Hankin and Abramson, 2002). A small additional group (n=10) who met the same eligibility criteria, but who were at lower risk of having an episode of depression, was recruited simultaneously and completed the same protocol. Thus, a total of 157 young people participated in the study.

Individuals participated in up to six laboratory visits taking place at approximately six-month intervals across 2.5 years of study. Blood was obtained via antecubital venipuncture at each lab visit. To control for circadian and dietary variations, sessions always occurred between 8:00 am and 11:00 am, following an overnight fasting period. The blood was used to assess multiple aspects of inflammation, described below. Participants also took part in several survey- and interview-based assessments of health and psychosocial functioning. Subjects gave written consent to participate. For those younger than 18, consent was also obtained from a parent or guardian. All study procedures were approved by the University of British Columbia's Research Ethics Board.

2.2. Measures

2.2.1. Interpersonal violence exposure

At each study visit, participants took part in a UCLA Life Stress Interview (LSI) - Adolescent Version (Adrian and Hammen, 1993) with trained study personnel. The LSI is a semi-structured interview that captures both chronic and episodic stressors that occurred over the previous six months. The interviewer asks a series of open-ended questions about different areas of the participant's life (e.g., romantic relationships, friendships, home and family life, school, health, and finances). In each domain, the interviewer rates the level of chronic, ongoing stress. The interviewer also collects details of episodic stressors, which are defined as specific events with a discrete onset and offset. To judge the objective impact of episodic stressors, our team made a consensus rating for each event after being briefed on its details by the primary interviewer. Impact ratings ranged from 1, no long-term impact, to 5, severe long-term impact. The ratings explicitly consider the context in which each event occurred. For example, if a participant's grandfather had a heart attack, the impact rating would depend on factors such as the closeness of the relationship between the participant and the grandfather, whether she visited him in the hospital, and whether she had previous experience coping with serious family illnesses. Following convention (Hammen et al., 2000; Miller and Chen, 2010), we considered episodic stressors rated 2.5 or higher, reflecting moderate long-term impact, to be major events. Before beginning the interview, participants were informed by the interviewer that their responses during the interview would be kept confidential, except in the case that there was a disclosure of child abuse or of harm to self or to others.

In 2021, the LSI interviews were re-coded for interpersonal violence exposure. Coding was done at the interview level, meaning that each LSI interview at each study visit was assigned a code of 1 or 0 according to the whether any of the major episodic stressors (those rated 2.5 or

higher) described in it involved interpersonal violence or not, respectively. Interpersonal violence was operationalized according to the World Health Organization's definition as "…the intentional use of physical force or power, threatened or actual, by a person or a small group of people against another person or small group that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation" (World Health Organization, 2014, p. 82). This included experiences such as family violence (e.g., intimate partner violence), community violence (e.g., physical or sexual assault, violent crime, gang violence), as well as other forms of violence (e.g., teen dating violence, bullying/harassment, being in a physical fight). Individuals were considered to have been exposed to interpersonal violence if they were either directly victimized, were a witness to a violent event, or if someone close to them (e.g., a family member) was directly victimized.

Individuals had up to six codes for violence exposure - one for each study visit at which they completed the LSI. One study team member (E.D.F.) coded all LSI interviews that had a major life event (178 interviews). For reliability purposes, a second study team member (R.W-T.) independently coded 50% of these interviews, selected at random, which is a standard approach to coding narrative data (Syed and Nelson, 2015). Interrater reliability on these double-coded interviews was excellent (Cohen's kappa=0.95; percent agreement = 98.88%).

2.2.2. Low-grade inflammation

The extent of low-grade inflammatory activity was quantified via serum levels of C-reactive protein (CRP) and interleukin-6 (IL-6), the two most commonly measured biomarkers of this phenomenon. Blood was drawn into Serum-Separator Tubes (Becton-Dickinson, Oakville, ON). After serum was harvested by centrifugation, it was frozen at −30 C. CRP was measured by high-sensitivity chemiluminescence on an Immulite 2000 (Diagnostic Products Corporation, Los Angeles, CA). This assay has a minimum detection threshold of 0.20 mg/L and intra-assay variability of 2.2%. IL-6 was measured in duplicate by commercially available high-sensitivity ELISA (R&D Systems; Minneapolis, MN). These kits have a minimum detection threshold of 0.039 pg/ml. Intra-assay variability for duplicate pairs was < 10%. Prior to analysis, CRP and IL-6 values were natural log transformed to correct for positive skew in their distributions at each study visit.

2.2.3. Interleukin-6 production to microbial challenge

To assess how aggressively participants' monocytes responded to microbial challenge, we cultured them with a bacterial stimulus, lipopolysaccharide (LPS), and measured production of IL-6. Whole blood was drawn into lithium-heparin Vacutainers (Becton-Dickinson, Oakville, ON), diluted 10:1 with saline, and then incubated with LPS (50 ng/ml; Sigma; Saint Louis, MO) for 6 h at 37°C in 5% CO_2. The supernatants were collected and frozen at −80°C until analysis. IL-6 was assayed in duplicate by ELISA with kits that have a minimum detectable threshold of 0.7 pg/ml and inter- and intra-assay variability for duplicate pairs was below 5% (DuoSet ELISA Development Systems; R&D Systems). Stimulated IL-6 was normally distributed and therefore not log transformed.

2.2.4. Glucocorticoid sensitivity

To measure sensitivity to signals that regulate inflammation, we quantified IL-6 production in cells that had been co-incubated with LPS and cortisol. At high levels cortisol conveys anti-inflammatory messages to monocytes, and this assay measures their ability to dampen IL-6 production when signaled to do so. Blood was diluted 10:1 with saline and dispensed into 6-well culture plates with LPS (50 ng/ml), along with one of five doses of hydrocortisone (final concentrations: 0, $2.76*10^{-5}$, $2.76*10^{-6}$, $2.76*10^{-7}$, $2.76*10^{-8}$ M HC; Sigma Chemicals; St. Louis, MO, USA). After six hours of incubation at 37°C in 5% CO^2, the supernatants were collected and frozen until analysis. IL-6 levels were quantified in duplicate using the same ELISA kits (DuoSet ELISA Development Systems; R&D Systems). A specimen-specific inhibition slope was then estimated, where higher values reflected more sensitivity to glucocorticoid inhibition (Chiang et al., 2019).

2.2.5. Monocyte count

Because monocyte cells are the major cellular source of these inflammatory processes, we quantified their presence in circulation. This was done by automated complete blood count with differential on an ADVIA 70 Hematology System (Holiston, MA).

2.3. Analysis plan

The current analysis was pre-registered on the Open Science Framework on February 18, 2021 (https://osf.io/vba48). The pre-registration details our a-priori analytic plan. Deviations from the original pre-registered plan are described in the Online Supplement. We used fixed-effects (FE) panel regression models (Allison, 2009) with STATA version 16 (StataCorp, LLC) to estimate within-person associations between interpersonal violence exposure and each of the indicators of inflammatory activity. Each FE model additionally included a dummy code for timepoint of observation. We also undertook several sensitivity analyses to assess the sensitivity of model estimates to various model specifications.

3. Results

3.1. Descriptive analyses

Table 1 displays descriptive statistics of the sample. At study entry, participants were 17 years old, on average (M=17; SD = 1.3). Approximately 48% of participants identified their ethnic background as European, 43% identified as South or East Asian, and 8% identified as another ethnicity. Descriptive information concerning body mass index, parent education, and average values of the inflammatory biomarkers are also shown in Table 1.

Of the 157 participants, n=17 had been exposed to violence during the 2.5-year study period. There were no group differences between those who had experienced violence (n=17) and those who had not (n=140) in terms of age at the baseline visit, ethnic background, body mass index, and average levels of the inflammatory outcomes (all p-values > 0.05). Of the 17 participants, 11 had been exposed to a single

Table 1
Descriptive statistics of the sample (N=157).

	N (%) / M (SD)
Exposed to interpersonal violence	17 (10.8%)
Age at study entry (years)	17 (1.3)
Body mass index at study entry	21.7 (2.6)
Ethnic background	
South or East Asian	68 (43.3%)
European	76 (48.4%)
Other	13 (8.3%)
Parent education	
High school or less	23 (14.7%)
Some college or Associates degree	53 (33.8%)
Bachelor's degree	48 (30.6%)
Advanced degree	29 (18.5%)
Missing data	4 (2.5%)
Inflammatory biomarkers	
Circulating C-reactive protein (CRP; mg/L)	0.82 (1.40)
Circulating interleukin-6 (IL-6; pg/ml)	0.73 (.67)
Monocyte count (x 10^9 cells per L)	0.40 (.12)
IL-6 production after LPS stimulation (pg/ml)	47506 (16977)
Glucocorticoid sensitivity	0.56 (0.04)

Note. Descriptive statistics for inflammatory biomarker data shown here are from across all study visits. Values for circulating CRP and IL-6 are shown here in their raw units, however, prior to analysis, these values were natural log transformed at each study visit to correct for positive skew in their distributions. LPS = lipopolysaccharide

violent event across the duration of the study and 6 had experienced more than one event. Types of violence included exposure to violent crime in the community, sexual violence, physical fight, intimate partner violence, family violence, threatened or harassed, bullying, and gang violence. A total of 24 LSI interviews included an event coded as violence.

3.2. Fixed-effects regression models

Fixed-effects panel regression analyses were run separately for each of the five outcomes. The results are displayed in Fig. 1. Results suggested that individuals' immune cells produced more IL-6 following LPS stimulation (b=9145.77, p=0.002, 95% CI = 3238.68, 15,052.86) and were less sensitive to inhibition by glucocorticoids (b= −0.02, p=0.01, 95% CI = −0.03, −0.004) at visits when they had experienced violence in the previous six months compared to visits when they had not. Consistent with this observation, individuals also had higher numbers of circulating monocytes at visits when they experienced violence compared to visits when they had not (b=0.04, p=0.03, 95% CI = 0.004, 0.08). In contrast to these observations, there was no within-person association between violence exposure and markers of low-grade inflammation, as reflected in circulating levels of IL-6 (b=0.06, p=0.57, 95% CI = −0.16, 0.29) or CRP (b= −0.16, p=0.35, 95% CI = −0.50, 0.18). Figures S1-S5 in the Online Supplement depict individual-level scatterplots.

3.3. Sensitivity analyses

One possible explanation for the observed within-person associations is that they reflect a more general stress process. If this were the case, we might also expect to observe similar within-person associations when coding for individuals' exposure to major episodic stressors in general and not just violence specifically. We tested this in a sensitivity analysis where all LSI interviews that included at least one episodic stressor rated on impact as >= 2.5 were coded as 1 (otherwise coded as 0). Fixed-effects regression models were re-run to investigate within-person associations between major episodic stressors and variation in the inflammatory outcomes. In this sensitivity analysis, none of the within-person associations between major episodic stressors and inflammatory outcomes were statistically significant (Table S1 in the Online Supplement). The implication of these null findings is not that violence is the only type of interpersonal stress associated with these inflammatory patterns, rather, the findings provide further evidence that violence may be one specific form of severe interpersonal stress that may promote inflammation. The range of stressors coded here as major episodic stressors varied in terms of their severity, chronicity/duration, and proximity in time to individuals' lab visits, and collectively were not associated with the inflammatory outcomes at the within-person level.

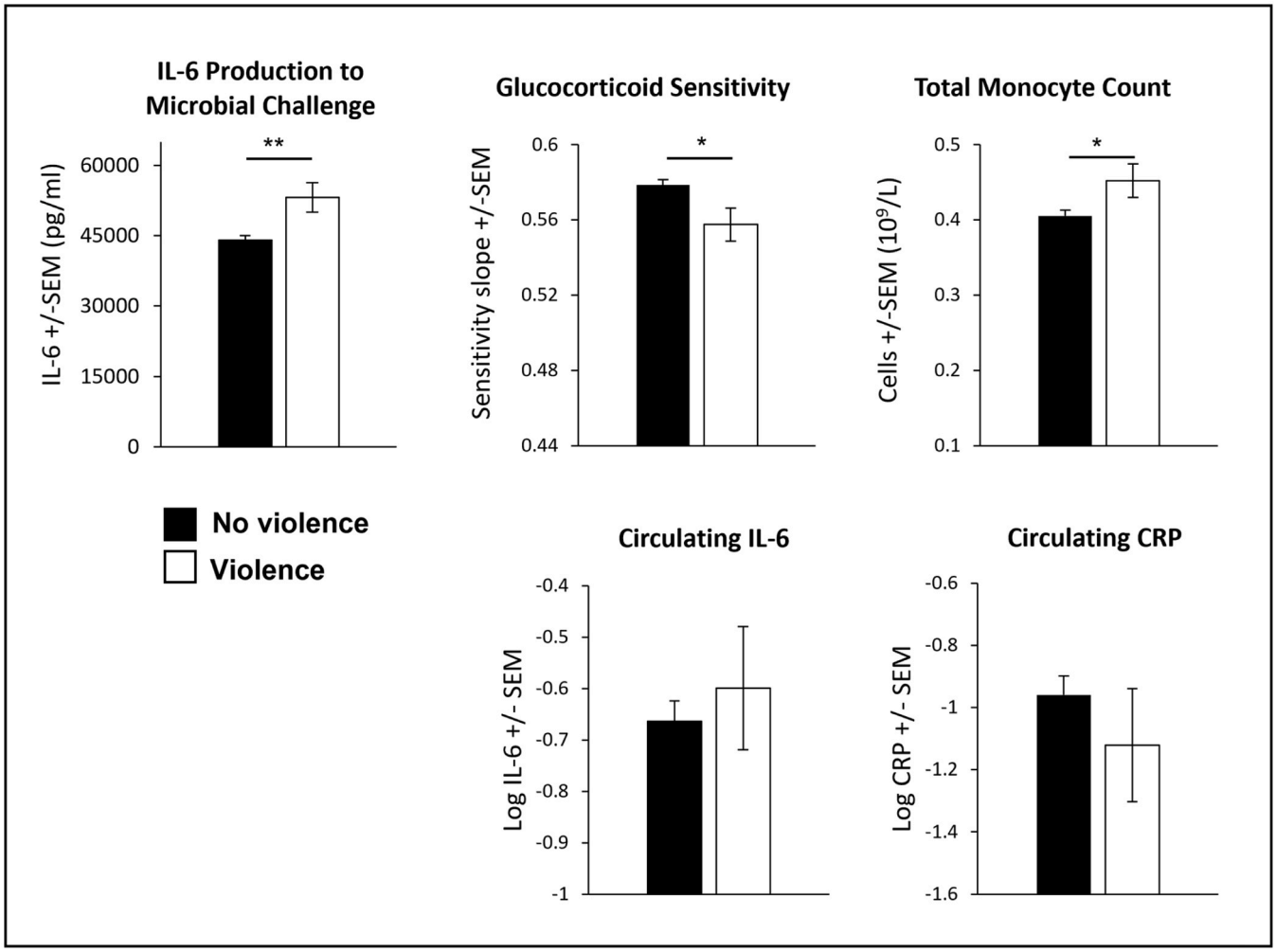

Fig. 1. Within-person associations between violence exposure and inflammatory activity. **Note.** Fixed-effects (within-person) panel regression analyses comparing pro-inflammatory activity after violence exposure vs. no violence exposure. The mean values depicted in Fig. 1 reflect levels of inflammatory activity at study visit 1. However, the difference between violence and no violence in each model (i.e. the violence effect) is constant across study visits. Note: **= $p < 0.01$; *= $p < 0.05$; SEM= standard error of the mean; IL-6= interleukin-6; CRP=C-reactive protein.

Another possibility is that the observed within-person associations are not due to violence, per se, but rather, to omitted time-varying variables that increase both an individual's likelihood of being exposed to violence and their inflammatory activity. This could include contextual variables such as changes in young peoples' relationship with their parents and changes in family function more broadly, as well as changes in the financial/economic circumstances of families. As a test of whether these contextual factors might be confounding the observed within-person associations between violence exposure and inflammatory activity, we re-ran the fixed-effects models and included interviewer ratings of chronic strain in young peoples' family relationships and interviewer ratings of household conditions/finances as additional time-varying covariates (see Table S2 [family relationships] and Table S3 [family finances] in the Online Supplement). These additional time-varying covariates were not significantly associated with within-person variation in any of the inflammatory outcomes nor did their inclusion in the models substantively change the coefficients for violence exposure.

Lastly, we considered whether the within-person associations observed for IL-6 production to LPS and glucocorticoid sensitivity were primarily due to more inflammatory cells (e.g. monocytes) being present in the context of violence. When the fixed-effects analyses were re-run including monocyte count as a time-varying covariate, the within-person associations between violence and IL-6 production as well as glucocorticoid sensitivity remained statistically significant, although the strength of coefficients was reduced in these models (Table S4 in the Online Supplement). These findings suggest that violence was associated with a functional difference on a per-cell basis, as well as an increase in the number of circulating inflammatory cells.

4. Discussion

Exposure to violence early in life increases the risk that young people will develop mental and physical health problems (Margolin and Gordis, 2004; Suglia et al., 2015; Wright et al., 2017). Chronic stress-related upregulation of innate immune system activity and low-grade inflammation have been hypothesized to partially underlie this increased health risk (Finegood and Miller, 2021), although questions remain about the association between violence and inflammatory activity in young people.

The current study advances insights in two primary ways. The first is that we utilized panel data and fixed-effects modeling to examine the association between violence exposure and inflammatory activity; testing this association within persons as opposed to between persons, to rule out all person-level confounds as alternative explanations. Findings suggest that it is unlikely that the associations between violence and aspects of inflammatory activity in young people are confounded by, for example, earlier features of the family environment and socioeconomic context (at least those aspects that are unchanging across the study period) or by heritable genetic traits. A strength of the panel data design and fixed effects analysis is that they provide a strong basis for inference where there are inherent challenges in utilizing a true experimental design. Having said this, the current study eliminates many, but not all, alternative explanations, so strong causal inferences are not permitted. There have been, however, a number of experimental animal studies that do provide evidence of causal effects of violence on inflammatory activity (Weber et al., 2017) and many of these studies observe patterns consistent with our findings.

The second way that this study advances insights is that, in addition to measuring biomarkers of low-grade inflammation, we also considered the prevalence and functioning of monocytes. These cells play a central role in the body's innate immune response to pathogens and injuries (Nahrendorf, 2018), and our findings indicate that after violence, young people had higher counts of circulating monocytes. Further, their immune cells exhibited pro-inflammatory tendencies after violence, indicated by heightened reactivity to bacterial stimulation and lower sensitivity to inhibitory signals from glucocorticoids. As noted above, these patterns are generally consistent with findings from experimental rodent studies of social defeat (Weber et al., 2017), a psychosocial stress paradigm involving physical conflict that is violent in nature. Social defeat increases the prevalence of circulating monocytes, particularly the prevalence of "classical" pro-inflammatory monocytes that are more resistant to glucocorticoids. These cells migrate into tissues where disease has developed or damage has occurred, including the brain and the lungs, visceral fat depots and coronary artery walls; and in animal models of psychosocial stress, the mobilization of these cells increases anxiety-like behavior (Weber et al., 2017), contributes to airway pathology following viral infection (Sheridan et al., 2006), and accelerates the progression of atherosclerosis, which underlies many ischemic strokes and heart attacks (Nahrendorf, 2018).

Although we observed evidence of violence-related variation in monocyte prevalence and function, this pattern was not evident for the biomarkers of low-grade inflammation: circulating levels of IL-6 and CRP. There are several potential explanations for this divergence in findings. One possibility is that circulating levels of IL-6 and CRP change more gradually, across a longer developmental timecourse, in response to interpersonal stress than does the regulation of inflammation at the cellular level. Stress-induced changes in circulating IL-6 and CRP may be too small to be detected within 6-month assessment intervals. And given that the sample was young and healthy, it may not be until later in life, when stress-induced changes in the cellular regulation of inflammation have been sustained in the longer-term, that increases in circulating IL-6 and CRP reflecting a low-grade inflammatory state are detectable.

Another important consideration that could also explain the divergent findings is that circulating levels of IL-6 and CRP are non-specific biomarkers that partially reflect processes other than innate immune functions and low-grade inflammation. For example, both IL-6 and CRP levels rise in response to conditions outside of inflammation (e.g., physical exercise, the accumulation of body fat), and IL-6, for example, is released by multiple types of cells and tissues (e.g., immune cells, adipose tissue, skeletal muscle; Del Giudice and Gangestad, 2018, for review).

4.1. Limitations and conclusions

The current study is limited in certain respects. The most significant limitation from a statistical standpoint is that violence was a relatively infrequent experience for individuals in the sample. Of n=157, n=17 were exposed to violence during the study period, meaning that the within-person associations we observed are based on data from 17 individuals. Thus, one concern might be that the estimates we observed are less likely to replicate in other samples. This concern is somewhat alleviated by the fact that our fixed-effects estimates are based on up to six waves of data per individual and, as a result, our study likely captures individuals' average levels of inflammatory activity with high accuracy. Having said this, future studies are needed to confirm the generalizability of our findings.

Other limitations given the low variation in violence exposure are that we were unable to explore possible moderators of violence effects (e.g., individual- and family-level factors that buffer against violence). And we were unable to explore whether different types of exposures (e.g., direct vs. indirect exposure) differed in the strength with which they were associated with inflammatory activity. A final consideration is that the kinds of experiences that individuals were exposed to varied in terms of severity, and given the low variation in violence overall, we considered all exposures to be of equal weight.

A final consideration is that while fixed-effects models do control for unobserved time-invariant variables, they do not control for unobserved time-varying variables such as changes in the family, neighborhood, and peer environment, each of which could plausibly covary with changes in violence exposure, and act as omitted third variables. We addressed this in sensitivity analyses, although, given our inability to fully eliminate

these and other alternative explanations, the associations we observed should not be interpreted as causal. Despite this, our findings add necessary and incremental evidence for the hypothesis that interpersonal violence exposure up-regulates innate immune system activity during adolescence and young adulthood in ways that could increase mental and physical health risk across life. Findings indicated that aspects of the cellular regulation of inflammation may be sensitive to changes in violence exposure, even over a relatively brief time period, which suggests that these features of immunobiology might also be sensitive to interventions aimed at reducing the health burden of violence exposure in young people.

CRediT authorship contribution statement

Rachel Weissman-Tsukamoto: Formal analysis, Investigation, Writing – original draft, Writing – review & editing. **Phoebe Lam:** Data curation, Methodology, Visualization, Writing – original draft, Writing – review & editing. **Eric Finegood:** Conceptualization, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing. **Edith Chen:** Conceptualization, Data curation, Investigation, Methodology, Writing – original draft, Writing – review & editing. **Gregory E. Miller:** Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Resources, Software, Supervision, Visualization, Writing – original draft, Writing – review & editing.

Declaration of Competing Interest

None.

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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.2024.107022.

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