Journal of Child & Adolescent Trauma
https://doi.org/10.1007/s40653-023-00594-2

ORIGINAL ARTICLE

Childhood Adversity and Adult Inflammation: Exploring the Mediating Role of Emotion Regulation in the MIDUS II Study

Christina Burns[1,2] · **Christina Hejl**[1] · **Yvette Z. Szabo**[1,3]

Accepted: 30 November 2023

Abstract

The present study furthers understanding of how childhood adversity connects to inflammation and, in turn, poor health. Using the publicly available Midlife in the United States II (MIDUS II) dataset, we test a recent theoretical model that suggests emotion regulation is a potential mechanism of associations between adversity and inflammation. We examined the indirect effects of various types of adversity (e.g., stressful events, maltreatment, threat, and deprivation) on inflammation via two emotion regulation strategies (i.e., expressive suppression and reappraisal). Participants included 1096 adults without a history of cancer or HIV/AIDS who had completed the initial MIDUS II follow up and a sub-study examining biomarkers. Participants completed self-report measures inquiring about psychosocial factors including stressful life events, childhood trauma, and emotion regulation as well as provided blood samples. Bivariate correlation indicated that multiple forms of childhood adversity were associated with both C-reactive protein and fibrinogen. Deprivation, as measured by a stressful life events scale, was positively associated with both reappraisal and suppression. Tests of indirect effects indicated that deprivation was positively associated with fibrinogen through both emotion regulation strategies, particularly for female participants. Our findings partially support recent theory positing emotion regulation as a pathway through which childhood adversity may impact inflammation in adulthood. Further, deprivation may be particularly critical in understanding how adversity is connected to maladaptive emotion regulation and inflammation. Emotion regulation may be an important treatment target to mitigate the negative impact of childhood adversity on health and well-being.

Keywords Childhood adversity · Reappraisal · Expressive suppression · Emotion regulation · Fibrinogen · C-reactive protein · Inflammation

Childhood adversity refers to events and experiences that negatively impact development and functioning, such as poverty, early life stress, abuse, and neglect (Felitti et al., 1998). Childhood adversity has been linked to poor mental health outcomes (McLaughlin et al., 2010), more physical health problems (Danese & Tan, 2014) as well as interpersonal difficulties (Poole et al., 2018) and increased healthcare utilization (Bellis et al., 2017; Chartier et al., 2010). While childhood adversity covers a wide range of experiences, childhood maltreatment (e.g., abuse and neglect) is a specific form of adversity that has been recognized as a prevalent, significant, and costly public health risk (CDC, 2019; Renna et al., 2021). The CDC has estimated lifetime healthcare cost to be over $200,000 per survivor (CDC, 2019). In order to best allocate prevention and intervention resources, greater understanding of how childhood adversity contributes to these negative health outcomes is needed.

The present study focuses on connections between childhood adversity and inflammation, one proposed pathway through which trauma impacts health (Kendall-Tackett, 2009). Inflammatory markers, including cytokines (e.g., interleukin- 6[IL-6]) and acute phase proteins (e.g., C-reactive protein (CRP) and fibrinogen), are released in response to infection or injury. Critically, these markers of inflammation can also increase in response to stress (Marsland et al., 2017). Excessive activation and increased circulation of these makers can negatively impact overall

✉ Yvette Z. Szabo
yszabo@calstatela.edu

[1] Department of Veterans Affairs, VISN 17 Center of Excellence for Research on Returning War Veterans, Waco, TX, USA

[2] Department of Education, Health, & Behavior, University of North Dakota, Grand Forks, ND, USA

[3] Department of Psychology, California State University, Los Angeles, Los Angeles, CA, USA

Published online: 03 January 2024

health (Coussens & Werb, 2002; Lopez-Candales et al., 2017; Yilmaz et al., 2011). Chronic or dysregulated inflammation has been associated with a range of physical and mental health conditions including cardiovascular disease (Lopez-Candales et al., 2017), cancer (Shacter & Weitzman, 2002), diabetes (Tsalamandris et al., 2019), depression (Beurel et al., 2020), posttraumatic stress disorder, and anxiety (Michopoulos et al., 2017). Many studies have suggested childhood adversity is associated with higher inflammation in adulthood (Baumeister et al., 2016; Chiang et al., 2022; Coelho et al., 2014; Danese & McEwen, 2012; Renna et al., 2021). However, it remains unclear whether different forms of adversity impact inflammation in unique ways, and the mechanisms linking childhood adversity to adult inflammation are still being explored.

Adversity is a broad construct often derived of several subtypes (e.g., childhood maltreatment or early life stress). Much of the previous research examining associations between childhood adversity, development and adult functioning has primarily focused on either a specific type of adversity, like physical abuse, or on a total score capturing multiple forms of adversity (i.e., cumulative risk). However, these approaches are limited because they do not take two things into account: 1) various types of adversity often co-occur, and 2) looking solely at cumulative scores suggest different forms of adversity impact development in similar ways (Lambert et al., 2017; McLaughlin & Sheridan, 2016; Sheridan & McLaughlin, 2014). An emerging approach to address these limitations is to examine childhood adversity as the subdomains of threat and deprivation. Threat can be defined as the experience of actual, threatened, or witnessed physical harm, violence, or death, and is consistent with definitions of trauma and abuse (McLaughlin et al., 2014). In contrast, deprivation is consistent with neglectful experiences, institutional rearing, poverty, and lack of expected social or cognitive stimulation (McLaughlin et al., 2014). Notably, previous studies have demonstrated that threat and deprivation have different effects on biological aging, neural development (Colich et al., 2020; Sheridan et al., 2017; Sumner et al., 2019), emotion regulation, and executive functioning (Lambert et al., 2017; Machlin et al., 2019; Sheridan et al., 2017). The present study builds on previous research by examining multiple measures of childhood adversity.

A secondary aim of this work is to examine whether other factors, that may be more responsive to treatment, may account for associations between childhood adversity and inflammation. Previous research has supported the idea that the associations between childhood maltreatment and inflammation are both direct and indirect (i.e., through other factors; Baumeister et al., 2016; Brown et al., 2021; Miller et al., 2011; Renna et al., 2021; Schrepf et al., 2014). Mathur and colleagues (2022) recently proposed that emotion regulation, which includes the conscious and unconscious processes used to manage emotion (Gross, 1998), is an understudied potential mediator connecting childhood adversity and inflammation. This proposed theory is plausible due to previous findings of associations between emotion dysregulation and elevated inflammation (Appleton et al., 2011, 2013; Miller et al., 2011; Renna, 2021) as well as existing research suggesting childhood maltreatment is connected to emotion dysregulation in adulthood (Bradley et al., 2011; Charak et al., 2019; Janiri et al., 2021; Poole et al., 2018; Tinajero et al., 2020). The present study tests this proposed theory by examining two commonly studied emotion regulation strategies: reappraisal and expressive suppression. Reappraisal is an adaptive strategy which involves reinterpretation of thoughts or events to reduce the influence of negative emotions and has been found to act as buffer to negative mental health outcomes (Gross & John, 2003; McRae et al., 2012; Miu et al., 2022). In contrast, expressive suppression is a maladaptive emotion regulation strategy that involves effortful inhibition of emotional expression (Gross & John, 2003). Findings in samples of midlife adults and trauma-exposed veterans indicate that expressive suppression is associated with elevated inflammation (Ellis et al., 2019; Khan et al., 2020). Additionally, several studies have found connections between childhood adversity and maladaptive emotion regulation, including expressive suppression (Gruhn & Compas, 2020; Heleniak et al., 2016; Weissman et al., 2019), as well as associations with emotion reactivity, a form of early occurring emotion regulation (Gruhn & Compas, 2020; McLaughlin et al., 2010; Sheridan et al., 2020). However, a gap in the literature is the test of whether emotion regulation strategies explain the relationship between childhood adversity and inflammation.

The present study builds on existing research connecting childhood adversity and inflammation by conducting secondary data analysis on a publicly available dataset from a nationally recruited longitudinal study of health and well-being. Our aims were to 1) explore how different domains of childhood adversity (e.g., stressful events, maltreatment, threat, and deprivation) are related to emotion regulation and inflammation and 2) to examine the indirect effects of childhood adversity and inflammation via two emotion regulation strategies (e.g., expressive suppression and reappraisal). This manuscript differs from previous MIDUS II studies examining childhood adversity and inflammation (Hostinar et al., 2015, 2017; Lee et al., 2017; Schrepf et al., 2014; Slopen et al., 2010) in its focus on different domains of childhood adversity, its focus on CRP and fibrinogen independently, as well as the focus on emotion regulation. Additionally, as part of our exploratory analysis, we included two measures of self-control (e.g., cognitive control and burden of consciousness), a construct which is closely related to emotion regulation (Gross, 1998; Paschke et al., 2016). While self-control

is not the focus of this paper, including these less studied measures will allow us to explore potential pathways through which childhood adversity may impact functioning. Based on previous findings, we hypothesized that expressive suppression, but not reappraisal, would be positively associated with both childhood adversity and inflammation. Further, we predicted that expressive suppression would partially account for the relationship between childhood adversity and inflammation.

Methods

Participants

Participants included a subsample from the second phase of a national longitudinal study of health and wellbeing, Midlife in the United States (MIDUS II; Ryff et al., 2021). The first phase of MIDUS recruited English speaking, non-institutionalized adults ages 25–74 between 1995 and 1996 using random digit dialing. The second phase was completed between 2004 and 2006, which included a follow-up on MIDUS participants (Project 1; $N = 3,487$). To increase diversity in MIDUS II, an additional African American sample (N = 592) was recruited from Milwaukee, Wisconsin, who completed questionnaires and procedures parallel to those in Project 1. In the present study, Project 1 and the Milwaukee sub-project will be collectively referred to as baseline. Participants who completed baseline measures were invited to participate in several sub-projects of MIDUS II, including the MIDUS II Biomarker Project (Project 4; $N = 1,255$; Ryff et al., 2019). Informed consent was obtained from participants for each project they participated in. Participants received financial compensation for their participation.

The present study analyzed data from 1096 adults aged 34–84 who participated in both baseline and Project 4. We further excluded individuals with a history of HIV, AIDS, or Cancer due to the influence these conditions have on inflammation ($n = 156$) as well as those who did not complete the childhood trauma questionnaire ($n = 3$). A small portion of participants did not provide a blood sample ($n = 19$), but were included in the analyses not including inflammation.

Procedure

In terms of procedures relevant to the present study, participants completed a telephone interview and were mailed packet of self-administered questionnaires related to health, wellbeing, and functioning at baseline. Project 4 participants were recruited via recruitment letters and study brochures to participate in a two-day overnight study session at one of three General Clinical Research Centers (at University of California Los Angeles, University of Wisconsin, and Georgetown University). On day one, participants completed a physical exam provided their medical history, and completed a questionnaire packet. A 12-h fasting blood draw was taken at approximately 7:00 am the following morning for assay of inflammatory markers.

Measures

Childhood Adversity

The different measures of childhood adversity are summarized in Table 1.

Stressful Events At baseline, participants completed a 27-item Stressful Life Events Scale (SLE), which was adapted from a life stress measure (Turner & Wheaton, 1995) for use in the MIDUS studies. For the first 7 items, participants selected each stressful event they had experienced as a child or teenager up to age 19 (e.g., one or both parents used drugs so often it regularly caused problems). Each item endorsed by the participant was given a score of 1. For the remaining 20 items, participants were instructed to indicate which events they had experienced, at any point in their lifetime (e.g., a parent died), as well as their age at the time of the event. To remain consistent with the 7 childhood/adolescent items, and current definitions of adolescence (Canadian Pediatric Society, 2003; World Health Organization, 2022) events which occurred at age 19 or younger were included in our conceptualization of early stressful life events (i.e., given a score of 1 if it was endorsed and occurred prior to age 20). For the present study, exposure to each of the 7 childhood/adolescent stressful events and endorsement of the remaining items prior to age 20 were summed to represent total *early stressful life events* (eSLE) score. For analysis, the eSLE was recoded to 0, 1, 2 and 3 + to improve distribution as there were low rates of exposure above three events.

Childhood Maltreatment At project 4, participants completed the 28-item Childhood Trauma Questionnaire (CTQ; Bernstein, 1994), a validated measure of childhood trauma which assesses the experience of emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect, as well as a 3-item subscale for minimization/denial which was not included in analysis. Responses are measured on a 5-point Likert scale of 1 = *never true* to 5 = *always true*, with total scores on the CTQ ranging from 25–125. For the present study, a *CTQ total* score was used for primary analysis to represent childhood maltreatment.

Threat and Deprivation Finally, building on theory, we also used three separate measures of deprivation and threat.

Table 1 Descriptions for measures of adversity

Measure of adversity	Description	Potential scores
eSLE	A score of exposure (yes/no) to any of the 27 SLE items occurring in childhood through late adolescence	0, 1, 2, 3 +
CTQ total	A frequency score of childhood maltreatment derived from physical abuse, sexual abuse, emotional abuse, physical neglect and emotional neglect subscales of the CTQ	25–125
CTQ threat	A frequency measure of threat derived from the physical abuse, sexual abuse and emotional abuse subscales of the CTQ	15–75
SLE threat	A score of exposure (yes/no) to 9 items that capture threat (i.e. parental alcohol/substance abuse, assault, combat, disaster) occurring in childhood through late adolescence	0, 1, 2, 3 +
Total threat	A score of exposure (yes/no) to CTQ emotional abuse screen (yes/no using a cut score), physical or sexual threat (i.e. yes/no to CTQ physical/sexual abuse screen OR yes/no to SLE physical/sexual assault), and the remaining 7 SLE threat items occurring in childhood through late adolescence	0, 1, 2, 3 +
CTQ deprivation	A frequency measure of deprivation derived from the emotional neglect and physical neglect subscales of the CTQ	10–50
SLE deprivation	A score of exposure (yes/no) to 8 items that capture deprivation (i.e. dropout/expulsion from school, parental death or divorce, jail/detention, and use of welfare) occurring in childhood through late adolescence	0, 1, 2, 3 +
Total deprivation	A score of exposure (yes/no) to the 8 SLE deprivation items occurring in childhood through late adolescence, CTQ physical neglect screening and CTQ emotional neglect screening	0, 1, 2, 3 +

Note. *eSLE* = early stressful life events, *CTQ* = Childhood Trauma Questionnaire, *SLE* = stressful life events

Three separate measures were incorporated due to variance between frequency and exposure measures in the available MIDUS measures. Our first measure of threat and deprivation was a frequency score created by summing the emotional neglect and physical neglect subscales of the CTQ to represent *CTQ deprivation*, and summing the physical abuse, sexual abuse, and emotional abuse subscales to represent *CTQ threat*.

Next, we created a score of the number of tactics the individual was exposed to using the SLE, which inquiries about whether an item happened or not. Based on previous research conceptualizing threat as experiences of, or exposure to, physical harm or violence (Banihashemi et al., 2021; Busso et al., 2017; Carrera et al., 2020; Heleniak & McLaughlin, 2020; Milojevich et al., 2019; Sheridan & McLaughlin, 2014), our measure of *SLE threat* was a single score composed of yes/no responses to items related to threatening or potentially traumatic experiences. Based on other research conceptualizing deprivation as poverty, lack of expected social or cognitive stimulation, and institutionalization (Banihashemi et al., 2021; Busso et al., 2017; Carrera et al., 2020; Heleniak & McLaughlin, 2020; Milojevich et al., 2019; Sheridan & McLaughlin, 2014), our score of *SLE deprivation* was a single score comprised of yes/no responses to associated items.

Lastly, an alternate measure of threat and deprivation scores were calculated using both the CTQ and SLE. First, participants were assigned a 1 for each subscale of the CTQ that they screened positive for (e.g., physical abuse, sexual abuse, emotional abuse, emotional neglect, and physical neglect) using previously established cut scores (Bernstein & Fink, 1998; Hori et al., 2021). Since there are also items inquiring about physical abuse and sexual abuse on the SLE and because abuse is often underreported, endorsing this on either the SLE or screening positive on the CTQ was considered indicative of physical threat or sexual threat. The CTQ emotional abuse screening, sexual threat, physical threat, and the remaining SLE threat items were summed to create a single score for *total threat*. CTQ screening for emotional and physical neglect and each of the SLE deprivation items were summed to create a single score for *total deprivation*. For analysis, all exposure measures were recoded to 0, 1, 2 and 3 + to improve distribution as there were low rates of exposure greater than 3.

Emotion Regulation

At project 4, participants completed a self-control questionnaire that included a shortened version of the emotion regulation questionnaire (ERQ; Gross & John, 2003; Markus & Kitayama, 1991). Participants rated the degree to which they agreed with 2 items measuring reappraisal ("I control my emotions by changing the way I think about the situation I'm in," and "when I'm faced with a stressful situation, I make myself think about it in a way that helps me stay calm") and 2 items measuring expressive suppression ("When I am feeling negative emotions I make sure not to express them" and "I keep my emotions to myself"). Responses to each item ranged from 1 = *strongly disagree* to 7 = *strongly agree*. For the present study, the two items were summed to create an

expressive suppression measure (α = .71). There was poor response consistency between the two items measuring reappraisal (α = .53). Previous reports in the MIDUS II study examining reappraisal have also reported low correlations between the item responses (Ellis et al., 2019), therefore we used a single-item assessment of *reappraisal* ("when I'm faced with a stressful situation, I make myself think about it in a way that helps me stay calm").

Additionally, the self-control scale included a 6-item subscale for *cognitive control* (e.g., "It is important to me to be able to think, feel, and act differently depending on the needs and demands of the situation"; α = .59), as well as a 7 item subscale measuring *burden of consciousness* (e.g., "It is important to me that I not bother others; α = .64). Analysis with these subscales were considered exploratory.

Inflammation

The present study considered two inflammatory markers that were measured in plasma: CRP and fibrinogen. Both CRP and Fibrinogen were measured using an immunonepholometric assay measured by the BNII nephelometer from Dade Behring (Ryff et al., 2021). Fibrinogen values are reported in mg/dL and assays were reported to have excellent CVs: inter-assay CV: 2.6% and intra-assay CV: 2.7%. CRP is presented in ug/mL and was also reported to have excellent CVs: inter-assay CV: 2.1 – 5.7% and intra-assay CV: 2.3 – 4.4%.

Covariates

Demographics As part of the baseline questionnaires, participants reported on their date of birth, education, sex, race, and ethnicity.

Depression As part of project 4, participants completed the 20-item Center for Epidemiological Studies Depression Inventory (CES-D; Randolff, 1977). For each item (e.g., "I thought my life had been a failure" and "I could not get "going"), participants reported how often they experienced that symptom in the past week on a scale ranging from 1 = *rarely or none of the time* to 4 = *most or all of the time*. A total depression score was calculated in the parent study by summing all items (α = .89).

Medical History As part of project 4, participants completed a standardized medical history interview that included participants health related symptoms and conditions in the past 12 months. Additionally, participants reported all medications they were currently taking, which was used to create a total number of medications score, and body mass index (BMI) was calculated using measurements of height and weight.

Data Analysis

All analyses were conducted using IBM SPSS Statistics 20. Two people were missing CTQ data for the sexual abuse subscale only. We imputed these as 5 (i.e., the lowest possible score) to keep these individuals in the dataset. Descriptive analysis revealed that the CTQ total, eSLE, SLE threat, SLE deprivation, expressive suppression, as well as both CRP and fibrinogen were significantly skewed. Log transformation did not fully normalize these variables, so primary analyses use Spearman correlations with non-transformed variables. For the multivariate analyses, the adversity variables were log transformed to reduce skewness. Expressive suppression was made worse by log transformation, so non-log transformed expressive suppression was used in analysis. To reduce the influence of several biologically plausible but outlying biomarker levels, individuals with biomarker levels more than or less than 3.29 standard deviations from the mean were replaced with a value representing 3.29 standard deviations from the mean (Tabachnick & Fidell, 2007). After, CRP was log transformed. Though fibrinogen was positively skewed, log transformation worsened and negatively skewed this variable, therefore we used non-log transformed fibrinogen in analysis.

Because our interest was in pathways through which adversity may impact inflammation, we considered indirect effects when we observed an association between the predictor (i.e., childhood adversity) and the proposed mediators (i.e., expressive suppression or reappraisal) as well as between the mediator and the outcome (i.e., inflammation; Shrout & Bolger, 2002). The first two steps were conducted using bivariate Spearman correlations. Then, statistical mediation models were conducted using PROCESS for SPSS which uses 5000 bootstrap samples for confidence intervals (Hayes, 2022). The effect is statistically significant if the 95% confidence interval (CI) does not include zero. These models were adjusted for demographics of age (continuous) and sex (0 = male, 1 = female), depression symptoms (continuous) and health variables of number of medications (continuous), chronic medical conditions (continuous) and BMI (continuous). These were selected based on recommendations for research with inflammation in health research (O'Connor et al., 2009; Szabo & Slavish, 2021). Due to small amounts of missing data for the covariates, n is smaller for adjusted models and n is clearly noted for all models.

Table 2 Demographics and sample characteristics

Characteristic	Mdn(IQR)	M(SD)	% (*n*)
Gender			
Male			43.9% (*n* = 481)
Female			56.1% (*n* = 615)
Age		53.67(11.37)	
Race			
White			77.8% (*n* = 850)
Black/African American			17.8% (*n* = 195)
American Indian/Alaskan Native			1.6% (*n* = 17)
Asian/ Asian American			.3% (*n* = 3)
Other			2.5% (*n* = 27)
Missing			.4% (*n* = 4)
Education			
No school/ some grade school			.2% (*n* = 2)
Eighth grade/ junior high school			.9% (*n* = 10)
Some high school/ no diploma			4.5% (*n* = 49)
GED			1.3% (*n* = 14)
Graduated from high school			20.8% (*n* = 228)
1–2 years of college no degree			17.6% (*n* = 193)
3 or more years of college no degree			4.5% (*n* = 49)
Graduated from 2 year college or associates degree			7.7 (*n* = 84)
Graduated with bachelor's degree			20.8% (n = 228)
Some graduate school			4.0% (n = 44)
Master's degree			13.2% (n = 145)
Doctorate or other professional degree			4.3% (n = 47
Missing			.3% (n = 3)
CTQ total	33(15)		
eSLE	1(2)		
Total deprivation	1(2)		
Total threat	1(2)		
Suppression	10(3)		
Single item reappraisal	5(1)		

The names for level of education options were slightly modified from the MIDUS II questionnaire

Note. eSLE = early stressful life events, *CTQ* = Childhood Trauma Questionnaire, *Mdn* = median, *IQR* = interquartile range, *M* = mean, *SD* = Standard deviation

Results

Sample demographics and characteristics are presented in Table 2. Associations between various measures of childhood adversity, emotion regulation, self-control, and inflammation are illustrated in Table 3.

Associations Between Childhood Adversity and Inflammation When examining associations between inflammation and childhood adversity, CTQ total, CTQ threat, CTQ deprivation, as well as total threat and total deprivation, were positively associated with both fibrinogen and CRP. The measures of eSLE and SLE deprivation, were positively associated with CRP only. There were no significant associations between SLE threat and any measure of inflammation.[1]

Associations Between Childhood Adversity and Emotion Regulation In terms of associations between childhood adversity and emotion regulation, SLE deprivation was positively

[1] The present study examines the indices of threat and deprivation due to new theoretical importance. Given historical norm to use subscales, we present exploratory correlations with CTQ subscales in Supplemental Table 1 in supplementary materials. We do not report on associations between subscales of the CTQ and CRP as Schrepf and colleagues (2014) presented those findings on a sub sample of MIDUS II participants.

Table 3 Correlations between emotion regulation, childhood adversity and inflammation

Variable	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Reappraisal	.797**	.032	.454**	-.054	-.005	-.013	.064*	-.035	-.059	.006	-.006	.106**	.032
2. Suppression		.096**	.561**	-.050	.006	-.004	.071*	-.013	-.084**	.017	-.012	.078*	.037
3. Burden of consciousness			.181**	.058	.009	.046	-.011	.055	.046	.068*	.035	-.023	-.022
4. Cognitive control				-.125**	-.048	-.053	.023	-.074*	-.146**	-.043	-.074*	.041	-.030
5. CTQ total					.359**	.330**	.237**	.861**	.906**	.737**	.692**	.082**	.089**
6. eSLE						.629**	.675**	.293**	.336**	.511**	.593**	.028	.073*
7. SLE threat							.130**	.309**	.272**	.642**	.315**	.014	.033
8. SLE deprivation								.176**	.251**	.271**	.712**	.048	.093**
9. CTQ threat									.604**	.794**	.469**	.073*	.086**
10. CTQ deprivation										.552**	.762**	.064*	.063*
11. Total threat											.511**	.065*	.080**
12. Total deprivation												.068*	.088**
13. Fibrinogen													.535**
14. CRP													

Note. CTQ = Childhood Trauma Questionnaire, *eSLE* = early stressful life events, *SLE* = stressful life events

*$p < .05$; **$p < .01$

associated with expressive suppression ($r = .071$, $p < .05$) and reappraisal ($r = .064$, $p < .05$). In contrast, CTQ deprivation was negatively associated with expressive suppression ($r = -.082$, $p < .01$) but not reappraisal. For our exploratory self-control measures, Total threat was positively associated with burden of consciousness ($r = .068$, $p < .05$). Additionally, CTQ total ($r = -.125$, $p < .01$), CTQ deprivation ($r = -.146$, $p < .01$), CTQ threat ($r = -.074$, $p < .05$), and total deprivation ($r = -.074$, $p < .05$) were negatively associated with cognitive control. There were no significant associations between total eSLE, CTQ total, total deprivation, or any measure of threat with either measure of emotion regulation.

Associations Between Emotion Regulation and Inflammation Reappraisal ($r = .106$, $p < .01$) and expressive suppression ($r = .078$, $p < .05$) were positively associated with fibrinogen, but not CRP. There were no associations between cognitive control or burden of consciousness and either measure of inflammation.

Indirect Effects of Adversity on Inflammation Through Emotion Regulation To test whether emotion regulation accounts for associations between childhood adversity and inflammation, a final set of linear regression models were conducted. Based on the preliminary correlations, the only pathways examined were 1) SLE deprivation → reappraisal → fibrinogen, 2) SLE deprivation → expressive suppression → fibrinogen and 3) CTQ deprivation → expressive suppression → fibrinogen (all shown in Table 4).

When both SLE deprivation and reappraisal were included in the model with covariates, reappraisal ($b = 6.01$, $p = .002$), but not SLE deprivation ($b = 13.03$, $p = .33$) was a significant predictor of fibrinogen. There was a significant indirect effect of SLE deprivation on fibrinogen through reappraisal, $b = 4.01$, CI [.94, 8.24], $p < .05$. This suggests that greater exposure to deprivation as measured by the SLE was associated with greater reappraisal, which in turn was associated with higher fibrinogen. As shown in the final model, greater BMI, older age and more frequent depression symptoms were all associated with greater fibrinogen, as was female sex.

When both SLE deprivation and expressive suppression were included in the model with covariates, expressive suppression ($b = 2.39$, $p = .03$), but not SLE deprivation ($b = 14.65$, $p = .28$) was a significant predictor of fibrinogen. There was a significant indirect effect of SLE deprivation on fibrinogen through expressive suppression $b = 2.38$, [.05, 6.00], $p < .05$. This suggests greater exposure to deprivation as measured by the SLE was associated with greater expressive suppression, which in turn was associated with higher fibrinogen. As shown in the final model, greater BMI, age and depression symptoms were all associated with greater fibrinogen, as was female sex.

Table 4 Linear Regression Models Examining Deprivation as a Predictor of Fibrinogen

Final model of SLE deprivation and reappraisal predicting fibrinogen $R^2 = .15$, $F(8, 1063) = 23.93$, $p < .001$			
Predictor	B	SE	p
Constant	91.53	20.99	<.001
SLE deprivation	13.03	13.41	.33
Reappraisal	6.01	1.92	.002
CES-D	.87	.33	.009
Age	1.06	.23	<.001
Sex	28.95	4.98	<.001
Total number of medications	-.83	.61	.17
BMI	4.0	.37	<.001
Number of health conditions	.11	1.25	.93
N = 1072. Indirect effect of SLE deprivation on fibrinogen through suppression, b = 4.01, SE = 1.88, 95% confidence interval [.94, 8.24], $p < .05$			
Final model of SLE deprivation and suppression predicting fibrinogen $R^2 = .15$, $F(8, 1063) = 23.12$, $p < .001$			
Constant	97.03	21.88	<.001
SLE deprivation	14.65	13.43	.28
Suppression	2.39	1.13	.03
CES-D	.79	.33	.02
Age	1.06	.23	<.001
Sex	29.08	5.00	<.001
Total number of medications	-.82	.61	.18
BMI	3.98	.37	<.001
Number of health conditions	.09	1.26	.94
N = 1072. Indirect effect of SLE deprivation on fibrinogen through suppression, b = 2.38, SE = 1.53, 95% confidence interval [.05, 6.00], $p < .05$			
Final model of CTQ deprivation and suppression predicting fibrinogen $R^2 = .10$, $F(8, 1063) = 23.35$, $p < .001$.			
Constant	66.16	29.14	.02
CTQ deprivation	27.63	16.54	.10
Suppression	2.57	1.12	.02
CES-D	0.66	.34	.05
Age	1.04	.23	<.001
Sex	28.99	4.99	<.001
Total number of medications	-.87	.61	.16
BMI	4.00	.37	<.001
Number of health conditions	.06	1.27	.96
N = 1072. Indirect effect of CTQ deprivation on fibrinogen through suppression, b = -1.50, SE = 1.42, 95% confidence interval [-4.72, 1.02], $p < .05$			

Note. *CTQ* = Childhood Trauma Questionnaire, *SLE* = stressful life events, *CES-D* = Center for Epidemiological Studies Depression Scale, *BMI* = body mass index

When both CTQ deprivation and expressive suppression were included in the model with covariates, expressive suppression ($b = 2.57$, $p = .02$), but not CTQ deprivation ($b = 27.63$, $p = .10$), was a significant predictor of fibrinogen. However, there was no evidence of a significant indirect effect. As shown in the final model, greater BMI, depression, and age were all associated with greater fibrinogen, as was female sex.

Sex Differences Because sex was a significant predictor in all mediation models and there is existing research documenting gender and sex differences in emotion and inflammation (Cartier et al., 2009; Gardener et al., 2013; Kwon et al., 2013; Lasselin et al., 2018; McRae et al., 2008; Nolen-Hoeksema, 2012;), we reran the two models with significant indirect effects by sex (see Supplemental Tables 2 and 3). The association with the SLE deprivation

and expressive suppression was observed in females only. In the final model, expressive suppression but not SLE deprivation predicted fibrinogen. However, there was no indirect effect of SLE deprivation through suppression on fibrinogen. The association with SLE deprivation and reappraisal was also observed for female participants. Reappraisal was associated with fibrinogen in both models. In addition, there was evidence of an indirect effect of deprivation on inflammation through reappraisal ($b = 7.13$, [1.05, 14.67], $p < .05$) in female participants only.

Discussion

This study aimed to better understand the relationship between childhood adversity and adult inflammation. Our use of the MIDUS II dataset allowed us to examine multiple forms of childhood adversity (e.g., early stressful life events, childhood maltreatment, threat, and deprivation) and their associations with emotion regulation and inflammation. Extending previous research, we observed associations between various forms of childhood adversity and greater inflammation. Specific forms of childhood adversity were differentially associated with emotion regulation, and we observed some early evidence of emotion regulation as one pathway through which childhood adversity relates to inflammation. The present study extends our understanding of potential contributors to poor health outcomes among those exposed to childhood adversity.

Our findings are in line with previous literature connecting childhood adversity to elevated inflammation in adulthood, research conducted using the MIDUS II dataset and independent samples (Baumeister et al., 2016; Chiang et al., 2022; Hostinar et al., 2015, 2017; Lee et al., 2017; Renna et al., 2021; Schrepf et al., 2014; Slopen et al., 2010). Of note, previous studies using MIDUS II data have documented associations between the CTQ total and subscales as well as an 8 item adverse childhood experiences measure derived from the CTQ and additional MIDUS II items, with composite inflammatory scores and CRP (Hostinar et al., 2015; Lee et al., 2017; Schrepf et al., 2014). In addition, one study from the MIDUS II study found an association between early life adversity (measured using childhood specific SLE items, and additional self-report items related to relationship/conflict with parents) with both fibrinogen and CRP (Slopen et al., 2010). Our findings extend this research by utilizing the CTQ and SLE measures to assess associations between domains of adversity (e.g., threat and deprivation) and specific markers of inflammation (e.g., fibrinogen and CRP). Specifically, we found that our measures of threat and deprivation as measured by the CTQ, as well as our total scores of threat and deprivation, were positively associated with both markers of inflammation. Two additional measures of adversity, derived from the SLE, were associated with CRP only. One possible explanation is that CRP is sensitive to early exposure to stress whereas greater frequency of exposure, as measured by the CTQ, may contribute to a global inflammatory state. However, the variation in statistical significance among the specific measures of adversity may be due to the types of measures used. Notably, all associations were in the expected direction and generally suggest associations exist between childhood adversity and elevated inflammation as measured by acute phase proteins, which could have important implications for health. Chronic inflammation has been associated with a range of physical health problems (Lopez-Candales et al., 2017; Rodríguez-Hernández et al., 2013; Yilmaz et al., 2011). Of note, associations observed were small in magnitude. This underscores the need to examine potential mediating factors more closely to determine whether they have meaningful associations with health.

The present study extends previous research by testing a theoretical model suggesting that childhood adversity impacts inflammation in part through emotion regulation (Mathur et al., 2022). In support of this, we observed associations with childhood adversity and a range of emotion regulation strategies. Partially supporting our hypotheses, we observed a positive relationship between SLE deprivation and expressive suppression. However, CTQ deprivation was negatively associated with expressive suppression (i.e., in the opposite direction than hypothesized). Together, these findings suggest that specific aspects of deprivation may uniquely impact emotion regulation. The items that comprise the CTQ deprivation measure captured more neglectful experiences (Lambert et al., 2017), whereas the SLE deprivation measure also captures aspects of social and cognitive deprivation. This could imply social or cognitive stimulation play an important role in emotion regulation and that being deprived of socially or cognitively enriching experiences may lead to greater use of maladaptive strategies. Furthermore, this pattern of findings differs from existing conceptual models and previous findings linking threat, but not deprivation, to difficulties in emotion regulation (Lambert et al., 2017; McLaughlin et al., 2015; Milojevich et al., 2019; Sheridan et al., 2020). While the use of different measures may partially explain these differences, more research with individuals who have only experienced threat or only experienced deprivation is needed to determine how they uniquely influence emotion regulation. Notably, there are many factors which may contribute to experiences of cognitive or social deprivation in childhood such as financial hardship, institutional rearing, neglectful caregiving environments (Mclaughlin et al., 2014), as well as societal and geographic factors such as social marginalization or living in a rural area (Kurani et al., 2022; Pearce et al., 2010). Recognizing these factors could be critical for

those working closely with youth to improve early intervention promoting adaptive emotion regulation.

Interestingly, reappraisal was also positively associated with SLE deprivation, which was inconsistent with our hypotheses and previous research (Khan et al., 2021; McRae et al., 2012; Miu et al., 2022). This could be explained by our use of a single item measure of reappraisal. While single item measures of psychosocial constructs can be useful and valid (Matthews et al., 2022), it is possible that this item alone did not capture reappraisal well. The item we selected specifically asked about whether individuals reappraise stressful situations to keep calm and was part of a broader emotional control subscale, which measured both expressive suppression and reappraisal. It is possible that this item better reflects emotional control. Recent evidence has demonstrated individuals make greater attempts to control their emotions or reappraise stressful situations if they have been exposed to moderate levels of stress in childhood (Schweizer et al., 2016; Shapero et al., 2015; Zeier et al., 2021). Another explanation is that individuals with high levels of childhood adversity may have maintained their resilience by using reappraisal. In support of this possible interpretation, the MIDUS II sample is largely healthy, evidenced by low rates of chronic health conditions and low rates of depression, therefore it is plausible. Future studies may consider exploring emotional control as a resiliency factor within the MIDUS II sample. However, more research is needed to support these post hoc interpretations of an unexpected finding.

The present study is, to our knowledge, the first to explore associations between childhood adversity, cognitive control and burden of consciousness, two forms of self-control. Previous studies have documented difficulties in task-based measures of cognitive control in samples of maltreated children and adolescence (e.g., Kim-Spoon et al., 2021; McLaughlin et al., 2015; Thompson et al., 2014). Our findings extend this research by demonstrating that adults with a history of childhood adversity, as measured by the CTQ, reported lower engagement in cognitive control as measured by a self-reported behavioral measure of self-control. Additionally, our findings indicated that there was a positive relationship between total threat and burden of consciousness, which captures the extent to which individuals attempt not to burden others. Prior research has suggested that those exposed to threat in childhood are more sensitive to threat cues (McLaughlin et al., 2015), which has been associated with emotion dysregulation (Thompson et al., 2014). This may indicate that those who experienced greater levels of threat may make greater attempts to control how others perceive them to reduce potential threats. Future researchers may consider testing the predictive validity of both measures of self-control for health and well-being among those exposed to childhood adversity. However, there are cultural differences to consider as those with collectivist cultural backgrounds may report high rates of burden of consciousness without any negative impact on wellbeing (Markus & Kitayama, 1991; Srirangarajan et al., 2020).

When examining the relationship between emotion regulation and inflammation, we found that the use both of expressive suppression and reappraisal were associated with higher levels of fibrinogen. The positive relationship between expressive suppression and fibrinogen supports our hypothesis and extends recent findings connecting elevated inflammation and maladaptive emotion regulation strategies (Appleton et al., 2013; Ellis et al., 2019; Khan et al., 2020; Lopez et al., 2020; Mathur et al., 2022). Conversely, the positive relationship between reappraisal and fibrinogen was surprising as previous studies have indicated this adaptive emotion regulation strategy may serve as buffer against heightened inflammatory activity (Appleton et al., 2013; Ellis et al., 2019; Mathur et al., 2022). It is worth noting that studies reporting on associations between reappraisal and inflammation have primarily focused on CRP or cytokines such as IL-6, and findings have not consistently linked reappraisal to lower inflammation (Khan et al., 2020; Lopez et al., 2020). As such, our findings could indicate that fibrinogen, which has been understudied in research connecting emotion regulation and inflammation, is particularly sensitive to psychological efforts to control emotion. More research using a variety of inflammatory markers, as well as measures that examine expressive suppression and reappraisal as separate constructs, would allow us to better understand how emotion regulation influences inflammatory processes. Furthermore, with few exceptions (i.e., one study examining veterans with PTSD; Khan et al., 2020), studies examining emotion regulation and inflammation have been limited to community samples. Examining association between maladaptive emotion regulation strategies in clinical samples could be an important next step in understanding how trauma and childhood adversity influence factors related to health.

A sizable contribution of this paper is the testing of indirect associations between childhood adversity and inflammation through emotion regulation, above the contributions of several covariates. Greater deprivation as measured by the SLE, but not the CTQ, was associated with greater use of expressive suppression, which in turn was associated with higher levels of fibrinogen. Cognitive or social deprivation, which was included in the SLE measure of deprivation but not the CTQ, may have influence over emotion regulation, which in turn elevates risk for dysregulation in inflammatory processes. Surprisingly, there was also a significant indirect effect of SLE deprivation on fibrinogen through reappraisal, such that greater deprivation was associated with greater

engagement with reappraisal, which in turn was associated with higher fibrinogen. This may suggest that deprivation is uniquely related to efforts to control emotion, which in turn is related to fibrinogen. Exploratory models stratified by sex suggested that this remained significant in female participants but not male participants. This is somewhat surprising given that previous research suggests men are more likely to engage in expressive suppression (Fladung & Kiefer, 2016; Gross & John, 2003; Kring et al., 1994; Kwon et al., 2013) and that woman, compared to men, have greater emotional reactivity (Domes et al., 2010; Gardener et al., 2013). MIDUS II asks participants to self-report their sex, which conflates this variable with gender. Further research focusing on hormonal sex differences and the impact of gender on these variables is needed. Notably our findings also considered the contributions of a range of covariates, including symptoms of depression. Taken together, our findings provide evidence that emotion regulation is one potential pathway through which certain forms of childhood adversity are connected to elevated inflammation, partially supporting the hypotheses laid out by Mathur and colleagues (2022). Extending this line of research could inform prevention and intervention efforts to lower the risk of negative health outcomes associated with inflammation and childhood adversity. As an example, previous studies have noted improved mental health among youth following interventions specifically targeting emotion regulation such as dialectical behavioral therapy skills (DBT; Asarnow et al., 2021; Zapolski & Smith, 2017). Similarly, treatments focusing on emotion regulation have been developed for those with childhood abuse related psychopathology such as the skills training in affect and interpersonal regulation (STAIR; Cloitre et al., 2010). Additionally, research has supported the use of school-based mindfulness and social emotional learning programs to improve both mental health and academic outcomes (Pedrini et al., 2022; Phillips & Mychailyszyn, 2022), which might be particularly important for those who have experienced adversity in the form of deprivation. Our findings may also have important implications for screening for deprivation in childhood for both practitioners and research, such as the potential use of measures that capture cognitive and social deprivation, for example, the Multidimensional Neglectful Behavior Scale (MNBS; Kaufman Kantor et al., 2004). Further research will inform implications for practice and screening.

Limitations and Future Directions

The present study is a useful first step to clarify associations between different measures of childhood adversity with emotion regulation and their association with inflammation. However, there are a number of limitations that should be considered when interpreting these results and offer opportunity for future research to build on these findings. Notably, the sample utilized in the present study was primarily healthy and white/Caucasian, which potentially limits generalizability to clinical and minoritized samples. Additionally, this sample was not recruited for the experience of childhood adversity and other reports from this sample suggest low rates of adversity as measured by the CTQ (Lee et al., 2017). This highlights the need for research with more variability in exposure to adversity to better understand how frequency of exposure influences inflammation and overall health.

In regard to measurement, the self-report retrospective measures of childhood trauma and stressful life events may have been subject to response bias due to memory problems or other factors. It is also important to note that our measures of adversity were taken at different timepoints (i.e., SLE at baseline and CTQ at project 4). Because baseline included many more self-report measures, it is also possible that participants underreported exposure to stressful events in early life due to response fatigue. Additionally, the CTQ and SLE were not designed to capture specific aspects of deprivation, particularly poverty and institutional rearing, or forms of threat such as exposure to community or domestic violence. Furthermore, researchers have critiqued the use of the CTQ neglect subscales when measuring deprivation noting that the emotional neglect items measure family cohesion which could be influenced by many factors other than neglect, and that the physical neglect items have poor reliability (Lambert et al., 2017). It is important for future researchers to consider measures that adequately capture unique aspects of threat and deprivation such as the Screen for Adolescent Exposure to Violence (SAVE; Hastings & Kelley, 1997), HOME environment questionnaire (Frankenburg & Coons, 1986), or a retrospective enriching early-life activities inventory (Chan et al., 2018). Lastly, we utilized a non-validated short form of the ERQ to conceptualize suppression and reappraisal. Using the full ERQ, or other validated measures of emotion regulation, such as the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2003), or more objective measures of emotion regulation such as functional neuroimaging tasks may improve our understanding of how emotion regulation impacts inflammation.

This investigation offers partial support of a model proposed by Mathur and colleges (2022). However, emotion regulation is not the only pathway through which childhood adversity may impact inflammation. For example, studies using data from MIDUS II have provided evidence for the mediating role of emotional eating, BMI, anxiety, (Schrepf et al., 2014), recent life stress (Hostinar et al., 2015), and frontal brain asymmetry (Hostinar et al., 2017) on the relationship between childhood trauma and inflammation. Additionally, among community adults sleep deprivation strengthened the relationship between childhood adversity

and inflammation, which in turn was associated with hypertension (Petrov et al., 2016). In addition to examining other mediators simultaneously, there is a need to understand for whom these associations may be strongest. The present study suggests that the experience of deprivation, particularly in the domains of cognitive and social stimulation, is critical. Furthermore, sex stratified analyses suggested that associations between deprivation and suppression might be observed only in female adults and encourage further inquiry in this area.

Conclusion

In sum, the present study tests a recent proposal that childhood adversity impacts inflammation in adulthood through emotion regulation. We observed some support for both associations between childhood adversity and emotion regulation, as well as expressive suppression specifically, and inflammation. In addition, we observed associations for both childhood adversity and emotion regulation with inflammation alone. This encourages further study of emotion regulation, and other factors, as mediators of associations of adversity on health. Critically, emotion regulation is amendable to intervention and often improves following psychosocial intervention (Gratz & Tull, 2010; Sloan et al., 2017; Spidel et al., 2018). This offers promise as one pathway to mitigate the harm of childhood adversity on health and well-being.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s40653-023-00594-2.

Acknowledgements Data used for this research was provided by the longitudinal study titled "Midlife in the United States," (MIDUS) which was supported by a grant from the National Institute on Aging (P01-AG020166) and managed by the Institute on Aging, University of Wisconsin.

Authors' Contribution Y. Szabo wrote the local IRB protocol. C. Burns and Y. Szabo processed and prepared the data for analysis, analyzed the data, and interpreted the results. C. Burns wrote the first draft of the manuscript. Y. Szabo and C. Hejl critically reviewed and contributed to subsequent drafts of the manuscript. All authors read and approved the final version of the manuscript.

Funding This present study was supported by resources from the Central Texas Veterans Healthcare System and the VISN 17 Center of Excellence for Research on Returning War Veterans. Dr. Szabo's work on this manuscript was partially supported by Career Development Award Number IK1RX003122 from the United States (U.S.) Department of Veterans Affairs Rehabilitation Research and Development (RR&D) Service. The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs or the U.S. government.

Data Availability MIDUS data for are made publicly available through the Inter-university Consortium for Political and Social Research (ICPSR) website. https://www.icpsr.umich.edu/web/ICPSR/series/203.

Declarations

Ethics Approval All procedures performed in the parent studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. MIDUS data collection was reviewed and approved by the Education and Social/Behavioral Sciences Institutional Review Board at the University of Wisconsin. MIDUS Biomarker data collection protocols were reviewed and approved by the IRBs at each of the General Clinical Research Centers (at University of California Los Angeles, University of Wisconsin, and Georgetown University). The analyses described here were also approved by the Central Texas Veterans Health Care System Research Committees (protocol number 2021–006).

Informed Consent The parent studies obtained written informed consent for all participants.

Competing Interests The authors have no relevant financial or non-financial interests to disclose.

References

Appleton, A. A., Buka, S. L., Loucks, E. B., Gilman, S. E., & Kubzansky, L. D. (2013). Divergent associations of adaptive and maladaptive emotion regulation strategies with inflammation. *Health Psychology, 32*(7), 748–756. https://doi.org/10.1037/a0030068

Appleton, A. A., Buka, S. L., McCormick, M. C., Koenen, K. C., Loucks, E. B., Gilman, S. E., & Kubzansky, L. D. (2011). Emotional functioning at age 7 years is associated with C-reactive protein in middle adulthood. *Psychosomatic Medicine, 73*(4), 295–303. https://doi.org/10.1097/PSY.0b013e31821534f6

Asarnow, J. R., Berk, M. S., Bedics, J., Adrian, M., Gallop, R., Cohen, J., Korslund, K., Hughes, J., Avina, C., Linehan, M. M., & McCauley, E. (2021). Dialectical Behavior Therapy for Suicidal Self-Harming Youth: Emotion Regulation, Mechanisms, and Mediators. *Journal of the American Academy of Child & Adolescent Psychiatry, 60*(9), 1105-1115.e4. https://doi.org/10.1016/j.jaac.2021.01.016

Banihashemi, L., Peng, C. W., Verstynen, T., Wallace, M. L., Lamont, D. N., Alkhars, H. M., Yeh, F., Beeney, J. E., Aizenstein, H. J., & Germain, A. (2021). Opposing relationships of childhood threat and deprivation with stria terminalis white matter. *Human Brain Mapping, 42*(8), 2445–2460. https://doi.org/10.1002/hbm.25378

Baumeister, D., Akhtar, R., Ciufolini, S., Pariante, C. M., & Mondelli, V. (2016). Childhood trauma and adulthood inflammation: A meta-analysis of peripheral C-reactive protein, interleukin-6 and tumour necrosis factor-α. *Molecular Psychiatry, 21*(5), 642–649. https://doi.org/10.1038/mp.2015.67

Bellis, M., Hughes, K., Hardcastle, K., Ashton, K., Ford, K., Quigg, Z., & Davies, A. (2017). The impact of adverse childhood experiences on health service use across the life course using a retrospective cohort study. *Journal of Health Services Research & Policy, 22*(3), 168–177. https://doi.org/10.1177/1355819617706720

Bernstein, D. P. (1994). Measure of child abuse and neglect. *American Journal of Psychiatry, 151*(8), 1132–1136.

Bernstein, D. P., & Fink, L. (1998). *Childhood Trauma Questionnaire: A retrospective self-report manual* San Antonio, TX: The Psychological Corporation.

Beurel, E., Toups, M., & Nemeroff, C. B. (2020). The bidirectional relationship of depression and inflammation: Double trouble.

Neuron, 107(2), 234–256. https://doi.org/10.1016/j.neuron.2020.06.002

Bradley, B., Westen, D., Mercer, K. B., Binder, E. B., Jovanovic, T., Crain, D., Wingo, A., & Heim, C. (2011). Association between childhood maltreatment and adult emotional dysregulation in a low-income, urban, African American sample: Moderation by oxytocin receptor gene. *Development and Psychopathology, 23*(2), 439–452. https://doi.org/10.1017/S0954579411000162

Brown, M., Worrell, C., & Pariante, C. M. (2021). Inflammation and early life stress: An updated review of childhood trauma and inflammatory markers in adulthood. *Pharmacology Biochemistry and Behavior, 211*, 173291. https://doi.org/10.1016/j.pbb.2021.173291

Busso, D. S., McLaughlin, K. A., & Sheridan, M. A. (2017). Dimensions of adversity, physiological reactivity, and externalizing psychopathology in adolescence: Deprivation and threat. *Psychosomatic Medicine, 79*(2), 162–171. https://doi.org/10.1097/PSY.0000000000000369

Canadian Pediatric Society. (2003). Age limits and adolescents. *Pediatrics & Child Health, 8*(9), 577–578. https://doi.org/10.1093/pch/8.9.577

Carrera, P., Jiménez-Morago, J. M., Román, M., & León, E. (2020). Differential associations of threat and deprivation with emotion understanding in maltreated children in foster care. *Child & Family Social Work, 25*(4), 973–982. https://doi.org/10.1111/cfs.12783

Cartier, A., Côté, M., Lemieux, I., Pérusse, L., Tremblay, A., Bouchard, C., & Després, J. (2009). Sex differences in inflammatory markers: What is the contribution of visceral adiposity? *The American Journal of Clinical Nutrition, 89*(5), 1307–1314. https://doi.org/10.3945/ajcn.2008.27030

CDC. (2019). *Preventing child abuse & neglect*. https://www.cdc.gov/violenceprevention/pdf/CAN-factsheet.pdf

Chan, T., Parisi, J. M., Moored, K. D., & Carlson, M. C. (2018). Variety of enriching early-life activities linked to late-life cognitive functioning in urban community-dwelling African Americans. *The Journals of Gerontology: Series B, 7*(8), 1345–1355. https://doi.org/10.1093/geronb/gby056

Charak, R., Villarreal, L., Schmitz, R. M., Hirai, M., & Ford, J. D. (2019). Patterns of childhood maltreatment and intimate partner violence, emotion dysregulation, and mental health symptoms among lesbian, gay, and bisexual emerging adults: A three-step latent class approach. *Child Abuse & Neglect, 89*, 99–110. https://doi.org/10.1016/j.chiabu.2019.01.007

Chartier, M. J., Walker, J. R., & Naimark, B. (2010). Separate and cumulative effects of adverse childhood experiences in predicting adult health and health care utilization. *Child Abuse & Neglect, 34*(6), 454–464. https://doi.org/10.1016/j.chiabu.2009.09.020

Chiang, J. J., Lam, P. H., Chen, E., & Miller, G. E. (2022). Psychological stress during childhood and adolescence and its association with inflammation across the lifespan: A critical review and meta-analysis. *Psychological Bulletin, 148*(1–2), 27–66. https://doi.org/10.1037/bul0000351

Coelho, R., Viola, T. W., Walss-Bass, C., Brietzke, E., & Grassi-Oliveira, R. (2014). Childhood maltreatment and inflammatory markers: A systematic review. *Acta Psychiatrica Scandinavica, 129*(3), 180–192. https://doi.org/10.1111/acps.12217

Cloitre, M., Stovall-McClough, K. C., Nooner, K., Zorbas, P., Cherry, S., Jackson, C. L., ... & Petkova, E. (2010). Treatment for PTSD related to childhood abuse: A randomized controlled trial. *American Journal of Psychiatry, 167*(8), 915–924.

Colich, N. L., Rosen, M. L., Williams, E. S., & McLaughlin, K. A. (2020). Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. *Psychological Bulletin, 146*(9), 721–764. https://doi.org/10.1037/bul0000270

Coussens, L. M., & Werb, Z. (2002). Inflammation and cancer. *Nature, 420*, 860–867. https://doi.org/10.1038/nature01322

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior, 106*(1), 29–39. https://doi.org/10.1016/j.physbeh.2011.08.019

Danese, A., & Tan, M. (2014). Childhood maltreatment and obesity: Systematic review and meta-analysis. *Molecular Psychiatry, 19*(5), 544–554. https://doi.org/10.1038/mp.2013.54

Domes, G., Schulze, L., Böttger, M., Grossmann, A., Hauenstein, K., Wirtz, P. H., Heinrichs, M., & Herpertz, S. C. (2010). The neural correlates of sex differences in emotional reactivity and emotion regulation. *Human Brain Mapping, 31*(5), 758–769. https://doi.org/10.1002/hbm.20903

Ellis, E. M., Prather, A. A., Grenen, E. G., & Ferrer, R. A. (2019). Direct and indirect associations of cognitive reappraisal and suppression with disease biomarkers. *Psychology & Health, 34*(3), 336–354. https://doi.org/10.1080/08870446.2018.1529313

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine, 14*(4), 245–258. https://doi.org/10.1016/S0749-3797(98)00017-8

Fladung, A.-K., & Kiefer, M. (2016). Keep calm! Gender differences in mental rotation performance are modulated by habitual expressive suppression. *Psychological Research Psychologische Forschung, 80*(6), 985–996. https://doi.org/10.1007/s00426-015-0704-7

Frankenburg, W. K., & Coons, C. E. (1986). Home screening questionnaire: Its validity in assessing home environment. *The Journal of Pediatrics, 108*(4), 624–626. https://doi.org/10.1016/S0022-3476(86)80853-8

Gardener, E. K. T., Carr, A. R., MacGregor, A., & Felmingham, K. L. (2013). Sex differences and emotion regulation: An event-related potential study. *PLoS ONE, 8*(10), e73475. https://doi.org/10.1371/journal.pone.0073475

Gratz, K. L., & Roemer, L. (2003). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment, 26*(1), 41–54.

Gratz, K. L., & Tull, M. T. (2010). Emotion regulation as a mechanism of change in acceptance- and mindfulness-based treatments. In *Assessing mindfulness and acceptance processes in clients: Illuminating the theory and practice of change.* (pp. 107–133). Context Press/New Harbinger Publications.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*(3), 29.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362. https://doi.org/10.1037/0022-3514.85.2.348

Gruhn, M. A., & Compas, B. E. (2020). Effects of maltreatment on coping and emotion regulation in childhood and adolescence: A meta-analytic review. *Child Abuse & Neglect, 103*, 104446. https://doi.org/10.1016/j.chiabu.2020.104446

Hastings, T. L., & Kelley, M. L. (1997). Development and validation of the screen for adolescent violence exposure (SAVE). *Journal of Abnormal Child Psychology, 25*(6), 511–530. https://doi.org/10.1023/A:1022641916705

Hayes, A. F. (2022). *Introduction to mediation, moderation, and conditional process analysis* (3rd ed.). Guildford Press.

Heleniak, C., Jenness, J. L., Vander Stoep, A., McCauley, E., & McLaughlin, K. A. (2016). Childhood maltreatment exposure and disruptions in emotion regulation: A Transdiagnostic

pathway to adolescent internalizing and externalizing psychopathology. *Cognitive Therapy and Research, 40*(3), 394–415. https://doi.org/10.1007/s10608-015-9735-z

Heleniak, C., & McLaughlin, K. A. (2020). Social-cognitive mechanisms in the cycle of violence: Cognitive and affective theory of mind, and externalizing psychopathology in children and adolescents. *Development and Psychopathology, 32*(2), 735–750. https://doi.org/10.1017/S0954579419000725

Hori, H., Itoh, M., Lin, M., Yoshida, F., Niwa, M., Hakamata, Y., Matsui, M., Kunugi, H., & Kim, Y. (2021). Childhood maltreatment history and attention bias variability in healthy adult women: Role of inflammation and the BDNF Val66Met genotype. *Translational Psychiatry, 11*(1), 122. https://doi.org/10.1038/s41398-021-01247-4

Hostinar, C. E., Davidson, R. J., Graham, E. K., Mroczek, D. K., Lachman, M. E., Seeman, T. E., van Reekum, C. M., & Miller, G. E. (2017). Frontal brain asymmetry, childhood maltreatment, and low-grade inflammation at midlife. *Psychoneuroendocrinology, 75*, 152–163. https://doi.org/10.1016/j.psyneuen.2016.10.026

Hostinar, C. E., Lachman, M. E., Mroczek, D. K., Seeman, T. E., & Miller, G. E. (2015). Additive contributions of childhood adversity and recent stressors to inflammation at midlife: Findings from the MIDUS study. *Developmental Psychology, 51*(11), 1630–1644. https://doi.org/10.1037/dev0000049

Janiri, D., Moccia, L., Dattoli, L., Pepe, M., Molinaro, M., De Martin, V., Chieffo, D., Di Nicola, M., Fiorillo, A., Janiri, L., & Sani, G. (2021). Emotional dysregulation mediates the impact of childhood trauma on psychological distress: First Italian data during the early phase of COVID-19 outbreak. *Australian & New Zealand Journal of Psychiatry, 55*(11), 1071–1078. https://doi.org/10.1177/0004867421998802

Kaufman Kantor, G., Holt, M. K., Mebert, C. J., Straus, M. A., Drach, K. M., Ricci, L. R., MacAllum, C. A., & Brown, W. (2004). *Multidimensional neglectful behavior scale–child report (MNBS-CR)*. APA PsycTests. https://doi.org/10.1037/t20630-000 Database record.

Kendall-Tackett, K. (2009). Psychological trauma and physical health: A psychoneuroimmunology approach to etiology of negative health effects and possible interventions. *Psychological Trauma: Theory, Research, Practice, and Policy, 1*(1), 35–48. https://doi.org/10.1037/a0015128

Khan, A. J., Maguen, S., Straus, L. D., Nelyan, T. C., Gross, J. J., & Cohen, B. E. (2021). Expressive suppression and cognitive reappraisal in veterans with PTSD: Results from the mind your heart study. *Journal of Affective Disorders, 283*, 278–284. https://doi.org/10.1016/j.jad.2021.02.015

Khan, A. J., O'Donovan, A., Neylan, T. C., Gross, J. J., & Cohen, B. E. (2020). Suppression, but not reappraisal, is associated with inflammation in trauma-exposed veterans. *Psychoneuroendocrinology, 122*, 104871. https://doi.org/10.1016/j.psyneuen.2020.104871

Kim-Spoon, J., Herd, T., Brieant, A., Peviani, K., Deater-Deckard, K., Lauharatanahirun, N., Lee, J., & King-Casas, B. (2021). Maltreatment and brain development: The effects of abuse and neglect on longitudinal trajectories of neural activation during risk processing and cognitive control. *Developmental Cognitive Neuroscience, 48*, 100939. https://doi.org/10.1016/j.dcn.2021.100939

Kring, A. M., Smith, D. A., & Neale, J. M. (1994). Individual differences in dispositional expressiveness: Development and validation of the Emotional Expressivity Scale. *Journal of Personality and Social Psychology, 66*(5), 934–949. https://doi.org/10.1037/0022-3514.66.5.934

Kurani, S., Webb, L., Cadet, K., et al. (2022). Area-level deprivation and adverse childhood experiences among high school students in Maryland. *BMC Public Health, 22*, 811. https://doi.org/10.1186/s12889-022-13205-w

Kwon, H., Yoon, K. L., Joormann, J., & Kwon, J.-H. (2013). Cultural and gender differences in emotion regulation: Relation to depression. *Cognition & Emotion, 27*(5), 769–782. https://doi.org/10.1080/02699931.2013.792244

Lambert, H. K., King, K. M., Monahan, K. C., & McLaughlin, K. A. (2017). Differential associations of threat and deprivation with emotion regulation and cognitive control in adolescence. *Development and Psychopathology, 29*(3), 929–940. https://doi.org/10.1017/S0954579416000584

Lasselin, J., Lekander, M., Axelsson, J., & Karshikoff, B. (2018). Sex differences in how inflammation affects behavior: What we can learn from experimental inflammatory models in humans. *Frontiers in Neuroendocrinology, 50*, 91–106. https://doi.org/10.1016/j.yfrne.2018.06.005

Lee, C., Coe, C. L., & Ryff, C. D. (2017). Social disadvantage, severe child abuse, and biological profiles in adulthood. *Journal of Health and Social Behavior, 58*(3), 371–386. https://doi.org/10.1177/0022146516685370

Lopez, R. B., Brown, R. L., Wu, E.-L.L., Murdock, K. W., Denny, B. T., Heijnen, C., & Fagundes, C. (2020). Emotion regulation and immune functioning during grief: Testing the role of expressive suppression and cognitive reappraisal in inflammation among recently bereaved spouses. *Psychosomatic Medicine, 82*(1), 2–9. https://doi.org/10.1097/PSY.0000000000000755

Lopez-Candales, A., Burgos, P. M. H., Hernandez-Suarez, D. F., & Harris, D. (2017). Linking chronic inflammation with cardiovascular disease: From normal aging to the metabolic syndrome. *Journal of Natural Sciences*, 22.

Machlin, L., Miller, A. B., Snyder, J., McLaughlin, K. A., & Sheridan, M. A. (2019). Differential associations of deprivation and threat with cognitive control and fear conditioning in early childhood. *Frontiers in Behavioral Neuroscience, 13*, 80. https://doi.org/10.3389/fnbeh.2019.00080

Markus, H. R., & Kitayama, S. (1991). Culture and self: Implications for cognition, emotion, and motivation. *Psychological Review, 98*(2), 224–253.

Marsland, A. L., Walsh, C., Lockwood, K., & John-Henderson, N. A. (2017). The effects of acute psychological stress on circulating and stimulated inflammatory markers: A systematic review and meta-analysis. *Brain, Behavior, and Immunity, 64*, 208–219. https://doi.org/10.1016/j.bbi.2017.01.011

Mathur, A., Li, J. C., Lipitz, S. R., & Graham-Engeland, J. E. (2022). Emotion regulation as a pathway connecting early life adversity and inflammation in adulthood: A conceptual framework. *Adversity and Resilience Science, 3*(1), 1–19. https://doi.org/10.1007/s42844-022-00051-3

Matthews, R., Pineault, L., & Hong, Y.-H. (2022). Normalizing the use of single-item measures: Validation of the single-item compendium for organizational psychology. *Journal of Business and Psychology*, *37*. https://doi.org/10.1007/s10869-022-09813-3

McLaughlin, K. A., Kubzansky, L. D., Dunn, E. C., Waldinger, R., Vaillant, G., & Koenen, K. C. (2010). Childhood social environment, emotional reactivity to stress, and mood and anxiety disorders across the life course. *Depression and Anxiety, 27*(12), 1087–1094. https://doi.org/10.1002/da.20762

McLaughlin, K. A., Peverill, M., Gold, A. L., Alves, S., & Sheridan, M. A. (2015). Child maltreatment and neural systems underlying emotion regulation. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(9), 753–762. https://doi.org/10.1016/j.jaac.2015.06.010

McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood adversity. *Current Directions in Psychological Science, 25*(4), 239–245. https://doi.org/10.1177/0963721416655883

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as

distinct dimensions of early experience. *Neuroscience & Biobehavioral Reviews, 47*, 578–591. https://doi.org/10.1016/j.neubiorev.2014.10.012

McRae, K., Jacobs, S. E., Ray, R. D., John, O. P., & Gross, J. J. (2012). Individual differences in reappraisal ability: Links to reappraisal frequency, well-being, and cognitive control. *Journal of Research in Personality, 46*(1), 2–7. https://doi.org/10.1016/j.jrp.2011.10.003

McRae, K., Ochsner, K. N., Mauss, I. B., Gabrieli, J. J. D., & Gross, J. J. (2008). Gender differences in emotion regulation: An fMRI study of cognitive reappraisal. *Group Processes & Intergroup Relations, 11*(2), 143–162. https://doi.org/10.1177/1368430207088035

Michopoulos, V., Powers, A., Gillespie, C. F., Ressler, K. J., & Jovanovic, T. (2017). Inflammation in fear- and anxiety-based disorders: PTSD, GAD, and beyond. *Neuropsychopharmacology, 42*(1), 254–270. https://doi.org/10.1038/npp.2016.146

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*(6), 959–997. https://doi.org/10.1037/a0024768

Milojevich, H. M., Norwalk, K. E., & Sheridan, M. A. (2019). Deprivation and threat, emotion dysregulation, and psychopathology: Concurrent and longitudinal associations. *Development and Psychopathology, 31*(3), 847–857. https://doi.org/10.1017/S0954579419000294

Miu, A. C., Szentágotai-Tătar, A., Balázsi, R., Nechita, D., Bunea, I., & Pollak, S. D. (2022). Emotion regulation as mediator between childhood adversity and psychopathology: A meta-analysis. *Clinical Psychology Review, 93*, 102141. https://doi.org/10.1016/j.cpr.2022.102141

Nolen-Hoeksema, S. (2012). Emotion regulation and psychopathology: The role of gender. *Annual Review of Clinical Psychology, 8*(1), 161–187. https://doi.org/10.1146/annurev-clinpsy-032511-143109

O'Connor, M.-F., Bower, J. E., Cho, H. J., Creswell, J. D., Dimitrov, S., Hamby, M. E., Hoyt, M. A., Martin, J. L., Robles, T. F., Sloan, E. K., Thomas, K. S., & Irwin, M. R. (2009). To assess, to control, to exclude: Effects of biobehavioral factors on circulating inflammatory markers. *Brain, Behavior, and Immunity, 23*(7), 887–897. https://doi.org/10.1016/j.bbi.2009.04.005

Paschke, L. M., Dörfel, D., Steimke, R., Trempler, I., Magrabi, A., Ludwig, V. U., Schubert, T., Stelzel, C., & Walter, H. (2016). Individual differences in self-reported self-control predict successful emotion regulation. *Social Cognitive and Affective Neuroscience, 11*(8), 1193–1204. https://doi.org/10.1093/scan/nsw036

Pearce, J. R., Richardson, E. A., Mitchell, R. J., & Shortt, N. K. (2010). Environmental justice and health: The implications of the socio-spatial distribution of multiple environmental deprivation for health inequalities in the United Kingdom: Environmental justice and health. *Transactions of the Institute of British Geographers, 35*(4), 522–539. https://doi.org/10.1111/j.1475-5661.2010.00399.x

Pedrini, L., Meloni, S., Lanfredi, M., & Rossi, R. (2022). School-based interventions to improve emotional regulation skills in adolescent students: A systematic review. *Journal of Adolescence, 94*(8), 1051–1067. https://doi.org/10.1002/jad.12090

Petrov, M. E., Davis, M. C., Belyea, M. J., & Zautra, A. J. (2016). Linking childhood abuse and hypertension: Sleep disturbance and inflammation as mediators. *Journal of Behavioral Medicine, 39*(4), 716–726. https://doi.org/10.1007/s10865-016-9742-x

Poole, J. C., Dobson, K. S., & Pusch, D. (2018). Do adverse childhood experiences predict adult interpersonal difficulties? The role of emotion dysregulation. *Child Abuse & Neglect, 80*, 123–133. https://doi.org/10.1016/j.chiabu.2018.03.006

Phillips, S., & Mychailyszyn, M. (2022). The Effect of school-based mindfulness interventions on anxious and depressive symptoms: A meta-analysis. *School Mental Health, 14*, 455–469. https://doi.org/10.1007/s12310-021-09492-0

Randolff, L. S. (1977). The CES-D scale: A self-report depression scale for resarch in the gernal population. *Applied Psychological Measurement, 1*(3), 385–401.

Renna, M. E. (2021). A review and novel theoretical model of how negative emotions influence inflammation: The critical role of emotion regulation. *Brain, Behavior, & Immunity - Health, 18*, 100397. https://doi.org/10.1016/j.bbih.2021.100397

Renna, M. E., Peng, J., Shrout, M. R., Madison, A. A., Andridge, R., Alfano, C. M., Povoski, S. P., Lipari, A. M., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2021). Childhood abuse histories predict steeper inflammatory trajectories across time. *Brain, Behavior, and Immunity, 91*, 541–545. https://doi.org/10.1016/j.bbi.2020.11.012

Rodríguez-Hernández, H., Simental-Mendía, L. E., Rodríguez-Ramírez, G., & Reyes-Romero, M. A. (2013). Obesity and inflammation: Epidemiology, risk factors, and markers of inflammation. *International Journal of Endocrinology, 2013*, 1–11. https://doi.org/10.1155/2013/678159

Ryff, C. D., Almeida, D. M., Ayanian, J. Z., Carr, D. S., Cleary, P. D., Coe, C., Davidson, R. J., Krueger, R. F., Lachman, M. E., Marks, N. F., Mroczek, D. K., Seeman, T. E., Seltzer, M. M., Singer, B. H., Sloan, R. P., Tun, P. A., Weinstein, M., & Williams, D. R. (2021). *Midlife in the United States (MIDUS 2), 2004-2006*. Inter-university Consortium for Political and Social Research [distributor]. https://doi.org/10.3886/ICPSR04652.v8

Ryff, C. D., Seeman, T., & Weinstein, M. (2019). *Midlife in the United States (MIDUS 2): Biomarker project, 2004-2009*. Inter-university Consortium for Political and Social Research [distributor]. https://doi.org/10.3886/ICPSR29282.v9

Schrepf, A., Markon, K., & Lutgendorf, S. K. (2014). From childhood trauma to elevated C-reactive protein in adulthood: The role of anxiety and emotional Eating. *Psychosomatic Medicine, 76*(5), 327–336. https://doi.org/10.1097/PSY.0000000000000072

Schweizer, S., Walsh, N. D., Stretton, J., Dunn, V. J., Goodyer, I. M., & Dalgleish, T. (2016). Enhanced emotion regulation capacity and its neural substrates in those exposed to moderate childhood adversity. *Social Cognitive and Affective Neuroscience, 11*(2), 272–281. https://doi.org/10.1093/scan/nsv109

Shacter, E., & Weitzman, S. A. (2002). Chronic inflammation and cancer. *Oncology, 16*(2), 217–226.

Shapero, B. G., Hamilton, J. L., Stange, J. P., Liu, R. T., Abramson, L. Y., & Alloy, L. B. (2015). Moderate childhood stress buffers against depressive response to proximal stressors: A multiwave prospective study of early adolescents. *Journal of Abnormal Child Psychology, 43*(8), 1403–1413. https://doi.org/10.1007/s10802-015-0021-z

Sheridan, M. A., & McLaughlin, K. A. (2014). Dimensions of early experience and neural development: Deprivation and threat. *Trends in Cognitive Sciences, 18*(11), 580–585. https://doi.org/10.1016/j.tics.2014.09.001

Sheridan, M. A., Peverill, M., Finn, A. S., & McLaughlin, K. A. (2017). Dimensions of childhood adversity have distinct associations with neural systems underlying executive functioning. *Development and Psychopathology, 29*(5), 1777–1794. https://doi.org/10.1017/S0954579417001390

Sheridan, M. A., Shi, F., Miller, A. B., Salhi, C., & McLaughlin, K. A. (2020). Network structure reveals clusters of associations between childhood adversities and development outcomes. *Developmental Science, 23*(5), e12934. https://doi.org/10.1111/desc.12934

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods, 7*(4), 422–445. https://doi.org/10.1037/1082-989X.7.4.422

Sloan, E., Hall, K., Moulding, R., Bryce, S., Mildred, H., & Staiger, P. K. (2017). Emotion regulation as a transdiagnostic treatment construct across anxiety, depression, substance, eating and borderline personality disorders: A systematic review. *Clinical Psychology Review, 57*, 141–163. https://doi.org/10.1016/j.cpr.2017.09.002

Slopen, N., Lewis, T. T., Gruenewald, T. L., Mujahid, M. S., Ryff, C. D., Albert, M. A., & Williams, D. R. (2010). Early life adversity and inflammation in African Americans and whites in the midlife in the United States survey. *Psychosomatic Medicine, 72*(7), 694–701. https://doi.org/10.1097/PSY.0b013e3181e9c16f

Spidel, A., Lecomte, T., Kealy, D., & Daigneault, I. (2018). Acceptance and commitment therapy for psychosis and trauma: Improvement in psychiatric symptoms, emotion regulation, and treatment compliance following a brief group intervention. *Psychology and Psychotherapy: Theory, Research and Practice, 91*(2), 248–261. https://doi.org/10.1111/papt.12159

Srirangarajan, T., Oshio, A., Yamaguchi, A., & Akutsu, S. (2020). Cross-cultural nomological network of gratitude: Findings from midlife in the United States (MIDUS) and Japan (MIDJA). *Frontiers in Psychology, 11*, 571. https://doi.org/10.3389/fpsyg.2020.00571

Sumner, J. A., Colich, N. L., Uddin, M., Armstrong, D., & McLaughlin, K. A. (2019). Early experiences of threat, but not deprivation, are associated with accelerated biological aging in children and adolescents. *Biological Psychiatry, 85*(3), 268–278. https://doi.org/10.1016/j.biopsych.2018.09.008

Szabo, Y. Z., & Slavish, D. C. (2021). Measuring salivary markers of inflammation in health research: A review of methodological considerations and best practices. *Psychoneuroendocrinology, 124*, 105069. https://doi.org/10.1016/j.psyneuen.2020.105069

Tabachnick, A. B., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Allyn & Bacon.

Thompson, K. L., Hannan, S. M., & Miron, L. R. (2014). Fight, flight, and freeze: Threat sensitivity and emotion dysregulation in survivors of chronic childhood maltreatment. *Personality and Individual Differences, 69*, 28–32. https://doi.org/10.1016/j.paid.2014.05.005

Tinajero, R., Williams, P. G., Cribbet, M. R., Rau, H. K., Silver, M. A., Bride, D. L., & Suchy, Y. (2020). Reported history of childhood trauma and stress-related vulnerability: Associations with emotion regulation, executive functioning, daily hassles and pre-sleep arousal. *Stress and Health, 36*(4), 405–418. https://doi.org/10.1002/smi.2938

Tsalamandris, S., Antonopoulos, A. S., Oikonomou, E., Papamikroulis, G.-A., Vogiatzi, G., Papaioannou, S., Deftereos, S., & Tousoulis, D. (2019). The role of inflammation in diabetes: Current concepts and future perspectives. *European Cardiology Review, 14*(1), 50–59. https://doi.org/10.15420/ecr.2018.33.1

Turner, R. J., & Wheaton, B. (1995). Checklist measurment of stressful life events. In S. Cohen, R. Kessler, & L. Underwood Gordon (Eds.). In *Measuring stress. A guide for health and social sciences* (pp. 29–58). Oxford University Press.

Weissman, D. G., Bitran, D., Miller, A. B., Schaefer, J. D., Sheridan, M. A., & McLaughlin, K. A. (2019). Difficulties with emotion regulation as a transdiagnostic mechanism linking child maltreatment with the emergence of psychopathology. *Development and Psychopathology, 31*(3), 899–915. https://doi.org/10.1017/S0954579419000348

World Health Organization. (2022). *Adolescent health*. https://www.who.int/health-topics/adolescent-health#tab=tab_1

Yilmaz, M. I., Stenvinkel, P., Sonmez, A., Saglam, M., Yaman, H., Kilic, S., Eyileten, T., Caglar, K., Oguz, Y., Vural, A., Cakar, M., Altun, B., Yenicesu, M., & Carrero, J. J. (2011). Vascular health, systemic inflammation and progressive reduction in kidney function; clinical determinants and impact on cardiovascular outcomes. *Nephrology Dialysis Transplantation, 26*(11), 3537–3543. https://doi.org/10.1093/ndt/gfr081

Zapolski, T. C. B., & Smith, G. T. (2017). Pilot study: Implementing a brief DBT skills program in schools to reduce health risk behaviors among early adolescents. *The Journal of School Nursing, 33*(3), 198–204. https://doi.org/10.1177/1059840516673188

Zeier, P., Meine, L. E., & Wessa, M. (2021). It's worth the trouble: Stressor exposure is related to increased cognitive reappraisal ability. *Stress and Health,* smi.3101. https://doi.org/10.1002/smi.3101

Supplemental Table 1

Correlations Between CTQ Subscales, Inflammation, and Emotion Regulation

Variable	Physical abuse	Sexual abuse	Emotional abuse	Physical neglect	Emotional neglect
Fibrinogen	.083**	.085**	.054	.068*	.055
Reappraisal	-.021	.021	-.056	-.001	-.079**
Suppression	.005	.039	-.039	-.017	-.101**

Note. CTQ = Childhood trauma questionnaire, *$p < .05$, **$p < .01$.

Supplemental Table 2

Exploratory Model of SLE Deprivation and Suppression Predicting Fibrinogen by Sex

SLE deprivation and suppression predicting fibrinogen in males R^2 = .08 , *F* (7,469) = 6.19, *p* <.001			
Predictor	B	SE	p
Constant	150.18	32.70	<.001
SLE deprivation	36.93	19.59	.06
Suppression	2.28	1.70	.18
CES-D	.41	.49	.41
Age	.96	.35	.007
Total number of medications	-.72	1.02	.41
BMI	3.19	.66	<.001
Number of health conditions	3.16	2.04	.12
N = 476. Indirect effect of SLE deprivation on fibrinogen through suppression, *b* = -.08, *SE* = 1.57, 95% confidence interval [-3.43, 3.41], *p* > .05			
SLE deprivation and suppression predicting fibrinogen in females R^2 = .16 , *F* (7, 588) = 16.24, *p* <.001			
Predictor	B	SE	p
Constant	136.04	26.82	<.001
SLE deprivation	-6.85	18.56	.71
Suppression	3.08	1.53	.04
CES-D	1.11	.44	.01
Age	1.15	.32	<.001
Total number of medications	-.83	.77	.29
BMI	4.40	.46	<.001
Number of health conditions	-1.80	1.62	.27
N = 596. Indirect effect of SLE deprivation on fibrinogen through suppression, b = 5.71, SE = 3.31, 95% confidence interval [-.03, 12.99], *p* > .05			

Note. CTQ = Childhood Trauma Questionnaire, SLE = stressful life events, CES-D = Center for Epidemiological Studies Depression Scale, BMI = body mass index.

Supplemental Table 3

Exploratory Model of SLE Deprivation and Reappraisal Predicting Fibrinogen by Sex

SLE deprivation and reappraisal predicting fibrinogen in males $R^2 = .09$, $F(7,468) = 6.72$, $p < .001$			
Predictor	B	SE	p
Constant	138.86	31.68	<.001
SLE deprivation	36.07	19.52	.07
Reappraisal	6.61	2.91	.02
CES-D	.55	.50	.27
Age	.94	.35	.008
Total number of medications	-.65	1.02	.52
BMI	3.22	.66	<.001
Number of health conditions	3.16	2.03	.12
N = 476. Indirect effect of SLE deprivation on fibrinogen through reappraisal, b = .78, SE = 2.27, 95% confidence interval [-3.75, 5.74], p > .05			
SLE deprivation and reappraisal predicting fibrinogen in females $R^2 = .17$, $F(7, 588) = 16.62$, $p < .001$			
Predictor	B	SE	p
Constant	136.87	24.96	<.001
SLE deprivation	-8.27	18.52	.66
Reappraisal	6.45	2.58	.01
CES-D	1.12	.44	.01
Age	1.10	.31	.48
Total number of medications	-.88	.77	.26
BMI	4.40	.46	<.001
Number of health conditions	-1.67	1.60	.30
N = 596. Indirect effect of SLE deprivation on fibrinogen through reappraisal, b = 7.13, SE = 3.52, 95% confidence interval [1.05, 14.67], p < .05			

Note. CTQ = Childhood Trauma Questionnaire, SLE = stressful life events, CES-D = Center for Epidemiological Studies Depression Scale, BMI = body mass index.4