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Psychological Pathways From Childhood Maltreatment to Inflammation Among Midlife and Older Adults

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Objective: Childhood abuse and neglect have been linked to increased inflammation in adulthood. Psychological pathways that lead from abuse and neglect to inflammation have been seldom identified. Abuse and neglect may impact traits such as trait anger and trait anxiety, which in turn, influence emotion regulation thus leading to a psychological cascade that results in heightened levels of inflammation. ***Method:*** Trait anxiety and trait anger, and suppression and reappraisal were examined as pathways between abuse and neglect and inflammatory biomarkers, including interleukin-6, C-reactive protein, and tumor necrosis factor alpha. Longitudinal data from the Midlife Development in the United States study were used to analyze a sample of 317 adults within a structural equation modeling framework. ***Results:*** Neglect was associated with elevated levels of C-reactive protein through higher levels of trait anger and higher levels of emotional suppression. The effects of abuse were not significant. ***Conclusions:*** Findings of the study indicate that experiences of childhood neglect, but not abuse, appear to initiate a psychological cascade that results in elevated C-reactive protein. Neglect leads to raised levels of trait anger which, in turn, increases emotional suppression.

Clinical Impact Statement
The study suggests that a history of childhood neglect, through trait anger, increases emotional suppression, which leads to raised levels of C-reactive protein. Utilizing interventions that focus on effective emotion regulation in those with higher levels of trait anger may have physiological and physical health benefits via reducing inflammatory biomarkers.

Keywords: childhood abuse, childhood neglect, emotion regulation, inflammation

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The question of how psychosocial trauma in childhood ultimately influences physiological dysfunction decades later remains largely unanswered. Childhood abuse and neglect have known, prospective associations with a variety of physical morbidity as well as physiological biomarkers (Widom et al., 2012), notably inflammatory biomarkers (Danese et al., 2007). Over one third of adults in the United States have experienced abuse or neglect in childhood (Cammack & Hogue, 2017), making the heightened levels of inflammation associated with maltreatment exceptionally common. In fact, Danese contends that approximately 10% of cases of low-grade inflammation is attributable to maltreatment (Danese et al., 2007). Inflammation becomes increasingly salient as adults move into middle and later adulthood and increases risk for cognitive, psychological, and behavioral impairment, increased morbidity, and premature mortality (Ferrucci & Fabbri, 2018).

There are several notable limitations to existing research. Although prospective research has documented linkages from abuse and neglect to inflammation (e.g., Danese et al., 2007), there are few longitudinal studies investigating links from abuse and neglect to inflammation over time (see Renna et al., 2020, for exception). Additionally, mediators are rarely tested longitudinally, and doing so could serve as the basis of trauma-informed and potentially modifiable point of prevention and intervention (Caldwell et al., 2019; Hodgson et al., 2021). Consistent with Renna (2021), we propose that childhood

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abuse and neglect increase adults' general disposition to experience anger and anxiety (Fitzgerald & Williams, 2024), which will hamper adults' ability to implement effective emotional regulation strategies and increase the likelihood of maladaptive regulation strategies, leading to greater inflammation over time. Inflammation is a particularly important biomarker due to associations with cardiovascular health and premature mortality (Henein et al., 2022; Proctor et al., 2015). We used a sample of adults in the United States to longitudinally examine the relationships from childhood maltreatment to inflammatory biomarkers (interleukin-6 [IL6], C-reactive protein [CRP], tumor necrosis factor alpha [TNF-α]) via trait anger and anxiety and emotion regulation (suppression and reappraisal) strategies in a cascade model.

Childhood Maltreatment

There are several commonly used ways to measure and operationalize maltreatment, all of which have their benefits and drawbacks. One approach is to examine the effects of individual maltreatment types separately, which provides information about the unique effects versus shared effects of different forms of maltreatment on health (Cecil et al., 2017), but much of the variation accounted for by the individual subtypes overlaps across subtypes and fails to consider the cumulative impact of adversity. A second method to operationalize maltreatment is the utilization of a cumulative maltreatment score (e.g., dose–response relationship), which refers to the accumulated impact of abuse and neglect (Scott-Storey, 2011). This approach makes strong assumptions that each of the subtypes equally contributes to distal outcomes (Scott-Storey, 2011). A third approach is to utilize a dimensional model which groups maltreatment types together based on common underlying features. McLaughlin et al. (2021) proposed that maltreatment subtypes can be categorized into threat (e.g., abuse) and deprivation (e.g., neglect). Underpinning this approach is an assumption that abuse and neglect operate differently on mechanisms that potentiate distal health outcomes (McLaughlin et al., 2021). Notably, abuse includes acts inflicted upon the child that cause harm or injury, while neglect is a failure to meet the child's basic needs. McLaughlin et al. highlighted that experience-driven plasticity plays a significant role in the differential outcomes resulting from threat versus deprivation. Through myelination and pruning, experience-driven plasticity shapes neural circuits and behavior according to environmental stimuli (i.e., unnecessary neural connections are eliminated while those responding to frequent environmental input are strengthened). Threat versus deprivation experiences have been demonstrated to have different outcomes regarding neural structure and function, development, and cognitive abilities (McLaughlin et al., 2019, 2021). Most existing studies have utilized either a cumulative or individual approach, and parceling out the effects of threat and deprivation could provide novel insights into how these discrete types of maltreatment uniquely impact adult health, which will be examined in the present study.

Childhood Abuse and Neglect and Inflammation

One of the negative health outcomes childhood abuse and neglect has been consistently linked to are elevated inflammatory biomarkers, including IL6, CRP, and TNF-α (Bitsika et al., 2021; Coelho et al., 2014; Kerr et al., 2021). Studies utilizing a dimensional model to examine the contributions of abuse versus neglect to increases in inflammatory biomarkers are very limited (see Fanning et al., 2015, for exception), but evidence from studies examining individual types of maltreatment demonstrates a noticeable trend where abuse tends to be more consistently associated with biomarkers than neglect (Bitsika et al., 2021; Grosse et al., 2016; Matthews et al., 2014; Müller et al., 2019; Munjiza et al., 2018). For example, research has linked all types of abuse with higher levels of IL6 (Grosse et al., 2016; Müller et al., 2019; Munjiza et al., 2018), but only some evidence suggested that physical neglect was a predictor (Müller et al., 2019; Munjiza et al., 2018). Regarding CRP, Bitsika et al. (2021) found that types of abuse (physical and emotional), but not neglect, were related to increased levels. Matthews et al. (2014) also identified emotional abuse, and in addition, emotional neglect as predictors of elevated CRP. Fanning et al. (2015) utilized a dimensional model of abuse and neglect and demonstrated that only abuse but not neglect was associated with CRP. This study, however, did not include possible mediators between dimensions of maltreatment and biomarkers. TNF-α, previously better known for its diverse roles in cancer and neurological processes (Aggarwal et al., 2012), has been recognized as a major component of systemic inflammation within the fields of immunology and physiology, however, it has not been consistently incorporated in social science studies investigating links of abuse and neglect with inflammation until more recently and thus has received less attention than IL6 and CRP. Fewer studies have examined maltreatment subtypes and TNF-α, and none of the subtypes have been shown to predict TNF-α by themselves (Hartwell et al., 2013) which may indicate the need for using a dimensional model to examine whether experiences of threat or deprivation, as opposed to individual subtypes, assert an influence on TNF-α. Taken together, research has underscored that no single form of abuse is a consistent predictor of inflammatory biomarkers, yet the construct of abuse and its associated qualities (i.e., threat) have more consistent associations with inflammation than neglect.

Psychological Pathways From Childhood Abuse and Neglect to Inflammation

Decades of research indicate that childhood maltreatment potentiate neurobiological changes (e.g., volume and functioning of brain structures). While abuse and neglect can co-occur, due to the differences between the characteristics of abuse and neglect (i.e., threat vs. deprivation), there may be differential effects on neurobiological changes that have implications for trait anger and anxiety as well as inflammation. Dimensional models acknowledge that threat and deprivation experiences can simultaneously coexist, but have unique attributes whose impacts on development should be disentangled and cannot be assumed to be the same (McLaughlin et al., 2021).

Experiences of threat in early life are associated with changes in the amygdala, medial prefrontal cortex, and hippocampus which can be linked to both increased trait anger and trait anxiety. Alterations in the amygdala may predispose survivors to experiencing increased anger and aggression (Blair, 2012). These underlying neurobiological mechanisms provide support for social learning theory suggesting that children learn through observing, imitating, and modeling adults' behaviors, and the anger experienced with abuse may be a learned trait that carries on into adulthood (Bandura et al., 1961). The neurobiological changes also lead to increased sensitivity to perceiving anger in others, accuracy in identifying others' facial expressions of anger, and attentional bias toward threat cues (McLaughlin et al., 2019). Due to increased sensitivity and bias toward threat, individuals who were

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abused are predisposed to have higher levels of trait anxiety (Günther et al., 2020). As a result of underlying neurobiological changes, adults who have experienced greater abuse are more sensitive to developing higher levels of trait anger and trait anxiety.

In contrast, deprivation (e.g., neglect) is characterized by reduced volume and activation of the frontoparietal regions (i.e., lateral prefrontal cortex and posterior parietal cortex) responsible for cognitive resources causing difficulties with the cognitive control of emotions (McLaughlin et al., 2021). While threat has also been linked to executive function dysregulation in some studies (Maged Hamza et al., 2023), other studies provide greater support for associations of neglect and not abuse with poor executive functioning (Hildyard & Wolfe, 2002; Kirke-Smith et al., 2012; Kotch et al., 2008; Nikulina & Widom, 2013). Diminished cognitive control is a consequence of the inability of the more advanced brain regions (e.g., prefrontal cortex) to override or downregulate anger, which leads to higher trait anger (Wilkowski & Robinson, 2008). Further, reduced prefrontal activity and prefrontal attentional control functions are also related to increased trait anxiety (Bishop, 2009). Based on the neurobiological impacts of deprivation, experiencing neglect can predispose adults to both higher levels of trait anger and trait anxiety. One notable distinction between those who were abused versus neglected is that while experiences of abuse lead to a bias toward angry and threatening facial expressions, neglect results in diminished ability to recognize and distinguish emotional expressions, and has also been linked to avoidant reactions to social threat and angry facial expressions in others (Brüne et al., 2013; Pollak et al., 2000; Russo et al., 2015). Thus, neglect is less likely to lead to hypervigilance toward threat, and adults who were abused may experience greater increases in trait anger and anxiety due to more frequent perceptions of threat than those who were neglected.

Those higher in trait anxiety and trait anger may be at risk for increased inflammation due to greater struggles implementing emotion regulation strategies. Trait anxiety and trait anger are characterized by negative emotionality, or the tendency of experiencing frequent, prolonged negative emotions (Mincic, 2015; Spielberger, 1988). Negative emotionality increases vulnerability for using maladaptive emotion regulation strategies (e.g., suppression) as opposed to adaptive emotion regulation (e.g., cognitive reappraisal). Maladaptive emotion regulation strategies are ways of managing emotions that are ineffective at truly modifying negative affect and therefore lead to experiencing increased negative affect for longer periods of time (Aldao et al., 2010). Although maladaptive emotion regulation strategies can be viewed as adaptive responses to strong and overwhelming emotions that temporarily relieve stress (Gross, 1998), they tend to perpetuate negative emotions and lead to worse psychological outcomes over time which makes them ultimately maladaptive (Aldao et al., 2010). The combination of more frequent maladaptive regulation strategies and less frequent positive regulation strategies perpetuates negative emotions (Mennin et al., 2005). Supporting recent theory about emotion regulation strategies serving as a mechanism from emotionality to inflammatory biomarkers (Renna, 2021), a systematic review conducted by Moriarity et al. (2023) supports the notion that emotion regulation influences inflammation. In general, poor emotion regulation is linked to higher inflammation and better emotion regulation skills are associated with lower inflammation (Moriarity et al., 2023). Emotional suppression is characterized by reduced external expression of emotions and is usually considered to be a maladaptive emotion regulation strategy, while cognitive reappraisal is the process of generating new interpretations of stressful experiences and is an adaptive method for managing emotions (Gross, 1998; Mennin et al., 2005). Several studies found a significant relationship between emotional suppression and higher levels of CRP (Appleton et al., 2013; Khan et al., 2020). In contrast, cognitive reappraisal has been associated with lower IL6, CRP, and TNF-α (Appleton et al., 2013; Chen et al., 2015; Jones et al., 2023; Shahane et al., 2023).

The Present Study

The present study aimed to examine associations between childhood abuse and neglect and IL6, CRP, and TNF-α through trait anger and anxiety, and suppression and reappraisal in a cascade model. We hypothesized that abuse and neglect would be positively associated with both trait anger and trait anxiety, but abuse would have stronger associations with trait anger and trait anxiety than neglect. We expected that both trait anger and trait anxiety would be positively associated with emotional suppression and negatively associated with reappraisal. Finally, we hypothesized that suppression would be positively associated with all three biomarkers and reappraisal would be negatively associated with all of them. Several covariates were included in the study, such as marital status, educational achievement, age, gender, smoking status, vigorous exercise, body mass index, and inflammatory biomarkers at the second wave of the Midlife Development in the United States study (MIDUS 2). Details about the covariates and reasons for their inclusion in the study can be found in the Supplemental Material.

Method

Data for the present study are from the study of MIDUS, a longitudinal study that has been sponsored by the John D. and Catherine T. MacArthur Foundation since 1995 (https://www.icpsr.umich.edu/web/ICPSR/series/203). Utilizing telephone interviews and mailed self-administered questionnaire, the data have been collected approximately every 9 years including 1995–1996 (MIDUS 1), 2004–2006 (MIDUS 2), and 2013–2014 (MIDUS 3). The MIDUS study originally recruited 7,108 adults from four subpopulations including (a) a national random digit dialing (RDD) sample (n = 3,487); (b) city oversamples in the United States (n = 757); (c) siblings of individuals from the RDD sample (n = 950); and (d) a national RDD sample of twin pairs (n = 1,914). The first follow-up wave (MIDUS 2) retained 4,963 participants and the second follow-up wave (MIDUS 3) retained 3,294 individuals. In addition to the telephone interviews and self-administered questionnaire, following MIDUS 2 and MIDUS 3, there were several subprojects including biomarker and neuroscience studies to obtain additional report data as well as biological data. The MIDUS 2 biomarker (n = 1,255) was conducted 2005–2009 and consisted of 1,054 adults from the MIDUS 2 and 201 adults from an independent sample of racial minorities recruited from Milwaukee. The Milwaukee sample is a sample of African Americans recruited from areas of the city of Milwaukee, Wisconsin stratified according to the proportion of the African American population (areas with high concentrations were sampled at higher rates than areas with low concentrations). Following the MIDUS 2 biomarker study, a subsample of participants were recruited to participate in a neuroscience subproject. The neuroscience project contains data from 331 respondents who participated in the biomarker project at University of Wisconsin–Madison: (a) longitudinal sample including participants of the core MIDUS sample from

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city oversamples, sibling RDD, and twin RDD (n = 223) and (b) Milwaukee subsample of racial minorities (n = 108). MIDUS 3 biomarker (n = 787) was conducted 2017–2022.

Participants

We included participants (n = 317) if they participated in the MIDUS 2, MIDUS 2 biomarker, and MIDUS 2 Neuroscience project and included 132 from the national RDD sample, indicated as (a) above; one from the sibling RDD sample, indicated as (c) above; 87 from the twin RDD sample, indicated as (d) above; and 97 from the Milwaukee subsample. Of the 317 participants, 127 participants participated in the MIDUS 3 biomarker. The present study included n = 317 participants with a mean age of M = 53.31 (SD = 11.43). 54.3% of participants were female and 60.3% of the sample reported being married. 34.3% of the sample had a high school degree or less, 29.5% reported some college without a bachelor's degree, 18.4% had a bachelor's degree, and 17.8% had at least some graduate school.

Measures

Childhood Maltreatment

Childhood maltreatment was assessed using the Childhood Trauma Questionnaire, a well-validated measure of retrospective reports of childhood abuse and neglect with acceptable internal consistency and test–retest reliability and concurrent validity (Bernstein et al., 2003). The Childhood Trauma Questionnaire uses 25 items assessing emotional, physical, and sexual abuse, as well as physical and emotional neglect prior to the age of 18. Items are scored on a 5-point Likert scale, ranging from 1 (*never*) to 5 (*very frequently*). Childhood maltreatment was operationalized for this study using two latent constructs including abuse (scores of the emotional, physical, and sexual abuse subscale) and neglect (scores of the physical and emotional neglect subscales). Higher scores on the subscales indicate greater abuse or neglect. Reliability of the latent factors was measured with Coefficient H, a maximum reliability estimate based on factor loadings for optimally weighted scales (Hancock & Mueller, 2001); abuse = .84 and neglect = .80.

Trait Anger

The present study used the Spielberger Trait Anger Inventory (Spielberger, 1983) to measure trait anger. The scale has previously demonstrated good internal consistency and test–retest reliability, and construct and concurrent validity (Forgays et al., 1997) and consists of 15 items rated on a 4-point Likert type scale where 1 = *almost never*, 2 = *sometimes*, 3 = *often*, 4 = *almost always*. The items were summed together for an overall indicator of trait anger. Example item includes "I am quick tempered." Scores could range from 15 to 60. Trait anger was collected at the MIDUS 2 biomarker study. McDonald's omega (ω) = .83.

Trait Anxiety

Trait anxiety was measured using the Spielberger Trait Anxiety Inventory (Spielberger, 1983). The Spielberger Trait Anxiety Inventory demonstrates acceptable internal consistency and test–retest reliability, and construct and concurrent validity (Spielberger, 1983) and consists of 20 items scored on 4-point Likert type scale where 1 = *almost never*, 2 = *sometimes*, 3 = *often*, 4 = *almost always*. Among the items, seven were positively worded and reverse coded. The items were then summed together for an overall indicator of trait anxiety. Example item includes "I take disappointments so keenly that I can't put them out of my mind." Scores could range from 20 to 80. Trait anxiety was collected at the MIDUS 2 biomarker study, ω = .90.

Emotion Regulation

Emotional regulation was assessed using the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). Research has established that the ERQ has strong psychometric properties including internal consistency, criterion validity, and invariance across gender and race (Melka et al., 2011; Preece et al., 2020). The ERQ is a 10-item scale assessing two dimensions of emotion regulation: (a) cognitive reappraisal and (b) expressive suppression. Respondents answered each item on a 7-point Likert type scale ranging from 1 (*strongly disagree*) to 7 (*strongly agree*). For reappraisal, ω = .77; and for suppression, ω = .76.

Inflammatory Biomarkers

Inflammatory biomarkers were assessed with three common indicators of inflammation: IL6, CRP, and TNF-α. The biomarkers IL6, CRP, and TNF-α were obtained from blood samples the morning after a 12-hr fast which occurred during the participants' overnight stay at one of three clinical labs (University of California, Los Angeles, Madison, Georgetown). IL6 and TNF-α concentrations were measured using blood serum and enzyme linked immunosorbent assay and CRP concentrations were measured using blood plasma and immunonephelometry.

Covariates

We controlled for numerous covariates including marital status (married/unmarried), educational achievement, age, gender (male/female), smoking status (yes/no), vigorous exercise (no/yes), body mass index, and inflammatory biomarkers at MIDUS 2 (see measurement details in Supplemental Material).

Statistical Analysis

Missing Data

Modern missing data techniques were employed to address missingness with the study. 63.5% of participants were missing data on the ERQ and 60% were missing on MIDUS 3 biomarker inflammation data. We employed modern missing data techniques proposed by Enders (2025). Specifically, we utilized full information maximum likelihood estimation (Enders, 2025), which assumes that data are missing at random. Data that are missing at random has identifiable patterns of missingness, but the patterns are correlated (explained by) with variables within the data set (e.g., auxiliary variables). In other words, data are conditionally missing. The inclusion of auxiliary variables in the prediction of missingness increases statistical power, adjusts standard errors, and provides more accurate test statistics (Baraldi & Enders, 2010; Enders, 2008, 2025) even when there is a considerable amount of missing data (Baraldi & Enders, 2010). We identified family support and strain, subjective

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evaluations of mental health, depression, stress, problem-focused and emotion-focused coping, hope, positive affect, and gratitude as auxiliary variables. Details of our missing data analytic procedures can be found in Supplemental Material.

Structural Equation Modeling

The first analytic step was to generate means, standard deviations, and correlations. Next, we utilized structural equation modeling to test the hypotheses in Mplus Version 8.10. Childhood abuse and neglect were modeled as latent variables and all other variables were modeled as observed. Statistics include the chi-square statistic, comparative fix index (CFI), standardized root-mean-square residual, and root-mean-square error of approximation (RMSEA). Although Hu and Bentler (1999) proposed cutoff scores, the cutoffs are overgeneralized and misapplied (West et al., 2023), and may not accurately represent a good fitting model (e.g., Kenny et al., 2015; Van Laar & Braeken, 2021). To evaluate model-data fit, omnibus measures were used (e.g., RMSEA) to detect overall model-data fit as well as examination of the residual matrix (expected–observed covariance matrix) to identify local misfit. While modification indices can provide information on model misspecification, they are not theoretically derived and therefore were not used to adjust model fit. To address multivariate nonnormality and enhance interpretability and generalizability, we employed robust maximum likelihood and employed cluster corrected standard errors to address biased standard errors due to nonnormality and correlated errors among twins (McNeish et al., 2017; Muthén & Muthén, 1998/2017).

Two other notable issues are worthy of brief discussion. First, inflammatory biomarkers are often log-transformed to address nonnormality (e.g., Bitsika et al., 2021) which creates several problems including decreased external validity (e.g., the transformed marginal distribution no longer represents the data from participants), log-transformation does not directly address nonnormality (e.g., transformations affect the marginal distribution, but the normality assumption applies to the residuals), and the associations with other variables are fundamentally changed as a result of log-transformations (e.g., for variables that have a positive skew and are leptokurtic such as inflammation, the correlation with other variables can actually increase). We utilized robust maximum likelihood to address nonnormality and used the raw data. To demonstrate how log-transformations can lead to different findings, however, we also estimated the same model with log-transformed biomarker values as a sensitivity analysis. Second, due to prevalence rates of missing data, which can be largely addressed with modern missing data analysis, we still had a substantial amount of missing data for emotion regulation and inflammatory biomarkers and would not have sufficient power for testing the likely small, but meaningful (Carey et al., 2023) serial indirect effects. Results should be understood as cascading, not statistically indirect, effects.

Results

Bivariate and Descriptive Results

Results of bivariate correlations, means, and standard deviations are presented in Table 1 and more information is provided in Supplemental Material. Using cutoff points proposed by Walker et al. (1999), we found that 19.7% adults reported emotional abuse, 14.3% sexual abuse, 18.6% physical abuse, 16.2% emotional neglect, and 27.8% physical neglect.

Structural Equation Modeling

The model demonstrated adequate fit: $\chi^2(80) = 134.10$, $p < .001$, CFI = .96, RMSEA = .05 (90% confidence interval [.03, .06]) and examination of the residual matrix indicates that there was not substantial evidence of local misfit (e.g., no outlying z scores in residual matrix). Figure 1 presents the results of the model. We found that childhood neglect ($\beta = .29$, $p = .03$), but not abuse ($\beta = -.01$, $p = .98$), was associated with higher levels of trait anger. Neither childhood abuse ($\beta = .09$, $p = .40$) nor neglect ($\beta = .03$, $p = .09$) was associated with higher levels of trait anxiety. Adults who reported higher levels of trait anger also tended to report higher levels of emotional suppression ($\beta = .29$, $p = .005$) but not reappraisal ($\beta = -.15$, $p = .20$). Trait anxiety was not associated with either reappraisal ($\beta = .11$, $p = .34$) or suppression ($\beta = -.09$, $p = .32$). Childhood abuse and neglect were not hypothesized to be predictive of emotional suppression and reappraisal, and constraining the paths to be zero did not reduce model-data fit, providing support for the effects being only indirect. Regarding the prediction of CRP, adults who were higher on emotional suppression reported higher levels of CRP ($\beta = .25$, $p = .03$), but

Table 1

Correlations, Means, and Standard Deviations of Main Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	*M* (*SD*)
1. Emotional abuse	—												7.82 (4.06)
2. Physical abuse	.69**	—											6.86 (3.03)
3. Sexual abuse	.39**	.46**	—										6.28 (3.45)
4. Emotional neglect	.66**	.49**	.23**	—									9.46 (4.40)
5. Physical neglect	.53**	.56**	.32**	.64**	—								6.87 (2.68)
6. Trait anger	.16**	.14*	.15**	.11*	.11	—							23.65 (5.31)
7. Trait anxiety	.23**	.21**	.16**	.28**	.27**	.47**	—						33.86 (8.24)
8. Suppression	−.15	−.11	−.22*	.03	−.01	−.00	.14	—					3.44 (1.21)
9. Reappraisal	−.01	.06	−.03	.07	−.03	.03	−.04	.01	—				4.98 (0.91)
10. TNF-α	.18*	.16	.05	.06	.05	−.00	−.02	−.16	−.14	—			2.42 (0.80)
11. IL6	.17	.34**	.12	−.03	.06	.02	.05	−.23	.01	.48**	—		3.56 (3.33)
12. CRP	.10	.14	.04	.04	−.01	−.04	−.08	.19	−.06	.37**	.39**	—	3.17 (3.52)

Note. TNF-α = tumor necrosis factor alpha; IL6 = interleukin-6; CRP = C-reactive protein.
* $p < .05$. ** $p < .01$.

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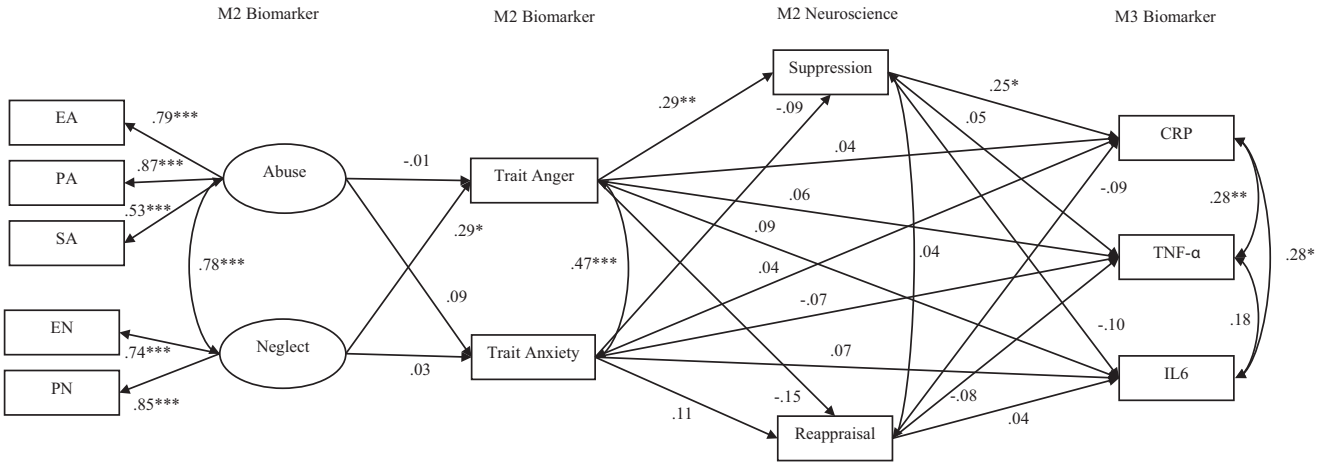
Figure 1

Standard Coefficients of the Structural Equation Modeling Examining Pathways From Childhood Abuse and Neglect to Inflammatory Biomarkers



Note. Control variables in the model include body mass index, gender, age, initial levels of IL6, TNF-α, CRP, education, exercise, smoking, and marital status. EA = emotional abuse; PA = physical abuse; SA = sexual abuse; EN = emotional neglect; PN = physical neglect; CRP = C-reactive protein; TNF-α = tumor necrosis factor alpha; IL6 = interleukin-6.
$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

reappraisal (β = −.09, p = .30), trait anxiety (β = .04, p = .66), and trait anger (β = −.14, p = .09) were not significantly associated with CRP. Regarding TNF-α, the model was not effective in accounting for variation as suppression (β = .05, p = .61), reappraisal (β = −.08, p = .31), trait anxiety (β = .06, p = .45), and trait anger (β = .06, p = .38) were each nonsignificant. Likewise, with IL6, suppression (β = −.10, p = .21), reappraisal (β = .04, p = .47), trait anxiety (β = −.06, p = .48), and trait anger (β = .09, p = .19) were nonsignificant. Parameter estimates of the covariates are reported in Supplemental Material.

As a sensitivity analysis, we reestimated the model with log-transformed biomarker values and differences in findings are reported here for transparency. Most main findings remained the same and paths predicting TNF-α and IL6 remained nonsignificant. Regarding CRP, however, results changed. Emotional suppression no longer predicted CRP (β = .21, p = .05), on the other hand, cognitive reappraisal became a significant predictor of lower levels of CRP (β = −.20, p = .02). All other parameter estimates are reported in Supplemental Material.

Discussion

Childhood abuse and neglect are known risk factors for increased levels of inflammatory biomarkers in adulthood (Bitsika et al., 2021; Coelho et al., 2014; Kerr et al., 2021). Recently, researchers have shifted their attention toward identifying psychological pathways through which early experiences can impact physiological functioning. The current exploratory study examined the cascading effects from childhood abuse and neglect to inflammatory biomarkers (IL6, CRP, TNF-α) via trait anger and anxiety and suppression and reappraisal. We found that neglect was associated with elevated levels of CRP via higher levels of trait anger and higher levels of emotional suppression; however, the effects of abuse were not significant.

Results of our exploratory study suggest that childhood neglect may be a developmental antecedent of psychological sequelae that ultimately influence biomarkers associated with morbidity and mortality. We found that those who reported more severe neglect also tended to report higher levels of trait anger. Those who were neglected may be particularly at risk for increased trait anger due to alterations in brain structures (McLaughlin et al., 2021) such as parts of the lateral prefrontal cortex and posterior parietal cortex responsible for the cognitive control of emotions (Zanto & Gazzaley, 2013). Limited cognitive control of emotions has been suggested to lead to limited ability to control anger and increased levels of trait anger (Wilkowski & Robinson, 2008). Several studies linked cumulative experiences of maltreatment to trait anger in adulthood (Berthelot et al., 2014; Win et al., 2021) and preliminary research has demonstrated a positive relationship between subtypes of maltreatment and trait anger (de Bles et al., 2023), while our study is the first (to our knowledge) to examine and demonstrate a positive relationship between cumulative childhood neglect and trait anger.

The findings also suggest that those higher in trait anger also report higher levels of emotional suppression. Suppression may require less cognitive resources compared with other higher order emotion regulation strategies (i.e., reappraisal) and thus may be a more "affordable" strategy when dealing with frequent, trait-level aroused emotional states with limited cognitive resources available (Goldin et al., 2008). Those low on trait anger have been previously found to automatically recruit cognitive control resources to downregulate hostile reactions, whereas people with high trait anger generally do not engage cognitive resources to reduce anger (Wilkowski & Robinson, 2010). Further, emotional suppression was linked to higher levels of CRP. Rather than decreasing negative emotions, suppression extends negative emotionality through psychological and physiological processes (Renna, 2021). Previous research has demonstrated that suppression and prolonged negative emotions increase inflammatory responses, including

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the production of CRP (Appleton et al., 2013; Khan et al., 2020; Renna, 2021).

In contrast to our hypotheses and findings of previous studies (e.g., Midei et al., 2010; Tracy et al., 2021), abuse was not associated with either trait anxiety or trait anger in the present study. There are several possible explanations for why we did not find the expected associations. First, there is a large time gap between experiences of childhood abuse and reports of trait anxiety and anger in our sample of midlife and older adults. Our finding may reflect domain-specific differences between abuse and neglect (McLaughlin et al., 2021), such that a history of childhood abuse does not have a prolonged effect on affective traits several decades later through the pathways tested in the present study, and there may be protective effects (e.g., moderators) that attenuate the long-term effects of abuse specifically. For example, corrective experiences in individuals' lives (e.g., therapy) and in social relationships (e.g., support from romantic partners, family, friends) may buffer the impact of abuse on affective traits. Indeed, therapeutic intervention can produce meaningful changes in personality traits and trait-level emotional stability (Roberts et al., 2017), and supportive, positive relationships moderate the associations between childhood trauma and negative affective outcomes in adulthood (Allen et al., 2023; Evans et al., 2013; King et al., 2022). Regarding trait anger, previous studies have identified subgroups of abuse survivors (Rivera et al., 2018), and specific subgroups may be more or less likely to experience trait anger in adulthood. While Bandura's social learning theory would suggest that adults who were abused in childhood would report higher levels of trait anger (Bandura et al., 1961), there may also be a different subgroup of adults who avoid anger due to their experiences of abuse. Due to a fear of "losing control" and becoming perpetrators of abuse when angry, the latter subgroup may completely dissociate feelings of anger. If there are latent subgroups in our sample, the opposing tendencies would cancel each other out in our aggregated analyses resulting in an inability to detect the effects of abuse on trait anger.

Further, trait anger did not predict cognitive reappraisal. The use of different emotion regulation strategies can depend on the specific emotions and the context in which emotions are evoked (Renna, 2021; Southward et al., 2019). Cognitive reappraisal is more effectively utilized in predictable and calm situations than unpredictable contexts (Imburgio & MacNamara, 2019). It may be that those higher in trait anger utilize cognitive reappraisal in contexts that are not highly emotionally arousing and when cognitive control resources are more easily accessible to them, but tend to use maladaptive strategies (i.e., suppression) in situations that involve intense negative emotions.

Last, IL6 and TNF-α were not predicted by either suppression or reappraisal. Prior research has ubiquitously transformed inflammation data, which alters the structural relationships between variables in the model and inflammation (Becker et al., 2019; Feng et al., 2014). The present study did not transform biomarker data for several reasons. First, we chose to maintain the natural distribution of inflammatory biomarkers that represents the general population. Transformed distributions that do not exist in the real world greatly reduce generalizability and threaten external validity. Second, nonlinear transformations such as log-transformations pose threats to validity, because it is unclear what log-transformed inflammation values theoretically represent (Becker et al., 2019; Feng et al., 2014). Third, nonlinear transformations change the characteristics of distributions and may increase Type II error. Therefore, our operationalization of inflammatory biomarkers produces more accurate results, but due to the use of nontransformed data, our study may not demonstrate the same relationships as previous studies that used log-transformed biomarker values. Another explanation is that inflammatory processes involve the simultaneous activation of pro- and antiinflammatory systems, and different inflammatory biomarkers can be influenced by a variety of physiological processes. For example, IL6 and TNF-α levels may be suppressed by cortisol, while CRP production can remain elevated through the upregulation of antiinflammatory processes (Del Giudice & Gangestad, 2018; Garbers et al., 2012). We were unable to account for the effects of cortisol, thus it is possible that suppression leads to the increase of pro-inflammatory biomarkers but we were unable to demonstrate relationships due to antiinflammatory responses that follow elevated levels of pro-inflammatory biomarkers.

Limitations and Future Direction

One of the main limitations of the study is that it had substantial proportions of missing data, specifically, 63.5% on the ERQ and 60% on MIDUS 3 biomarker inflammation data. While we used modern missing data analysis methods, future research should endeavor on designs that increase sample size to replicate our findings. Several parameters in the model demonstrated meaningful effect sizes, but the standard errors were somewhat large, which may be a function of missing data that could not be sufficiently addressed by modern methods (Kang, 2013). Second, our sample is a primarily White, middle class sample which limits generalizability to other populations. Prior research has demonstrated that racial minorities and populations of lower socioeconomic status are at increased risk for inflammation (Lam et al., 2021). Further, we did not use poststratification weights provided in the MIDUS for our sample and our sample is not nationally representative. Third, we used retrospective reports of childhood abuse and neglect, and results need to be replicated with studies utilizing prospective or multiinformant reports, which provide unique information (Hardt & Rutter, 2004). Fourth, we only examined the impacts of emotional suppression and cognitive reappraisal, but other emotion regulation strategies may also influence inflammation. Future studies should consider other emotion regulation strategies, such as avoidance, rumination, problem-solving responses, mindfulness, and the ability to identify, accept, and flexibly respond to emotions (Gratz & Roemer, 2004). Fifth, COVID-19 interrupted the data collection process in the MIDUS 3 biomarker for a small percentage of participants, so findings may be subject to Type 1 or 2 errors due to the period effect of COVID-19. Last, the present study was a between-person investigation and a within-person design could provide additional information, for example, associations between emotion regulation strategies and inflammatory biomarkers may be significant at the within-person level (Renna, 2021).

Conclusion

Experiences of childhood neglect, but not abuse, appear to initiate to a psychological cascade that results in elevated CRP. Neglect leads to raised levels of trait anger which, in turn, increases emotional suppression. Those higher in trait anger may benefit from interventions aimed at effective emotion regulation. Maladaptive emotion regulation, such as suppression, can be a risk factor for elevated inflammatory biomarkers.

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