HHS Public Access

Author manuscript

Psychoneuroendocrinology. Author manuscript; available in PMC 2021 September 08.

Published in final edited form as:

Psychoneuroendocrinology. 2020 December ; 122: 104871. doi:10.1016/j.psyneuen.2020.104871.

Suppression, but not reappraisal, is associated with inflammation in trauma-exposed veterans

A.J. Khan[a,b,*], **A. O'Donovan**[a,b], **T.C. Neylan**[a,c], **J.J. Gross**[d], **B.E. Cohen**[a,c]

[a]San Francisco Veterans Affairs Health Care System, San Francisco, CA, United States

[b]Department of Psychiatry, University of California, San Francisco, CA, United States

[c]School of Medicine, University of California, San Francisco, CA, United States

[d]Department of Psychology, Stanford University, Stanford, CA, United States

Abstract

Background: Emotion dysregulation can elicit inflammatory activity. The current study examined whether specific maladaptive and adaptive emotion regulation strategies were associated with inflammatory markers in trauma-exposed veterans, above and beyond PTSD.

Methods: In a cohort study, 606 participants exposed to a Criterion A trauma and recruited from Veteran Health Administration facilities completed fasting blood draws, the Emotion Regulation Questionnaire, and the Clinician Administered PTSD Scale-IV. Inflammation was assessed with high sensitivity C-reactive protein (hsCRP), white blood cell count (WBC), and fibrinogen levels. An inflammation index was created by summing standardized log-transformed levels of the three biomarkers. Our primary linear regression models were adjusted for sex, age, race, education, income, creatinine, and PTSD.

Results: Suppression, but not cognitive reappraisal, was significantly associated with higher levels of the inflammatory index ($\beta = 0.14$, $p = 0.001$). Parallel analyses for the individual inflammatory markers also showed suppression, but not reappraisal, was significantly associated with higher hsCRP ($\beta = 0.11$, $p = 0.01$), WBC ($\beta = 0.11$, $p = 0.01$), and fibrinogen ($\beta = 0.10$, $p = 0.02$).

Conclusions: Emotional suppression is related to elevated systemic inflammation independent of PTSD. Cognitive reappraisal is unrelated to inflammation. Findings suggest over-utilization of maladaptive, rather than underutilization of adaptive, emotion regulation strategies may be associated with systemic inflammation in trauma-exposed veterans.

[*]Corresponding author at: San Francisco VA Health Care System 4150 Clement Street, San Francisco, CA, 94121, United States. Amanda.Khan@va.gov (A.J. Khan).

Declaration of Competing Interest

The authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.psyneuen.2020.104871.

Keywords

Emotion regulation; Inflammation; Trauma; PTSD; Veterans; Immunology

1. Introduction

Most people will experience a traumatic event in their lifetime (e.g., McLaughlin et al., 2013). Trauma exposure can change individuals' perceptions of themselves, how they relate to others, and their reactivity and responsivity to the environment (Badour and Feldner, 2013; Scher et al., 2017). Although trauma exposure can lead to posttraumatic growth, it can also lead to posttraumatic stress sequelae including posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) (Cloitre et al., 2005; O'Donnell et al., 2004). The mediators involved in the pathway from trauma exposure to psychiatric illness involve both psychological and biological mechanisms (King et al., 1999; Roth, 2014). Emotion regulation and inflammation are two such mechanisms (e.g., Ehring and Quack, 2010; O'Donovan et al., 2017), but little is known about their associations with one another in trauma-exposed populations.

Emotion regulation (ER) refers to the set of processes involved in changing the onset, duration, intensity, and/or type of emotion elicited by a stimulus (Gross, 1999). ER is consistently found to be involved in the etiology and maintenance of trauma sequelae (see Ehring and Quack, 2010). For example, above and beyond the effects of negative affect (Bradley et al., 2011), so-called "maladaptive" strategies such as expressive suppression are implicated in the etiology of PTSD (Ehring and Quack, 2010). Specifically, individuals with PTSD may over-utilize maladaptive strategies and under-utilize more effective "adaptive" strategies (Boden et al., 2013) such as cognitive reappraisal, which entails changing one's thoughts about the meaning of a stimulus (Gross, 1998). However, research has demonstrated stronger associations between maladaptive ER strategies and psychopathology including PTSD than adaptive ER strategies (Aldao et al., 2010; Lee et al., 2015; Seligowski et al., 2015). Suppression, or the active inhibition of emotion-expressive behavior (Gross, 1998), is theorized to contribute to the development and maintenance of PTSD by inhibiting emotional processing (Foa and Kozak, 1986; Sijbrandij et al., 2013). Suppression also has physiologic consequences, often causing increased sympathetic activation (e.g., Gross and Levenson, 1997). Sustained activation of the sympathetic nervous system (SNS) may not only exacerbate PTSD symptoms, it can also increase inflammatory activity (Michopoulos et al., 2017).

Inflammation has been identified as a pathophysiologic mechanism involved in the etiology and maintenance of both mental and physical health sequelae of trauma (Baumeister et al., 2016; Passos et al., 2015; Sumner et al., 2019; Tursich et al., 2014). Trauma exposure and PTSD are associated with elevated markers of systemic inflammation including high-sensitivity C-reactive protein (hsCRP), white blood cell count (WBC), and fibrinogen (Danese et al., 2007; O'Donovan et al., 2017). More broadly, pervasive emotion dysregulation and chronic psychosocial stress are consistently associated with inflammation (e.g., Crowell et al., 2015; Hansel et al., 2010). However, it is largely unclear if and to what

extent specific emotion regulation *strategies* play a role in inflammation in trauma-exposed samples.

Few studies have investigated the relationship between specific ER strategies and inflammation. Rather, the majority of the scant research examining this relationship has instead focused on emotional dysregulation in general. For example, in a longitudinal study in children, difficulties in emotional self-regulation predicted higher C-reactive protein (CRP) concentrations in middle adulthood (Appleton et al., 2011). In a cross-sectional study of African American women with Type-2 diabetes, difficulty regulating emotions was associated with elevated CRP independent of body mass index (BMI), PTSD, and MDD (Powers et al., 2016). To our knowledge, only one prior study has examined the relationship between specific ER strategies and inflammation. In a cross-sectional study of community-dwelling adults, Appleton et al. (2013) found expressive suppression was associated with elevated CRP whereas cognitive reappraisal was associated with significantly lower levels of CRP. While these findings provide important preliminary evidence, additional assessment of the relationship between specific ER strategies and other indices of inflammation is needed. Most importantly, it remains unknown if these findings extend to trauma-exposed samples (i.e., a risk factor for maladaptive ER and elevated inflammation) and whether this relationship remains after accounting for psychiatric diagnosis.

The current study sought to examine the relationship between expressive suppression and cognitive reappraisal with inflammation in trauma-exposed Veteran Health Administration (VHA) patients. In the study by Appleton et al. (2013) described above, the effect size for reappraisal was smaller than for suppression. Moreover, maladaptive ER use is consistently more strongly associated with psychiatric illness than adaptive strategies (Aldao et al., 2010). Thus, we hypothesized that expressive suppression, but not cognitive reappraisal, would be associated with inflammation, above and beyond the effects of biological covariates and PTSD diagnosis. Specifically, we expected greater use of suppression to be positively associated with inflammation.

2. Method

2.1. Participants and procedures

The Mind Your Heart Study (MYH) is a prospective cohort study investigating the long-term relationship between physical health outcomes and PTSD in VHA patients. The current study used cross-sectional baseline data that was collected from 746 MYH participants between February 2008 and June 2010. Participants were recruited from two Northern California VA Health Care Systems. In line with primary study aim, VHA patients with a PTSD diagnosis were intentionally over-sampled. Participants were recruited through three methods: via flyers posted in VA clinics, provider referrals, and mailed letters sent to patients who were seen in medical clinics within the past five years and received International Classification of Diseases (9th revision; ICD-9) codes for PTSD diagnosis, as well as to patients of a similar-age also seen in the medical clinics but without a PTSD diagnosis. Exclusion criteria included inability to walk one block or myocardial infarction within the prior six months due to cardiac testing performed at baseline. Patients were also excluded if they lacked a stable living address or had plans to move in the next three years.

Given the primary aim to examine ER and inflammation in a trauma-exposed sample, further inclusion criterion for primary analyses was exposure to a Criterion A trauma as defined by DSM-IV criteria for PTSD (American Psychiatric Association, 2000); of the participants, 94 veterans did not experience a criterion A trauma. In-person interviews and self-report questionnaires were conducted and blood draws were performed, all at VHA facilities. If participants reported acute illness, appointments were rescheduled. All participants provided written informed consent. The study was approved by the Institutional Review Board at the University of California and the San Francisco VA Health Care System Research and Development Committee.

2.2. Measures

2.2.1. Demographics—All demographic data (i.e., sex, age, race, ethnicity, education, combined household past year income, marital status, employment status) and military variables (e.g., branch of service, war zone exposure, rank, deployment data, time served) were collected via self-report questionnaires. Because of changes in study protocol, military variables were only collected at baseline for the last 145 participants and during second year follow-up by telephone interview for participants that were still retained in the study at that point (see Table 1 for full demographics including missing data percentages).

2.2.2. Diagnoses—Current (past month) and lifetime PTSD diagnostic criteria and severity was assessed using the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995) based on the criteria for Diagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV-TR; American Psychiatric Association, 2000). The CAPS is the gold standard diagnostic interview for PTSD and measures the frequency (0 = never or none, 4 = almost daily, more than 80 % of the time) and severity (0 = none, 4 = extreme) of each symptom on a four-point scale. The CAPS demonstrates excellent test-reliability and internal consistency (Weathers et al., 2001). All interviews were conducted by master's level clinicians under the supervision of a licensed clinical psychologist with PTSD assessment expertise. The PTSD group included individuals with either full PTSD or partial PTSD, which is associated with significant impairment in functioning (Weathers et al., 2001). Of the three DSM-IV symptom clusters, re-experiencing represents the most conceptually distinct cluster from other psychiatric illnesses and thus partial PTSD participants were required to meet full criteria for this cluster. Partial PTSD participants were required to meet diagnostic threshold for one other cluster, in addition to the other CAPS criteria as well as the lower PTSD threshold total severity score ≥ 40 (Weathers et al., 2001). This approach allowed for inclusion of patients who had qualifying traumatic events and substantial PTSD symptoms, but missed exact diagnostic criteria because of their symptom distribution. Based on these criteria, individuals were categorized into two groups: Current PTSD or No Current PTSD. Of the 246 veterans meeting criteria for Current PTSD, 19 (7.72 %) met criteria for only partial PTSD.

2.2.3. Emotion regulation questionnaire—The Emotion Regulation Questionnaire (ERQ; Gross and John, 2003) is a 10-item self-report assessment of the typical use of expressive suppression (4 items) and cognitive reappraisal (6 items). Both expressive suppression (ES) and cognitive reappraisal (CR) items assess ER strategy use for both

positive emotions and negative emotions. Respondents rate how much they agree with each item on a seven-point Likert scale (1 = strongly disagree, 7 = strongly agree), with higher scores indicating greater use of the respective ER strategy. Items are summed to create a total score (range: 7–70) and two orthogonal subscale scores (ES range: 4–28; CR range: 6–42). The ERQ is widely used in psychopathology research and demonstrates good reliability and convergent validity (Gross and John, 2003). Internal consistency in the current sample was good (expressive suppression $\alpha = 0.72$, cognitive reappraisal $\alpha = .83$).

2.2.4. Inflammatory markers—Participants provided fasting morning venous blood samples to measure levels of three widely-used biomarkers of inflammation (Pearson et al., 2003). HsCRP, an acute phase inflammatory protein, was measured using a BNII nephelometer (Siemens Health Care Diagnostics, Tarrytown, New York) with interassay coefficients of variation < 5 %. WBC count, or the number of WBC per blood volume, was determined using the Beckman Coulter LH 750 analyzer (Beckman Coulter, Inc., California) with an interassay coefficient of variation < 2 %. Finally, for fibrinogen, a clotting protein, serum concentrations were obtained by the Clauss assay with coefficients of variation < 3 %.

2.2.5. Covariates—Participants completed several self-report questionnaires to determine health behaviors that may additionally impact inflammation levels (Cohen et al., 2009). Tobacco use was assessed using an item asking participants whether they currently smoke cigarettes. The Alcohol Use Disorders Identification Test-consumption (AUDIT-C; Bush et al., 1998) was used to identify possible problematic alcohol use, using the recommended cut-off scores of 3 for women and 4 for men. Physical activity was assessed using an item asking participants how often they engaged in physical activity (defined as at least 15–20 min of brisk walking, swimming, general conditioning, or recreational sports) over the past month on a 6-point Likert scale (0 =Not at all active, 5 = Extremely active (5 or more times a week)). In line with prior studies (e.g., Cohen et al., 2009), activity level was dichotomized as physically inactive (Likert scores 0–1) and physically active (Likert scores 2–5). This has shown to be a reliable methodology for assessing physical activity (Jackson et al., 2007). Using an item from the Pittsburgh Sleep Quality Index (Buysse et al., 1989), participants rated their subjective overall sleep quality for the past month on a 5-point Likert scale (4 = very bad, 0 = very good). Sleep quality was dichotomized (good = 0, poor = 1) as poor sleep (Likert scores 3–4) and good sleep (Likert scores 0–2). Body mass index (BMI) was also assessed by a trained technician and calculated by the participants' weight divided by the square of their height (kg/m^2). Kidney function was also assessed as elevations in inflammatory markers can be associated with impaired renal function. Serum creatinine levels were obtained through fasting morning venous blood samples. Self-reported depression severity was assessed using the 9-item Patient Health Questionnaire (PHQ-9; Spitzer et al., 1999).

Additionally, history of health conditions and disease events were assessed through self-report. Participants provided yes/no responses to whether a doctor or nurse had diagnosed them with the condition. The conditions were then grouped into eight overarching disease taxonomies: cardiovascular (heart attack, angina, congestive heart failure, other heart disease or stroke), diabetes, liver (hepatitis, cirrhosis, or liver disease), renal, AIDS/HIV, chronic

lung disease (obstructive lung disease or other lung disease), neurological (Parkinson's, dementia, other neurologic disease), and cancer. Dummy variables were created for each participant based on endorsement of at least one disorder within each category. A medical comorbidity index severity score was then created summing the counts across categories.

2.3. Statistical analyses

Several preliminary analyses were conducted to exclude invalid data and to account for missing data in variables of interest. Participants with missing, incomplete, or invalid CAPS and ERQ data were excluded from analyses (N = 22). For all inflammatory analyses, participants with any missing inflammatory markers were also excluded (N = 12). The final Criterion A trauma-exposed sample used for all analyses was 606 veterans.

Preliminary analyses were conducted to determine descriptive statistics and frequencies for demographic variables. For all analyses, hsCRP, WBC count, and fibrinogen were log-transformed to produce normal distributions. Using a previously published method to create an inflammatory index (O'Donovan et al., 2012), log-transformed inflammatory markers were standardized into z-scores and summed. Bivariate Pearson correlations were conducted to determine associations between each ER strategy and inflammatory index (primary outcome) and individual biomarkers (secondary outcomes). Partial correlations were also performed to examine associations adjusting for PTSD severity.

Two multiple linear regressions were then conducted to examine the association between expressive suppression and cognitive reappraisal and the inflammatory index; the first regression adjusted for sociodemographic and biological covariates (age, sex, ethnicity, education, income, and creatinine) and the second regression adjusted additionally for PTSD diagnostic status. Six parallel multiple linear regressions were performed to test for the association between emotion regulation strategies and each inflammatory biomarker separately (hsCRP, WBC, fibrinogen), adjusting first for biological covariates only then in addition to PTSD. For all regressions, sex (Male = 0, Female = 1), race (White = 0, Racial/Ethnic Minority = 1), annual household income (<$20,000 = 0, ≥$20,000 = 1), and PTSD diagnostic status (No Current PTSD = 0, Current PTSD = 1) were dummy coded. To examine the potential role of health behavior mediators, sensitivity analyses were conducted further adjusting for additional covariates that were associated with inflammatory biomarkers at $p < .05$. All analyses were run using SPSS, Version 22.

3. Results

3.1. Demographics and preliminary analyses

The sample identified primarily as male (93.7 %), Non-Hispanic (89.3 %), and White (58.9 %) with an average age of 58.0 years (*SD* = 11.2; see Table 1 for full demographics). Approximately 41 % of trauma-exposed veterans met for current PTSD (n = 246). Consistent with prior research (e.g., Gross and John, 2003), suppression and reappraisal were not significantly correlated with each other ($r = .01$, p = 0.82), confirming these are independent ER strategies. Bivariate correlations revealed expressive suppression was significantly positively associated with CAPS past month total severity score ($r = .30$,

$p < .001$). Expressive suppression was also significantly positively associated with the inflammatory index ($r = .17$, $p < .001$) and all inflammatory biomarkers (hsCRP $r = .12$, $p = .003$; WBC $r = .12$, $p = .005$; fibrinogen $r = .15$, $p < .001$). Adjusting for PTSD severity, suppression remained significantly positively correlated with inflammatory index ($r = .14$, $p < .001$) and all inflammatory biomarkers (hsCRP $r = .11$, $p = .01$; WBC $r = .09$, $p = .02$; fibrinogen $r = .13$, $p = .002$).

Cognitive reappraisal was significantly negatively associated with CAPS total severity scores although correlations were very small ($r = -.09$, $p = .02$). Cognitive reappraisal was not significantly correlated with any inflammatory biomarkers or the inflammatory index. Partial correlations and adjustment for PTSD severity revealed a similar pattern. See Table 2 for bivariate Pearson correlations between sociodemographic and health behavior variables with emotion regulation and inflammation markers.

3.2. Primary outcome

Adjusting for sex, age, race, education, income, and creatinine ($R = 0.29$, $F(8, 581) = 6.40$, $p < .001$), multiple linear regression revealed expressive suppression, but not cognitive reappraisal, was associated with significantly higher levels of the inflammatory index ($\beta = 0.15$, $p < 0.001$). After additionally adjusting for PTSD diagnostic status (see Table 3 for fully adjusted models, ($R = 0.29$, $F(9, 580) = 5.97$, $p < .001$))[1], expressive suppression, but not cognitive reappraisal, remained significantly positively associated with inflammatory index ($\beta = 0.14$, $p = 0.001$).

Subsequent parallel analyses revealed a similar pattern across all inflammatory biomarkers. When adjusting for sociodemographic and biological covariates only, expressive suppression was associated with significant increases in hsCRP ($R = 0.23$, $F(8, 581) = 3.80$, $p < .001$; $\beta = 0.11$, $p = 0.006$), WBC ($R = 0.19$, $F(8, 581) = 2.50$, $p = .01$; $\beta = 0.12$, $p = 0.003$), and fibrinogen ($R = 0.27$, $F(8, 581) = 5.71$, $p < .001$; $\beta = 0.10$, $p = 0.01$). After further adjusting for PTSD (Table 2)[2], expressive suppression remained significantly positively associated with elevations in hsCRP ($R = 0.23$, $F(9, 580) = 3.64$, $p < .001$; $\beta = 0.11$, $p = 0.01$), WBC ($R = 0.18$, $F(9, 580) = 2.19$, $p = .02$; $\beta = 0.11$, $p = 0.02$), and fibrinogen ($R = 0.27$, $F(9, 580) = 5.16$, $p < .001$; $\beta = 0.10$, $p = 0.03$).

3.3. Sensitivity analyses

Five separate sensitivity analyses were performed (see Table 4 and Supplementary Table 1). First, fully adjusted linear regressions (i.e., sex, age, race, education, income, creatinine, PTSD) were repeated with additional adjustment for health behaviors (physical activity, problematic drinking, smoking, BMI, poor sleep), all of which were significantly associated with at least one inflammatory marker at $p < .05$. Results, although attenuated, remained largely unchanged. Specifically, suppression, not reappraisal, remained significantly

[1]This pattern of results remained the same when repeated with CAPS past month total severity. Overall model was significant ($F(9, 580) = 5.90$, $p < .001$) and suppression, but not reappraisal remained a significantly positively associated with inflammatory index ($\beta = 0.13$, $p = 0.002$).

[2]Although slightly attenuated, patterns of results remained the same when repeated with CAPS past month total severity. Suppression, but not reappraisal remained significantly positively associated with hsCRP ($\beta = 0.10$, $p = 0.017$), WBC ($\beta = 0.10$, $p = 0.02$), and fibrinogen ($\beta = 0.09$, $p = 0.035$).

associated with inflammatory index (β = 0.11, p = 0.007), WBC (β = 0.11, p = 0.01), and fibrinogen (β = 0.08, p = 0.046). However, the effect of suppression on hsCRP was no longer significant (β = 0.06, p = 0.15). Second, fully adjusted linear regressions were also repeated omitting veterans with hsCRP > 10 mg/L (N = 23) in order to rule out the possibility of inflammation due to acute infection or injury. Suppression, but not reappraisal, continued to be positively associated with inflammation index (β = 0.11, p = 0.008), hsCRP (β = 0.09, p = 0.03), and fibrinogen (β = 0.08, p = 0.05), however the effect on WBC only approached significance (β = 0.08, p = 0.08). Third, fully adjusted linear-regression models were repeated excluding participants taking daily or as needed oral corticosteroids or immunosuppressants (N=11). Analyses revealed similar results; suppression, not reappraisal, remained significantly associated with inflammatory index (β = 0.14, p = 0.001), hsCRP (β = 0.09, p = 0.02), WBC (β = 0.11, p = 0.008), and fibrinogen (β = 0.11, p = 0.01). Additionally, given overlap of depression and PTSD in the current sample, linear regressions were repeated adjusting for depression severity in addition to sociodemographic variables. Results revealed a similar pattern; suppression, but not reappraisal, was significantly positively associated with inflammatory index (β = 0.14, p = 0.002), hsCRP (β = 0.09, p = 0.037), WBC (β = 0.12, p = 0.006), and fibrinogen (β = 0.10, p = 0.02). Depression severity was not significantly associated with inflammation index, WBC, or fibrinogen (ps > 0.05), but was significantly associated with hsCRP (β = 0.09, p = 0.05), Finally, fully adjusted linear regressions were also repeated adjusting for comorbid medical disease severity score. The pattern of findings was similar; suppression, but not reappraisal was significantly associated with inflammatory index (β = 0.14, p = 0.001), hsCRP (β = 0.10, p = 0.01), WBC (β = 0.11, p = 0.01), and fibrinogen (β = 0.10, p = 0.02).

4. Discussion

In this large cohort of trauma-exposed veterans, we found that expressive suppression, but not cognitive reappraisal, was associated with inflammation independent of PTSD. Greater use of suppression was associated with higher hsCRP, WBC, and fibrinogen. These findings demonstrate a relationship between two recognized risk factors for PTSD, maladaptive emotion regulation and systemic inflammation. Avoidance-related ER strategies have a stronger relationship with psychiatric illness than do approach-related strategies (Aldao et al., 2010) and the current study extends these findings to inflammatory markers.

Our hypothesis that expressive suppression would be significantly positively associated with inflammation was supported. In fully adjusted models, a one standard deviation increase in suppression was associated with a 14 % increase in inflammatory index and 10–11 % increases in hsCRP, WBC, and fibrinogen, respectively. This finding is in line with the only other study examining suppression and CRP (Appleton et al., 2016) and effect sizes in fully adjusted models, although modest, were similar suggesting emotional suppression is associated with systemic inflammation. Our findings also build on this foundational work by examining these relationships in a trauma-exposed sample of veterans, adjusting for PTSD, and demonstrating the relationship of specific ER strategies with other markers of systemic inflammation including WBC and fibrinogen.

Our hypothesis that cognitive reappraisal would not be significantly associated with inflammation was supported. This finding is in contrast to the results reported by Appleton et al. (2016). This difference may at least be partially explained by differences in samples; they utilized a community-dwelling sample of civilians whereas the current sample was comprised of veterans exposed to a Criterion A trauma. However, Appelton and colleagues reported that unlike suppression, reappraisal was no longer significantly related to CRP after accounting for BMI. Similarly, we found suppression was no longer associated with hsCRP after adjusting for BMI, problematic drinking, smoking, physical activity, and poor sleep. These findings indicate that health behaviors may play a role in the relationship between ER and CRP. Longitudinal analyses will be needed to test this hypothesis.

Collectively, the current findings suggest that for trauma-exposed individuals, greater use of maladaptive emotion regulation (ER) strategies is associated with elevated inflammation. This finding is above and beyond the effects of PTSD, thus maladaptive ER use is associated with inflammation even after accounting for negative affect. In contrast, greater use of adaptive ER strategies is not associated with lower inflammation, suggesting their use may not confer a protective effect. This pattern is consistent with research showing stronger associations between maladaptive ER strategies and psychopathology than adaptive ER (Aldao et al., 2010), between social strain and inflammation than social support (Yang et al., 2015), and between pessimism and inflammation than optimism (O'Donovan et al., 2009). These findings highlight the potency of negative *intra*personal and *inter*personal regulation processes on the mind and body (Hofmann, 2014).

Suppression, although directed at outward emotional expression, causes a rebound effect leading to both increased PTSD symptoms and greater SNS activation (e.g., Gross and Levenson, 1997). Continued over-reliance on suppression may thus perpetuate inflammation and allostatic load through stress-induced SNS activation (Crowell et al., 2015; Vumma et al., 2017). In turn, elevated inflammation may influence functioning of both the brain and peripheral bodily systems via a cascade of effects on neurotransmitter metabolism, neuroendocrine function, catecholamines, and parasympathetic outflow pathways (Elenkov et al., 2000; Michopoulos et al., 2017; Miller et al., 2009).

Suppression is also associated with significant psychosocial costs. Specifically, suppression is associated with a stronger sense of inauthenticity, greater disruptions to social relationships, fewer social supports, and less interpersonal closeness (e.g., Butler et al., 2003; Gross and John, 2003). These psychosocial consequences of suppression may also promote inflammation either through increases in perceived stress and its biological byproducts or through the removal or absence of protective factors that can serve as a buffer (e.g., Cole et al., 2015). Although longitudinal research is needed, targeting over-reliance on suppression and other avoidance-related strategies to regulate negative emotions may help prevent acute inflammatory states following trauma from becoming chronic. It will be critical for future research to determine if maladaptive emotion regulation and inflammation are sequential or concurrent, perhaps transactional, mediators of PTSD and other trauma sequelae.

5. Limitations

Although the current study has several strengths including a large sample size, the use of a structured interview to determine PTSD diagnosis and criterion A trauma exposure, adjustment for numerous covariates, and incorporation of multiple sensitivity analyses, several important limitations should be noted. First, the current study utilized a cross-sectional design and therefore causality cannot be inferred between inflammation and ER strategy. Second, ER, sleep, problematic drinking, and physical activity were assessed using a self-report measure, which may be susceptible to biases. ER strategy use was also limited to just two strategies, which precluded investigating other important strategies such as rumination and acceptance. Similarly, only three inflammatory markers were assessed, all of which were peripheral. Future research should examine how ER is related to the numerous other mediators involved in systemic inflammation including such as tumor necrosis factor-α. Because this study began in 2008, PTSD was also assessed based on DSM-IV criteria and therefore the current findings may differ when using DSM-5 criteria for PTSD.

Additionally, the current sample was comprised entirely of VHA patients, limiting the generalizability to other military personnel. Also, the majority of the sample identified as male and White, limiting the generalizability of the current findings to women and racial/ethnic minorities. Although the current sample did include a small sample of women, we had insufficient power to explore gender differences. Future research investigating how gender impacts associations between different ER strategies and inflammation is important, given variations in hormonal functioning, trauma exposure, and socialization. Finally, this study focused on PTSD and thus psychiatric comorbidities other than PTSD and depression were not adjusted for and may have influenced inflammation levels. Further, our pattern of findings remained the same when adjusting for comorbid medical disease severity. However, these health conditions were gathered via self-report and therefore the exact influence of emotion regulation in the context of chronic medical conditions remains unclear.

6. Conclusions

Emotional suppression has deleterious effects on psychological and social functioning. In a large cohort of trauma-exposed veterans, suppression was associated with elevations in systemic inflammation beyond the effects of PTSD. However, cognitive reappraisal was not related to any marker of systemic inflammation, suggesting it may not serve as a protective factor in decreasing pro-inflammatory states. Maladaptive ER strategies may thus have a stronger effect on systemic inflammation than adaptive strategies. Future research is needed to better understand the complex, dynamic relationship between psychological and biological risk factors for disease development and course following trauma exposure.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We wish to thank the Mind Your Heart Study participants for their continued participation in this project and gratefully acknowledge the contributions of Dr. Mary Whooley, the Mind Your Heart Study staff, and co-investigators.

Role of funding source

This work was supported by the Northern California Institute for Research and Education (W81XWH-11-2-0189), the Department of Defense (W81XWH-11-2-0189), the National Heart Lung and Blood Institute (K23 HL 094765-0), the Irene Perstein Foundation, the American Heart Association (AHA: 12CRP11810021), and departmental funds from the University of California, San Francisco(UCSF: 111583A). AK is supported by the VA Office of Academic Affiliations in conjunction with the VA Advanced MIRECC Fellowship. AOD is supported by a U.S. National Institute of Mental Health K01 Career Development Award (K01MH109871). These funding sources did not have any role in the conduct of the research or the preparation of this article. The views expressed in this article are those of the authors and do not necessarily reflect the views of the Department of Veterans Affairs or the United States Government.

References

Aldao A, Nolen-Hoeksema S, Schweizer S, 2010. Emotion regulation strategies across psychopathology: a meta-analysis. Clin. Psychol. Rev. 30, 217–237. [PubMed: 20015584]

American Psychiatric Association, 2000. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. American Psychiatric Publishing, Arlington, VA.

Appleton AA, Buka SL, McCormick MC, Koenen KC, Loucks EB, Gilman SE, Kubzansky LD, 2011. Emotional functioning at age 7 years is associated with C-reactive protein in middle adulthood. Psychosom. Med. 73 (4), 295–303. [PubMed: 21536835]

Appleton AA, Buka SL, Louchs SE, Gilman SE, Kubzansky LD, 2013. Divergent associations of adaptive and maladaptive emotion regulation strategies with inflammation. Health Psychol. 32 (7), 748–756. [PubMed: 23815767]

Badour CL, Feldner MT, 2013. Trauma-related reactivity and regulation of emotion: associations with posttraumatic stress symptoms. J. Behav. Ther. Exp. Psychiatry44 (1), 69–76. [PubMed: 22922079]

Baumeister D, Akhtar R, Ciufolini S, Pariante CM, Mondelli V, 2016. Childhood trauma and adulthood inflammation: a meta-analysis of peripheral C-reactive protein, interleukin-6 and tumour necrosis factor-α. Mol. Psychiatry21, 642–649. [PubMed: 26033244]

Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, Keane TM, 1995. The development of a clinician-administered PTSD scale. J. Trauma. Stress8 (1), 75–90. [PubMed: 7712061]

Boden MT, Westermann S, McRae K, Kuo J, Alvarez J, Kulkarni MR, Gross JJ, Bonn-Miller MO, 2013. Emotion regulation and posttraumatic stress disorder: a prospective investigation. J. Soc. Clin. Psychol. 32 (3), 296–314.

Bradley B, DeFife J, Guarnaccia C, Phifer J, Fani N, Ressler K, 2011. Emotion dysregulation and negative affect: association with psychiatric symptoms. J. Clin. Psychiatry72, 685–691. [PubMed: 21658350]

Bush K, Kivlahan DR, McDonell MB, Fihn SD, Bradley KA, 1998. The AUDIT alcohol consumption questions (AUDIT-C): an effective brief screening test for problem drinking. Ambulatory Care Quality Improvement Project (ACQUIP). Alcohol Use Disorders Identification Test. Arch. Intern. Med. 158 (16), 1789–1795. [PubMed: 9738608]

Butler EA, Egloff B, Wilhelm FH, Smith NC, Erickson EA, Gross JJ, 2003. The social consequences of expressive suppression. Emotion3 (1), 48–76. [PubMed: 12899316]

Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ, 1989. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. J. Psychiatr. Res. 28 (2), 193–213.

Cloitre M, Miranda R, Stovall-McClough KC, Han H, 2005. Beyond PTSD: emotion regulation and interpersonal problems as predictors of functional impairment in survivors of childhood abuse. Behav. Ther. 36 (2), 119–124.

Cohen BE, Marmar CR, Neylan TC, Schiller NB, Ali S, Whooley MA, 2009. Posttraumatic stress disorder and health-related quality of life in patients with coronary heart disease: findings from the Heart and Soul Study. Arch. Gen. Psychiatry66, 1214–1220. [PubMed: 19884609]

Cole SW, Capitanio JP, Chun K, Arevalo JMG, Ma J, Cacioppo JT, 2015. Myeloid differentiation architecture of leukocyte transcriptome dynamics in perceived social isolation. PANAS112 (49), 15142–15147.

Crowell SE, Puzia ME, Yaptangco M, 2015. The ontogeny of chronic distress: emotion dysregulation across the life span and its implications for psychological and physical health. Curr. Opin. Psychol. 3, 91–99.

Danese A, Pariante CM, Caspi A, Taylor A, Poulton R, 2007. Childhood maltreatment predicts adult inflammation in a life-course study. PNAS104 (4), 1319–1324. [PubMed: 17229839]

Ehring T, Quack D, 2010. Emotion regulation difficulties in trauma survivors: the role of trauma type and PTSD symptom severity. Behav. Ther. 41, 587–598. [PubMed: 21035621]

Elenkov IJ, Wilder RL, Chrousos GP, Vizi ES, 2000. The sympathetic nerve—an integrative interface between two supersystems: the brain and the immune system. Pharmacol. Rev. 52 (4), 595–638. [PubMed: 11121511]

Foa EB, Kozak MJ, 1986. Emotional processing of fear: exposure to corrective information. Psychol. Bull. 99, 20–35. [PubMed: 2871574]

Gross JJ, 1998. Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. J. Pers. Soc. Psychol. 74 (1), 224–237. [PubMed: 9457784]

Gross JJ, 1999. Emotion regulation: past, present, future. Cogn. Emot. 13 (5), 551–573.

Gross JJ, John OP, 2003. Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. J. Pers. Soc. Psychol. 85, 348–362. [PubMed: 12916575]

Gross JJ, Levenson RW, 1997. Hiding feelings: the acute effects of inhibiting negative and positive emotion. J. Abnorm. Psychol. 106, 95–103. [PubMed: 9103721]

Hansel A, Hong S, Camara RJ, von Kanel R, 2010. Inflammation as a psychophysiological biomarker in chronic psychosocial stress. Neurosci. Biobehav. Rev. 35 (1), 115–121. [PubMed: 20026349]

Hofmann SG, 2014. Interpersonal emotion regulation model of mood and anxiety disorders. Cognit. Ther. Res. 38 (5), 483–492.

Jackson AW, Morrow JR Jr., Bowles HR, Fitzgerald SJ, Blair SN, 2007. Construct validity evidence for single response items to estimate physical activity levels in large sample studies. Res. Q. Exerc. Sport78 (2), 24–31. [PubMed: 17479571]

King DW, King LA, Foy DW, Keane TM, Fairbank JA, 1999. Posttraumatic stress disorder in a national sample of female and male Vietnam veterans: risk factors, war-zone stressors, and resilience-recovery variables. J. Abnorm. Psychol. 108, 164–170. [PubMed: 10067002]

Lee DJ, Witte TK, Weather FW, Davis MT, 2015. Emotion regulation strategy use and posttraumatic stress disorder: associations between multiple strategies and specific symptom clusters. J. Psychopathol. Behav. Assess. 37, 533–544.

McLaughlin KA, Koenen KC, Hill ED, Petukhova M, Sampson NA, Zaslavsky AM, Kessler RC, 2013. Trauma exposure and posttraumatic stress disorder in a national sample of adolescents. J. Am. Acad. Child Adolesc. Psychiatry52 (8), 815–830.e14. [PubMed: 23880492]

Michopoulos V, Powers A, Gillespie CF, Ressler KJ, Jovanovic T, 2017. Inflammation in fear- and anxiety-based disorders: PTSD, GAD, and beyond. Neuropsychopharmacology42, 254–270. [PubMed: 27510423]

Miller AH, Maletic V, Raison CL, 2009. Inflammation and its discontents: the role of cytokines in the pathophysiology of major depression. Biol. Psychiatry65, 732–741. [PubMed: 19150053]

O'Donnell ML, Creamer M, Pattison P, 2004. Posttraumatic Stress Disorder and depression following trauma: understanding comorbidity. Am. J. Psychiatry161, 507–514. [PubMed: 14992977]

O'Donovan A, Lin J, Tillie J, Tillie JM, Dhabhar FS, Wolkowitz OM, Epel E, 2009. Pessimism correlates with leukocyte telomere shortness and elevated interleukin-6 in post-menopausal women. Brain Behav. Immun. 23 (4), 446–449. [PubMed: 19111922]

O'Donovan A, Neylan TC, Metzler T, Cohen BE, 2012. Lifetime exposure to traumatic psychological stress is associated with elevated inflammation in the Heart and Soul Study. Brain Behav. Immun. 26 (4), 642–649. [PubMed: 22366689]

O'Donovan A, Ahmadian AJ, Neylan TC, Pacult MA, Edmondson D, Cohen BE, 2017. Current posttraumatic stress disorder and exaggerated threat sensitivity associated with elevated inflammation in the Mind your Heart Study. Brain Behav. Immun. 60, 198–205. [PubMed: 27765647]

Passos IC, Vasconcelos-Moreno MP, Costa LG, Kunz M, Brietzke E, Quevedo J, Kauer-Sant'Anna M, 2015. Inflammatory markers in post-traumatic stress disorder: a systematic review, meta-analysis, and meta-regression. Lancet Psychiatry2, 1002–1012. [PubMed: 26544749]

Powers A, Michopoulos V, Conneely K, Gluck R, Dixon H, Wilson J, Gillespie CF, 2016. Emotion dysregualtion and inflammation in African-American women with type 2 diabetes. Neural Plasticity, 2016, 8926840. [PubMed: 27493807]

Roth TL, 2014. How traumatic experiences leave their signature on the genome: an overview of epigenetic pathways in PTSD. Front. Psychiatry5, 93. [PubMed: 25132824]

Scher CD, Suvak MK, Resick PA, 2017. Trauma cognitions are related to symptoms up to 10 years after cognitive behavioral treatment for posttraumatic stress disorder. Psychol. Trauma Theory Res. Pract. Policy9 (6), 750–757.

Seligowski AV, Lee DJ, Bardeen JR, Orcutt HK, 2015. Emotion regulation and posttraumatic stress symptoms: a meta-analysis. Cogn. Behav. Ther. 44 (2), 87–102. [PubMed: 25421727]

Sijbrandij M, Engelhard IM, Lommen MJ, Leer A, Baas JM, 2013. Impaired fear inhibition learning predicts the persistence of symptoms of posttraumatic stress disorder (PTSD). J. Psychiatr. Res. 47 (12), 1991–1997. [PubMed: 24090716]

Spitzer RL, Kroenke K, Williams JB, 1999. Validation and utility of a self-report version of PRIME-MD: the PHQ primary care study. JAMA282, 1737–1744. [PubMed: 10568646]

Sumner JA, Nishimi KM, Koenen KC, Roberts AL, Kubzansky LD, 2019. Posttraumatic stress disorder and inflammation: untangling issues of bidirectionality. Biol. PsychiatryS006–3223 (19), 31855–31864.

Tursich M, Neufeld RWJ, Frewen PA, Harricharan S, Kibler JL, Rhind SG, Lanius RA, 2014. Association of trauma exposure with proinflammatory activity: a transdiagnostic meta-analysis. Transl. Psychiatry4, e413. [PubMed: 25050993]

Vumma R, Johansson J, Venizelos N, 2017. Proinflammatory cytokines and oxidative stress decrease the transport of dopamine precursor tyrosine in human fibroblasts. Neuropsychobiology75, 178–184. [PubMed: 29339668]

Weathers FW, Keane TM, Davidson JR, 2001. Clinician-Administered PTSD Scale: a review of the first ten years of research. Depress. Anxiety13, 132–156. [PubMed: 11387733]

Yang YC, Schorpp K, Harris KM, 2015. Social support, social strain and inflammation: evidence from a national longitudinal study of U.S. Adults. Soc. Sci. Med. 107, 124–135.

Table 1

Demographic, military, psychological, and biological variables by PTSD group.

Variable	Whole Sample (N = 606)	Current PTSD (N = 246)	No Current PTSD (N = 360)	Statistic (t, χ^2)
	N (%) or Mean (*SD*)			
Age M(*SD*)	58.01 (*11.17*)	58.04 (*10.16*)	58.00 (*11.82*)	−0.05
Range	24–88	24–85	26–88	
Missing	2 (.00 %)	0 (0%)	2 (.01 %)	
Gender				8.56**
Male	568 (93.7 %)	222 (90.2 %)	346 (96.1 %)	
Female	38 (6.3 %)	24 (9.8 %)	14 (3.9 %)	
Race				8.23
White/Caucasian	357 (58.9 %)	148 (60.2 %)	209 (58.1 %)	
Black/AA	131 (21.6 %)	50 (20.3 %)	81 (22.5 %)	
Latinx	48 (8.1 %)	18 (7.3 %)	31 (8.6 %)	
Asian/PI	43 (7.1 %)	14 (5.7 %)	29 (8.1 %)	
Other	17 (2.8 %)	12 (4.9 %)	5 (1.4 %)	
Missing	9 (1.5 %)	4 (1.6 %)	5 (1.4 %)	
Ethnicity				0.80
Hispanic/Latinx	57 (9.4 %)	20 (8.1 %)	37 (10.3 %)	
Non-Hispanic	541 (89.3 %)	223 (90.7 %)	318 (88.3 %)	
Missing	8 (1.3 %)	3 (1.2 %)	5 (1.4 %)	
Marital status				8.77
Married	224 (37.0 %)	96 (39.0 %)	128 (35.6 %)	
Never married	160 (26.4 %)	51 (20.7 %)	109 (30.3 %)	
Divorced	175 (28.9 %)	74 (30.1 %)	101 (28.1 %)	
Widowed	21 (3.5 %)	11 (4.5 %)	10 (2.8 %)	
Separated	24 (4.0 %)	13 (5.3 %)	11 (3.1 %)	
Missing	2 (0.3 %)	1 (0.4 %)	1 (0.3 %)	
Education				8.51
<HS diploma	22 (3.6 %)	11 (4.5 %)	11 (3.1 %)	
HS diploma	108 (17.8 %)	52 (21.1 %)	56 (15.6 %)	
Some college	298 (49.2 %)	11 (45.5 %)	186 (51.7 %)	
College Degree	102 (16.8 %)	43 (17.5 %)	59 (16.4 %)	
Graduate Degree	75 (12.4 %)	28 (11.4 %)	47 (13.1 %)	
Missing	1 (0.2 %)	0 (0%)	1 (0.3 %)	
Income <$20,000	189 (31.2 %)	70 (28.5 %)	119 (33.1 %)	1.53
Missing	4 (0.7 %)	1 (0.4 %)	3 (0.8 %)	
Paid Employment	180 (29.7 %)	57 (23.2 %)	123 (34.25 %)	8.59**
Missing	1 (0.2 %)	0 (0%)	1 (0.3 %)	
Service branch				6.09
Air force	56 (9.2 %)	17 (6.9 %)	39 (10.8 %)	

Variable	Whole Sample (N = 606)	Current PTSD (N = 246)	No Current PTSD (N = 360)	Statistic (t, χ^2)
Army	277 (45.7 %)	116 (47.2 %)	161 (44.7 %)	
Marines	74 (12.2 %)	36 (14.6 %)	38 (10.6 %)	
Navy	102 (16.8 %)	36 (14.6 %)	66 (18.3 %)	
Coast guard	6 (1.0 %)	2 (0.8 %)	4 (1.1 %)	
Multiple	17 (2.8 %)	6 (2.4 %)	11 (3.1 %)	
Missing	74 (12.2 %)	33 (13.4 %)	41 (11.4 %)	
Era Served				8.46
World War II	14 (2.3 %)	3 (1.2 %)	11 (3.1 %)	
Korea	20 (3.3 %)	7 (2.8 %)	13 (3.6 %)	
Vietnam	313 (51.7 %)	151 (61.4 %)	162 (45.0 %)	
Gulf	35 (5.8 %)	14 (5.7 %)	21 (5.8 %)	
OEF/OIF	24 (4.0 %)	8 (3.3 %)	16 (4.4 %)	
Multiple/Other	22 (3.6 %)	7 (2.8 %)	15 (4.2 %)	
Missing	178 (29.4 %)	56 (22.8 %)	122 (33.9 %)	
Physically Active	422 (69.6 %)	148 (60.2 %)	271 (76.1 %)	17.58**
Current smoking	155 (25.6 %)	62 (25.2 %)	93 (25.8 %)	0.03
Poor Sleep	252 (41.6 %)	149 (60.6 %)	103 (28.6 %)	61.44**
Problematic Drinking	240 (39.6 %)	87 (36.6 %)	153 (43.7 %)	3.01
CAPS Severity Score	32.77 (31.72)	66.58 (18.89)	9.67 (11.57)	
PHQ-9 Score	7.60 (6.02)	11.26 (5.84)	5.09 (4.72)	−13.78**
ERQ				
Suppression	12.16 (*3.21*)	13.12 (*3.22*)	11.50 (*3.04*)	−6.29**
Reappraisal	21.25 (*4.27*)	21.09 (*4.73*)	21.26 (*3.93*)	0.74
BMI (kg/m^2)	29.27 (5.62)	30.25 (5.94)	28.59 (5.30)	−3.59**
Creatinine	1.04 (*0.42*)	1.08 (*0.54*)	1.01 (*0.31*)	−2.11*
Disease Severity Index	1.11 (1.08)	1.33 (1.11)	0.96 (1.03)	−4.13**
Range	0–6.00	0–5.00	0–6.00	
hsCRP (mg/L)	2.83 (*5.27*)	2.78 (*3.39*)	2.86 (*6.26*)	−2.00*
Range	0.16–88.00	0.16–22.70	0.16–88.00	
WBC (×10^9/L)	6.54 (*2.03*)	6.82 (*2.22*)	6.35 (*1.86*)	−2.65**
Range	2.40–18.20	3.10–18.20	2.40–14.30	
Fibrinogen (mg/dL)	336.79 (*71.89*)	344.60 (*71.43*)	331.46 (*71.82*)	−2.33*
Range	91–761	178–653	91–761	
Inflammatory Index	0.08 (*2.26*)	0.43 (*2.20*)	−0.15 (*2.27*)	−3.09**

Note.

*$p \leq .05$

**$p \leq .01$.

Chi-square (χ^2 statistics) and independent t-tests are presented for categorical and continuous variables, respectively. PTSD status based on Clinician Administered PTSD Scale-IV criteria. AA = African American. PI = Pacific Islander. Physically Active = dichotomized (active, inactive)

self-report of current physical activity level. Current smoking = dichotomized self-report current tobacco smoker. Poor Sleep = dichotomized past month overall sleep quality item from Pittsburgh Sleep Quality Index. Problematic Drinking = dichotomized threshold criteria from Alcohol Use Disorders Identification Test-Consumption. CAPS = Clinician Administered PTSD Scale past month total severity score. PHQ-9 Total Score = Patient Health Questionnaire total depression severity score. ERQ = Emotion Regulation Questionnaire. BMI = Body Mass Index calculated as participant weight divided by the square of height. Inflammatory biomarkers are raw data, but the *p*-values are based on log-transformed data. Disease Severity Index = Severity score of summed counts across medical disease endorsement categories. HsCRP = high sensitivity C-reactive protein. WBC = white blood cell count. Inflammatory Index = sum of z-scored log-transformed inflammatory markers.

Table 2

Pearson correlations between sociodemographic and health behaviors with emotion regulation and inflammation.

	Suppression	Reappraisal	Inflammation Index	hsCRP	WBC	Fibrinogen
Sex	−0.01	−0.01	0.06	0.06	0.02	0.06
Age	**0.09** *	0.03	**0.12** **	0.08	0.06	**0.14** **
Education	−.07	0.00	**−0.14** **	**−0.10** **	**−0.09** *	**−0.12** **
Race	0.08	**0.09** *	−0.02	−0.02	−0.00	−0.03
Income	−0.04	−0.05	−0.08	**−0.09** *	−0.04	−0.05
Creatinine	0.08	0.04	**0.13** **	**0.11** **	**0.05**	**0.13** **
BMI	**0.11** **	−0.05	**0.30** **	**0.36** **	**0.14** **	**0.18** **
Inactivity	**0.13** **	0.05	**−0.13** **	**−0.20** **	**−0.01**	**−0.09** *
Drinking	−0.00	−0.02	−0.06	−0.02	**−0.09** *	−0.03
Smoking	−0.02	0.01	**0.14** **	0.07	**0.17** **	0.08
Poor Sleep	**0.19** **	−0.04	**0.10** **	**0.08** *	**0.11** **	0.04

Note.

* $p < .05$

** $p \leq .01$.

All inflammatory markers are log transformed. Inflammatory index is standardized summation of log-transformed markers. Suppression & Reappraisal = Emotion Regulation Questionnaire subscale scores.

Table 3

Fully adjusted linear regressions predicting inflammatory index and per biomarker.

Variable	B	SE	β	t	95 % CI Lower	95 % CI Upper	Statistic
Inflammatory Index							$\Delta R^2 = 0.02$ **
Sex	**0.84**	**0.39**	**0.09**	**2.17** *	**0.08**	**1.61**	$\Delta F = 5.17$ **
Age (years)	**0.02**	**0.01**	**0.11**	**2.71** **	**0.01**	**0.04**	
Education	**−.28**	**0.09**	**−0.13**	**−2.96** **	**−0.47**	**−0.09**	
Race	−0.08	0.08	−0.04	−1.01	−0.25	0.08	
Income	−0.25	0.20	−0.05	−1.24	−0.66	0.15	
Creatinine	**0.62**	**0.22**	**0.12**	**2.84** **	**0.19**	**1.03**	
PTSD	0.24	0.19	0.05	1.27	−0.13	0.62	
Suppression	**0.10**	**0.03**	**0.14**	**3.21** **	**0.04**	**0.15**	
Reappraisal	0.00	0.02	0.01	0.12	−0.04	0.04	
High-Sensitivity CRP							$\Delta R^2 = 0.01$ *
Sex	**0.18**	**0.09**	**0.09**	**2.02** *	**0.01**	**0.36**	$\Delta F = 3.44$ *
Age (years)	0.00	0.00	0.07	1.65	−0.00	0.00	
Education	**−0.04**	**0.02**	**−0.09**	**−2.01** *	**−0.09**	**−0.00**	
Race	−0.02	0.02	−0.04	−0.88	−0.05	0.02	
Income	−0.07	0.05	−0.06	1.48	−0.16	0.02	
Creatinine	**0.12**	**0.05**	**0.11**	**2.42** **	**0.02**	**0.22**	
PTSD	0.02	0.04	0.03	0.61	−0.06	0.11	
Suppression	**0.02**	**0.01**	**0.11**	**2.47** **	**0.00**	**0.03**	
Reappraisal	0.00	0.01	0.03	0.85	−0.01	0.01	
White Blood Cell Count							$\Delta R^2 = 0.01$ *
Sex	0.00	0.02	0.01	0.17	−0.04	0.05	$\Delta F = 3.21$ *
Age (years)	0.00	0.00	0.06	1.40	0.00	0.00	
Education	−0.01	0.01	−0.07	−1.50	−0.02	0.00	
Race	−0.00	0.01	−0.00	−0.19	−0.01	0.01	
Income	−0.01	0.01	−0.03	−0.64	−0.03	0.00	

Variable	B	SE	β	t	95 % CI Lower	95 % CI Upper	Statistic
Creatinine	0.01	0.01	0.03	0.77	−0.02	0.04	
PTSD	0.02	0.01	0.07	1.54	−0.01	0.04	
Suppression	**0.00**	**0.00**	**0.11**	**2.41** *	**0.00**	**0.01**	
Reappraisal	−0.00	0.00	−0.03	−0.82	−0.00	0.00	
Fibrinogen							$\Delta R^2 = 0.01$
Sex	**0.04**	**0.02**	**0.11**	**2.64** **	**0.01**	**0.07**	$\Delta F = 2.56$
Age (years)	**0.00**	**0.00**	**0.13**	**2.99** **	**0.00**	**0.00**	
Education	**−0.01**	**0.00**	**−0.13**	**−3.09** **	**−0.02**	**−0.00**	
Race	−0.00	0.00	−0.05	−1.17	−0.01	0.00	
Income	−0.01	0.01	−0.03	−0.63	−0.02	0.01	
Creatinine	**0.03**	**0.01**	**0.12**	**2.89** **	**0.01**	**0.04**	
PTSD	0.01	0.01	0.03	0.75	−0.01	0.02	
Suppression	**0.00**	**0.00**	**0.10**	**2.24** *	**0.00**	**0.01**	
Reappraisal	0.00	0.00	0.01	0.25	−0.00	0.00	

Note.

* $p < .05$

** $p \leq .01$.

Results reflect Step 2 of regression. All inflammatory markers are log transformed. Inflammatory index is standardized summation of log-transformed markers. PTSD = Posttraumatic stress disorder diagnostic status based on past month Clinician Administered PTSD Scale –IV. Suppression & Reappraisal = Emotion Regulation Questionnaire subscale scores.

Psychoneuroendocrinology. Author manuscript; available in PMC 2021 September 08.

Table 4

Sensitivity linear regressions predicting inflammatory index and biomarkers adjusted for health behaviors.[a]

Variable	B	SE	β	t	95 % CI Lower	95 % CI Upper	Statistic
Health Behaviors							
Inflammatory Index							$\Delta R^2 = 0.01$ *
BMI	**0.12**	**0.02**	**0.30**	**7.69** **	**0.09**	**0.15**	$\Delta F = 3.87$ *
Inactivity	−0.25	0.19	−0.05	−1.29	−0.62	0.13	
Drinking	−0.04	0.17	−0.01	−0.22	−0.38	0.30	
Smoking	**0.80**	**0.21**	**0.17**	**4.21** **	**0.47**	**1.29**	
Poor Sleep	0.08	0.18	0.02	0.42	−0.28	0.44	
Suppression	**0.08**	**0.03**	**0.11**	**2.74** **	**0.02**	**0.13**	
Reappraisal	0.01	0.02	0.02	0.41	−0.03	0.05	
High-Sensitivity CRP							$\Delta R^2 = 0.01$
BMI	**0.03**	**0.00**	**0.35**	**9.06** **	**0.02**	**0.04**	$\Delta F = 2.33$
Inactivity	**−0.15**	**0.04**	**−0.14**	**−3.61** **	**−0.24**	**−0.07**	
Drinking	0.03	0.04	0.03	0.89	−0.04	0.11	
Smoking	**0.01**	**0.05**	**0.09**	**2.14** *	**0.01**	**0.19**	
Poor Sleep	0.02	0.04	0.02	0.38	−0.06	0.01	
Suppression	0.01	0.01	0.06	1.39	−0.00	0.02	
Reappraisal	0.01	0.00	0.06	1.62	−0.00	0.02	
White Blood Cell Count							$\Delta R^2 = 0.01$ *
BMI	**0.00**	**0.00**	**0.13**	**3.20** **	**0.00**	**0.01**	$\Delta F = 3.66$ *
Inactivity	0.02	0.01	0.06	1.52	−0.01	0.04	
Drinking	−0.02	0.01	−0.07	−1.65	−0.04	0.00	
Smoking	**0.06**	**0.01**	**0.20**	**4.62** **	**0.03**	**0.08**	
Poor Sleep	0.01	0.01	0.05	1.15	−0.01	0.03	
Suppression	**0.00**	**0.00**	**0.11**	**2.55** **	**0.00**	**0.01**	
Reappraisal	−0.00	0.00	−0.04	−0.98	−0.00	0.00	
Fibrinogen							$\Delta R^2 = 0.01$

Variable	B	SE	β	t	95 % CI Lower	95 % CI Upper	Statistic
BMI	**0.00**	**0.00**	**0.19**	**4.66** **	**0.00**	**0.00**	ΔF = 2.08
Inactivity	−0.01	0.01	−0.03	−0.83	−0.02	0.01	
Drinking	0.00	0.01	0.01	0.34	−0.01	0.02	
Smoking	**0.02**	**0.01**	**0.10**	**2.37** *	**0.00**	**0.04**	
Poor Sleep	−0.01	0.01	−0.03	−0.62	−0.02	0.01	
Suppression	**0.00**	**0.00**	**0.09**	**2.00** *	**0.00**	**0.01**	
Reappraisal	0.00	0.00	0.01	0.32	−0.00	0.00	

Note.

*$p \leq .05$

**$p \leq .01$.

Results reflect Step 2 of regression. All inflammatory markers are log transformed. Inflammatory index is standardized summation of log-transformed markers. Suppression & Reappraisal = Emotion Regulation Questionnaire subscale scores.

[a]All models adjusted for age, sex, race, education, income, creatinine, PTSD diagnostic status.