HHS Public Access

Author manuscript

Stress. Author manuscript; available in PMC 2020 February 07.

Published in final edited form as:

Stress. 2018 November ; 21(6): 548–555. doi:10.1080/10253890.2018.1490724.

Emotion regulation moderates the association between chronic stress and cardiovascular disease risk in humans: a cross-sectional study

Brita Roy, MD[1,*], **Carley Riley, MD**[2,3], **Rajita Sinha, PhD**[4]

[1.] Assistant Professor, Section of General Internal Medicine, Yale School of Medicine, 367 Cedar St., ESH-A, Suite 406A, New Haven, CT 06510; (203) 785-5564; brita.roy@yale.edu

[2.] Assistant Professor, Department of Pediatrics, University of Cincinnati College of Medicine, 3230 Eden Avenue, Cincinnati, OH 45267

[3.] Attending Physician, Division of Critical Care Medicine, Department of Pediatrics, Cincinnati Children's Hospital Medical Center, 3333 Burnet Avenue, Cincinnati, OH 45229; (312) 208-0767; carley.riley@cchmc.org

[4.] Yale Stress Center and Departments of Psychiatry, Neuroscience and Child Study, Yale School of Medicine, 2 Church Street S, New Haven, CT 06519; (203) 737-5805; rajita.sinha@yale.edu

Abstract

Chronic stress is a risk factor for incident cardiovascular disease. Emotion regulation is the ability to modulate one's state or behavior in response to a given situation or stressor, and may mitigate the effect of chronic stress on cardiovascular disease (CV) risk. Data from a cohort of 754 community-dwelling young to middle-aged adults who were assessed between 2007–12 on stress, emotion regulation and CV risk measures were used to test the hypothesis that emotion regulation mitigates the effect of chronic stress on CV risk. Emotion regulation was measured using the Difficulties in Emotion Regulation Scale (DERS). We created a composite stress score using data from the Cumulative Adversity Interview and the Perceived Stress Scale. Our outcomes included blood pressure, body mass index, and insulin resistance separately and combined into a composite CV risk score. Covariates included age, sex, race, years of education, and smoking status. We used multivariable logistic regression to evaluate associations between stress measures and CV risk among participants and the impact of emotion regulation (DERS scores) on this association. We found that composite stress interacted significantly with the DERS score to affect CV risk (p=0.007). A median split of the DERS scores indicated that CV risk was associated with the composite stress score in the fully adjusted model (ß=0.206; p=0.005) among participants with low emotion regulation, but not among those with high emotion regulation (ß=0.048; p=0.59). Chronic stress was associated with CV risk only among participants with poor emotion regulation. Emotion regulation is a teachable skill, and may play a role in preventing CV disease.

Lay Summary:

[*]Corresponding author.

Emotion regulation is the ability to modify one's reaction to a negative or stressful event, and is a teachable skill. Effective emotion regulation dampens the negative effect of chronic stress on the body, which may reduce risk for cardiovascular disease.

Keywords

emotional intelligence; emotion regulation; chronic stress; psychological stress; cardiovascular disease risk; primary prevention

Introduction

Cardiovascular (CV) disease remains the leading cause of death in the United States, with almost 800,000 deaths attributed to CV disease in 2013 (Bauer, Briss, Goodman, & Bowman, 2014). Underlying cardiometabolic risk factors for CV disease include diabetes mellitus, hypertension, and obesity, which remain highly prevalent. Fortunately, approximately 88% of CV disease is preventable through adoption of healthy behaviors and control of these cardiometabolic risk factors (Bauer et al., 2014; Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005).

Chronic toxic stress is when an individual is "chronically exposed to uncontrollable stressors, such as a chaotic environment, abuse or neglect, in the absence of adequate social or emotional support" (Adler & Stewart, 2010). Chronic toxic stress has been found to be an additional risk factor for CV disease (Rozanski, Blumenthal, & Kaplan, 1999). Individuals with high perceived chronic stress are less likely to engage in healthy behaviors that control cardiometabolic risk factors, such as maintaining a healthy diet rich in fruits and vegetables, engage in regular physical activity, and refrain from smoking (DeSteno, Gross, & Kubzansky, 2013). In addition, chronic toxic stress activates the neuro-hormonal "stress response," including chronic inappropriate stimulation of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis, as well as low-grade inflammation (Boehm & Kubzansky, 2012; van Eck, Berkhof, Nicolson, & Sulon, 1996). These responses are known to further independently increase risk of hypertension, diabetes, and obesity, and thus contribute to the lifetime risk of developing CV disease (Black & Garbutt, 2002; Das & O'Keefe, 2008; Pradhan, Manson, Rifai, Buring, & Ridker, 2001; Sinha & Jastreboff, 2013).

Emotion regulation is the ability of individuals to control their emotional experience and expression, especially the control of or response to negative emotions (Cole, Michel, & Teti, 1994; Kopp, 1989). It is a multi-dimensional process through which individuals may effortfully or automatically monitor, evaluate, and modify their emotion experience to environmental demands, including stressful events (Bargh & Williams, 2007; Campbell-Sills & Barlow, 2007; Gratz & Roemer, 2004; Rottenberg & Gross, 2007). Individuals with effective emotion regulation may be able to attenuate the neuro-hormonal stress response despite undergoing stressful life events. In addition, individuals with high emotion regulation may have increased ability to adopt and maintain healthy behaviors such as regular physical activity, eating a diet rich in fruits and vegetables, and refraining from smoking. Importantly, emotion regulation is a teachable skill, and curricula are emerging to improve one's ability to actively monitor and moderate emotional responses to stressful

situations (Rimm-Kaufman et al., 2014; Rivers, Brackett, Reyes, Elbertson, & Salovey, 2013). Further, an individual's emotion regulation is measurable. Deficits in emotion regulation can be assessed using validated instruments, such as the Difficulties in Emotion Regulation Scale (DERS) (Gratz & Roemer, 2004). The DERS includes six subscales: lack of emotional awareness, lack of emotional clarity, non-acceptance of emotional response, limited access to emotion regulation strategies, impulse control difficulties, and difficulties engaging in goal-oriented behavior.

To better understand the role emotion regulation may have in mitigating the association between stress and CV risk, we used data from a unique cohort of community-dwelling young adults to examine the effect of emotion regulation as measured by the DERS on the association between chronic stress and risk factors for CV disease. We hypothesized that emotion regulation moderates the effects of stress on CV disease risk such that high emotion regulation would mitigate the association between chronic stress and three risk factors for CV disease, including blood pressure, body mass index, and insulin resistance.

Methods:

Participants

We used data from the initial examination of a cohort of 754 adults residing in the greater New Haven, CT area, volunteering to participate in a study examining the role of stress and self-control in addictive behaviors at the Yale Stress Center to perform this cross-sectional study. Participants were recruited between December 2007 and May 2012 through advertisements in local newspapers and flyers in community centers and organizations. Participants between 18–50 years of age and able to read English at or above a sixth-grade level were eligible to participate. Participants were excluded if he or she had a substance use disorder (except nicotine), were pregnant, or had a medical condition that would preclude participation in the study.

Procedures

The Yale University Institutional Review Board approved the study. Study procedures were performed at the Yale Stress Center. Potential participants completed an initial screening over the telephone or in-person to determine eligibility. Following screening, eligible participants met with a research assistant for a 2-hour intake session to obtain informed consent and begin assessments. Participants also had a separate morning biochemical evaluation session after fasting overnight. Participants were compensated for study participation.

Measures

Independent Variable: Chronic Stress—The Cumulative Adversity Index (CAI) is a 140-item multifaceted interview-based assessment of life events and subjective stress (Turner & Lloyd, 1995). Trained interviewers asked all participants in our cohort about specific stressful events during their lifetime, in addition to the occurrence, timing and frequency of these events. The CAI is comprised of four domains: major life events, life trauma, recent life events, and chronic stress. We summed the major life events, life trauma,

and recent life events subscales to create the cumulative adverse life events (CALE) score (Ansell, Rando, Tuit, Guarnaccia, & Sinha, 2012; Turner & Wheaton, 1995). The chronic stress (CS) subscale does not quantify adverse events, but rather, it elicits the participants' subjective experience of stress in specific social roles or domains of life and in the context of specific adverse events. The CAI has been reported to have good reliability, validity, and internal consistency (Cronbach's alpha 0.83) (Abravanel & Sinha, 2014; Dohrenwend, 2006; Hammen, 2005).

Because the items in the CS inquire about stress from a set list of specific experiences that participants may or may not have experienced, we also used the perceived stress scale (PSS), a widely-used assessment of stress, to measure participant's current level of general global subjective stress (Sheldon Cohen, Kamarck, & Mermelstein, 1983; Sharp, Kimmel, Kee, Saltoun, & Chang, 2007). The PSS is a measure of the degree to which respondents feel life events are unpredictable, uncontrollable, and overwhelming. It consists of a 10-item scale that has good internal consistency and reliability (Cronbach's alpha 0.85), and has been validated in many populations, including community-dwelling adults (Cohen & Williamson, 1988; Ezzati et al., 2014). Overall, we found significant but modest associations between the CALE, CS and PSS (Table 2). Thus, to assess the overall association of life stressors with CV risk, including number of adverse life events as well as specific chronic stress and global subjective stress, we created a composite stress score by summing the normalized CALE, CS and PSS scores.

Moderating Variable: Emotion Regulation—We used the DERS to assess emotion regulation (Gratz & Roemer, 2004). The DERS is a 41-item measure that is comprised of six domains with six corresponding subscales: lack of emotional awareness, lack of emotional clarity, non-acceptance of emotional responses, limited access to emotion regulation strategies, impulse control difficulties, and difficulties engaging in goal-oriented behavior. Participants respond to each item on a scale ranging from "almost never applies" (1 point; 0–10% of the time) to "almost always applies" (5 points; 91–100% of the time). The DERS has good psychometric properties, including good reliability in our cohort (Cronbach's alpha 0.84) (Abravanel & Sinha, 2014). High scores on the DERS correspond to lower emotion regulation, or greater emotion dysregulation.

Covariates—Participants were also asked to report their age, sex, race/ethnicity, years of education obtained, and smoking status during the initial examination. Age was categorized into five non-overlapping groups (18–25 years, 26–35 years, 36 to 45 years, 46 to 55 years, and over 55 years). Race/ethnicity was categorized as White, Black, Hispanic, Asian, and Native American/Pacific Islander. Years of education was categorized as <12 years, equal to 12 years, 13–16 years, and >17 years. Smoking status was recorded as current smoker or not a current smoker.

Outcomes: Cardiometabolic CV Risk Factors—Data were collected on three cardiometabolic CV risk factors in the Yale Stress Center cohort: systolic blood pressure (SBP), diastolic blood pressure (DBP), body mass index, (BMI) and insulin resistance (HOMA-IR). SBP, DBP and BMI were recorded during a separate physical health examination. A research nurse or trained research staff member measured sitting blood

pressure and participant's weight and height. Blood pressure was recorded as the average of three readings during the initial assessment. BMI was calculated using each participant's measured height and weight during the initial assessment.

On a separate day from intake and baseline assessments and after overnight fasting, a subgroup of willing participants that were not on any medications (N=528) came to the laboratory at 7:30 AM, at which time circulating plasma insulin and glucose levels were drawn and tested in duplicate. All samples were immediately placed on ice, spun, and separated by centrifugation. Separated plasma was stored at −80°C. The homeostatic model assessment of insulin resistance (HOMA-IR) was used as a surrogate measure for insulin resistance and calculated as [glucose (mg/dL) × insulin (μU/mL)]/405 (Matthews et al., 1985; Wallace, Levy, & Matthews, 2004). Acutely ill patients and those on pharmacologic treatments for any illnesses were excluded from the fasting blood draw to eliminate any possible effects of medications and active illness on hormones and serum markers.

In order to assess the effect of our independent variables on CV disease risk, we adapted the American Heart Association's method for calculating an ideal CV health score (Lloyd-Jones et al., 2010) to create a composite CV risk score based on the risk markers that were available in the current sample. Points were allocated for low, moderate, and high risk for each CV risk factor according to standard thresholds and then summed to calculate a composite CV risk score. For the subgroup with HOMA-IR values, we assigned 0 points for low risk (SBP <=120, DBP <=80, BMI<=24 and HOMA-IR<=2.5); 1 point each for moderate risk (SBP 121–139, DBP 81–89, BMI 25–30, and HOMA-IR 2.6–3.8); and 2 points each for high risk (SBP >=140, DBP>=90, BMI>30, and HOMA-IR>3.8). We performed a secondary analysis using data from all participants, including those who were ineligible or unwilling to return for a fasting blood draw to assess HOMA-IR levels. For the full group, we calculated a modified CV risk score without HOMA-IR: we assigned 0 points for low risk (SBP <=120, DBP <=80, and BMI<=24); 1 point each for moderate risk (SBP 121–139, DBP 81–89, and BMI 25–30); and 2 points each for high risk (SBP >=140, DBP>=90, and BMI>30).

Statistical Analysis

We included data from 754 participants who completed enrollment and all stress measures (e.g., PSS and DERS) in this analysis. For our main analyses, we excluded 226 participants that had missing HOMA-IR values due to ineligibility for fasting blood draws. We performed a secondary analysis among the full sample using the modified CV risk score as our outcome (calculated without HOMA-IR).

We initially described demographic characteristics, cardiometabolic risk factors, and levels of emotion regulation and stress. We compared differences in these baseline characteristics between participants with high and low emotion regulation using Pearson's Chi-square.

We used linear regression and Pearson's correlations to examine the associations and standardized effect sizes between each stress measure, as well as our composite stress measure, and each cardiometabolic risk factor (*i.e.*, BP, BMI, and HOMA-IR). We used ordinal logistic regression to assess associations with our composite CV risk score. Initially,

we examined unadjusted associations. We then examined associations adjusted for age, sex, race/ethnicity, years of education, and smoking status. We repeated these steps in a secondary analysis using data from the full cohort, including those that did not have HOMA-IR levels, with the modified CV risk score as the outcome.

To explore the hypothesized moderating effect of emotion regulation, we tested an interaction term between the continuous DERS score and our composite stress score on its relationship with CV risk. We also examined the marginal effects of DERS on the slopes of coefficients of the composite stress score predicting CV risk in unadjusted and adjusted models. If the interaction term was significant with discernible changes in slope, we planned to split the sample by median DERS score, and assess unadjusted and adjusted associations between chronic stress and CV risk within each strata. In exploratory analyses examining the effect modification of each emotion regulation domain, the same approach was planned for each of six DERS subscale scores.

All analyses were performed using Stata SE 14.1 (College Station, TX). All tests were two-tailed with alpha set at 0.05.

Results:

Our sample of 754 participants had a mean (SD) age of 29.7 (9.2) years, was 55% female, 72% white, 26% current smokers, and obtained 15.2 (2.4) years of education (Table 1). Those who were excluded due to missing HOMA-IR values were older (mean age 34 years), less likely to be white (67%), and more likely to be a current smoker (33%); sex and years of education were similar between the groups with and without HOMA-IR. Our full sample had mean (SD) systolic BP 123.1 (15.2) mmHg, diastolic BP 75.8 (11.4) mmHg, BMI 27.6 (5.7) kg/m^2, and HOMA-IR 3.17 (2.06), with a mean composite CV risk score of 2.4 (1.7) and modified composite CV risk score of 1.7 (1.2).

Our median DERS score was 66; participants were categorized as having high emotion regulation if DERS<=66 and low emotion regulation if DERS>66. Participants with high emotion regulation had more years of education and were less likely to smoke (Table 1). Participants with high emotion regulation had lower scores for each stress measure compared to participants with low emotion regulation. The CS and the PSS scores were correlated (r=0.42) (Table 2). The DERS score was also correlated with our composite stress score (r =0.51).

Standardized effect sizes and unadjusted and adjusted associations between each stress measure and risk factor for CV disease are shown in Tables 2 and 3. CALE scores were positively associated with all CV risk factors in unadjusted models, and associated with SBP, DBP, and BMI after adjustment for age, sex, race/ethnicity, education, and smoking status (SBP: ß =0.22, p=0.006; DBP: ß =0.14, p=0.017; BMI: ß =0.134, p<0.001). CS scores were positively associated with BMI and HOMA-IR in unadjusted analyses (BMI: ß =0.20, p<0.001; HOMA-IR: ß =0.33, p=0.006), and associated with BMI only after full adjustment (ß =0.11, p=0.001). Higher PSS scores were associated with higher BMI in unadjusted and adjusted models (unadjusted: ß =0.142, p=0.017; adjusted: ß =0.049, p=0.041). The

composite stress score was associated with higher BMI and HOMA-IR in unadjusted analyses (BMI: ß =0.05, p<0.001; HOMA-IR: ß =0.092, p=0.006); the association with HOMA-IR was attenuated in the adjusted model (BMI: ß =0.355, p<0.001; HOMA-IR: ß =0.077, p=0.064).

In the full sample, all stress measures were significantly associated with our composite CV risk score (Table 3). The interaction term between DERS and our composite chronic stress score was significant in predicting the composite and modified CV risk scores (p=0.007), and marginal effects of DERS ranged from −0.017 (95% CI: −0.027, −0.006) to 0.012 (95% CI: 0.004, 0.20), signifying the DERS modifies the association between chronic stress and CV risk. Stratified analyses revealed no association between the composite stress score and the composite CV risk score among the subgroup with high emotion regulation (ß=0.081, p=0.36). Among participants with low emotion regulation, the composite stress score was associated with the CV risk score in unadjusted (ß=0.185, p=0.006) and adjusted models (ß=0.206, p=0.005). Stratified results were similar using the modified CV risk score.

Among participants with high emotion regulation, neither the CS, the PSS, nor the composite stress score was associated with the CV risk score. Among participants with low emotion regulation, CS, PSS, and composite stress were all associated with higher composite CV risk scores in fully adjusted models (CS: ß =0.042, p=0.021; PSS: ß =0.032, p=0.014; Composite: ß =0.206, p=0.005). Results were similar in secondary analyses using the modified CV risk score as the outcome among the larger sample.

Exploratory Analyses using DERS Subscales

Higher composite stress scores were associated with higher CV risk scores among participants above the 50th percentile in non-acceptance of emotional response (ß=0.155, p=0.007), limited access to emotion regulation strategies (ß=0.179, p=0.003), impulse control difficulty (ß=0.165, p=0.002), and difficulty engaging in goal oriented behavior (ß=0.113, p=0.04), in adjusted analyses (Table 4). Opposite trends were seen in the other two subscales, with higher composite stress scores associated with higher CV risk scores among participants below the 50th percentile in lack of emotional awareness (ß=0.131, p=0.011) and lack of emotional clarity (ß=0.15, p=0.003).

Discussion

In our sample of young to middle aged community-dwelling adults, we confirmed associations between cumulative adverse life events and chronic and perceived stress with cardiometabolic risk factors for CV disease, including BMI and insulin resistance. We found a significant interaction exists between chronic stress and emotion regulation, and the positive association between chronic stress and CV risk was attenuated among those with more effective emotion regulation. Our results suggest that high emotion regulation may lessen the physiologic impact of chronic stress on CV risk factors. These findings support our hypothesis that emotion regulation moderates the association between chronic stress and CV risk.

Of note, the young to mid-life age of our sample may have precluded us from finding associations with SBP or DBP. However, the fact that we identified linkages with BMI and HOMA-IR among such a young sample of community-dwelling adults with emotion dysregulation, with a mean age of 29 years, has potential long-term implications. While our study is cross-sectional and no causal interpretations can be made, our findings that chronic stress was associated with CV risk factors among those with poor emotion regulation suggests that improving emotion regulation strategies earlier in life, during childhood, adolescence, and young adulthood may have long-term benefit in the prevention of morbidity and mortality from early onset of CV disease.

Effective emotion regulation promotes positive psychologic well-being by improving coping mechanisms in the setting of negative events and by fostering healthy reappraisal and reflection in the setting of positive events (Eid & Larsen, 2008; Quoidbach, Berry, Hansenne, & Mikolajczak, 2010). As such, our study adds to the growing body of literature suggesting that positive psychologic well-being is protective of incident CV disease (Boehm & Kubzansky, 2012; Kubzansky, Park, Peterson, Vokonas, & Sparrow, 2011; Rozanski et al., 1999; Tindle et al., 2009). Characteristics of positive psychologic well-being, including greater life satisfaction and emotional vitality were associated with reduced risk of coronary heart disease after adjusting for relevant risk factors in a large cohort study (Boehm, Peterson, Kivimaki, & Kubzansky, 2011; Boehm, Peterson, Kivimaki, & Kubzansky, 2011). Results from a smaller cross-sectional study showed that higher emotional intelligence was associated with lower incidence of coronary heart disease (Vlachaki & Kassotaki, 2013). Our findings provide a potential mechanism to strengthen positive psychologic well-being, which may in turn mitigate the effect of chronic stress on CV risk.

Other positive psychologic factors such as optimism and positive affect, as well as emotion regulation, have been associated with lower levels of cortisol and serum markers of inflammation, such as interleukin-6 (IL-6) and C-reactive protein (CRP) (Appleton, Buka, Loucks, Gilman, & Kubzansky, 2013; Gianaros et al., 2014; Roy et al., 2010). These serum markers of inflammation and cortisol have been found to be elevated with chronic, toxic stress (Boehm & Kubzansky, 2012; van Eck et al., 1996). Higher C-reactive protein levels were associated with maladaptive emotion regulation and higher adult BMI in a life-course model (Appleton, Loucks, Buka, Rimm, & Kubzansky, 2013). Similarly, Gianaros, *et al.* (2014), reported greater reappraisal of negative stimuli was associated with lower levels of interleukin-6 (IL-6), a serum marker of inflammation (Gianaros et al., 2014). In this study, IL-6 was a mediator in the relationship between emotional reappraisal and preclinical atherosclerosis, supporting the hypothesized biologic disease pathway. Measures of positive psychologic well-being are also associated with better health behaviors, such as not smoking, eating more fruits and vegetables, and engaging in physical activity (Giltay, Geleijnse, Zitman, Buijsse, & Kromhout, 2007), which also contribute to lower lifetime CV risk. In our sample, higher emotion regulation was associated with not smoking, but other health-related behaviors were not assessed. Taken together, these studies support the hypothesized neuro-hormonal biologic mechanism between more effective emotion regulation, lower chronic stress, better health behaviors, and lower CV risk.

Our exploratory analyses assessing correlations between chronic stress and CV risk within high and low subgroups of each domain of emotion regulation reveal mixed results. Chronic stress and CV risk were positively associated among subgroups with better emotional awareness and clarity, and among subgroups with worse acceptance of emotional response, access to emotion regulation strategies, impulse control difficulties, and engaging in goal-oriented behavior. These subgroups are not mutually exclusive, making these results difficult to interpret, and more studies are needed to understand how these emotion regulation subscales relate to each other in influencing chronic stress and CV risk.

Notably, in comparison to other psychologic factors that are considered to be trait personality factors, such as neuroticism or optimism, emotion regulation is a teachable skill. Emotion regulation is a key aspect of mindfulness-based stress reduction skills training for adults, targeting emotion regulation abilities such as acceptance, awareness, clarity and impulse control, as assessed in the DERS (Baer, 2003; Goldin & Gross, 2010). Curricula are also emerging to train children and adolescents in effective strategies to recognize and regulate emotions (Rimm-Kaufman et al., 2014; Rivers et al., 2013), and evidence that learning skills to effectively regulate emotions at a young age may reduce the cumulative risk for CV disease over the life course (Boehm, Vie, & Kubzansky, 2012; McEwen & Seeman, 1999). Participants in our study with high emotion regulation had >1 standard deviation lower perceived stress scores than participants with low emotion regulation, though the difference in cumulative adverse life events was not as great between the two groups (<1 standard deviation). This suggests that developing emotion regulation during childhood or adolescence may reduce perceived stress in response to adverse events, thereby mitigating the neuro-hormonal stress response (Das & O'Keefe, 2008; Juster, McEwen, & Lupien, 2010). Further study is needed to evaluate this hypothesis, as emerging evidence from longitudinal studies is mixed on the 10-year effect on CV risk. Appleton, *et al.* (2013), report that increased proneness to distress increases the likelihood for a CV event over the next 10 years by 4.2% for women and 8.4% for men (Appleton, Loucks, et al., 2013). However, inappropriate self-regulation as measured by psychologist observation was not associated with 10-year CV risk. Whether teaching specific stress and emotion regulation skills in high risk adults may normalize the neurohormonal response to reduce CV risk remains to be tested.

Levels of chronic stress are reported to be higher among low socioeconomic status and racial/ethnic minority populations, at least partially due to higher levels of financial insecurity and exposure to traumatic events (Adler & Newman, 2002). Higher rates of chronic stress in these segments of the US population may contribute to observed disparities in rates of CV disease incidence (Adler & Newman, 2002; Shonkoff, Boyce, & McEwen, 2009). Targeted interventions to improve emotion regulation skills within these populations to moderate the biologic effect of chronic stress may reduce CV health disparities in the US, and should be tested. Currently, most curricula to improve skills in emotion regulation are limited to higher socioeconomic areas. Focused instruction on specific domains of emotion regulation as measured by DERS such as acceptance of emotional response, increased access to emotion regulation strategies, improved impulse control, and a focus on goal-directed coping could be important to improve emotion regulation skills and reducing CV risk. Strategies utilizing mindfulness approaches such as mindfulness-based stress reduction

(Gratz & Tull, 2010) as well as acceptance and commitment therapy and dialectical behavior therapy (Gratz, Tull, & Wagner, 2005) are all programs that include such types of skills building and may be used to increase sustainable emotion regulation and reduce CV risk within these vulnerable populations.

Our study is a cross-sectional analysis, limiting causal inference and assessment of directionality. However, our results support prior longitudinal studies that demonstrate an association between positive psychologic well-being and lower incident CV disease. Though we only measured smoking as a health-related behavior, high emotion regulation was associated with lower BMI, suggesting that emotion regulation may also be associated with other health-related behaviors like adhering to a healthy diet and regular physical activity. The sample we used for this study was recruited via voluntary participation in response to local community advertisement, which may have resulted in selection bias. However, the mean age and percent white are similar to those reported for the city of New Haven, CT. Exclusion of non-English speaking adults may limit generalizability to minority and immigrant populations. Additionally, exclusion of adults with substance use disorder in our cohort may have biased our sample towards having better emotion regulation, and thus our median DERS score may be lower than in the general population. If anything, this bias results in more conservative estimates than expected in the population including those with substance use disorders. Finally, given the cross-sectional nature of the study, we did not conduct advanced mediational and moderated mediational analyses that may be more suitable for longitudinal data sets. Thus, our findings warrant further exploration of the moderating effect of emotion regulation on the relationship between chronic stress and CV risk in larger, longitudinal cohorts of community dwelling adults.

Conclusions

In our study, higher emotion regulation mitigated the association between chronic stress and CV risk in a sample of young to middle aged, community-dwelling adults. Because emotion regulation is a teachable skill, these results may have important implications for reducing the incidence of CV disease and attenuating disparities in CV disease risk over the life course. Further testing in prospective cohorts is warranted.

Acknowledgements

We would like to acknowledge the assistance of Adam Hong, MA in data extraction, cleaning and organization.

Declaration of Interests

The current study was supported in part by National Institutes of Health grants UL1-DE019586 and PL1-DA024859 (Rajita Sinha, PI). BR was supported by grant number K12HS023000 from the Agency for Healthcare Research and Quality and BR and CR were also supported by the Robert Wood Johnson Foundation Clinical Scholars Program during part of the study period. The content is solely the responsibility of the authors and the funders had no role in study design, data collection and analyses, decision to publish, or preparation of the manuscript. BR and CR are consultants for the Institute for Healthcare Improvement, BR sits on the advisory board for DataHaven, and RS is on the Scientific Advisory Board of Embera Neurotherapeutics.

All authors were involved in the initial conceptualization of the study, its design, interpretation of the data, and drafting of the manuscript. All authors approved the final manuscript. An abstract based on these results was presented at the Society of General Internal Medicine 38th Annual Meeting in Toronto, Canada, April 2015.

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Table 1.
Demographics of the Yale Stress Center cohort (N=754), 2007–12.

Characteristics of the entire cohort (full sample), as well as the high and low emotion regulation subgroups are shown. P-value compares differences between the high and low emotion regulation subgroups.

	Full Sample	High Emotion Regulation	Low Emotion Regulation	p-value
Age (years (SD))	29.7 (9.2)	30.3 (9.2)	29.0 (9.2)	0.06
Female (%)	55	56	54	0.67
Race				0.21
White (%)	72	68	74	
Black (%)	23	27	20	
Hispanic (%)	1	1	1	
Asian (%)	4	4	4	
Native Am/Pacific Is (%)	0.3	0	1	
Education (years (SD))	15.2 (2.4)	15.4 (2.5)	14.9 (2.1)	0.005
Current smoker (%)	26	19	33	<0.001
Systolic blood pressure (mmHg (SD))	123 (15)	123 (14)	123 (15)	0.97
Diastolic blood pressure (mmHg (SD))	75 (11)	75 (11)	74 (11)	0.86
Body mass index (kg/m^2 (SD))	27 (5.7)	27 (5.5)	28 (5.8)	0.29
HOMA-IR (SD)	3.2 (2.1)	3.1 (1.9)	3.3 (2.2)	0.42
Composite CV Risk (SD)	2.9 (1.8)	2.8 (1.8)	2.9 (1.8)	0.51
Cumulative adverse life events	10.7 (6.8)	9.8 (6.5)	11.5 (6.9)	0.001
Chronic stress	10.1 (6.1)	8.5 (5.2)	11.7 (6.4)	<0.001
Perceived stress scale	21 (8.7)	16.8 (6.5)	25.7 (8.3)	<0.001
Composite stress score	−0.14 (2.3)	−1.1 (1.9)	0.75 (2.3)	<0.001

Table 2.

Pearson's correlation coefficients between each measure of stress and cardiometabolic CV risk factor, Yale Stress Center cohort 2007–12, N=528. Bolded numbers denote significant correlations ($p<0.05$).

	CALE	CS	PSS	COMP	SBP	DBP	BMI	HOMA
CALE	**1.00**							
CS	**0.439**	**1.00**						
PSS	**0.267**	**0.417**	**1.00**					
COMP	**0.740**	**0.815**	**0.740**	**1.00**				
SBP	**0.164**	0.019	0.001	0.054	**1.00**			
DBP	**0.173**	0.034	−0.023	0.034	0.789	**1.00**		
BMI	**0.237**	**0.195**	**0.079**	**0.219**	**0.329**	**0.338**	**1.00**	
HOMA	**0.127**	**0.119**	**0.084**	**0.144**	**0.234**	**0.220**	**0.501**	**1.00**

Abbreviations: CALE = cumulative adverse life events scale; CS = chronic stress scale; PSS = perceived stress scale; Comp = composite stress scale; SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index; HOMA = homeostasis model assessment

Table 3.

Associations between measures of stress and cardiovascular risk factors among Yale Stress Center cohort, 2007–12.

	SBP (n=628)		DBP (n=627)		BMI (n=687)		HOMA-IR (n=562)	
	ß	p-value	ß	p-value	ß	p-value	ß	p-value
Cumulative Life Events								
Unadjusted	0.36	<0.001	0.28	<0.001	0.19	<0.001	0.04	0.002
Adjusted *	0.22	0.006	0.14	0.017	0.134	<0.001	0.02	0.14
Chronic Stress Score								
Unadjusted	0.007	0.614	0.018	0.34	0.203	<0.001	0.327	0.006
Adjusted *	0.01	0.914	−0.38	0.6	0.111	0.001	0.022	0.15
Perceived Stress Scale								
Unadjusted	0.0004	0.986	−0.018	0.558	0.142	0.017	0.347	0.047
Adjusted *	0.012	0.85	−0.188	0.7	0.049	0.041	0.016	0.127
Composite Stress Score								
Unadjusted	−0.001	0.88	−0.004	0.448	0.05	<0.001	0.092	0.006
Adjusted *	0.177	0.513	−0.072	0.724	0.355	<0.001	0.077	0.064

*Adjusted for age, race/ethnicity, sex, education, and smoking status.

Abbreviations: SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index; HOMA-IR = homeostasis model assessment

Table 4.

Association between stress scores and composite CV risk score, stratified by median emotion regulation score, Yale Stress Center cohort 2007–12, N=528.

	Full Sample		High Emotion Regulation		Low Emotion Regulation	
	ß	**p-value**	**ß**	**p-value**	**ß**	**p-value**
Cumulative Life Events						
Unadjusted	0.054	<0.001	0.056	0.002	0.049	0.003
Adjusted *	0.034	0.005	0.033	0.064	0.033	0.052
Chronic Stress Score						
Unadjusted	0.034	0.009	0.036	0.083	0.033	0.064
Adjusted *	0.028	0.029	0.018	0.38	0.042	0.021
Perceived Stress Scale						
Unadjusted	0.020	0.027	−0.004	0.80	0.038	0.004
Adjusted *	0.020	0.021	0.007	0.67	0.032	0.014
Composite Stress Score						
Unadjusted	0.145	<0.001	0.081	0.36	0.185	0.006
Adjusted *	0.116	0.003	0.048	0.59	0.206	0.005

*Adjusted model includes age, sex, race, educational attainment, and smoking.

Table 5.

Associations between composite stress score and cardiovascular risk score, stratified by median DERS subscale score, Yale Stress Center cohort 2007–12.

	Unadjusted		Adjusted[b]	
DERS Subscale[a]	**ß**	**p-value**	**ß**	**p-value**
Lack of emotional awareness				
Below median	0.173	<0.001	0.131	0.011
Above median	0.101	0.069	0.090	0.139
Lack of emotional clarity				
Below median	0.188	<0.001	0.150	0.003
Above median	0.127	0.026	0.109	0.114
Non-acceptance of emotional response				
Below median	0.145	0.005	0.100	0.071
Above median	0.158	0.002	0.155	0.007
Limited access to emotion regulation strategies				
Below median	0.130	0.034	0.093	0.140
Above median	0.180	0.001	0.179	0.003
Impulse control difficulties				
Below median	0.165	0.007	0.077	0.245
Above median	0.126	0.008	0.165	0.002
Difficulties engaging in goal-oriented behavior				
Below median	0.169	0.004	0.124	0.048
Above median	0.148	0.002	0.113	0.040

[a] A domain score above the median reflects poorer functioning with that domain (e.g., greater lack of emotional awareness).

[b] Adjusted model includes age, sex, race, educational attainment, and smoking.