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War and Remembrance: Combat Exposure in Young Adulthood and Memory Function Sixty Years Later

Michael D. Nevarez, M.D.¹, Johanna C. Malone, Ph.D.¹, Dorene M. Rentz, Psy.D.^{2,3}, and Robert J. Waldinger, M.D.¹

¹Harvard Study of Adult Development, Massachusetts General Hospital, Harvard Medical School, Boston, MA

²Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA

³Center for Alzheimer Research and Treatment, Department of Neurology, Brigham & Women's Hospital, Harvard Medical School, Boston, MA

Abstract

Objective—Identifying adaptive ways to cope with extreme stress is essential to promoting long-term health. Memory systems are highly sensitive to stress, and combat exposure during war has been shown to have deleterious effects on cognitive processes, such as memory, decades later. No studies have examined coping styles used by combat veterans and associations with later-life cognitive functioning. Defenses are coping mechanisms that manage difficult memories and feelings, with some more closely related to memory processes (e.g., suppression, repression). Utilizing a longitudinal database, we assessed how reliance on certain defense mechanisms after World War II combat exposure could affect cognitive health 60 years later.

Method—Data spanning 75 years were available on 71 men who had post-war assessment of combat exposure, defense mechanism ratings (ages 19–50), and late-life neuropsychological testing. Interaction models of combat exposure with defenses predicting late-life memory were examined.

Results—In bivariate analyses, greater reliance on suppression correlated with worse memory performance ($r = -0.30$, $p = 0.01$), but greater reliance on repression did not. Greater reliance on suppression strengthened the link between combat exposure and worse memory in late life ($R^2 =$

Corresponding author: Michael D. Nevarez (mnevarez@partners.org).

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Location of work and address for reprints: Massachusetts General Hospital, Harvard Study of Adult Development, 151 Merrimac St, 2nd Floor, Boston, MA 02114

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0.24, $p < .001$). In contrast, greater reliance on repression attenuated the link between combat exposure and poorer late-life memory ($R^2 = 0.19$, $p < .001$).

Conclusions—Results suggest that coping styles may affect the relationship between early-adult stress and late-life cognition. Findings highlight the importance of understanding how coping styles may impact cognitive functioning as people move through adult life.

Keywords

cognition; memory; repression; suppression; combat; coping

1. Introduction

Understanding adaptive ways to cope with extreme stress is essential to promoting the long-term psychological, physical, and cognitive health of those exposed to potentially traumatic events. This matter is of particular relevance to our most recent war veterans returning from decade-long campaigns in Iraq and Afghanistan that have marked America's longest sustained combat operations to date. Does the use of specific coping styles utilized after combat exposure affect long-term well-being decades after war and, if so, how? Utilizing a unique longitudinal database that spans more than 75 years, we aimed to identify how the reliance on subsequent defense mechanisms after combat exposure in World War II (WWII) could affect late-life cognitive health. To our knowledge, this is the first study to report on late-life cognitive outcomes and defense mechanisms in veterans exposed to combat in WWII.

For many soldiers, combat is a traumatic, life-threatening stressor for which no resource can fully compensate [1]. Exposure to combat has been found to predict not only earlier physical decline and mortality [1,2], but also compromised cognition [3]. Studies have shown deleterious effects of combat-related experiences on memory processes shortly after return from deployment [4] as well as long-term effects in later life [5,6]. However, little is known about how coping styles for handling combat exposure affects memory functioning. A wide range of studies report on adaptive coping styles and their impact on well-being with stressors such as traumatic brain injury [7], cancer [8], and peer violence [9]. However, no studies have examined specific coping styles used by combat veterans throughout the lifespan and cognitive functioning many decades later. This gap in our understanding is especially significant as pre-deployment interventions are being implemented to address combat stress, and the field lacks knowledge of how these strategies may affect psychological outcomes as veterans' age.

Shorter-term studies support the relevance of coping styles to post-war cognitive functioning. Jha and colleagues [10] piloted an effective protocol to decrease stress that preserved working memory capacity post-deployment, showing that this capacity was degraded in soldiers without the intervention. Moreover, there is significant interest in how soldiers might engage with their stressful internal experiences in more adaptive ways. For example, a prior study highlighted an intervention in Marine infantry using mindfulness, an adaptive approach that faces and acknowledges challenging internal experiences rather than avoiding them [11]. This study showed enhanced physiologic recovery after stressful

exposure in those who had completed an 8-week mindfulness program. However, the long-term consequences of this particular strategy are not known. Taking advantage of a rare longitudinal study that has followed WWII veterans over 6 decades after military service, we investigate possible links between reliance on specific post-combat defense mechanisms throughout adult life and late-life cognition.

Cognitive decline in later life is a significant contributor to morbidity and mortality [12,13], and studies suggest that individuals' interpretations of and responses to stressful events, rather than the events themselves, are associated with cognitive function [14]. We approached this inquiry from the premise that styles of coping with stress involve habitual patterns of processing thoughts and feelings in the brain. We hypothesized that such patterns, used over a lifetime, could impact related cognitive domains. The conceptual construct of coping refers to a broad range of ways that individuals manage stressful contexts [15], and defense mechanisms are a subset of habitual coping strategies. The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) defines defenses mechanisms (or coping styles) as automatic processes that protect the individual against anxiety and awareness of internal or external stressors, as well as mediate the individual's reaction to emotional conflicts [16]. Empirical research demonstrates that defenses develop in predictable ways across the lifespan, are aspects of normal psychological functioning, and are related to one's affective wellbeing [17].

When considering cognition, compromised memory is one of the first warning signs of cognitive decline and is highly associated with functional difficulties [18]. Moreover, memory function has been shown to be highly sensitive to stress [19,20]. In studying the early-adult predictors of late-life cognitive functioning, we were particularly interested in defense mechanisms that are closely related to memory processes – specifically, suppression and repression. Suppression involves the semiconscious decision to postpone attending to mental content once retrieved from memory [21,22]. By contrast, repression involves the unconscious exclusion of mental content from consciousness [22]. Neuroimaging studies support the notion that both suppression [23] and repression [24] are specifically related to memory-related brain structures (e.g., hippocampus, anterior cingulate cortex).

One way to conceptualize the adaptiveness of defenses is to categorize the extent to which they approach experience or avoid it. These two defense mechanisms represent opposite ends of this spectrum of emotion regulation strategies aimed at managing stressful experiences. Suppression is categorized as 'engaging' and more adaptive, because it allows for conscious recognition of reality but postpones processing the content. Repression is categorized as 'avoidant' and less adaptive, since it banishes uncomfortable reality from awareness without an opportunity for processing [25,26]. We hypothesized that suppression would relate to better late-life memory performance in the face of a significant life stressor (i.e., combat exposure), whereas repression would not. In addition, we hypothesized that other non-memory-specific defenses, whether adaptive (e.g., altruism) or less adaptive (e.g., displacement), would not be related to memory performance.

2. Method

2.1. Study Participants

Participants were 71 men drawn from a larger sample of 268 Caucasian male Harvard College sophomores, born between 1915 and 1924, in a study of male psychological health [27,28]. Selection criteria for the initial study included absence of both physical and mental illness and satisfactory freshman academic record [27]. Participants' IQs ranged from 110 to 150 ($M = 136.20$, $SD = 11.71$). The men were primarily of middle and upper socioeconomic status. On entry into the study, the men were assessed by internists, psychiatrists, psychologists, and anthropologists. Over the next 75 years, men completed questionnaires approximately every 2 years, their medical records were obtained every 5 years, and they were interviewed every 10–15 years. The subset of 71 participants selected for this study was based on the availability of data regarding combat exposure during WWII, defenses in mid-life, and neuropsychological testing in late life. The mean age during neuropsychological testing was 85.6 years ($SD = 1.3$), 87.3% were WWII veterans, and 38% of the 71 men had combat exposure during World War II. Using t-tests we determined that these 71 individuals did not differ significantly from the other participants in this cohort in terms of IQ, social class, quality of upbringing and nurturance by parents, defense maturity in young adulthood (age 20–35), self-reported use of alcohol and tobacco, and health outcomes related to cognition (e.g., stroke).

2.2. Memory

Memory was assessed using the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) [29]. The CERAD memory section has three parts: 1) the Word Recall Task where participants read 10 words aloud and recall any words they can remember over 3 trials; 2) the Delayed Recall section occurs after five-minutes where participants are asked to recall any words they can remember from the list; and 3) the Recognition Recall, where they are read a list of 20 words and asked to respond 'yes' if the word was on the list or 'no' if it was not. Higher scores on each CERAD task indicate better memory function. Validity for CERAD assessments is supported by good agreement with clinical diagnosis of dementia [29]. Inter-rater reliability ranges from .92 to 1.0; test-retest reliability ranges from .43 to .83 [29]. Data from these 3 tests were z-scored and averaged to arrive at a composite CERAD memory score that was used as an index of overall memory performance. Our group has previously shown CERAD measures to be associated with aspects of attachment [30] and psychosocial development [31] in this same cohort. These tests were performed as part of a larger neuropsychological test battery that included measures of attention, language, executive function, and visuospatial ability.

2.3. Combat Exposure

In 1946, a comprehensive questionnaire asking about participants' war experiences was sent to all 224 surviving men who had served in the military (8 were killed in WWII); 217 (97%) of the men completed the questionnaire. In the same year, the men were interviewed by the study staff about their wartime experiences [32]. Both the questionnaire and the interview focused on the effects of the war, the men's exposure to combat, and their descriptions of thoughts and bodily symptoms in the face of danger. Combat exposure was measured on two

domains that together form the Combat Exposure Scale: 1) an interview focused on the presence of specific combat experiences, and 2) a self-report of participants' perception and quantity of danger level they were exposed to. The first domain was based on men's responses to combat-related questions, the following six experiences were coded as present (1) or absent (0) for six possible points: 1) being under enemy fire, 2) firing at the enemy, 3) killing anyone, 4) seeing allies killed or wounded, 5) seeing the enemy killed or wounded, 6) being wounded. The second domain was based on the sustained danger scale [32], a self-report scale created by study staff prior to 1946. The top category, rating 7, represented danger at the height of action with the enemy (e.g., "being shelled heavily", "vessel hit", and "heavy combat with enemy") while the bottom category, rating 1, represented complete absence of combat-related danger. Added to the first domain were four possible points for sustained danger (0=no days spent in danger levels 6 or 7; 1= 1-21 days spent in danger levels 6 or 7; 2= 22 days or more spent in danger levels 6 or 7) and severity of danger (0 = no days spent in danger levels 6 or 7; 1 = 6 was scored as the highest level of danger; 2 = 7 was scored as the highest level of danger). The combination of these two domains resulted in the Combat Exposure Scale [33], a 10-point scale which assesses exposure to specific combat experiences and the extent to which a person was exposed to sustained combat danger. All the variables that created the Combat Exposure Scale were significantly inter-correlated at $p < .001$, except that being wounded was not significantly correlated with high level of perceived danger. In the current study, the coefficient alpha for this scale was 0.74. The Combat Exposure Scale is unique in that it combines both self-report and interview methods, and its two domains share similarity to two sub-scales of the widely used Deployment Risk and Resilience Inventory research tool (Subscale D: combat experiences, Subscale G: perceived threat) [34]. The Combat Exposure Scale has been supported in prior studies as a predictor of PTSD symptoms and early mortality following WWII [33], as well as a predictor of later life well-being in WWII veterans [35].

2.4. Defense Mechanisms

Defense mechanisms were identified using vignettes taken from multiple interview transcripts when the men were between the ages of 19 and 50. Participants were interviewed at three points in time by study staff members. During college, a staff psychiatrist conducted eight 1-hour interviews with each man. At age 30, men were interviewed by a trained anthropologist for approximately 2 hours. Interviews at age 45 to 50 were conducted by a psychiatrist or social worker, lasted 2 to 3 hours, and took place in participants' homes or at their workplaces. Interview protocols were designed to focus on challenges in participants' relationships, physical health, and work. Interviewers took notes during the interviews and wrote extensive summaries immediately following each meeting. Interviewers were instructed to elicit participants' descriptions of the behaviors that they used to cope with challenges [22]. This single measure spans 3 decades, and unfortunately our study does not have access to repeated measures during this timeframe in order to assess stability of defenses over this time period. However, studies have shown general stability of defense mechanisms across the lifespan, especially for adaptive defenses [36], and that once a defense mechanism is established, it continues to exist and is made available for use if needed [17].

Fifteen individual defenses (age 19–50) were assessed from the data described above by three independent raters who were blind to other information about participants. Raters identified vignettes illustrating coping styles during challenging times, and the number of identified vignettes per participant ranged from 10 to 30 per interview. Coders gave each vignette a single rating for the best-fitting defense. Ratings were based on a coding manual developed by Vaillant [37]. These codes were then tallied and given the following scores based on the frequency with which each particular defense appeared across all vignettes: 1 = no significant vignettes noted illustrating this defense; 2 = defense evident in at least 1 vignette; 3 = evident in at least 2 or 3 vignettes; and 4 = evident in at least 4 or more vignettes, or if no defense was rated this frequently, the two or three most frequently used individual defenses were rated 4. The defenses used in the present analysis were suppression and repression, which we hypothesized to be related to memory functioning, and altruism and displacement, which we hypothesized to be unrelated to memory and had appropriate base rates for analyses. Inter-rater reliability for these frequency ratings ranged from $r = .72$ to $.84$ [22]. These ratings have shown good external validity, predicting better physical health in late life, mental health, subjective well-being, and work satisfaction [28,38,39]. These defense mechanism ratings are described in greater detail in other studies [40,41].

2.5. Analysis

Distributions for variables were first examined for normality. The distribution for combat exposure was positively skewed due to higher levels of participants without combat and was therefore square root transformed prior to analyses. The distribution for suppression tended towards higher scores, but appeared acceptable (skewness = -1.16 , kurtosis = 0.89). Pearson correlations (2-tailed) among the defenses of suppression, repression, altruism, and displacement were first examined with the three individual memory scores as well as the composite memory score. These particular defenses were also examined to ensure appropriate base rates across the 4-point scale range. Based on these correlations, we conducted two hierarchical linear regression analyses to test whether combat exposure interacts with suppression and repression, respectively, to predict late-life memory. Analyses were conducted using IBM SPSS version 23 with moderation analyses utilizing bias-corrected bootstrapping methods via the PROCESS macro for SPSS v2.13.2 macro by Andrew F. Hayes [42].). To probe the interaction models we utilized the Johnson-Neyman technique [43,44] from the PROCESS macro to inform where in the distribution of the proposed moderator (i.e., suppression) combat exposure may have had an effect on late life memory. This technique has advantages over the selection of various values of the moderator to estimate the conditional effect, as it derives appropriate values of the moderator to be tested that better reflect a ‘region of significance’ of the effect of X on Y [42]. In addition, an output option from the PROCESS macro for visualizing the conditional effect of combat exposure on memory at values of the moderator was utilized to construct graphical representations for the models (Figures 1 and 2).

3. Results

3.1. Pearson Correlations

Means, standard deviations, and correlations among variables of interest are presented in Table 1. We found a statistically significant association between memory outcomes, suppression, and combat exposure, and the absence of such relationships with repression. In addition, suppression was correlated with each of the 3 CERAD sub-tests, lending support to our composite variable being a consistent measure. As expected, no such links were present for defenses hypothesized to be unrelated to memory (altruism, displacement). Greater reliance on suppression was inversely associated with use of repression in young adulthood (Table 1). Unusually, suppression and altruism (both adaptive defenses) were not positively correlated with each other, and repression and displacement (both non-adaptive defenses) were also not positively correlated with each other. Moreover, additional bivariate analyses revealed no significant correlations between the defenses examined in this study and other measures of attention and executive functioning (e.g., trail making tests). In addition, we performed bivariate analyses of the 11 remaining defense mechanisms not included in this study but available in our dataset (i.e., fantasy, projection, hypochondriasis, acting out, intellectualization, reaction formation, displacement, dissociation, humor, anticipation, sublimation) and no significant correlations were found between these defenses and the CERAD memory composite.

3.2. Interaction Models

We then examined possible interactions of combat exposure with defense mechanisms in predicting late-life memory. Table 2 reports on the results of two hierarchical linear regression analyses testing the interaction of suppression with combat exposure and repression with combat exposure, respectively, in predicting overall memory function (CERAD composite) in late life.

The overall model with suppression proposed as a moderator showed a main effect that explained 24% of the variance ($p < .001$) in late life memory function (Table 2). The regression coefficient for the product of suppression and combat was statistically different from zero ($-0.264, p < 0.01$), meaning that the effect of combat exposure on memory depended on the level of suppression. In this case, the moderation component of the model explained 7.8% of the variance (Table 2). The Johnson-Neyman technique revealed that for values of suppression (mean-centered) greater than -0.054 , the conditional effect of combat on memory was increasingly negative and remained statistically different from zero. For values of suppression less than or equal to -0.054 , the conditional effect was not statistically significant. Therefore, greater combat exposure significantly predicted worse late life memory only in those with who relied more on suppression, and in those who relied less on suppression there was no association between combat exposure and late life memory (Figure 1).

The overall model with repression proposed as a moderator showed a main effect that explained 19% of the variance ($p < .001$) in late life memory function (Table 2). The regression coefficient for the product of repression and combat was also statistically

different from zero ($-0.220, p < 0.05$), meaning that the effect of combat exposure on memory depended on the level of repression. In this case, the moderation component of the model explained 8.5% of the variance (Table 2). The Johnson-Neyman technique revealed that for values of repression (mean-centered) less than 0.201, the conditional effect of combat on memory was increasingly negative and remained statistically different from zero. For values of repression greater than or equal to 0.201, the conditional effect was not significant. Therefore, greater combat exposure significantly predicted worse late life memory only in those with less repression, and in those who relied more on repression there was no association between combat exposure and late life memory (Figure 2). This suggests repression may have been somewhat protective depending on the level of combat exposure.

4. Discussion

This study uses longitudinal data spanning 6 decades to examine how habitual emotion regulation strategies may interact with an earlier life stressor to predict cognitive health. This investigation seeks to contribute to our understanding of how particular coping styles may affect long-term well-being, even decades after a stressful life event. To our knowledge, this is the first study to explore late-life cognition related to earlier adult combat exposure and defense mechanisms in WWII veterans. We proposed that reliance on defense mechanisms that involve manipulation of memory would be linked with memory functioning in late life, and that they might buffer or exacerbate the effects of combat exposure in young adulthood.

Strengths of this investigation include the use of multiple methods (objective testing, self-reports, multiple assessors, validated defense ratings) in a cohort with data collection in multiple domains for over 75 years. Our investigation yielded three main results. First, higher levels of suppression in young adulthood were associated with poorer memory function in later life. Second, greater reliance on suppression after exposure to combat in young adulthood amplified negative associations between combat exposure and late-life memory functioning. Third, although combat exposure predicted poorer late-life memory functioning among the group of 71 men in this study, those individuals who relied on the use of repression to manage stressful situations were buffered from the effects of combat exposure on memory. Of particular note is the fact that these associations were present across decades of temporal separation between combat exposure and late-life cognitive testing.

Prior work that suggests the adaptive or maladaptive nature of certain defense mechanisms is robust, involving both experimental and observational studies [45], but has been largely cross-sectional, leaving open the question of any long-term consequences of reliance on particular defense mechanisms despite the short-term relief they may provide; the current investigation in a longitudinal cohort attempts to fill that gap. This study brings a novel neurocognitive perspective to how reliance on specific coping mechanisms may impact cognition across the adult lifespan. Binary classifications such as engaging/avoidant or mature/immature do not allow for a nuanced view of how habitual patterns of processing thoughts and feelings in the brain may be cognitively healthier for one individual but not for another based on prior experience. It also raises the possibility that an emotion regulation strategy which benefits an individual psychologically in the short run might have untoward

longer-term effects. These results have important implications for stress-related mental health research and evidence-based foundations for psychological treatment.

How might suppression exacerbate links between exposure to stress and memory? From a contemporary neurobiological view, suppression is an executive control process that prevents mental content from elaborating in awareness [21]. As expected, suppression was not associated with late life executive functioning (e.g., trail making tests), because the conscious strategy of suppression is itself thought to be under executive control [21]. The CERAD protocol taps memory functions similar to those involved in the use of suppression – that is, encoding, storage, and retrieval. Therefore, objective memory tasks such as those in the CERAD may be sensitive to the effects of suppression on memory.

A critical difference between suppression and repression is that repression does not involve manipulation of memory processes in a conscious way [22]. Repressed content is not brought to awareness and subject to manipulations of retrieval. Accordingly, in our bivariate results we did not find links between reliance on repression and performance on objective tests of conscious memory. As expected, another adaptive defense – altruism – was not associated with memory. Altruism involves dealing with emotional conflict or internal or external stressors by dedication to meeting the needs of others [16], and it does not act predominantly on neurocognitive processes involved in memory.

We speculate that the finding that consistent use of suppression in the face of a significant early life stressor predicted poorer memory may be related to the role of the hypothalamic-pituitary-adrenal (HPA) axis in the neurobiological health of memory structures. However, we recognize that this speculation was not tested in our study and would require further, more focused investigation. The HPA axis has direct connections to the hippocampus [46], and the memory-modulation hypothesis notes that stress and high cortisol levels can have impairing or enhancing effects on memory [47]. Human studies have confirmed the detrimental effects on memory retrieval of both experimentally-manipulated cortisol treatment [48–50] and psychosocially-induced stress [51]. In addition, fMRI investigations show that cortisol treatment leads to reduced activation of the hippocampus during memory retrieval and decreased activation of the prefrontal cortex [52]. Moreover, the retrieval of conscious negative words appears to be more affected (reduced) than retrieval of neutral words if both are presented in the setting of cortisol administration [53]. Thus, conscious emotionally-valenced material might be especially sensitive to the compromising retrieval effects of cortisol. Taken together, established models have been proposed to suggest that glucocorticoids enhance memory consolidation but may also impair memory retrieval.

In contrast to the acute effects of stress hormones described above, Lupien and colleagues found that chronic elevation in basal cortisol in older individuals is associated with reduction in hippocampal volume and memory deficits [54]. They concluded that basal cortisol elevation might cause hippocampal damage and impair hippocampus-dependent learning and memory in humans over time. With regard to our participants, the presence of a psychological threat may have stimulated two processes: 1) whether or not a physiologic stress response would be deployed, and 2) which psychological defense would be utilized. In those who predominantly used repression, we speculate that their physiologic stress

response may have been less since the threat wasn't conscious. In those who predominantly used suppression, perhaps a stress response, with more potential for cortisol exposure to brain structures, was concomitantly deployed since the threat was conscious. In addition, the relationship between a given defense mechanism and the reactivity of the stress response may have been affected if an extreme stressor significantly engaged the HPA axis earlier in life, such as combat exposure. This proposed speculation of a mechanism is only one possible explanation for our findings; other possibilities could include chronically higher levels of physical and/or psychological stress in those who relied on suppression, and/or poorer memory performance at baseline in those who happened to rely on suppression throughout their lives.

Although suppression is traditionally categorized as an adaptive coping mechanism that allows one to delay attending to difficult content, in certain contexts postponement of processing difficult content may set a path for adverse neurobiological consequences. This may be in line with current efforts at utilizing mindfulness in the military to engage with upsetting content more immediately in a more flexible manner that also validates difficulties rather than putting them aside for an undetermined later time point [10,11]. Correspondingly, in some contexts repression may be neurobiologically protective insofar as it may prevent threat perception and concomitant HPA production of cortisol.

The study has limitations that are important to note. Only 71 men were included in these analyses, a subset of the 268 in the original cohort who had survived and were willing and able to participate in neuropsychological testing in their 80s. The fact that all were white males from middle and upper-class backgrounds from one historical cohort limits the generalizability of our findings. In addition, our combat exposure scale is limited to what would be 'Criterion A' (nature of the trauma exposure) of Post-Traumatic Stress Disorder and therefore leaves unclear if any symptomatic response (i.e., Criterion B-E) was experienced and might have been addressed by defense mechanisms. Furthermore, we did not assess the particular types of psychological content (e.g., content related to combat) that triggered the use of suppression or repression, nor did we have measures of stress hormones or serial neuropsychological testing to see relationship trends between use of defenses, memory performance, and physiology over time. To our knowledge, this is the first investigation to show a potential untoward cognitive effect of suppression in the face of a major life stressor, and warrants replication to further validate this finding. Taken together, these findings constitute evidence that strategies of emotion regulation may affect the brain in specific ways that can be harmful or protective. This study is the first to our knowledge to study the long term cognitive effects of coping styles, and suggests the importance for further understanding of how particular styles of coping may impact cognitive functioning as people move through adult life. A potential important next step would be to examine the extent to which these findings could be supported within an in vivo context, perhaps with physiological measurement of stress utilizing different regulation strategies, and examine these effects over time.

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Figure 1. Suppression as a moderator of combat exposure level on mean score CERAD memory composite in late life

Figure 2. Repression as a moderator of combat exposure level on mean score CERAD memory composite in late life

Table 1

Means, Standard Deviations, and Pearson Correlations N=71

	1	2	3	4	5	6	7	8	9
1. Suppression	--								
2. Altruism	0.05	--							
3. Repression	-0.30*	0.15	--						
4. Displacement	-0.08	0.14	-0.17	--					
5. Word Recall	-0.27*	-0.14	0.03	0.12	--				
6. Delayed Recall	-0.25*	-0.11	-0.04	0.14	0.68**	--			
7. Word Recognition	-0.25*	-0.20	0.03	0.16	0.48**	0.67**	--		
8. CERAD Composite	-0.30*	-0.17	0.01	0.16	0.83**	0.92**	0.83**	--	
9. Combat Exposure Scale (SQRT transformed)	0.16	-0.04	-0.07	-0.21	-0.28*	-0.25*	-0.31**	-0.32**	--
Mean	3.28	2.23	2.37	2.44	17.56	4.46	8.45	10.16	0.79
SD	0.85	1.10	0.98	0.97	4.13	2.45	1.74	2.41	1.06

* p<.05,

** p<.01 (2-tailed)

Table 2

Regression Analyses Predicting CERAD Memory Performance

Model 1: Suppression (moderator)						
Model Summary	R^2	MSE	F	df1	df2	p
	0.24	0.51	7.01	3.00	67.00	< .001
Model	p					
Constant	b	SE b	t	1.798	0.077	
Suppression (centered)	0.154	0.086		-2.611	0.011	
Combat (SQRT; centered)	-0.248	0.095		-2.176	0.033	
Suppression $\times$ Combat	-0.186	0.086		-2.859	0.006	
Note: R^2 increase due to interaction is 0.078 ($p < .01$)						
Model 2: Repression (moderator)						
Model Summary	R^2	MSE	F	df1	df2	p
	0.19	0.55	6.39	3.00	67.00	< .001
Model	p					
Constant	b	SE b	t	1.446	0.153	
Repression (centered)	0.133	0.092		-0.188	0.851	
Combat (SQRT; centered)	-0.017	0.089		-2.593	0.012	
Repression $\times$ Combat	-0.250	0.096		2.480	0.016	

Note: R^2 increase due to interaction is 0.085 ($p < .05$)