HHS Public Access

Author manuscript

Aging Ment Health. Author manuscript; available in PMC 2023 May 01.

Published in final edited form as:

Aging Ment Health. 2022 May ; 26(5): 971–979. doi:10.1080/13607863.2021.1904829.

Subjective Memory in Adults Over 50 Years of Age: Associations with Affective and Physiological Markers of Emotion Regulation

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Abstract

Objectives: To examine associations among subjective memory reports, psychophysiological markers of emotion regulation, and cognitive performance in healthy adults over 50 years of age.

Method: A cross-sectional laboratory study was conducted with healthy, community-dwelling, non-depressed adults (*M* age = 60.4 years, *SD* = 8.4). The Metamemory in Adulthood (MIA) questionnaire provided reports of subjective memory capacity and stability (versus decline) and anxiety about memory. Poorer emotion regulation was marked by greater negative affect (NA) and lower high frequency heart rate variability (HF-HRV) responses to a challenging working memory task. Regression models were used to identify associations between subjective memory and emotion regulation markers, and structural equation modeling was used to explore whether emotion regulation mediated associations between subjective memory and objective task performance.

Results.—A total of 115 participants were included in the final sample. Subjective memory decline (indicated by lower scores on memory stability) was associated with lower HF-HRV response and worse working memory performance. Poorer subjective memory capacity and more anxiety about memory were both associated with greater negative affect in response to the working

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Disclosure statement

The authors have no conflicts.

memory task. There was an indirect effect of subjective memory capacity on working memory performance through negative affect response.

Conclusions.—The findings here suggest that worse subjective memory may signal reduced capacity for emotion regulation. Along with known cognitive risks of depression and anxiety, more subtle emotion regulation difficulties may be involved in pathways of poor cognitive aging.

Keywords

subjective memory; emotion regulation; affect; cognitive aging; older adults

Introduction

Subjective memory concerns in otherwise healthy adults are predictors of future cognitive decline and dementia (Mitchell et al., 2014), although findings are mixed and mechanisms remain elusive (Jessen et al., 2014). Notably, subjective memory concerns, including subjective memory decline and poorer subjective memory capacity, are associated with amyloid-beta deposition and neurodegeneration revealed by neuroimaging (Perrotin et al., 2012; Sun et al., 2015). Poorer subjective memory in cognitively normal older adults are also concurrently associated with poorer emotional well-being, including depression and anxiety (Balash et al., 2013; Norman et al., 2020; Tanaka et al., 2016), which increases risk for poorer cognitive aging (Beaudreau & O'Hara, 2008; Bunce et al., 2014; Perna et al., 2016). Many studies have examined associations between subjective memory and depressive symptoms and anxiety, including anxiety about memory (S. T. Chen et al., 2014; Irak & Capan, 2018; Verhaeghen et al., 2000b), as well as worry about everyday cognitive errors being signs of future dementia (Kinzer & Suhr, 2016; Lee et al., 2020). Subjective memory concerns may reflect depressive symptoms (Zlatar et al., 2018) or dementia worry (Kinzer & Suhr, 2016) more than objective cognitive difficulties. The degree to which subjective memory is associated with emotion regulation, however, is less understood. Identifying emotion mechanisms in cognitive decline and dementia can advance prevention and intervention efforts that target modifiable risk factors (Jessen et al., 2014; Marchant et al., 2020). In the current study, we examined whether subjective memory was associated with affect and physiological markers of emotion regulation in cognitively and emotionally healthy adults over 50 years of age.

Emotion regulation is the process of modifying, either consciously or unconsciously, the valence or intensity of emotion in response to environmental demands (Gross, 2015). Evidence suggests emotion regulation may represent a plausible mechanism, and potential modifiable treatment target, in pathways linking subjective memory to dementia risk. Self-perceptions of poor memory, in the absence of objective memory decline, are associated with a host of phenotypes reflecting poor capacity for emotion regulation, including trait neuroticism (Jenkins et al., 2019; Merema et al., 2013; Pearman & Storandt, 2004; Reid & Maclullich, 2006), greater anxiety and worry about memory (Irak & Capan, 2018; Mol et al., 2008; Verhaeghen et al., 2000a) or worry about having/developing dementia (Kinzer & Suhr, 2016; Lee et al., 2020), and greater negative affect reactivity in response to memory failure (Moraitou & Efklides, 2009). Conversely, subtle cognitive deficits in healthy older adults (Mather, 2012) and poorer cognitive aging (Charles, 2010) can underpin

emotion dysregulation. This suggests important bi-directional associations between emotion and cognitive dysregulation. Together, apart from clinical symptoms of depression, poor subjective memory may reflect underlying emotion regulation difficulties or dysfunction that, if untreated, may accelerate risk for cognitive decline.

Understanding how subjective memory relates to physiological markers of emotion regulation may reveal additional pathways to poor late-life cognitive outcomes. Contemporary psychophysiological models of adaptation and self-regulation suggest shared neural underpinnings of cognitive, emotion, and physiological regulation (Charles, 2010; Mather, 2012; Thayer & Lane, 2009). Specifically, brain regions and networks that support cognitive abilities also play a direct role in regulating emotion and activities of the autonomic nervous system (ANS) (Lane et al., 2009; Thayer & Lane, 2009). Adaptive, flexible regulation by the ANS can be non-invasively measured by high frequency heart rate variability (HF-HRV) (Gianaros et al., 2004; Hansel & von Kanel, 2008; Lane et al., 2009), a marker of cardiac control by the parasympathetic branch of the ANS (Berntson et al., 1997; Lane et al., 2009). Both higher resting HF-HRV, and greater responsivity of HF-HRV to psychosocial or changing environmental demands, are associated with better emotional well-being (Kogan et al., 2013; Rottenberg, Clift, et al., 2007), including more positive emotions and lower depressive and anxiety symptoms (Beauchaine, 2001; Kok & Fredrickson, 2010; Rottenberg, Chambers, et al., 2007), as well as better emotion regulation, including in non-depressed older adults (Mather & Thayer, 2018). Conversely, lower HF-HRV during acute laboratory stressors may indicate an individual's poorer capacity for ANS regulation in response to challenge (Kim et al., 2018), as greater parasympathetic cardiac control keeps heart rate from increasing to unnecessarily high levels during the challenge. Given the overlap of ANS and emotion regulation, lower HF-HRV in response to a challenge or stress may suggest a concurrently poorer capacity to regulate emotion.

Resting and dynamic changes in HF-HRV also covary with cognitive function in healthy adults. Better performance on measures of global cognition and executive function is associated with higher resting HRV, including HF-HRV (Frewen et al., 2013; Lin et al., 2014; Thayer et al., 2009), and greater HF-HRV responsivity during tasks is also associated with better task-related executive function and attention (Duschek et al., 2009; Mathewson et al., 2010). Cognitive challenges that diminish attentional control simultaneously reduce HF-HRV responsivity to changing environmental cues (i.e., emotionally negative stimuli) (Park et al., 2014), further underscoring the overlap in neural pathways regulating the ANS and cognitive abilities. No studies have yet investigated the possible role of HF-HRV in pathways linking subjective memory to cognitive risk.

Taken together, individuals reporting worse subjective memory and memory decline have phenotypic characteristics consistent with underlying emotion dysregulation, irrespective of objective and clinically meaningful memory deficits or impairment. Further, cognition, emotion, and ANS regulation have common neural underpinnings. Thus, poor subjective memory, if indeed a reflection of emotion regulation difficulties, may be associated with more anxiety about memory generally, but also less well-regulated negative affect, and concurrently poorer physiological regulation marked by less HF-HRV, in response to cognitive challenge.

To shed further light on cognitive-emotional pathways that may be tied to later life cognitive decline, we examined associations among subjective memory and affective and physiological markers of emotion regulation in cognitively healthy, non-depressed adults over age 50. Guided by evidence for the shared neural underpinnings of emotion and cognitive regulation, we hypothesized that in healthy middle and older aged adults, worse subjective memory signals emotion regulation difficulties that are concomitant with poorer cognitive performance. If so, integrated deficits in cognitive-emotion regulation may further indicate who, over time, may be most vulnerable to poor cognitive outcomes. We used archived data from a prior study of older adults' physiological responses to cognitive tests. The study had available measures of multiple domains of subjective memory from a well-validated questionnaire (Dixon et al., 1988), including subjective memory stability, capacity, and anxiety. Lower scores on subjective memory stability reflect self-perceptions of greater memory decline over time and age, whereas subjective memory capacity reflects one's self-perception of memory ability (poor versus good). These subscales afforded indicators of two domains relevant to the current recommended definition of subjective memory decline (Jessen et al., 2014), which includes perceptions of memory decline over time, as well as one's subjective memory capacity relative to similarly aged peers. We also had available a measure of anxiety about memory, providing an index of more trait-like worry about memory, which is also recommended for consideration in studies of subjective and objective memory (Jessen et al., 2014). Specifically, we tested whether worse subjective memory (more decline, less capacity, and greater anxiety), was associated with poorer emotion regulation (greater negative affect and lower HF-HRV responses). Next, we explored the potential role of emotion regulation in associations between subjective and objective memory. That is, we used mediational path modeling to test the hypothesis that worse subjective memory would be associated with poorer emotion regulation which, in turn, would be associated with worse performance on the working memory task.

Materials and Methods

Participants

Healthy men and women (N = 123), ages 50 years and older, were recruited through community advertisements for a study of stress and memory. Exclusion criteria included factors that could affect cognitive health as well as cardiovascular, metabolic, and immune measures which were of interest in the parent study: having immune, cardiovascular or endocrine–related health problems (e.g., pacemaker, stroke, recent surgeries, cancer, diabetes); current smoker; evidence of global cognitive impairment (using the Repeatable Battery of Neuropsychological Status (Randolph et al., 1998); overall RBANS composite score below 20th percentile for participant's age and education, or performance on two or more RBANS index scores below 20th percentile for participant's age and education); reported more than 14 alcoholic drinks per week; were more than 30% above ideal weight or more than 10% below; reported needle or blood phobias; reported a diagnosis or showed evidence of major depression (Geriatric Depression Scale score > 20; (Yesavage et al., 1982); reported current use of psychotropic medications, including anti-depressants or anxiolytics. Of the initial sample, 115 participants completed two in-laboratory sessions and constitute the sample for the current study. The mean age of the sample was 60 years

(*SD* = 8.4; range: 50 – 87 years). The sample was predominantly female (*N* = 66, 57.4%) and White, Non-Hispanic (93.9%); followed by African American (5.2%); White, Hispanic (.9%); and Pacific Islander or Alaska Native (.9%) participants. The internal review boards at Ohio University and the University of Rochester approved the study; all participants provided written informed consent prior to participation.

Procedure

Complete study procedures are detailed elsewhere (Heffner et al., 2012). After an initial session where informed consent, a demographics questionnaire, and screening for cognitive impairment were administered, those individuals remaining eligible to participate in the study were provided with questionnaires to complete at home (including the subjective memory measure of interest), and asked to return them approximately one week later at a laboratory study session. All study sessions started between 1:00 and 3:00 p.m. to control for diurnal variation in cognitive performance and physiological measures. Participants provided electrocardiography (ECG) and blood pressure measures (recorded throughout the session), completed self-report measures, and then sat quietly for a 30-min adaptation period. After a reading task (irrelevant to the current study), participants were administered a standard, neuropsychological auditory verbal learning test, and then a subsequent challenging test of working memory, the Auditory Consonant Trigram Task (ACT; (Strauss et al., 2006)), chosen by design as an acute, highly demanding cognitive task to provoke physiological arousal. Participants reported their current affect before and after the cognitive tests.

Measures

Subjective memory—Participants completed the Metamemory in Adulthood (MIA) questionnaire (Dixon et al., 1988), a measure of overall self-knowledge and self-perceptions about one's own memory skills and memory more generally. The questionnaire consists of 108 items comprising seven different subscales. Participants respond to items on a 5-point Likert scale. The three MIA scales used to operationalize subjective memory in this study were memory capacity (self-perceptions of one's own memory capacity; 17 items; Cronbach's alpha = .77), memory stability (perceived stability (versus decline) in memory capacity with time and age; 18 items; Cronbach's alpha = .91), and memory anxiety (anxiety about one's own memory capacity; 11 items; Cronbach's alpha = .85). For the memory anxiety subscale, we included 11 items that reflected anxiety about one's own memory performance to align conceptually with our interest in emotion regulation difficulties; we excluded 3 items related to more general knowledge about how anxiety affects memory function, as this was not the focus of the current study. Four of the MIA subscales (knowledge and use of memory strategies, knowledge of basic memory processes, motivation to perform well in memory tasks, and perceived sense of control over memory) were excluded from analyses, as these scales were unrelated to the aims of the current study.

High Frequency (HF)-HRV—To obtain HF-HRV, a Biopac MP100 (Biopac, Inc) was used to collect the electrocardiogram (ECG) signal (samples at 1000 Hz) using a standard lead II patient spot electrode configuration. Commercial software (MindWare Technologies, Inc) was used to process data as described previously (Berntson et al., 1997). In brief, a series of intervals between consecutive R waves – the interbeat intervals -- were inspected

and artifacts removed. The heart period time series was band-pass filtered (from 0.12 – 0.40 Hz) and underwent Fast Fourier transform power spectral analysis and scaled to $msec^2/Hz$. HF-HRV data were expressed in absolute units and then natural log transformed to better approximate normalization. The average HF-HRV across the last 5-minutes of the adaptation period, and the average HF-HRV across the 10-minute ACT task, were available for analyses.

Negative affect—The 10-item negative affect (NA) subscale of the 20-item Positive and Negative Affect Schedule (Watson et al., 1988), a well-validated and reliable measure of current state affect, was used to evaluate NA response to the cognitive tests, with NA measured prior to and immediately after (post) cognitive testing. Previous research shows support for its use with adults over the age of 50 (Kercher, 1992). Cronbach's alpha was .66 and .83 at baseline and post-task, respectively.

Working memory performance—The Auditory Consonant Trigram test (ACT; (Strauss et al., 2006)), also known as the Brown-Peterson test, is a challenging cognitive task that measures auditory working memory under high cognitive load. Individuals listen to a string of three consonants and are immediately requested to count backwards aloud by threes from different numbers during intervals of 0, 9, 18, and 36-sec. Individuals are then asked to recall all three letters. ACT total score, providing a measure of working memory performance, is calculated as the total number of correctly recalled letters across all trials.

Data analysis

Multiple regression was used to identify significant subjective memory predictors of NA and HF-HRV responses, operationalized as post-task NA or HF-HRV during ACT controlling for their respective baseline values in the models; a multiple regression model was likewise used to test subjective memory predictors of ACT performance. In all instances, the Mplus structural equation modeling software (Muthen & Muthen, 1998–2017) was used. Mplus makes use of the full information maximum likelihood (FIML) approach for the handling of missing data, using estimates throughout the model to provide more accurate estimates where data are missing. Full information estimation provides more realistic parameter estimates than other missing data techniques (e.g., listwise, pairwise, mean imputation) (Arbuckle, 1996), provided data is missing at random or missing completely at random. In each regression equation, MIA subscale scores for memory capacity, memory stability, and memory anxiety were included as predictors. When examining psychophysiological responses to cognitive challenge (as an indicator of emotion regulation), the baseline value of the dependent variable (for NA and HF-HRV) was entered as a predictor in the respective model. Age and education were included as covariates in all models. As a form of sensitivity, we also ran the models including gender as a further test of robustness of the findings.

To better understand the potential role of emotion regulation in links between subjective and objective memory, mediational path models were used to test whether NA or HF-HRV response (post-measure adjusted for baseline measure) mediated potential associations between MIA subscale scores and ACT total score. The indirect, mediational effects were

assessed using bias-corrected bootstrap confidence limits (BCCI) (MacKinnon et al., 2004). Significance of the indirect effects was assessed by whether or not the 95% confidence limit contains zero. This approach takes the non-normality of the multiplicative distribution into account (resulting in asymmetric confidence limits) and has been shown to provide the most accurate confidence limits and improved statistical power when compared with other approaches for detecting mediation (MacKinnon et al., 2004; Williams et al., 2008).

Results

Sample Characteristics and Correlations

Table 1 presents the means, standard deviations, and correlations among the study variables. Correlational analysis revealed that older age was associated with lower HF-HRV both at rest and during the ACT task, but showed only a small but non-significant association with ACT total score. Having more years of education was associated modestly with lower subjective memory anxiety, higher memory capacity, stability, ACT total score, and lower NA following the tasks. As expected, for NA and HF-HRV, their baseline and post-task values were moderately to largely correlated, respectively. Higher ACT total score was associated with lower NA following the tasks. In analysis of participants with data from both baseline and post-task NA, and HF-HRV during ACT, paired t-tests indicated a significant increase in NA from baseline to post-tasks (baseline $M = 10.94$, $SD = 1.67$; post-task $M = 12.91$; $SD = 3.52$; $t(109) = -6.77$, $p < .001$); HF-HRV during ACT was incrementally though non-significantly increased during ACT compared to baseline values (baseline $M = 5.08$, $SD = 1.18$; during ACT $M = 5.21$; $SD = .99$; $t(103) = -1.58$, $p = .12$). There were no gender differences observed (all p's > .10).

Associations between subjective memory and markers of emotion regulation, and working memory performance

Table 2 presents results from the multiple regression analyses for each of the three outcome variables of interest. Less anxiety about memory and greater subjective memory capacity were associated with lower NA following cognitive challenge, adjusted for baseline NA. For HF-HRV, more subjective memory stability was associated with higher HF-HRV during the ACT; greater perceived memory capacity showed a negative association with HF-HRV, although this did not reach significance. Perceiving memory stability was also associated with better ACT performance. As a form of sensitivity, we re-tested the linear regression models with gender included; the substantive findings were unchanged.

Mediation model of subjective memory and working memory performance via negative affect

We also explored a path model examining whether our affective and physiological markers of emotion regulation mediated relationships between subjective memory and memory anxiety, and ACT total score. Figure 1 illustrates the findings for mediation by NA. Accounting for the mediating effects of post NA controlling for pre NA, the strength of the direct association noted in the multiple regression between memory stability and ACT total score (beta = .25) was incrementally larger in this mediational model (beta = .27), although no longer statistically significant (p = .09). The remaining source variables

had no statistically significant direct effects on ACT total. Indirect effects, however, were found. Bias-corrected bootstrap confidence intervals indicated a significant indirect effect of subjective memory capacity on ACT score through post NA adjusted for pre NA (indirect effect = .048; 95% BCCI = .002 to .157). Because worse performance on the task was associated with post-task NA in unadjusted correlation analyses, we also tested an alternative mediational pathway to evaluate whether subjective memory had indirect effects on post-task NA through ACT performance. We did not find support for this alternative model, as none of the subjective memory source variables were significantly related to the mediating variable of ACT total score (see Data, Supplemental Digital Content 1, for full model results). A path model testing mediation by HF-HRV was not supported; similar to the model for NA as the mediator, the strength of the direct association between memory stability and ACT total score was similar in this mediational model (beta = .29) to that from the multiple regression, although also no longer statistically significant (p = .17).

Discussion

Subjective memory concerns are a risk factor for later cognitive decline, even in the absence of objectively-measured, clinically meaningful cognitive deficits. Little is known about the mechanisms of this association. We explored here whether subjective memory concerns -- including subjective memory decline, capacity, and anxiety -- may signal poorer capacity for emotion regulation, a possible underlying contributor to cognitive risk. We found among cognitively healthy, nondepressed adults ages 50 years and older that having poorer subjective memory capacity, but also more anxiety about memory, were each independently associated with greater negative affect in response to a cognitive challenge. In addition, greater subjective memory decline (as indicated by lower scores on memory stability) was associated with lower HF-HRV during a challenging working memory task. More generally, these novel findings suggest that poorer subjective memory, in a sample of cognitively healthy individuals without depression, may nonetheless reflect a lower capacity for emotion regulation.

Notably, however, negative affect and HF-HRV responses were each associated with different domains of subjective memory. Future work is necessary to determine the reliability of these findings; however, from basic studies of metamemory, self-reported negative mood and emotion appear reliably associated with self-evaluations of worse memory capacity in healthy samples (Efklides, 2016). How these findings relate to subjective memory decline, as a self-evaluation of one’s current performance relative to past performance, rather than self-evaluation of current memory performance abilities (good versus poor), is unknown. To index emotion regulation, we used a self-report measure of negative affect, and HF-HRV, a physiological marker of ANS regulation that has been associated with both emotional well-being and cognitive function. We found in supplementary analyses that resting HF-HRV was associated with lower negative affect response to the task in our sample (data not shown). This aligns with previous support for resting HF-HRV as an indicator of better capacity for emotion regulation (e.g., Mather & Thayer, 2018), and lends support for our emotion regulation indicators. Nonetheless, there may be differing pathways through which subjective memory domains relate to cognitive risk. We offer speculation based on our novel investigation that worse subjective

memory capacity, as well as anxiety about memory, may signify greater dysregulation of negative emotion domains, whereas subjective memory decline may signal greater ANS dysregulation, either of which may reflect or underlie cognitive risk. Similar to other calls for more rigorous understanding of subjective memory (e.g., Jessen et al., 2014), we suggest greater attention be devoted to how different subjective memory domains play a role in future cognitive decline.

We further explored whether subjective memory was associated with performance on the working memory task and if our psychophysiological markers explained any associations. We found that greater subjective memory decline (i.e., as indicated by lower scores on memory stability) related to worse performance on the working memory task. We did not find that HF-HRV during the ACT mediated this association, even though better subjective memory stability was moderately associated with higher HF-HRV during ACT in the regression model. In the full path model, negative affect response mediated the associations between subjective memory capacity and ACT performance. In further support of this pathway, we did not find evidence for an alternative pathway whereby poor subjective memory predicted worse working memory performance, which in turn resulted in more negative affect. It is important to underscore that negative affect response (operationalized as post negative affect adjusted for baseline negative affect in our path model) was found to statistically mediate – or explain – the relationship between subjective memory (capacity, memory anxiety) and ACT performance. To be able to assess whether negative affect response causally mediates this relationship (that is, as a temporal mediator), assessment of ongoing changes in negative affect during the ACT would be required. Nonetheless, given the observed shared neurobiological underpinnings of affect regulation and cognitive function, including working memory, our findings suggest that negative affect response is concomitant with poorer working memory capacity. Altogether, we suggest that subjective memory capacity and anxiety signal a poorer ability to regulate negative affect, which may explain -- or contribute to -- poorer working memory.

Overall, there may be a role for more subtle emotion dysregulation in understanding causal contributors to longer-term cognitive decline. A large body of research suggests depression explains associations between subjective and objective memory; however, some contradictory evidence exists (Brailean et al., 2019), and trait neuroticism may be more closely tied to subjective memory complaints than depression (Merema et al., 2013). Apart from depression, there may be value in considering more nuanced emotion mechanisms, as worse subjective memory may signal more subtle emotion regulation difficulties, and potential subtle cognitive deficits that underlie those difficulties (Mather, 2012).

There was no evidence from mediational analysis that HF-HRV response during the ACT mediated the association between subjective memory decline and working memory performance. Although exploratory in nature, this null finding may indicate that subjective memory decline operates through some other mechanism to explain its association with future cognitive decline. Alternatively, our cross-sectional findings cannot rule out a possible role of HF-HRV in the links between subjective memory decline and future cognitive decline. It is plausible that reduced self-regulatory capacity as indicated by HF-HRV may increase vulnerability to unhealthy cognitive aging. Future longitudinal work should address

whether increasingly worse subjective memory capacity or accelerated decline corresponds to declines in HF-HRV responsivity over time, and, subsequently, objective cognitive decline.

We did not observe any association between HF-HRV at rest or during ACT with ACT performance, replicating our prior findings (Lin et al., 2014). Others' work has shown that both resting HF-HRV and HF-HRV response to demanding tests of executive function are associated with test performance, supporting the common frontal cortex regulation of autonomic cardiac control and cognitive function (Thayer & Lane, 2009). Despite this, evidence suggest caveats to integrated central control of HF-HRV and cognition (Jennings et al., 2015). The current study used a task that required alterations between listening, albeit for a brief duration (listening for the trigrams for each trial), and vocalization (serial subtraction), which can impact HRV and is a limitation that was addressed by averaging HF-HRV across the task. Tasks that better control for movement and vocalization would allow for analysis of HF-HRV dynamics at a finer resolution, possibly revealing other ANS and cognition links. For example, we identified a pattern of nonlinear change in HF-HRV across 2-minutes of a computerized cognitive training that predicted better training outcomes (Q. Chen et al., 2020). Although HF-HRV and working memory performance were not associated in the current study, the current findings nevertheless suggest that subjective memory decline may provide a window into an individual's self- and emotion regulatory capacity, insofar as both HF-HRV during cognitive challenge, and better cognitive performance, each index this capacity (Thayer & Lane, 2009). As noted, longitudinal studies that examine correspondence between changes in subjective and objective memory, and changes in HF-HRV measures, would lend further understanding to the integrated nature of autonomic and cognitive regulation.

The current findings should be considered preliminary; however, they are novel and lend support for further examining whether and how components of emotion regulation contribute to the observed links between subjective memory and cognitive risk. It is notable that these associations were observed across cognitively and emotionally healthy middle and older aged adults. Identifying early indicators of future cognitive decline, before emotional and cognitive disturbances reach clinical significance, is sorely needed. Future studies designed to directly assess subjective perceptions of upcoming performance on cognitive tasks and subsequent objective performance on those tasks, alongside physiological and affective measures, may afford more direct evaluation of the links between subjective and objective memory performance, and emotion regulation. In addition, evidence suggests reciprocal influence of subjective memory decline and objective performance over time (Snitz et al., 2015). Thus, longitudinal models of the role of physiological and affective factors in cognitive change will need to consider this bi-directionality.

A limitation of the current study is its reliance on archival data from a prior study that was not designed to robustly address the current hypotheses of interest. The sample was healthy, well-educated, and predominantly non-Hispanic White, which limits generalizability. Further, the sample was on average around 60 years of age. Although a small noticeable decline in performance can begin at around age 50, greater cognitive decline is more prominent around age 70 years and older (Salthouse, 2009), which may help explain the

weak associations observed between age and ACT performance. As noted, the findings here should be used to guide future work that can more definitively advance understanding of pathways linking subjective memory and future cognitive impairment. Further, although we screened for depression, we did not screen or measure generalized anxiety. Thus, it is unknown whether anxiety about memory in the current sample reflected more general trait anxiety that may, in turn, affect emotion regulation and working memory performance. Future studies should clarify whether there are distinct roles for trait versus domain-specific anxiety about memory or dementia worry in pathways linking emotion dysregulation to cognitive outcomes. Because of interests in physiological stress regulation in the parent study, we used a challenging working memory task (ACT) that provokes physiological arousal (Heffner et al., 2012). Although we did find one of our subjective memory domains (i.e., decline) to predict working memory performance, a comprehensive assessment of memory function and other cognitive domains requires consideration for a complete picture of mechanisms linking subjective and objective memory decline.

In conclusion, we found evidence for psychophysiological correlates of subjective memory. Continued study of plausible emotion regulation pathways is needed to more fully understand whether and how subjective memory is predictive of future cognitive decline and dementia. Further understanding of these pathways may afford novel intervention targets to reduce risk for accelerated cognitive decline or dementia and poor emotional-being in later life.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments:

The research was supported by NIH/National Institute on Aging under grant R03 AG030029, and a research supplement to promote diversity in health-related research to parent award R01 AG049764.

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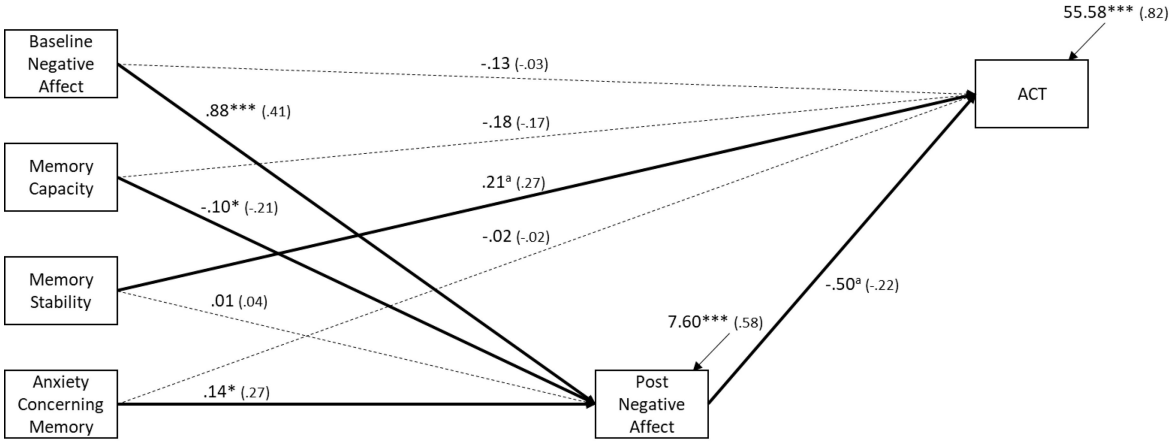

Indirect Effects:
Baseline Negative Affect -> Post Negative Affect -> ACT (95% BCCI = -1.182, -.056)
Memory Capacity -> Post Negative Affect -> ACT (95% BCCI = .002, .157)
Memory Stability -> Post Negative Affect -> ACT (95% BCCI = -.080, .023)
Anxiety Concerning Memory -> Post Negative Affect -> ACT (95% BCCI = -.214, .001)

Figure 1.

SEM model of post negative affect mediating baseline negative affect, memory capacity, memory stability, and anxiety concerning memory associations with ACT score (unstandardized regression parameters presented (standardized in parenthesis); significant paths bolded, non-significant paths dashed). Age and education effects omitted for clarity as were correlations among the source variables. *** $p < .001$; ** $p < .01$, * $p < .05$, [a] $p < .10$. ACT = Auditory Consonant Trigram Task; BCCI = Bias-corrected bootstrap confidence interval.

Table 1.

Study means, standard deviations, and correlations.

		N	mean	SD	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1.	Age, years	114	60.38	8.405	1.00									
2.	Education, years	115	15.13	3.249	−.121	1.00								
3.	Anxiety about Memory	106	30.96	6.994	−.013	−.236*	1.00							
4.	Memory Capacity	102	55.73	7.750	−.038	.238*	−.481**	1.00						
5.	Memory Stability	102	51.60	10.650	−.161	.211*	−.541**	.555**	1.00					
6.	ACT	112	43.49	8.277	−.182	.215*	−.224*	.133	.309**	1.00				
7.	Baseline Negative Affect	110	10.94	1.672	.017	−.151	.268**	−.136	−.093	−.145	1.00			
8.	Post Negative Affect	113	12.98	3.606	.007	−.243**	.458**	−.377**	−.243*	−.285**	.499**	1.00		
9.	HF-HRV at rest	106	5.08	1.160	−.249*	−.084	.064	−.056	.080	−.021	.151	−.083	1.00	
10.	HF-HRV during ACT	105	5.19	1.015	−.233*	.007	.044	−.060	.223*	.007	−.031	−.140	.725**	1.00

Note: ACT = Auditory Consonant Trigrams; HF-HRV = High frequency heart rate variability

** p < .01

* p < .05.

Missing data for subjective memory measures due to participants returning incomplete questionnaires; missing data for self-reported negative affect and HF-HRV due to technical error (e.g., missed self-report assessment, equipment failure).

Table 2.

Regression results predicting negative affect and HF-HRV response, and ACT score

	Post Negative Affect		HF-HRV during ACT		ACT score	
Predictor	***b***	***beta***	***b***	***beta***	***b***	***beta***
(Intercept)	5.13		1.66		46.29***	
Age (years)	.01	.01	−.00	−.03	−.13	−.13
Education	−.09	−.07	.01	.04	.49	.16[a]
Baseline negative affect	.88	.41***	--	--	--	--
Baseline HF-HRV	--	--	.59	.68***	--	--
Memory capacity	−.11	−.22*	−.02	−.14	−.15	−.14
Memory stability	.01	.04	.03	.27*	.20	.25*
Anxiety about memory	.14	.26**	.01	.08	−.13	−.12
Variance Explained (R^2)	.43		.58		.14	

Note: HF-HRV = high frequency heart rate variability; ACT = Auditory Consonant Trigrams; n ranges from 105–113 due to missing data for some variables.

*** p < .001

** p < .01

* p < .05

[a] p < .10.