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Cognitive and Vascular Risk Factors for Depression: Testing an Integrated Theoretical Framework

Rosanna Scott
University of Central Florida

COGNITIVE AND VASCULAR RISK FACTORS FOR DEPRESSION: TESTING AN INTEGRATED THEORETICAL FRAMEWORK

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ROSANNA G. SCOTT
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ABSTRACT

Objective: Theoretical models that have guided the study of later-life depression include the vascular depression hypothesis, depression-executive dysfunction syndrome, and the CaR-FA-X model. Evidence suggests these can be integrated into a single developmental model of disordered mood (and its associated overgeneral memory feature) in later-life to delineate a mechanism of the vascular depression effect and identify modifiable intervention targets.

Methods: In older adults, four serial mediation models evaluated the relationships between (1) vascular burden and depressive symptoms via executive control and rumination, and (2) vascular burden and autobiographical memory specificity (AMS) via executive control and rumination. In younger adults, four simple mediation models were conducted to compare results to older adults, including models assessing the relationships between (1) executive control and depressive symptoms via rumination, and (2) executive control and AMS via rumination. Bias-corrected bootstrapping was employed throughout.

Results: Older adult n=56; younger adult n=63. Older adult serial mediation models demonstrated significant individual relationships between a working memory measure and depressive symptoms, as well as between rumination and depressive symptoms. The vascular depression effect neared significance. No other direct or indirect effects were supported. In younger adults, rumination was significantly associated with depressive symptoms; all other hypothesized relationships were not significant.

Conclusions: Model 1, evaluating the impact of vascular burden on depressive symptoms in older adults via working memory and rumination, respectively, was the most effective in integrating vascular depression, DED, and CaR-FA-X. However, there was not support for a vascular depression mechanism. Null results in this sample could be attributable to inadequate

power or measurement error. Clinically, results promote interventions that target older adults presenting with depression, executive dysfunction, or rumination, independently or combined.

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INTRODUCTION

Mood Dysregulation in Later-Life

Depression is the second leading cause of disability worldwide (Ferrari et al., 2013), and confers even greater risk for older adults. Prevalence estimates for major depression in elderly community samples ranges from 1-4%; however, the prevalence of clinically significant depressive symptomatology ranges from 8% to 16% (Blazer, 2003). While depressive symptoms are typically accompanied by disability across the lifespan, they are associated with substantial functional impairment and ominous physical health implications in later-life (Bruce, Seeman, Merrill, & Blazer, 1994; Yochim, Mast, & Lichtenberg, 2003). For example, Yochim et al. (2003) found that individuals with depressed mood spent more days sick in bed and made more visits to a medical doctor compared to their non-depressed counterparts. In a sample of adults in their 70s, results indicated that high depressive symptoms were associated with an increased risk of onset of disability with activities of daily living (Bruce et al., 1994). Given the commonality and substantial negative impact of depressive symptoms in later-life, it is necessary to identify modifiable risk factors for mood dysregulation to prevent and/or prolong pathology onset and improve prognosis.

Proposed pathological models of depression relevant in later-life include the vascular depression hypothesis and its associated depression-executive dysfunction syndrome, as well as the CaR-FA-X model (i.e., capture, rumination, functional avoidance, and impaired executive control). Evidence suggests that these models can be integrated into a single developmental model of disordered mood in later-life.

Vascular Depression

Extant research indicates an interrelationship between cerebrovascular risk factors such as diabetes, hypertension, and cardiovascular disease, and depressive symptomatology from mid-life to later-life (Scott & Paulson, 2018). Alexopoulos and colleagues (1997) proposed the vascular depression hypothesis, stating that vascular disease can "predispose, precipitate, or perpetuate" depressive symptoms among older adults (p. 915). The vascular depression hypothesis posits that cerebrovascular burden (CVB) contributes to neurological decrement in areas of the brain's white matter beyond that expected in the typical aging brain. Specifically, it is hypothesized that these lesions are located in neural regions recruited in emotion regulation and executive functioning (Alexopoulos et al., 1997). Thus, the presence of cerebrovascular disease and its associated neurological decrement precipitate maladaptive cognitive changes, which, in the early stages of a broad disease process, are manifested as the emergence of depressive symptoms. The vascular depression theory is supported by two complementary lines of research: neuroradiological findings and studies of clinical indicators of CVB.

With respect to neuroradiological findings, prior research indicates that known cardiovascular risk factors such as hypertension and atherosclerosis are associated with increased white matter hyperintensity (WMH) burden, the clinical hallmark of CVB (Manolio et al., 1994). A systematic review by Herrmann, Le Masurier, and Ebmeier (2008) found that later-life and late-onset depression were characterized by greater quantity and intensity of WMH. Individuals with late-onset depression are 2.57 times more likely to have periventricular WMHs than healthy controls, and 4.51 times more likely when compared to individuals with early-onset depression (Herrmann et al., 2008). In their study, odds ratios for deep WMHs mirrored those for periventricular WMHs: for WMH severity, compared with healthy controls and individuals with early-onset depression, those with late-onset depression demonstrated more severe WMHs in

periventricular (d=0.9 and d=0.73) and deep (d=0.46 and d=0.87) white matter, respectively (Herrmann et al., 2008). Coffey et al. (1990) not only found hyperintensities in depressed individuals' white matter, but also in the thalamus and basal ganglia, and subcortical hyperintensity severity was related to the presence of cerebrovascular risk factors. Even more compelling, Sneed and colleagues (2008) found that presence of deep WMHs yielded perfect sensitivity (1.00) and near-perfect specificity (.95) for the vascular depression subtype.

A review of the literature indicates limbic-cortical-striatal-pallidal-thalamic (LCSPT; more generally, frontal-subcortical) circuit involvement in emotional behavior given its anatomical connectivity with visceral control structures that mediate emotional expression (e.g., the hypothalamus and periaqueductal grey; Price & Drevets, 2010). The relationship between limbic structures and mood is long standing; for example, mood disorders are common in patients with basal ganglia diseases, including Parkinson's disease, supranuclear palsy, Huntington's disease, Meige's disease, Wilson's disease, and basal ganglia calcification (Sobin & Sackeim, 1997). Moreover, functional neuroimaging studies of depressed patients have demonstrated decreased prefrontal cortex blood flow and metabolism, as well as abnormalities in blood flow and metabolism in the basal ganglia (Soares & Mann, 1997) and increased reactivity in the amygdala to negative stimuli (Hamilton et al., 2012). Per the vascular depression hypothesis, disruption of the LCSPT due to CVB-related WMHs produces pathological emotional symptoms (i.e., depressive symptomatology).

The second line of research supporting the vascular depression hypothesis includes studies of clinical indicators of cerebrovascular disease, including diabetes, hypertension, and cardiac disease. Scott and Paulson (2018) found that, in a large longitudinal sample of adults followed from mid-life into later-life, depressive symptomatology was significantly positively

associated with the number of endorsed cerebrovascular risk factors from years before, even after controlling for prior depressive symptoms and demographic variables. The relationship between self-reported cerebrovascular risk factors and depressive symptoms remains even after controlling for comorbid general medical burden, such as physical disability and decreased cognitive functioning (Mast et al., 2008; Mast, Neufeld, MacNeill, & Lichtenberg, 2004). Furthermore, previous studies have found a threshold effect for number of cerebrovascular risk factors on depressive symptomatology, such that individuals with two or more cerebrovascular risk factors are at significantly greater risk of developing depressive symptomatology in later-life and its associated negative health outcomes compared to those with zero or one cerebrovascular risk factor (Mast, MacNeill, & Lichtenberg, 2004; Yochim et al., 2003).

Depression-Executive Dysfunction Syndrome

Alexopoulos and colleagues (2002) expanded on the vascular depression hypothesis, reporting that striatofrontal dysfunction not only contributes to the development of depressive symptoms, but also to the development of concurrent executive dysfunction; this was termed the depression-executive dysfunction syndrome (DED). In a sample of older adults, significant WMH burden was more frequent in individuals with late-onset depression compared to those without depression or with early-onset depression, and significant WMH burden was associated with poorer executive function (Lesser, Boone, Mehringer, & Wohl, 1996). No significant differences were found between minimal and high WMH burden groups in any other cognitive domain. Neuroradiological support for DED parallels that for vascular depression, in that WMHs develop in deep and periventricular white matter (Coffey et al., 1990; Herrmann et al., 2008; Sneed et al., 2008). Given that the striatum is heavily interconnected with the frontal cortex,

lesions in striatofrontal structures and projections could conceivably result in disruption of mood regulation and executive functioning.

Comorbid depression and executive dysfunction are particularly perilous for older adults given studies showing that executive impairment predicts poor or delayed response to antidepressants for geriatric major depression (Kalayam & Alexopoulos, 1999). Concurrent depression and executive dysfunction also predict the chronicity, relapse, and recurrence of geriatric depressive symptomatology, for which memory impairment, disability, medical burden, social support, or number of previous episodes are not predictors (Alexopoulos et al., 2000). Given this information, Alexopoulos and colleagues (2002) identified the clinical presentation of DED in later-life, comparing depressed elders with and without concurrent executive dysfunction. In this sample, 53 of 126 participants (42%) with later-life depression exhibited executive dysfunction (i.e., a score on the Initiation/Perseveration domain of the Dementia Rating Scale that fell one standard deviation below the mean; Alexopoulos et al., 2002). Results indicated that DED is characterized by reduced verbal fluency, impaired visual naming, psychomotor retardation, loss of interest in activities, and paranoia (Alexopoulos et al., 2002). Of note, the degree of executive dysfunction in depressed participants was positively associated with level of impairment with instrumental activities of daily living (Alexopoulos et al., 2002). The researchers acknowledge that, due to the descriptive nature of the study, results cannot establish that depression and executive dysfunction are pathogenetically linked (Alexopoulos et al., 2002); however, the theory and data supporting DED are compelling.

CaR-FA-X

Autobiographical Memory

Autobiographical memory is the recollection of personally experienced past events, and is a fundament of one's sense of self (Williams et al., 2007). Generative retrieval of autobiographical memories occurs when one intentionally attempts recall. The generative retrieval process was originally conceptualized as a top-down, iterative, three-step approach to memory retrieval, in which the first step is to identify the retrieval specification; this involves a description of the target memory (elaboration of a memory cue) and verification criteria against which memories will be compared (Norman & Bobrow, 1979). In the second stage memory records are accessed and selected for comparison against the verification criteria (Norman & Bobrow, 1979). In the third stage of generative retrieval, memory records are assessed against the verification criteria; if no memory meets the verification criteria, a new iteration of the process begins, and the cycle continues until the criteria are met (Norman & Bobrow, 1979). Further, retrieval specifications can be updated during the retrieval process based on intermediate information that is encountered and informative retrieval errors (Norman & Bobrow, 1979).

Nearly two decades later, researchers expanded the conceptualization of generative retrieval, positing that elaboration of the memory cue (i.e., the description, included in the retrieval specifications; stage 1) and the verification stage (stage 3) are modulated by supervisory executive processes, likely housed in the networks of the frontal cortex (Burgess, 1996; Conway, 1996). Included in these executive processes is an analysis of the task demands, which informs a mental model from which verification criteria are developed (e.g., making a strategic self-disclosure to a coworker versus to a significant other likely leads to generation of different criteria that fit one's goals; Conway & Pleydell-Pearce, 2000). Similarly, verification criteria will vary dependent upon the task at hand, serving to inhibit irrelevant knowledge and inhibit access

to knowledge prohibited by self-goals (Conway & Pleydell-Pearce, 2000). In all, criteria from various sources are combined into a retrieval model, on which the memory search is carried out in the autobiographical memory knowledge base (a separate entity from the frontally mediated supervisory mechanisms), and these two entities interact dynamically to shape successive elaborated cues to meet the verification criteria in later iterations (Conway & Pleydell-Pearce, 2000).

Contemporary research has also begun to expand on the complexity of the autobiographical knowledge base, stating that *general* autobiographical knowledge is typically recalled more quickly, potentially because general events are the preferred level of entry into a particular knowledge base (Conway, 1996). This imparts a partonomic, hierarchical system of autobiographical memory (Conway & Pleydell-Pearce, 2000). Upon satisfaction of the verification criteria of a retrieval model, search of the knowledge base ceases, and the constructed memory must be effortfully maintained (Conway & Pleydell-Pearce, 2000).

Overgeneral Autobiographical Memory

The CaR-FA-X model, as proposed by Williams and colleagues (Williams et al., 2007), articulates a model of affective control comprised of three factors: (1) capture and rumination, (2) functional avoidance, and (3) impaired executive control, all of which are theorized to impact autobiographical memory specificity. Depressed mood has been demonstrated to impair an individual's ability to recall specific memories, resulting in overgeneral autobiographical memory (OGM). OGM has been shown to be more prevalent in individuals with depression compared to their non-depressed counterparts (Williams et al., 2007), and has also shown to predict the course of depression (i.e., depressed individuals with less specific memories are more likely to remain depressed; Hermans et al., 2008). Williams and colleagues (2007) reported that

depressed patients consistently demonstrate significantly greater OGM than their matched controls (Cohen's d = 1.12 across 11 studies); this effect has been replicated with multiple affective disorders. Per the CaR-FA-X model, OGM is a distinguishing feature of dysregulated mood as a result of the three aforementioned components. Despite the majority of literature on autobiographical memory specificity and depression being conducted on younger and middle-aged adults, some research suggests that this relationship is also evident in older adults. Latorre and colleagues (2013) found that, in a nonclinical sample of older adults, those with higher depression symptom scores exhibited less specific autobiographical memory retrieval and greater latency times for specific memory retrieval, which they proposed may indicate more difficulty with moving fluently through the memory hierarchy. More research is warranted to determine how CaR-FA-X variables interrelate with each other, autobiographical memory, and depressive symptoms in later-life.

Autobiographical memory specificity has primarily been studied using clinical populations and with the Autobiographical Memory Test (AMT; Williams & Broadbent, 1986), a cue word procedure where participants are explicitly instructed to provide specific memories. These data have been instrumental in establishing the relationship between OGM and depression. However, following multiple unpublished studies with nonclinical samples where use of the AMT led to low levels of OGM, Raes and colleagues (2007) assert that "possibly, the very extensive instructions, the provision of practice trials, and the repeated prompting for specificity in the standard AMT administration jointly mean that people with an overgeneral memory retrieval style in a nonclinical population still manage to obtain high specificity scores," (p.497), indicating that the AMT is insufficiently sensitive in this population. Raes and colleagues (2007) report unpublished findings, whereby OGM was not related to depressive symptoms or

rumination when measured by the AMT in nonclinical populations, which contrasts the consensus regarding OGM and pathological mood drawn from previous research. To address this, Raes et al. (2007) created a sentence completion task, where participants are given sentence stems that probe them to retrieve past experiences, but do not explicitly request specificity; this measure is the Sentence Completion for Events from the Past Test (SCEPT). In a series of studies, the SCEPT was shown to be more sensitive to OGM than the AMT, and OGM as measured by the SCEPT was significantly correlated with depressive symptoms and rumination. As predicted, these relationships were not evident with the AMT in this nonclinical sample (Raes et al., 2007). In a follow-up study, they compared nonclinical samples' responses on the standard SCEPT to a modified SCEPT where they were explicitly instructed to be specific. Results showed that the specificity instructions significantly decreased OGM in the sample, and OGM was no longer related to depressive symptoms or rumination, indicating that it is the function of explicit specificity instructions that makes the AMT insufficiently sensitive in nonclinical samples as opposed to the difference in task (Raes et al., 2007). This approach allows researchers to examine a participant's habitual memory *style*, rather than their *ability* to generate a specific autobiographical memory when prompted.

Capture & Rumination

The capture mechanism of the CaR-FA-X model is the phenomenon that individuals remain at more general levels of memory retrieval if the conceptual information activated during the retrieval attempt is related to one's personal concerns or self-representations (Sumner, Griffith, & Mineka, 2011). If this self-referent information is highly elaborated, an individual can be captured at this general level of memory representation, staying focused on this conceptual information rather than continuing down the hierarchy of autobiographical memory to more

specific event details (Sumner, 2012). Given that self-representations are strongly interconnected, activation of these self-representations can lead to subsequent activation of other self-representations, rather than the activation of episodic details, resulting in an individual moving across rather than down the hierarchy of autobiographical memory, known as mnemonic interlock (Williams, 1999). As a result, specific memory retrieval is disrupted, resulting in OGM. In one study comparing previously depressed to never-depressed individuals, the number of cues on the AMT that were relevant to the individual (i.e., a degree of self-reference) was negatively predictive of autobiographical memory specificity in those with a history of depression; this effect was not significant for their never-depressed counterparts (Crane, Barnhofer, & Williams, 2007). Similar results have been found in a sample of individuals with borderline personality disorder or a history of depression, such that they exhibited greater OGM when the memory specificity task cues were related to an individual's maladaptive attitudes and schemas (Spinhoven, Bockting, Kremers, Schene, & Williams, 2007). Such results support the notion that activation of basic maladaptive attitudes may impair specific memory retrieval in clinical populations. The relationship between cue self-relevance and OGM is less clear in nonclinical populations. Sumner, Griffith, and Mineka (2011) found contrasting results, such that greater cue self-relevance was associated with more specific memory retrieval in undergraduate participants. More research is needed to determine the role of the capture phenomenon in autobiographical memory retrieval later in the lifespan and in nonclinical populations.

Rumination is defined as thinking perseveratively about one's feelings and problems, and is known to exacerbate and prolong distress and depression through the following mechanisms: (1) enhancing the effects of depressed mood on thinking, increasing the likelihood that individuals will use negative thoughts and memories activated by their depressed mood to

understand current circumstances; (2) interfering with effective problem-solving, partially due to pessimistic and fatalistic thoughts; and (3) interfering with instrumental behaviors, exacerbating stressful circumstances (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Regarding rumination's impact on autobiographical memory specificity, a review by Sumner (2012) identified two components of rumination that are especially maladaptive: (1) utilization of an abstract, analytical, and evaluative processing style that focuses on the causes, meanings, and consequences of one's experience; and (2) processing negatively-valenced self-referential material. In studies where rumination is experimentally induced, analytical rumination (i.e., thinking about the causes, meanings, and consequences of one's feelings), as opposed to experiential rumination (e.g., focusing on the specific experience of one's feelings), is predictive of decreased autobiographical memory specificity, particularly in individuals with a history of depression or who are high on trait depressive rumination (Crane, Barnhofer, Visser, Nightingale, & Williams, 2007; Raes, Watkins, Williams, & Hermans, 2008; Sutherland & Bryant, 2007). Treynor, Gonzalez, and Nolen-Hoeksema (2003) identified a two-factor model of rumination: (1) reflection, defined as "a purposeful turning inward to engage in cognitive problem solving to alleviate one's depressive symptoms," and (2) brooding, defined as "a passive comparison of one's current situation with some unachieved standard" (p. 256). Brooding, akin to maladaptive analytical processing, has been shown to mediate the positive relationship between depressive symptoms and OGM; this relationship was not evident with reflection, which is more adaptive (Debeer, Hermans, & Raes, 2009). In addition, Sutherland and Bryant (2007) found that inducing rumination about negative affective content increased OGM in nonclinical dysphoric participants more than positively-valenced ruminative prompts, an effect not evident in participants endorsing low depressive symptoms. One explanation for this

effect is that negative rumination may be more likely to activate negative schemas of the self, increasing the likelihood of capture and OGM (Sumner, 2012).

Functional Avoidance

Functional avoidance, the second mechanism of the CaR-FA-X model, refers to when the retrieval of specific memories is passively avoided as a function of affect regulation (i.e., to prevent recalling details of negative memories; Williams et al., 2007). Functional avoidance is broken down into four key concepts: (1) it develops as a response to trauma, particularly that occurring in early life, (2) it is a cognitive avoidance strategy, (3) especially distressing memories are highly avoided, and (4) the avoidance of distressing memories reduces emotional distress (Sumner et al., 2011). Over time, passive avoidance of specific negative memories engenders an indiscriminate overgeneral retrieval style due to negative reinforcement (Williams et al., 2007). Per the CaR-FA-X theory, this protective short-term use of functional avoidance becomes maladaptive when it is applied indiscriminately over an extended period of time due to continuous negative reinforcement. Indiscriminate use of an overgeneral retrieval style would likely perpetuate capture and rumination, particularly if the avoided memories activate an individual's negative categorical self-representations. In addition, trauma literature (specifically cognitive processing theory) posits that avoidance of distressing memories prevents integration of distressing information into preexisting mental schemas, which occurs when an individual confronts information that is discordant with their existent models (Creamer, Burgess, & Pattison, 1992). While functional avoidance is included as a risk factor in the CaR-FA-X model and is broadly implicated as a maladaptive thinking style, it was not included in this study's conceptualization of an integrated framework for the development of depressed mood in older adults.

Impaired Executive Control

Executive control is an umbrella term for frontal processes involved in goal-directed action, including goal formation, planning, carrying out goal-directed plans, and effective performance (Jurado & Rosselli, 2007). A primary component of executive control is working memory, which entails a system that provides temporary storage and manipulation of information necessary for complex cognitive tasks (Baddeley, 1992). Included in working memory is the central executive, the attentional controller responsible for oversight and coordination of the two subsidiary systems: the visuospatial sketchpad and the phonological loop (Baddeley, 1992). A fourth component, the episodic buffer, is a limited capacity system assumed to hold multidimensional information between working memory, perception, and long-term memory (Baddeley, 2012); the episodic buffer awaits extensive empirical scrutiny. Executive control, including working memory, plays an integral role in the generative retrieval of specific memories, involving defining and holding the retrieval model in working memory, the inhibition of irrelevant autobiographical knowledge during the search (and avoiding capture during this process), and maintaining the identified memory in working memory (Conway & Pleydell-Pearce, 2000; Williams et al., 2007). Thus, impaired working memory and other executive functions are likely to directly interrupt generative retrieval of a specific memory, resulting in OGM.

Throughout early life development, growth and myelination of the prefrontal cortex relates to concurrent development of autobiographical memory and associated frontal/executive components (Nelson & Fivush, 2004). On the other end of the lifespan, OGM is more evident, and older adults demonstrate poorer performance on working memory tasks (Ros, Latorre, & Serrano, 2009). In a sample of nonclinical older adults, for example, Winthorpe and Rabbitt (1988) found that working memory capacity predicted autobiographical memory specificity even

after controlling for IQ. These results are consistent with the notion that executive processes (of which working memory is primary) are integral to generative memory retrieval, and that executive capacity decline is inherent in normal aging (De Luca et al., 2003; Park & Schwarz, 2000).

The role of inhibition of irrelevant information to protect against interference is critical to adequate memory retrieval. Piolino and colleagues (2010) found that age-related decline in updating (necessary for modifying the retrieval model) and inhibition (necessary for disregarding irrelevant information and preventing interference) were predictive of OGM, contributing to the demonstrated effect of lower memory specificity in older participants. Support for inhibitory control as a mechanism of OGM has also been demonstrated with children, as inhibitory control mediated the relationship between depressive symptoms and memory specificity in a sample of nine to 13-year-olds (Raes, Verstraeten, Bijttebier, Vasey, & Dalgleish, 2010). Of note, research has shown that the impact of executive dysfunction is not merely due to effects of low mood, as these effects remain even after controlling for depressive symptomatology (e.g., Dalgleish et al., 2007).

Extant research has typically examined the relationship between independent components of executive control (e.g., inhibition, working memory, or interference control) and autobiographical memory specificity. Given the wide reach of working memory abilities and the difficulty of examining various executive abilities in the absence of a working memory component, it is possible that the impact of executive control on autobiographical memory is entirely a function of decline in working memory capacity.

Vascular Aging: An Integrated Approach

Significant theoretical and empirical overlap exists between the vascular depression hypothesis, the depression executive dysfunction syndrome, and the CaR-FA-X model, which independently characterize constellations of symptoms and developmental models of depression. Informed by their independent bodies of research, these three models can be integrated into a single developmental model of depression in later-life, explaining the mechanisms involved in the relationship between vascular burden and depressed mood. The predictive effect of vascular burden on executive control would be the first path of the model. Williams (2007) presents the components of the CaR-FA-X model not only as independent predictors of OGM (and their established relationship with depression), but as interacting with one another to compound and perpetuate the relationship between these components and OGM. Thus, research implies that executive control may predict rumination, as their interaction is primarily conceptualized as a failure to inhibit interfering information, resulting in capture and rumination at categorical stages of memory retrieval. Thus, the second pathway of the model would be from executive control to rumination. The third path in the model would close the loop between rumination and both OGM (a proximal outcome) and depressive symptoms (a distal outcome). Support for this framework comes, in part, from Von Hippel and colleagues (2008), who found that rumination mediated the relationship between executive dysfunction and depressive symptoms in late-onset depression, but not early-onset depression. Recent work has also suggested that rumination moderates the relationship between vascular burden, measured by self-report, and depressive symptoms in older adults (Herrera, 2017). The proposed integrated model extends these results to delineate a comprehensive developmental model of depression in later-life due to vascular burden.

It is necessary to mention that Herrera's (2017) recent work identifying rumination as a moderator of the vascular depression effect evaluated a similar model to the current hypothesized

serial mediation model, presented above. His model included a series of analyses conducted to (a) examine the mediating effect of rumination and autobiographical memory specificity in the relationship between executive control and depression, and (b) examine the moderating impact of rumination and functional avoidance on the vascular depression effect. Rumination significantly moderated the vascular depression effect, while all other mediation and moderation analyses yielded null results. Multiple explanations for these null results are apparent. First, Herrera's study utilized the minimal instructions Autobiographical Memory Test, which has not been extensively studied in nonclinical samples of older adults, and which Herrera posits may lead to floor effects in individuals over age 70 due to age-related decline in executive capacity (2017). Further, Herrera's sample of healthy older adults was very well educated (mean years of formal schooling = 16.46), reported high income (mean income = $62,500, interquartile range = $42,500), and was predominantly White (90.39%). It is possible that this sample does not yield enough variability in the constructs of interest to adequately demonstrate the hypothesized relationships. Further, the analyzed sample size (n=45) is below that recommended for 80% power in simple mediation analyses using bias-corrected bootstrapping by Fritz and MacKinnon (2007), unless *a* and *b* paths in the model were expected to yield large effects (i.e., effect sizes greater than or equal to .59), in which case a sample of 34 participants would be adequately powered. Lastly, Herrera's work did not include measures specific to working memory capacity, which extant research has deemed integral to the process of generative memory retrieval.

The current study builds on Herrera's work, modifying aspects of his methods to better measure the constructs of interest and expanding on his theoretical framework by using analyses more appropriate for the theoretically informed model presented in the current study. Such modifications will include utilization of a measure of autobiographical memory specificity that

has been demonstrated as more sensitive to OGM in nonclinical populations, described below. The present study also primarily recruited from community dwelling older adults at: a primary care clinic, an independent living facility, a rural Florida mobile home/RV community, and two church communities. This sample better represents the central Florida community, demonstrating greater variability than that in previous studies using convenience samples. The present study also incorporated a measure of working memory capacity, allowing evaluation of components of executive control that have been deemed integral to generative memory retrieval in extant literature.

The Current Study: Hypotheses

The goal of this study was to examine the relationships between vascular burden, executive control, rumination, depressive symptoms, and autobiographical memory specificity in older adults. Primarily, this study aimed to identify mediating pathways through which vascular burden leads to depressive symptoms in later-life.

Older Adult Sample

Hypothesis 1: Main Effects

a) Vascular burden will be positively associated with depressive symptoms and rumination in older adults, and negatively associated with executive control (i.e., working memory and inhibition) and autobiographical memory specificity in older adults.
b) Executive control measures (i.e., working memory and inhibition) will be negatively associated with rumination and depressive symptoms, and positively associated with autobiographical memory specificity.

c) Rumination will be negatively associated with autobiographical memory specificity, and positively associated with depressive symptoms.

Hypothesis 2: Serial Mediation

a) The hypothesized significant relationship between vascular burden and depressive symptoms will be serially mediated by executive control and rumination, respectively, such that greater vascular burden will predict worse executive control, which will predict increased rumination, leading to greater depressive symptoms.
b) The hypothesized significant relationship between vascular burden and autobiographical memory specificity will be serially mediated by executive control and rumination, respectively, such that greater vascular burden will predict worse executive control, which will predict increased rumination, leading to lower autobiographical memory specificity.

Younger Adult Sample

Hypothesis 3: Main Effects

a) Executive control measures (i.e., working memory and inhibition) will be negatively associated with rumination and depressive symptoms, and positively associated with autobiographical memory specificity.
b) Rumination will be positively associated with depressive symptoms, and negatively associated with autobiographical memory specificity.

The younger adult sample was examined using simple mediation models (see 'Statistical Methodology', below). These analyses were employed to evaluate the mediating effect of rumination on the hypothesized significant relationships between (1) executive control measures

and depressive symptoms, and (2) executive control measures and autobiographical memory specificity. These simple mediation analyses were exploratory, thus directional hypotheses were not identified.

METHODS

Older Adult Participants

Participants were recruited via physician referral, flyers, email advertisements, and researcher presentations. Exclusionary criteria for this study included individuals younger than 65 years, being non-fluent in English, presence of moderate to severe cognitive decline (determined by a phone screening measure), inadequate vision for assessment purposes, severe mental illness, previous head trauma with known cognitive impairments, and history of stroke. Data collection was completed at UCF Health (6850 Lake Nona Blvd, Orlando, FL 32827) or an alternative public location agreed upon between the researcher and participant. Older adults were compensated $20 upon completion of their assessment.

There is limited extant literature on sample size and statistical power for mediation analyses. With respect to simple mediation (i.e., one mediating variable), Fritz and MacKinnon (2007) stated that the empirically estimated sample size needed for 80% probability of rejecting the null hypothesis in a simple mediation analysis using bias-corrected bootstrapping is 54 participants, assuming medium and large *a* (independent variable to mediating variable) and *b* (mediating variable to dependent variable) paths, or vice versa. Specific to serial mediation (i.e., two mediating variables), Briggs (2006) reported that a sample of 50 participants allows for detection of individual specific indirect effects with a power above .70, using bias-corrected bootstrapping and assuming the following conditions: (1) partial mediation of the total effect, (2) equal partitioning of the total indirect effect between the two mediators, and (3) a direct effect of .48. In this sample of 50 participants, power using bias-corrected bootstrapping exceeded .80 in detecting a significant total indirect effect (Briggs, 2006). Given that the present study is focused on the process through which two intervening variables influence the relationship between vascular burden and depressive symptoms, assessment of the total indirect effect is the primary

analysis of interest. Thus, it is reasonable to conclude that a sample of 54 is sufficient for testing models with (1) a single mediator, and (2) two mediators.

Younger Adult Participants

A separate sample of adults age 18-30 was collected to evaluate the relationships between executive control, rumination, autobiographical memory specificity, and depressive symptoms in younger adults. Data collection on younger adults allowed for cross-sectional examination of the impact of age on the proposed network of relationships between select CaR-FA-X variables, autobiographical memory specificity, and depressive symptoms. Based on the aforementioned literature on sample size and power, 54 participants age 18 to 30 years was estimated to be sufficient for simple mediation. Younger adult participants were recruited using the Sona research participation system at the University of Central Florida; students received course credit for their study participation. Exclusionary criteria for younger adults included severe mental illness, being non-fluent in English, inadequate vision for assessment purposes, and previous head trauma with known cognitive impairments.

Measures

Modified Telephone Interview of Cognitive Status (TICS): The TICS is a brief assessment of cognitive status that assesses orientation, concentration, short-term memory, mathematical skills, praxis, and language; this measure was developed on the commonly used TICS. The maximum score is 35 with high scores reflecting better functioning; scores below 11 suggest clinically significant cognitive impairment. Past work has identified factors demonstrating mental status and memory; this measure had a coefficient alpha of .69 for the mental status factor (Herzog & Wallace, 1997). The TICS and similar modified versions of the TICS have shown to be generally sensitive to cognitive impairment (Welsh, Breitner, & Magruder-Habib, 1993). Other modified

versions of the TICS that include a delayed word recall component have been shown to distinguish between cognitively impaired individuals (i.e., individuals with mild cognitive impairment or dementia) and cognitively normal individuals (Knopman et al., 2010).

Demographic Variables: Age, gender, marital status, ethnicity, and income were self-reported by the participant.

Geriatric Depression Scale: This 30-item scale is a self-report measure of depressive symptomatology, and has demonstrated high internal consistency (Cronbach's alpha = .92) and validity (sensitivity = 92.5%, specificity = 76.6% when using cutoff score of 11 for depression; Lach, Chang, & Edwards, 2010). Despite its name, the GDS has demonstrated high reliability in younger adults (alpha coefficient = .82 in adults aged 17 to 55 years; Rule, Harvey, & Dobbs, 1990).

Vascular Burden: Self-reported cerebrovascular risk factors. Participants were asked, "Has a doctor ever diagnosed you with . . . hypertension or high blood pressure; high cholesterol; diabetes; heart problems." Participants also self-reported past and present cigarette smoking (such that pack year data could be generated).

Executive Control:

- Stop-Signal: Assesses inhibitory control by establishing a prepotent response (clicking a button in response to presentation of a particular shape). Participants are then instructed to withhold their response for trials preceded by an auditory stimulus. The pace of the stop-signal task increases or decreases until the participant correctly inhibits responses to half of such trials. Dependent variables from this measure included stop-signal reaction time and accuracy; results were considered in context of mean reaction time and stop signal delay. Stopping in the stop-signal paradigm is associated with activation of a

frontal-basal-ganglia circuit (Verbruggen & Logan, 2008), overlapping with the circuitry associated with hypothesized degradation due to vascular burden, discussed above.

- Complex Span Tasks: Two automated complex span tasks (shortened operation span and symmetry span) were used to measure an individual's working memory capacity. For both complex span tasks, participants are given a sequence of to-be-remembered items, and they must complete a distractor task in between each to-be-remembered item. In operation span (to-be-remembered item=letters), participants make a judgment regarding the correctness of simple arithmetic strings for the distractor task; scores are calculated by summing the numbers of letters correctly recalled in serial position. In symmetry span (to-be-remembered item=red dot locations), a participant makes a judgment regarding the symmetry of displayed shapes across a vertical axis for the distractor task; scores are calculated by summing the number of red square locations correctly recalled in serial position (Draheim, Harrison, Embretson, & Engle, 2018). Automated complex span tasks have demonstrated good reliability and validity (Redick et al., 2012). To reduce participant burden, the shortened version of operation span was implemented. Research has shown that, when using one block of operation span and three blocks of symmetry span (whereas the full tasks have three blocks each), data predict 85.3% of the fluid intelligence factor predicted by using all three blocks for three complex span tasks combined (Foster et al., 2015).

Sentence Completion for Events from the Past Test (SCEPT): an 11-item sentence completion task that instructs participants to complete sentences "any way [they] want," selecting a different topic for each response. The SCEPT sentence stems lend themselves to referencing autobiographical memories, but this is purposefully not stated in the instructions. The SCEPT

has improved sensitivity to overgeneral responding in nonclinical samples compared to other measures (i.e., the AMT), and SCEPT scores are positively correlated with depression and rumination scores (Raes et al., 2007). The SCEPT has shown good interrater agreement (87%, K = .82; Raes et al., 2007).

Ruminative Responses Scale: A 22-item scale that measures the degree to which an individual engages in ruminative thinking. This measure has demonstrated good internal consistency (.90), test-retest reliability over a one-year period (r=.67), and acceptable convergent and predictive validity (Nolen-Hoeksema, Larson, & Grayson, 1999; Treynor et al., 2003).

Statistical Methodology

Variables

Regarding executive control measures, inhibition (measured by SSRT) was measured continuously. A working memory factor was initially proposed (derived from the two complex span tasks). However, the Kaiser-Meyer-Olkin (KMO) value of the generated factor, a measure that determines sampling adequacy for a factor by comparing correlation and partial correlation coefficients, was 0.50, indicative of unacceptable sampling adequacy for this factor's use (Hutcheson & Sofroniou, 1999). As such, the complex span task with the higher number of observations and better individual predictive validity of working memory (based on all blocks of all complex span tasks; Foster et al., 2015) was selected for the models—symmetry span score. A composite score for self-reported vascular burden was generated by summing an individual's endorsement of vascular risk factors (hypertension, diabetes, heart problems, high cholesterol, and cigarette pack years; each syndrome equals 1 point, pack years were broken down into quartiles for up to 1 point), ranging from 0 to 5. Measures of rumination, autobiographical

memory specificity, and depressive symptoms were examined independently as continuous variables.

Older Adult Sample

Primary Analyses

To evaluate individual relationships between vascular burden, executive control, rumination, depressive symptoms, and autobiographical memory specificity (hypotheses 1a through 1c), as well as the indirect relationships of vascular burden to depressive symptoms and autobiographical memory specificity by way of executive control measures and rumination (hypotheses 2a & 2b), serial mediation analyses were employed. Assuming X precedes M_1, M_1 precedes M_2, and M_2 precedes Y, serial mediation can be used to better understand causal explanations of an observed phenomenon (i.e., the positive relationship between vascular burden and depressive symptoms in later-life; Briggs, 2006). Based on empirical evidence, temporal precedence between impaired executive control and rumination is proposed given the likelihood that an individual with poorer executive control due to vascular burden will have greater difficulty inhibiting ruminative thoughts.

To evaluate hypotheses with depressive symptoms as the dependent variable, two serial mediation models were conducted using Model 6 of the *PROCESS* script for *SPSS* (see Figures 1 and 2 for models 1 and 2; Hayes, 2013). In general, output from *PROCESS* Model 6 serial mediation analysis includes main effects of X on M_1, X and M_1 on M_2, and X, M_1, and M_2 on Y, thus examining hypotheses 1a through 1c (i.e., the main effect of vascular burden on executive control, rumination, and depressive symptoms; the main effect of executive control on rumination and depressive symptoms; and the main effect of rumination on depressive symptoms). Output from *PROCESS* Model 6 also includes the total indirect effect, evaluating the

serial mediation of executive control and rumination on the relationship between vascular burden and depressive symptoms (hypothesis 2a). *PROCESS* Model 6 also evaluates the indirect effect of X on Y by way of M_1 while controlling for M_2, as well as the indirect effect of X on Y by way of M_2 while controlling for M_1.

Secondary Analyses

The two primary serial mediation analyses described above were also run using autobiographical memory specificity as the outcome variable, in place of depressive symptomatology (see Figures 3 and 4 for models 3 and 4). These models evaluated hypotheses 1a through 1c and 2b using Model 6 of the *PROCESS* script for *SPSS* (Hayes, 2013). In general, output from *PROCESS* Model 6 serial mediation analysis includes main effects of X on M_1, X and M_1 on M_2, and X, M_1, and M_2 on Y, thus examining hypotheses 1a through 1c (i.e., the main effect of vascular burden on executive control, rumination, and autobiographical memory specificity; the main effect of executive control on rumination and autobiographical memory specificity; and the main effect of rumination on autobiographical memory specificity). The total (serial) indirect effect and individual simple mediation effects are also provided from *PROCESS* Model 6, as previously explained.

Younger Adult Sample

Primary Analyses

To address hypotheses 3a, 3b, and to explore the relationships between executive control measures, rumination, and depressive symptoms in younger adults, two simple mediation models were employed using Model 4 of the *PROCESS* script for *SPSS* (see Figures 5 and 6 for models 5 and 6; Hayes, 2013). Output from *PROCESS* Model 4 simple mediation analysis includes main effects of X on M, X on Y, and M on Y, thus examining hypotheses 3a and 3b (i.e., the main

effect of executive control measures on rumination and depressive symptoms, and the main effect of rumination on depressive symptoms). Output from *PROCESS* Model 4 also includes the indirect effect, evaluating the mediation of rumination on the relationship between executive control measures and depressive symptoms.

Secondary Analyses

To explore the relationships between executive control measures, rumination, and autobiographical memory specificity in younger adults, two simple mediation models were employed using Model 4 of the *PROCESS* script for *SPSS* (see Figures 7 and 8 for models 7 and 8; Hayes, 2013). These models examined hypotheses 3a and 3b (i.e., the main effect of executive control measures on rumination and autobiographical memory specificity, and the main effect of rumination on autobiographical memory specificity). Output from *PROCESS* Model 4 also includes the indirect effect, evaluating the mediation of rumination on the relationship between executive control measures and autobiographical memory specificity.

RESULTS

Descriptive Statistics

Older adults (n=56) in this sample were, on average, 76.2 years old. The older adult sample was primarily female (75%), White (87.5%), and married (64.3%). On average, older adult participants had completed 15.34 formal years of education. The average number of vascular risk factors endorsed was 2.04 out of 5. The average number of depressive symptoms endorsed was 4.57 out of 30. The average number of specific responses on the SCEPT was 2.59 out of 11.

Younger adults (n=63) in this sample were, on average, 19.81 years old. The younger adult sample was primarily female (68.3%) and reported greater ethnic diversity (47.6% White, 20.6% Black, 22.2% Latin/x). All younger adult participants reported never having been married. On average, younger adult participants reported completing 13.06 formal years of education. The average number of depressive symptoms endorsed was 9.70 out of 30. The average number of specific responses on the SCEPT was 3.68 out of 11.

See Table 1 for descriptive characteristics of the sample.

Analyses with Older Adult Sample

Data were collected on 56 older adults. One older adult was excluded from models including SSRT because their score was an outlier (i.e., score was 1.5 IQR above the third quartile or below the first quartile). Thus, n=56 for models 1 and 3; n=55 for models 2 and 4.

Model 1 evaluated the relationship between vascular burden and depressive symptoms by way of symmetry span score and rumination, respectively. Variables in the total effect model accounted for 2.65% of the variance in depressive symptoms. Regarding the primary pathways of interest, the direct effect of vascular burden on depressive symptoms neared significance

(*b*=0.577, *SE*=0.311, *p*=.070); the total effect of vascular burden on depressive symptoms was not significant (*b*=0.523, *SE*=0.431, *p*=.230). The relationship between vascular burden and symmetry span score was not significant (*b*=0.113, *SE*=0.791, *p*=.887), nor was the relationship between symmetry span score and rumination (*b*=-0.102, *SE*=0.118, *p*=.394). Rumination was significantly positively associated with depressive symptoms (*b*=0.410, *SE*=0.062, *p*<.001). As for additional pathways, vascular burden was not significantly associated with rumination (*b*=-0.090, *SE*=0.0.688, *p*=.897). Symmetry span score was significantly negatively associated with depressive symptoms (*b*=-0.110, *SE*=0.054, *p*=.047). Indirect pathways showed that vascular burden was not significantly related to depressive symptoms by way of symmetry span score independently (*b*=-0.012, *SE*=0.104, *LLCI*=-0.284, *ULCI*=0.145), rumination independently (*b*=-0.037, *SE*=0.266, *LLCI*=-0.549, *ULCI*=0.504), or symmetry span score and rumination serially (*b*=-0.005, *SE*=0.056, *LLCI*=-0.179, *ULCI*=0.075). In sum, results indicated that symmetry span score and rumination were independently significantly associated with depressive symptoms in this older adult sample, and that the relationship between vascular burden and depressive symptoms neared significance. The hypothesized model 1 simple and serial mechanisms of a vascular burden – depressive symptoms relationship, however, were not significant. See Figure 1 for model 1 results.

Model 2 evaluated the relationship between vascular burden and depressive symptoms by way of SSRT and rumination, respectively. Variables in the total effect model accounted for 1.75% of the variance in depressive symptoms. Regarding the primary pathways of interest, the direct (*b*=0.383, *SE*=0.333, *p*=.254) and total (*b*=0.430, *SE*=0.443, *p*=.336) effects of vascular burden on depressive symptoms were not significant. The relationship between vascular burden and SSRT was not significant (*b*=11.514, *SE*=7.303, *p*=.121), nor was the relationship between

SSRT and rumination (*b*=-0.010, *SE*=0.013, *p*=.458). Rumination was significantly positively associated with depressive symptoms (*b*=0.433, *SE*=0.063, *p*<.001). As for additional pathways, vascular burden was not significantly associated with rumination (*b*=0.008, *SE*=0.731, *p*=.992), nor was SSRT significantly associated with depressive symptoms (*b*=0.008, *SE*=0.006, *p*=.193). Indirect pathways showed that vascular burden was not significantly related to depressive symptoms by way of SSRT independently (*b*=0.093, *SE*=0.127, *LLCI*=-0.034, *ULCI*=0.521), rumination independently (*b*=0.003, *SE*=0.302, *LLCI*=-0.546, *ULCI*=0.645), or SSRT and rumination serially (*b*=-0.050, *SE*=0.095, *LLCI*=-0.385, *ULCI*=0.033). In sum, results indicated that rumination was independently significantly associated with depressive symptoms in this older adult sample. The other hypothesized model 2 direct effects and the simple and serial mechanisms of a vascular burden – depressive symptoms relationship were not significant. See Figure 2 for model 2 results.

Model 3 evaluated the relationship between vascular burden and autobiographical memory specificity (AMS) by way of symmetry span score and rumination, respectively. Variables in the total effect model accounted for 4.05% of the variance in AMS. Regarding the primary pathways of interest, the direct (*b*=0.269, *SE*=0.179, *p*=.139) and total (*b*=0.281, *SE*=0.186, *p*=.137) effects of vascular burden on AMS were not significant. The relationship between vascular burden and symmetry span score was not significant (*b*=0.113, *SE*=0.791, *p*=.887), nor was the relationship between symmetry span score and rumination (*b*=-0.102, *SE*=0.118, *p*=.394). The relationship between rumination and AMS was also not significant (*b*=-0.048, *SE*=0.036, *p*=.185). As for additional pathways, vascular burden was not significantly associated with rumination (*b*=-0.090, *SE*=0.688, *p*=.897); the relationship between symmetry span score and AMS approached significance (*b*=0.060, *SE*=0.031, *p*=.060). Indirect pathways

showed that vascular burden was not significantly related to AMS by way of symmetry span score independently (*b*=0.007, *SE*=0.057, *LLCI*=-0.090, *ULCI*=0.159), rumination independently (*b*=0.004, *SE*=0.040, *LLCI*=-0.063, *ULCI*=0.105), or symmetry span score and rumination serially (*b*=0.001, *SE*=0.008, *LLCI*=-0.008, *ULCI*=0.029). In sum, results indicated that none of the hypothesized model 3 direct or indirect effects were significant in this older adult sample; however, the relationship between symmetry span and AMS approached significance. See Figure 3 for model 3 results.

Model 4 evaluated the relationship between vascular burden and AMS by way of SSRT and rumination, respectively. Variables in the total effect model accounted for 2.63% of the variance in AMS. Regarding the primary pathways of interest, the direct (*b*=0.181, *SE*=0.192, *p*=.350) and total (*b*=0.228, *SE*=0.190, *p*=.237) effects of vascular burden on AMS were not significant. The relationship between vascular burden and SSRT was not significant (*b*=11.514, *SE*=7.303, *p*=.121), nor was the relationship between SSRT and rumination (*b*=-0.010, *SE*=0.013, *p*=.458). Rumination also was not significantly associated with AMS (*b*=-0.053, *SE*=0.036, *p*=.154). As for additional pathways, vascular burden was not significantly associated with rumination (*b*=0.008, *SE*=0.731, *p*=.992), nor was SSRT significantly associated with AMS (*b*=0.004, *SE*=0.004, *p*=.324). Indirect pathways showed that vascular burden was not significantly related to AMS by way of SSRT independently (*b*=0.041, *SE*=0.058, *LLCI*=-0.023, *ULCI*=0.232), rumination independently (*b*=-0.0004, *SE*=0.044, *LLCI*=-0.098, *ULCI*=0.080), or SSRT and rumination serially (*b*=0.006, *SE*=0.015, *LLCI*=-0.003, *ULCI*=0.073). In sum, results indicated that none of the hypothesized model 4 direct or indirect effects were significant in this older adult sample. See Figure 4 for model 4 results.

Analyses with Younger Adult Sample

Data were collected on 63 younger adults. Two younger adults were excluded from models including SSRT because their scores were outliers (i.e., score was 1.5 IQR above the third quartile or below the first quartile). Two younger adults were excluded from models including symmetry span score because technical difficulties prevented them from completing the task. Thus, n=61 for all younger adult models.

Model 5 evaluated the relationship between symmetry span score and depressive symptoms by way of rumination. Variables in the total effect model accounted for 0.82% of the variance in depressive symptoms. The direct (b=0.046, SE=0.106, p=.668) and total (b=0.101, SE=0.145, p=.488) effects of symmetry span score on depressive symptoms were not significant. The relationship between symmetry span score and rumination was not significant (b=0.168, SE=0.302, p=.580). Rumination was significantly positively associated with depressive symptoms (b=0.330, SE=0.046, p<.001). The indirect effect of symmetry span score on depressive symptoms via rumination was not significant (b=0.055, SE=0.089, $LLCI$=-0.115, $ULCI$=0.237). In sum, results demonstrated a significant relationship between rumination and depressive symptoms. Otherwise, the hypothesized model 5 direct and indirect effects were not evident in this younger adult sample. See Figure 5 for model 5 results.

Model 6 evaluated the relationship between SSRT and depressive symptoms by way of rumination. Variables in the total effect model accounted for 0.01% of the variance in depressive symptoms. The direct (b=0.007, SE=0.016, p=.663) and total (b=-0.002, SE=0.021, p=.937) effects of SSRT on depressive symptoms were not significant. The relationship between SSRT and rumination was not significant (b=-0.029, SE=0.045, p=.517). Rumination was significantly positively associated with depressive symptoms (b=0.298, SE=0.047, p<.001). The indirect effect of SSRT on depressive symptoms via rumination was not significant (b=-0.009, SE=0.013,

LLCI=-0.036, *ULCI*=0.016). In sum, results demonstrated a significant relationship between rumination and depressive symptoms; no other hypothesized model 6 direct or indirect relationships were evident in this younger adult sample. See Figure 6 for model 6 results.

Model 7 evaluated the relationship between symmetry span score and AMS by way of rumination. Variables in the total effect model did not account for any of the variance in AMS. The direct (*b*=-0.0002, *SE*=0.031, *p*=.994) and total (*b*=-0.0001, *SE*=0.031, *p*=.998) effects of symmetry span score on AMS were not significant. The relationship between symmetry span score and rumination was not significant (*b*=0.168, *SE*=0.302, *p*=.580), nor was the relationship between rumination and AMS (*b*=0.001, *SE*=0.013, *p*=.937). The indirect effect of symmetry span score on AMS via rumination was not significant (*b*=0.0002, *SE*=0.005, *LLCI* =-0.008, *ULCI*=0.013). In sum, results indicated that none of the hypothesized model 7 direct or indirect relationships were significant in this younger adult sample. See Figure 7 for model 7 results.

Model 8 evaluated the relationship between SSRT and AMS by way of rumination. Variables in the total effect model accounted for 1.23% of the variance in AMS. The direct (*b*=-0.004, *SE*=0.005, *p*=.368) and total (*b*=-0.004, *SE*=0.005, *p*=.394) effects of SSRT on AMS were not significant. The relationships between SSRT and rumination (*b*=-0.029, *SE*=0.045, *p*=.517) and rumination and AMS (*b*=-0.009, *SE*=0.014, *p*=.509) were also not significant. The indirect relationship between SSRT and AMS via rumination was not significant (*b*=0.0003, *SE*=0.001, *LLCI*=-0.001, *ULCI*=0.003). In sum, results indicated that none of the hypothesized model 8 direct or indirect relationships were significant in this younger adult sample. See Figure 8 for model 8 results.

DISCUSSION

The primary purpose of the present study was to evaluate an integrated developmental model of depressive symptoms in later-life. Addressing a secondary goal, this study evaluated the same mechanism in the development of overgeneral autobiographical memory. Lastly, results from analyses with older adults were compared to mechanistic models of depressive symptoms and overgeneral autobiographical memory in younger adults to characterize changes in the mechanism as a function of age, if applicable.

Across models based on data from older adults, in which the outcome was depressive symptoms, vascular burden only approached significance (p=.070) in its relationship with depressive symptoms in the context of model 1. In this same model, working memory (as measured by symmetry span) was significantly negatively associated with depressive symptoms. Rumination was significantly positively related to depressive symptoms in all models with the older adult sample. All other hypothesized direct relationships, as well as the hypothesized mechanism through which vascular burden is associated with depressive symptoms, were not supported in this older adult sample. Similarly, only rumination was significantly associated with depressive symptoms in models representing younger adults; other direct and mechanistic relationships were not significant. Given the lack of a significant vascular depression effect, changes to any hypothesized vascular depression mechanisms as a function of age were not assessed.

The present results are consistent with the theoretical and clinical presentation of depression executive dysfunction syndrome as posited by Alexopoulos and colleagues (2002), such that lower scores on a working memory task were associated with greater depressive symptoms. Surprisingly, vascular burden was not significantly associated with depressive symptoms in this sample of older adults. Previous studies that have demonstrated the vascular

depression effect often do so in the context of hundreds or thousands of participants (e.g., Mast et al., 2008; Scott & Paulson, 2018; Yochim et al., 2003). When this project was proposed, the a priori target sample size for mediation was based on extant literature making the following assumptions: for simple mediation—a medium X→M relationship, and a large M→Y relationship; for serial mediation—partial mediation of the total effect, equal partitioning of the total indirect effect between the mediators, and a direct effect of .48 (Briggs, 2006). It is well established that one of the primary neuropsychological domains of impact from cerebrovascular burden is executive functioning (T O'Brien et al., 2003), as is the rumination – depression relationship (Nolen-Hoeksema, 2000). Further, odds ratios of vascular risk factors of depression suggest small-to-medium effects, and the magnitude of measurable depression risk varies as a function how the risk factor is operationalized (Mast et al., 2008). A power analysis suggested that the sample target size of 56 would have likely been adequate to demonstrate these effects; however, these relationships were not evident in this sample. In combination, it seems likely that this study was underpowered to identify the hypothesized vascular burden effect. Future studies seeking to characterize both the vascular depression effect and mechanisms by which those effects materialize may employ these findings in the estimation of necessary sample sizes.

With respect to the analysis of AMS among older adults, the proposed hypotheses were not supported. Working memory, as measured by symmetry span, approached significance in its relationship with older adult AMS (p=.060; model 3). Other hypothesized direct and indirect effects were not significant, for both older and younger adults. The lack of significant relationships with AMS is inconsistent with extant literature demonstrating a relationship between relevant CaR-FA-X variables and overgeneral autobiographical memory (e.g., Sutherland & Bryant, 2007; Winthorpe & Rabbitt, 1988). The SCEPT was selected to measure

AMS due to concerns that the Autobiographical Memory Test, a commonly used AMS measure, would lead to ceiling effects and low OGM sensitivity given its extensive practice and instructions for specificity (Raes et al., 2007). The present results suggest the opposite effect occurred with the SCEPT. One interpretation of these data is that omission of prompts for specificity and use of ambiguous stimuli contributed to a floor effect. The mean (2.59) and standard deviation (1.71) of the SCEPT scores (indicating 2.59 specific responses, on average, out of 11) indicate that there was not sufficient variability to evaluate the hypotheses involving AMS. Although SCEPT scores were higher by one point in younger adults compared to older adults, the limited variability and null results were also evident in younger adult models.

Inhibitory control, measured in this study using the SSRT, was not associated with rumination, depressive symptoms, or AMS in younger and older adult samples. One possible explanation is that the present theoretical framework was incorrect, and inhibition is not the component of executive function that is implicated in this network. A recent study by Respino and colleagues (2019) found no group differences between older adults with later-life depression and non-depressed older adults on a measure of inhibitory and interference control, the Stroop Color Word Test. By contrast, they did find significant group differences on other measures of executive functioning, including measure of attentional set shifting and self-reported dysexecutive behaviors (Respino et al., 2019). Another possible contributor to the lack of relationship between SSRT and depressive symptoms was the low endorsement of depressive symptoms in this sample. Similarly, low numbers of specific responses on the SCEPT may have contributed to null finding regarding AMS.

This study was built on previous work in the OLDeR Lab, from which recruitment methods and construct measurement were modified to address limitations. Compared to

Herrera's (2017) recent work collecting a convenience sample, the present sample was collected from multiple areas in central Florida (e.g., church, an independent living facility, and a mobile home/RV community), and the sample endorsed more vascular risk factors. Despite greater overall vascular burden, the current sample endorsed about 1.5 fewer depressive symptoms, on average, compared to Herrera's sample, with otherwise similar distribution. Similar to Herrera's sample, older adult participants in this study were well educated and primarily White. Future research should continue to diversify recruitment locations to improve generalizability of results and capture a more demographically diverse sample.

A primary limitation of the present study was the use of a single complex span task as an indicator of working memory. It was not the intent of the researcher, nor is it recommended by complex span task creators (Foster et al., 2015), to use a single task in representing this construct; we resorted to a single task given the unacceptability of the generated factor. Future research should seek to utilize multiple complex span tasks in generating a reliable factor with an adequate sample size to better represent the construct of working memory. As previously mentioned, future studies should also evaluate relationships between vascular burden, depressive symptoms, and potential mechanisms using a larger sample size, as the lack of a vascular depression effect and the unreliability of the generated working memory factor indicate inadequate power despite a priori assumptions. In addition, the SCEPT may not be an appropriate measure of AMS in older adults given the low scores in this sample. Future studies may consider employing multiple measures of AMS (e.g., the traditional Autobiographical Memory Test, the Minimal-Instructions Autobiographical Memory Test, and the SCEPT) to compare sensitivity of the measures to OGM in older adults. Finally, the present study was cross-sectional, and conclusions about causality cannot be drawn from results. Future studies should

evaluate the relationships between vascular and cognitive risk factors for depressive symptoms in later-life using longitudinal methods to assess these relationships across the lifespan.

Clinically, results provide continued support for established treatments targeting individuals with concurrent depression and executive dysfunction. Treatment for this population is paramount since the presence of dysexecutive features leads to worse depression prognosis (i.e., greater chronicity, relapse, and recurrence; Alexopoulos et al., 2000) and treatment response (Kalayam & Alexopoulos, 1999). Such treatments include ENGAGE (Alexopoulos et al., 2016) and problem solving therapy (Alexopoulos et al., 2015), which attribute pathological mood symptoms to disrupted cognitive systems such as the positive valence and other emotion regulation systems. ENGAGE and problem solving therapy have shown similar efficacy in treatment of later-life depression and associated disability (Alexopoulos et al., 2015). Results also identify rumination as a modifiable treatment target in older adults experiencing depressive symptoms. Deyo and colleagues (2009) suggest that mindfulness-based stress reduction, which teaches the practice of mindfulness meditation, could replace reactive, ruminative thought processes with more decentered, mindful thought observations. A second beneficial intervention for rumination is adaptive distraction, described by Hilt and Pollak (2012) as an activity that can alleviate negative mood long enough to promote problem-solving and terminate ruminative processes. Given the concrete nature of these interventions, it is feasible that they would be effective in the older adult population, even for those with concurrent depression and executive dysfunction. As such, a rumination-focused intervention could be an ancillary treatment to ENGAGE or problem solving therapy for older adults presenting with depressive symptoms, ruminative tendencies, and executive dysfunction.

Conclusions

This study evaluated a network of vascular and cognitive risk factors for depressive symptoms in later-life. Model 1, evaluating the impact of vascular burden on depressive symptoms in older adults by way of working memory and rumination, respectively, was the most effective in integrating vascular depression theory, depression-executive dysfunction syndrome, and the CaR-FA-X model. This model demonstrated a negative relationship between working memory and depressive symptoms, a positive relationship between rumination and depressive symptoms, and a near-significant vascular depression effect. However, there was not support for a vascular depression mechanism in this network. Null results in this sample could be attributable to inadequate power or measurement error. One must also consider the possibility that, though theoretically coherent, depressive symptoms due to vascular burden simply do not manifest through poor executive control and subsequent increased rumination. Future studies could evaluate these hypotheses using a larger, diverse, longitudinal sample and with additional measures of working memory.

APPENDIX A: TABLES

Table 1. *Descriptive statistics of the sample.*

	Older Adults (*n*=56)	Younger Adults (*n*=63)
	n (%)	*n* (%)
Gender		
Male	14 (25.0)	19 (30.2)
Female	42 (75.0)	43 (68.3)
Transgender	0 (0)	1 (1.6)
Marital Status		
Married	36 (64.3)	0 (0)
Widowed	11 (19.6)	0 (0)
Divorced	7 (12.5)	0 (0)
Never Married	2 (3.6)	63 (100)
Income		
$10,000-19,999	2 (3.6)	24 (38.1)
$20,000-29,000	5 (8.9)	6 (9.5)
$30,000-39,999	5 (8.9)	2 (3.2)
$40,000-49,999	5 (8.9)	5 (7.9)
$50,000-74,999	17 (30.4)	4 (6.3)
$75,000-99,999	10 (17.9)	7 (11.1)
$100,000-150,000	10 (17.9)	9 (14.3)
Over $150,000	2 (3.6)	6 (9.5)
Ethnicity		
White	49 (87.5)	30 (47.6)
Black	3 (5.4)	13 (20.6)
Latin/x	3 (5.4)	14 (22.2)
Asian	0 (0)	4 (6.3)
Pacific Islander	0 (0)	1 (1.6)
Other	1 (1.8)	1 (1.6)
	M (SD)	*M* (SD)
Age	76.2 (7.47)	19.81 (2.24)
Education in Years	15.34 (2.11)	13.06 (1.22)
Vascular Burden [a]	2.04 (1.23)	-
GDS Score [a]	4.57 (3.94)	9.70 (7.05)
SCEPT Score [a]	2.59 (1.71)	3.68 (1.59)

a. Vascular burden scores range from 0 to 5 (higher scores indicate greater burden); GDS scores range from 0 to 60 (higher scores indicate more depressive symptoms); SCEPT scores range from 0 to 11 (higher scores indicate more specific responses).

APPENDIX B: FIGURES

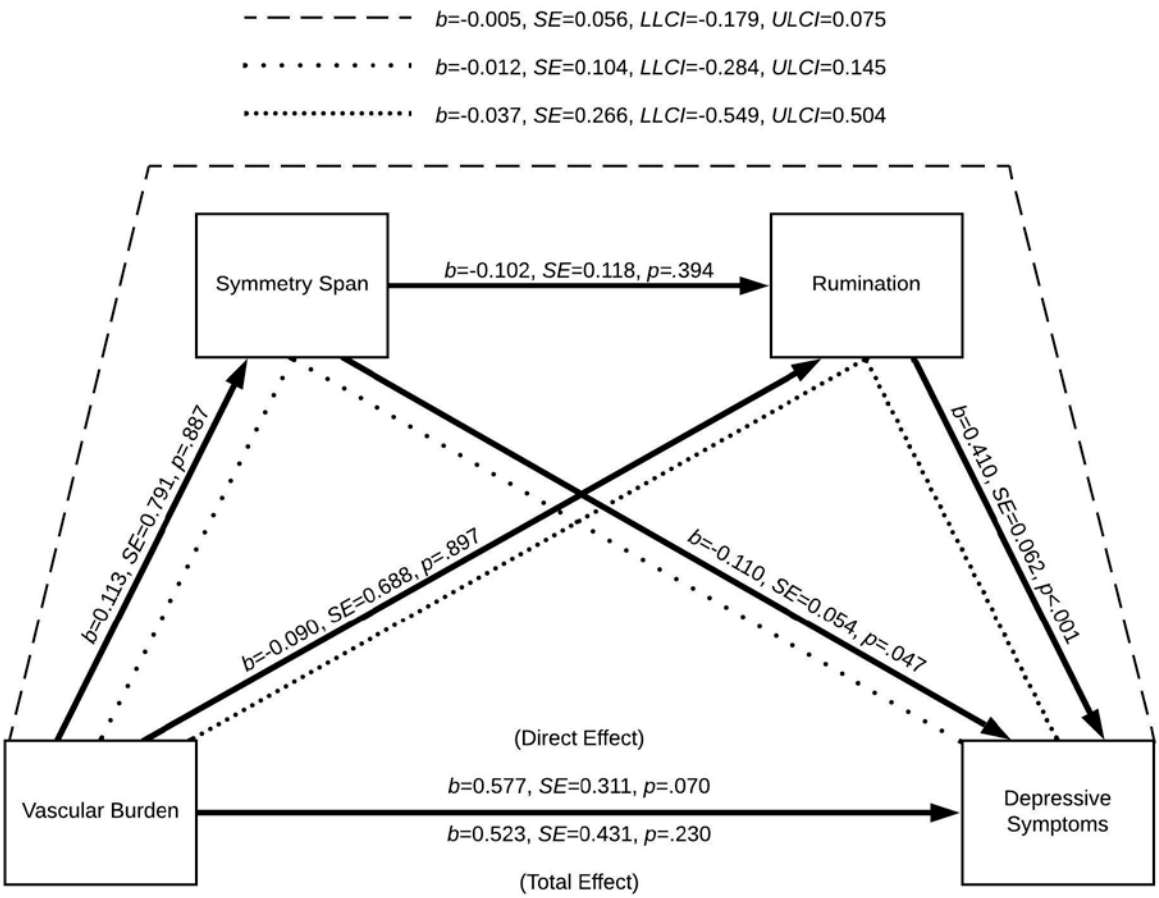


Figure 1. Model 1. Serial mediation model depicting the relationship between vascular burden and depressive symptoms in older adults via symmetry span scores and rumination, respectively.

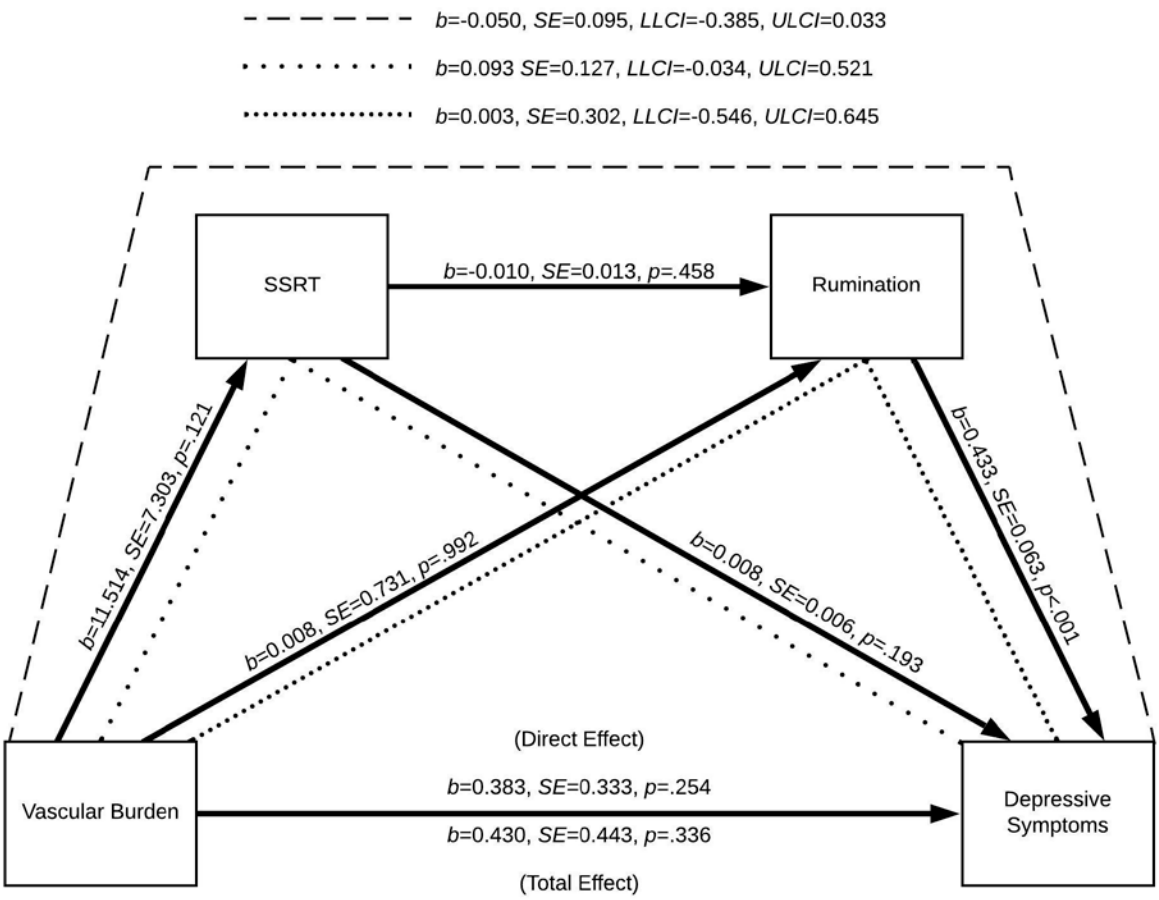


Figure 2. Model 2. Serial mediation model depicting the relationship between vascular burden and depressive symptoms in older adults via SSRT and rumination, respectively.

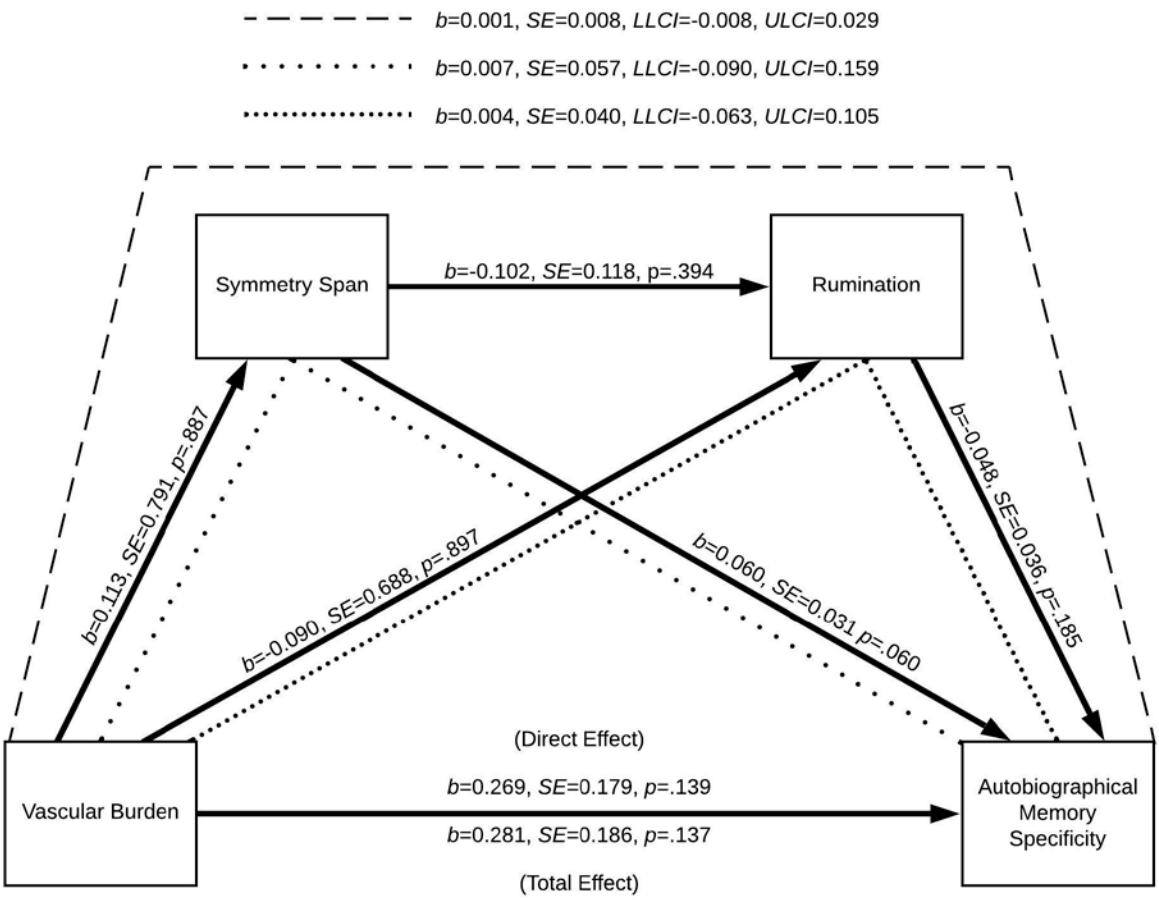


Figure 3. Model 3. Serial mediation model depicting the relationship between vascular burden and autobiographical memory specificity in older adults via symmetry span scores and rumination, respectively.

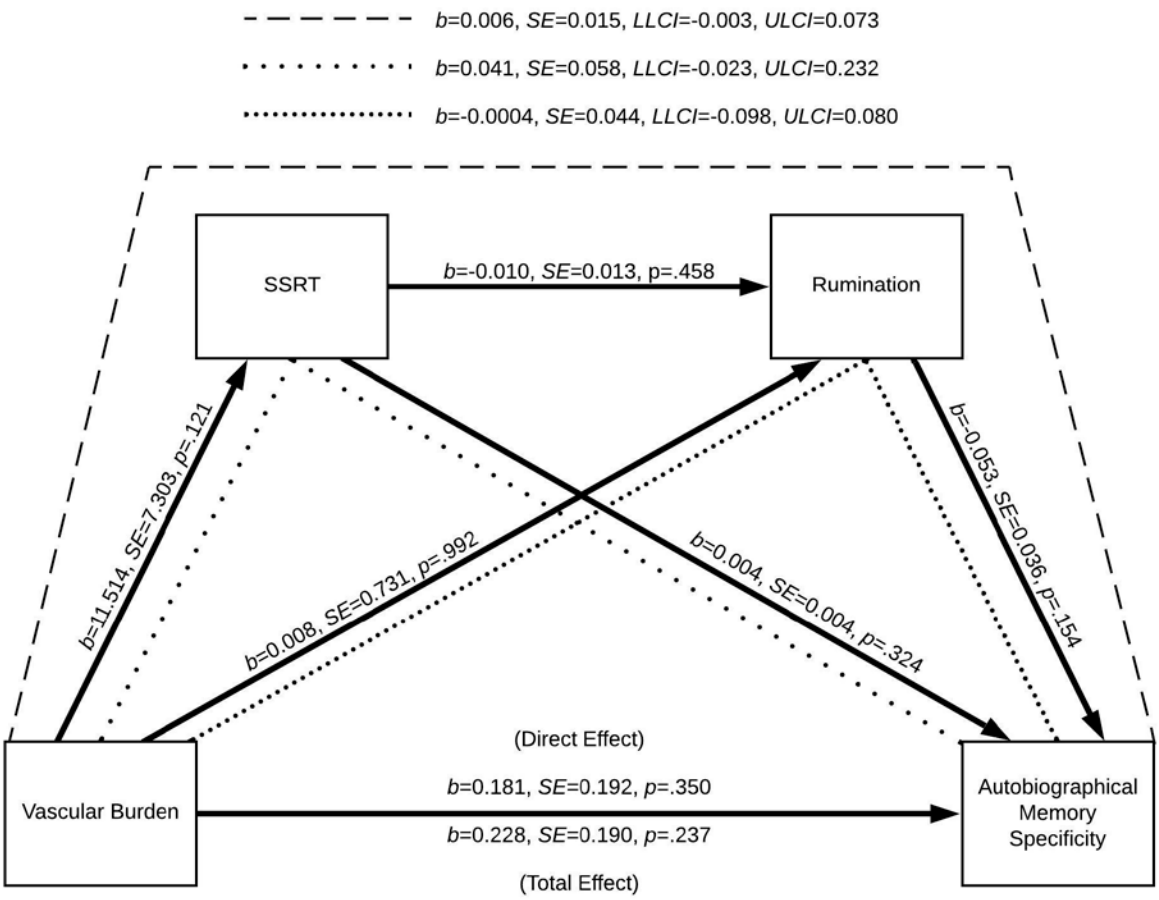


Figure 4. Model 4. Serial mediation model depicting the relationship between vascular burden and autobiographical memory specificity in older adults via SSRT and rumination, respectively.

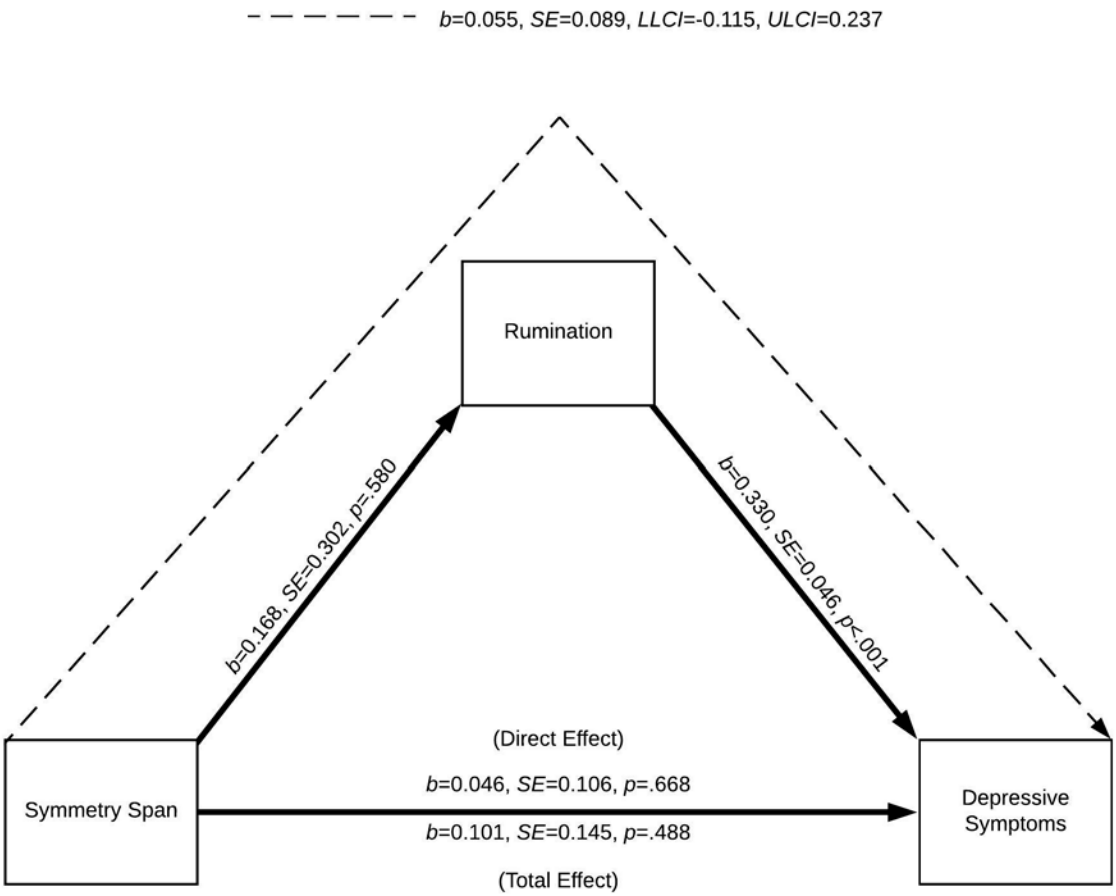


Figure 5. Model 5. Simple mediation model depicting the relationship between symmetry span scores and depressive symptoms in younger adults via rumination.

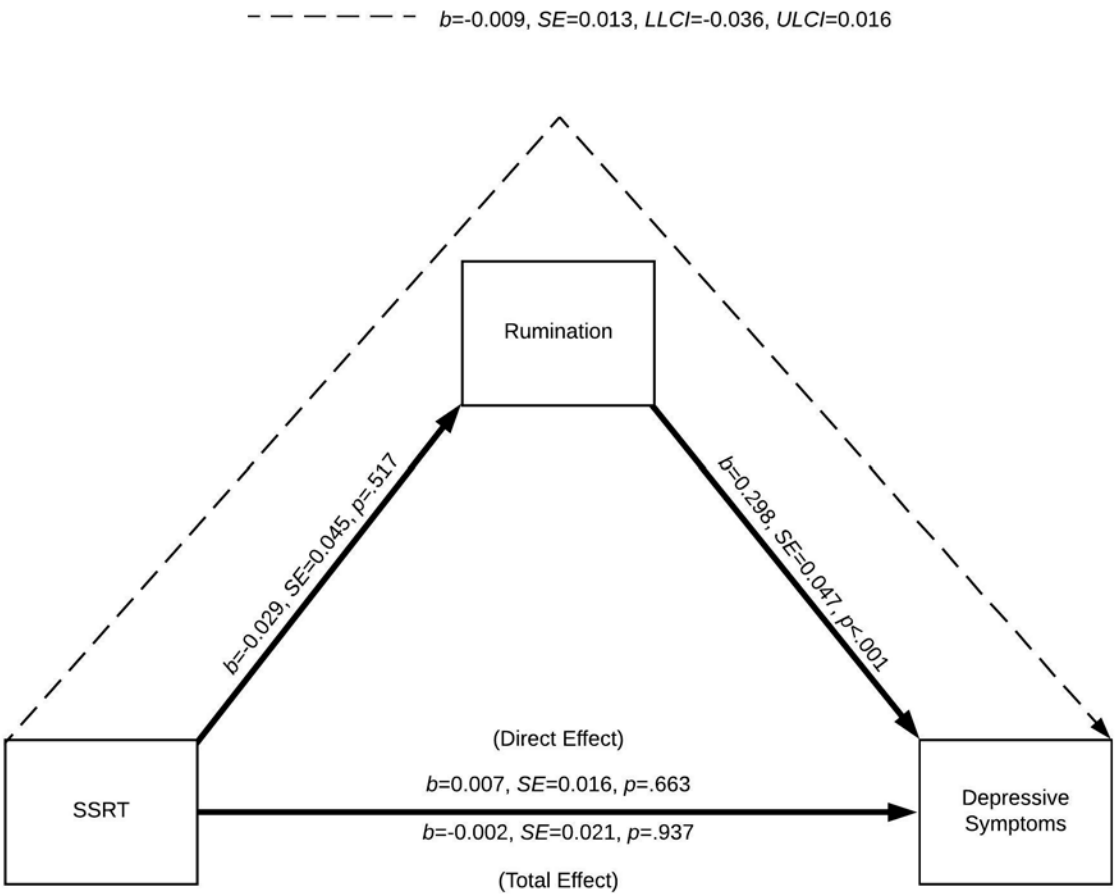


Figure 6. Model 6. Simple mediation model depicting the relationship between SSRT and depressive symptoms in younger adults via rumination.

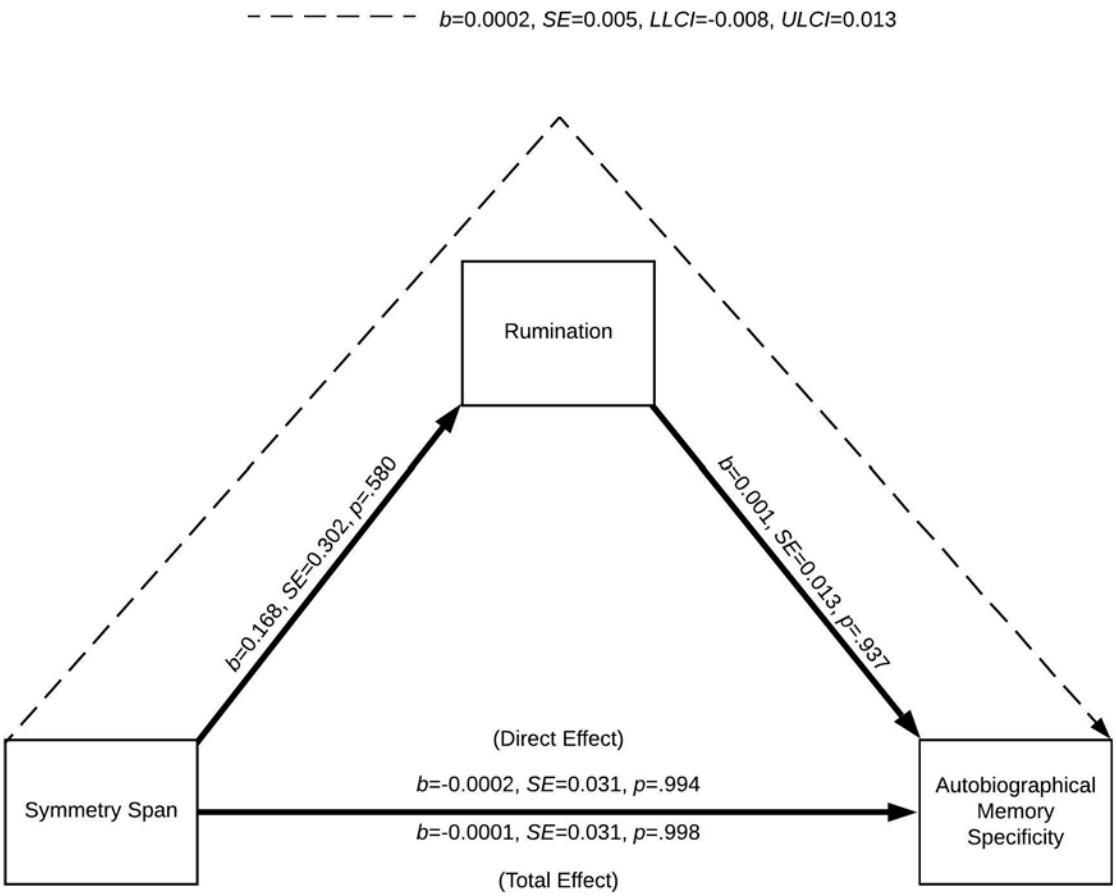


Figure 7. Model 7. Simple mediation model depicting the relationship between symmetry span scores and autobiographical memory specificity in younger adults via rumination.

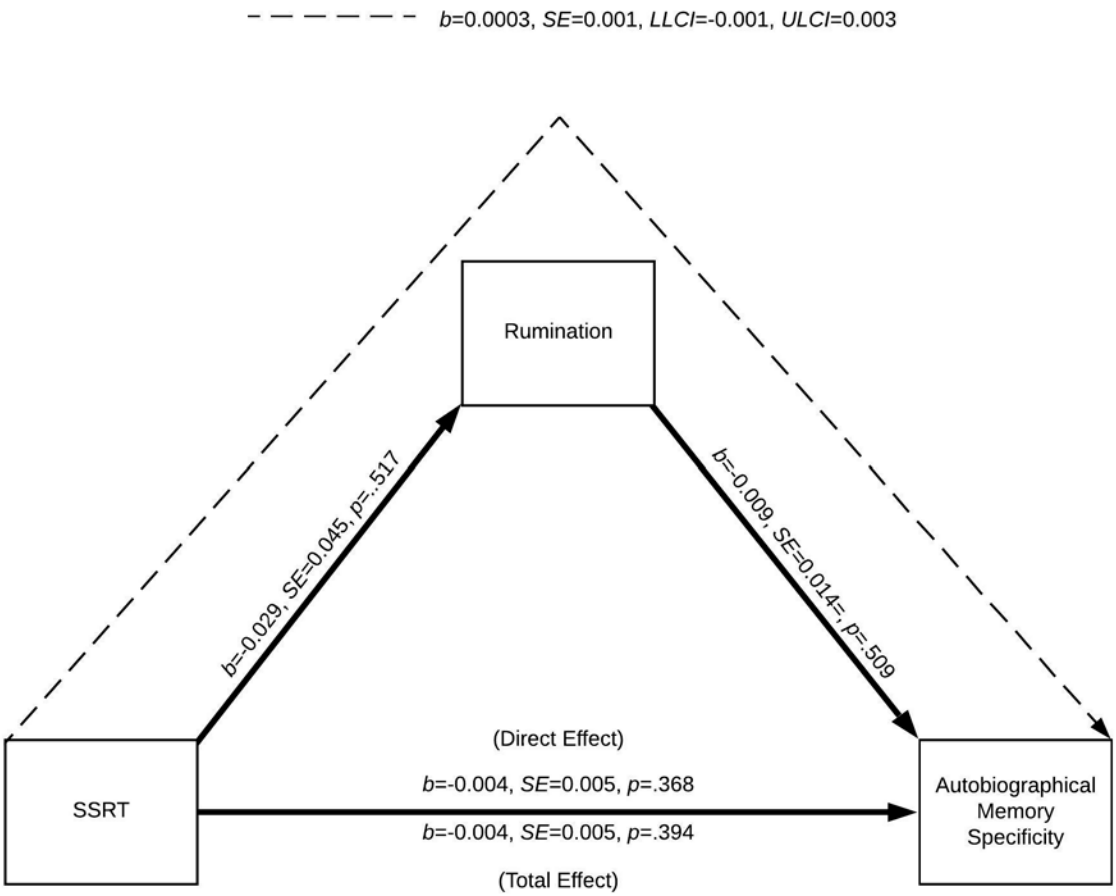


Figure 8. Model 8. Simple mediation model depicting the relationship between SSRT and autobiographical memory specificity in younger adults via rumination.

APPENDIX C: IRB APPROVAL FORM

University of Central Florida Institutional Review Board
Office of Research & Commercialization
12201 Research Parkway, Suite 501
Orlando, Florida 32826-3246
Telephone: 407-823-2901 or 407-882-2276
www.research.ucf.edu/compliance/irb.html

Approval of Human Research

From: **UCF Institutional Review Board #1**
FWA00000351, IRB00001138

To: **Rosanna G Scott** and Co-PIs **Daniel Lee Paulson and Mariana Beatriz Dangiolo**

Date: **August 31, 2018**

Dear Researcher:

On 08/31/2018 the IRB approved the following human participant research until 08/30/2019 inclusive:

Type of Review:	UCF Initial Review Submission Form Expedited Review Category #4 & 7; This approval includes a Waiver of Written Documentation of Consent
Project Title:	Evaluating an Integrated Developmental Model of Disordered Mood in Later-Life
Investigator:	Rosanna G Scott
IRB Number:	BIO-18-14207
Funding Agency:	University of Central Florida(UCF)
Grant Title:	N/A
Research ID:	N/A

The scientific merit of the research was considered during the IRB review. The Continuing Review Application must be submitted 30days prior to the expiration date for studies that were previously expedited, and 60 days prior to the expiration date for research that was previously reviewed at a convened meeting. Do not make changes to the study (i.e., protocol, methodology, consent form, personnel, site, etc.) before obtaining IRB approval. A Modification Form **cannot** be used to extend the approval period of a study. All forms may be completed and submitted online at https://iris.research.ucf.edu .

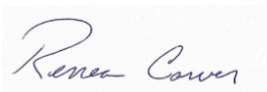
If continuing review approval is not granted before the expiration date of 08/30/2019, approval of this research expires on that date. When you have completed your research, please submit a Study Closure request in iRIS so that IRB records will be accurate.

Use of the approved, stamped consent document(s) is required. The new form supersedes all previous versions, which are now invalid for further use. Only approved investigators (or other approved key study personnel) may solicit consent for research participation. Participants or their representatives must receive a copy of the consent form(s).

All data, including signed consent forms if applicable, must be retained and secured per protocol for a minimum of five years (six if HIPAA applies) past the completion of this research. Any links to the identification of participants should be maintained and secured per protocol. Additional requirements may be imposed by your funding agency, your department, or other entities. Access to data is limited to authorized individuals listed as key study personnel.

In the conduct of this research, you are responsible to follow the requirements of the Investigator Manual.

This letter is signed by:



Signature applied by Renea C Carver on 08/31/2018 04:10:46 PM EDT

Designated Reviewer

REFERENCES

Alexopoulos, G.S., Kiosses, D.N., Klimstra, S., Kalayam, B., & Bruce, M.L. (2002). Clinical presentation of the "depression–executive dysfunction syndrome" of late life. *The American Journal of Geriatric Psychiatry, 10*(1), 98-106.

Alexopoulos, G.S., Meyers, B.S., Young, R.C., Campbell, S., Silbersweig, D., & Charlson, M. (1997). 'Vascular depression' hypothesis. *Archives of General Psychiatry, 54*(10), 915-922.

Alexopoulos, G.S., Meyers, B.S., Young, R.C., Kalayam, B., Kakuma, T., Gabrielle, M., . . . Hull, J. (2000). Executive dysfunction and long-term outcomes of geriatric depression. *Archives of General Psychiatry, 57*(3), 285-290.

Alexopoulos, G.S., Raue, P.J., Gunning, F., Kiosses, D.N., Kanellopoulos, D., Pollari, C., . . . Arean, P.A. (2016). "Engage" therapy: Behavioral activation and improvement of late-life major depression. *The American Journal of Geriatric Psychiatry, 24*(4), 320-326.

Alexopoulos, G.S., Raue, P.J., Kiosses, D.N., Seirup, J.K., Banerjee, S., & Arean, P.A. (2015). Comparing engage with PST in late-life major depression: a preliminary report. *The American Journal of Geriatric Psychiatry, 23*(5), 506-513.

Baddeley, A. (1992). Working memory. *Science, 255*(5044), 556-559.

Baddeley, A. (2012). Working memory: Theories, models, and controversies. *Annual Review of Psychology, 63*, 1-29.

Blazer, D.G. (2003). Depression in late life: Review and commentary. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 58*(3), M249-M265.

Briggs, N.E. (2006). *Estimation of the standard error and confidence interval of the indirect effect in multiple mediator models.* The Ohio State University.

Bruce, M.L., Seeman, T.E., Merrill, S.S., & Blazer, D.G. (1994). The impact of depressive symptomatology on physical disability: MacArthur Studies of Successful Aging. *American Journal of Public Health, 84*(11), 1796-1799. doi: 10.2105/AJPH.84.11.1796

Burgess, P.W. (1996). Confabulation and the control of recollection. *Memory, 4*(4), 359-412.

Coffey, C.E., Figiel, G.S., Djang, W.T., & Weiner, R.D. (1990). Subcortical hyperintensity on magnetic resonance imaging: A comparison of normal and depressed elderly subjects. *The American Journal of Psychiatry, 147*(2), 187-189.

Conway, M.A. (1996). Autobiographical knowledge and autobiographical memories. In D. C. Rubin (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 67-93). New York, NY: Cambridge University Press.

Conway, M.A., & Pleydell-Pearce, C.W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review, 107*(2), 261.

Crane, C., Barnhofer, T., Visser, C., Nightingale, H., & Williams, J.M.G. (2007). The effects of analytical and experiential rumination on autobiographical memory specificity in individuals with a history of major depression. *Behaviour Research and Therapy, 45*(12), 3077-3087.

Crane, C., Barnhofer, T., & Williams, J.M.G. (2007). Cue self-relevance affects autobiographical memory specificity in individuals with a history of major depression. *Memory, 15*(3), 312-323.

Creamer, M., Burgess, P., & Pattison, P. (1992). Reaction to trauma: A cognitive processing model. *Journal of Abnormal Psychology, 101*(3), 452.

Dalgleish, T., Williams, J.M.G., Golden, A.-M.J., Perkins, N., Barrett, L.F., Barnard, P.J., . . . Tchanturia, K. (2007). Reduced specificity of autobiographical memory and depression: The role of executive control. *Journal of Experimental Psychology: General, 136*(1), 23.

De Luca, C.R., Wood, S.J., Anderson, V., Buchanan, J.-A., Proffitt, T.M., Mahony, K., & Pantelis, C. (2003). Normative data from the CANTAB. I: Development of executive function over the lifespan. *Journal of Clinical and Experimental Neuropsychology, 25*(2), 242-254.

Debeer, E., Hermans, D., & Raes, F. (2009). Associations between components of rumination and autobiographical memory specificity as measured by a Minimal Instructions Autobiographical Memory Test. *Memory, 17*(8), 892-903.

Deyo, M., Wilson, K.A., Ong, J., & Koopman, C. (2009). Mindfulness and rumination: Does mindfulness training lead to reductions in the ruminative thinking associated with depression? *Explore, 5*(5), 265-271.

Draheim, C., Harrison, T., Embretson, S., & Engle, R. (2018). What item response theory can tell us about the complex span tasks. *Psychological Assessment, 30*(1), 116-129.

Ferrari, A.J., Charlson, F.J., Norman, R.E., Patten, S.B., Freedman, G., Murray, C.J.L., . . . Whiteford, H.A. (2013). Burden of depressive disorders by country, sex, age, and year: findings from the global burden of disease study 2010. *PLoS Medicine, 10*(11), e1001547.

Foster, J.L., Shipstead, Z., Harrison, T.L., Hicks, K.L., Redick, T.S., & Engle, R.W. (2015). Shortened complex span tasks can reliably measure working memory capacity. *Memory & Cognition, 43*(2), 226-236.

Fritz, M.S., & MacKinnon, D.P. (2007). Required sample size to detect the mediated effect. *Psychological Science, 18*(3), 233-239.

Hamilton, J.P., Etkin, A., Furman, D.J., Lemus, M.G., Johnson, R.F., & Gotlib, I.H. (2012). Functional neuroimaging of major depressive disorder: A meta-analysis and new integration of baseline activation and neural response data. *American Journal of Psychiatry, 169*(7), 693-703.

Hayes, A.F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*: Guilford Press.

Hermans, D., Vandromme, H., Debeer, E., Raes, F., Demyttenaere, K., Brunfaut, E., & Williams, J.M.G. (2008). Overgeneral autobiographical memory predicts diagnostic status in depression. *Behaviour Research and Therapy, 46*(5), 668-677.

Herrera, M. (2017). *Potential relation between vascular depression and autobiographical memory specificity in an older adult population according to the CaR-FA-X model.* (Masters of Science), University of Central Florida.

Herrmann, L.L., Le Masurier, M., & Ebmeier, K.P. (2008). White matter hyperintensities in late life depression: A systematic review. *Journal of Neurology, Neurosurgery & Psychiatry, 79*(6), 619-624.

Herzog, A.R., & Wallace, R.B. (1997). Measures of cognitive functioning in the AHEAD study. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 52B*, 37-48.

Hilt, L.M., & Pollak, S.D. (2012). Getting out of rumination: Comparison of three brief interventions in a sample of youth. *Journal of Abnormal Child Psychology, 40*(7), 1157-1165.

Hutcheson, G.D., & Sofroniou, N. (1999). *The multivariate social scientist: Introductory statistics using generalized linear models*: Sage.

Jurado, M.B., & Rosselli, M. (2007). The elusive nature of executive functions: A review of our current understanding. *Neuropsychology Review, 17*(3), 213-233.

Kalayam, B., & Alexopoulos, G.S. (1999). Prefrontal dysfunction and treatment response in geriatric depression. *Archives of General Psychiatry, 56*(8), 713-718.

Knopman, D.S., Roberts, R.O., Geda, Y.E., Pankratz, V.S., Christianson, T.J., Petersen, R.C., & Rocca, W.A. (2010). Validation of the telephone interview for cognitive status-modified in subjects with normal cognition, mild cognitive impairment, or dementia. *Neuroepidemiology, 34*(1), 34-42.

Lach, H.W., Chang, Y.P., & Edwards, D. (2010). Can older adults with dementia accurately report depression using brief forms? Reliability and validity of the Geriatric Depression Scale. *Journal of Gerontological Nursing, 36*(5), 30-37.

Latorre, J.M., Ricarte, J.J., Serrano, J.P., Ros, L., Navarro, B., & Aguilar, M.J. (2013). Performance in autobiographical memory of older adults with depression symptoms. *Applied Cognitive Psychology, 27*(2), 167-172.

Lesser, I.M., Boone, K.B., Mehringer, C.M., & Wohl, M.A. (1996). Cognition and white matter hyperintensities in older depressed patients. *The American Journal of Psychiatry, 153*(10), 1280.

Manolio, T.A., Kronmal, R.A., Burke, G.L., Poirier, V., O'Leary, D.H., Gardin, J.M., . . . Bryan, R.N. (1994). Magnetic resonance abnormalities and cardiovascular disease in older adults. The Cardiovascular Health Study. *Stroke, 25*(2), 318-327.

Mast, B.T., MacNeill, S.E., & Lichtenberg, P.A. (2004). Post-stroke and clinically-defined vascular depression in geriatric rehabilitation patients. *The American Journal of Geriatric Psychiatry, 12*(1), 84-92.

Mast, B.T., Miles, T., Penninx, B.W., Yaffe, K., Rosano, C., Satterfield, S., . . . Simonsick, E.M. (2008). Vascular disease and future risk of depressive symptomatology in older adults: Findings from the Health, Aging, and Body Composition study. *Biological Psychiatry, 64*(4), 320-326. doi: S0006-3223(08)00169-8 [pii] 10.1016/j.biopsych.2008.01.025

Mast, B.T., Neufeld, S., MacNeill, S.E., & Lichtenberg, P.A. (2004). Longitudinal support for the relationship between vascular risk factors and late-life depressive symptoms. *The American Journal of Geriatric Psychiatry, 12*(1), 93-101.

Nelson, K., & Fivush, R. (2004). The emergence of autobiographical memory: A social cultural developmental theory. *Psychological Review, 111*(2), 486.

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109*(3), 504.

Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology, 77*(5), 1061.

Nolen-Hoeksema, S., Wisco, B.E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*(5), 400-424.

Norman, D.A., & Bobrow, D.G. (1979). Descriptions: An intermediate stage in memory retrieval. *Cognitive Psychology, 11*(1), 107-123.

Park, D.C., & Schwarz, N. (2000). *Cognitive aging: A primer*: Psychology Press.

Piolino, P., Coste, C., Martinelli, P., Macé, A.-L., Quinette, P., Guillery-Girard, B., & Belleville, S. (2010). Reduced specificity of autobiographical memory and aging: Do the executive

and feature binding functions of working memory have a role? *Neuropsychologia, 48*(2), 429-440.

Price, J.L., & Drevets, W.C. (2010). Neurocircuitry of mood disorders. *Neuropsychopharmacology, 35*(1), 192-216.

Raes, F., Hermans, D., Williams, J.M.G., & Eelen, P. (2007). A sentence completion procedure as an alternative to the Autobiographical Memory Test for assessing overgeneral memory in non-clinical populations. *Memory, 15*(5), 495-507.

Raes, F., Verstraeten, K., Bijttebier, P., Vasey, M.W., & Dalgleish, T. (2010). Inhibitory control mediates the relationship between depressed mood and overgeneral memory recall in children. *Journal of Clinical Child & Adolescent Psychology, 39*(2), 276-281.

Raes, F., Watkins, E.R., Williams, J.M.G., & Hermans, D. (2008). Non-ruminative processing reduces overgeneral autobiographical memory retrieval in students. *Behaviour Research and Therapy, 46*(6), 748-756.

Redick, T.S., Broadway, J.M., Meier, M.E., Kuriakose, P.S., Unsworth, N., Kane, M.J., & Engle, R.W. (2012). Measuring working memory capacity with automated complex span tasks. *European Journal of Psychological Assessment*.

Respino, M., Jaywant, A., Kuceyeski, A., Victoria, L.W., Hoptman, M.J., Scult, M.A., . . . Murri, M.B. (2019). The impact of white matter hyperintensities on the structural connectome in late-life depression: Relationship to executive functions. *NeuroImage: Clinical, 23*, 101852.

Ros, L., Latorre, J.M., & Serrano, J.P. (2009). Working memory capacity and overgeneral autobiographical memory in young and older adults. *Aging, Neuropsychology, and Cognition, 17*(1), 89-107.

Rule, B.G., Harvey, H.Z.A., & Dobbs, A.R. (1990). Reliability of the Geriatric Depression Scale for younger adults. *Clinical Gerontologist, 9*(2), 37-43.

Scott, R., & Paulson, D. (2018). Cerebrovascular burden and depressive symptomatology interrelate over 18 years: Support for the vascular depression hypothesis. *International Journal of Geriatric Psychiatry, 33*(1), 66-74.

Sneed, J.R., Rindskopf, D., Steffens, D.C., Krishnan, K.R., & Roose, S.P. (2008). The vascular depression subtype: Evidence of internal validity. *Biological Psychiatry, 64*(6), 491-497. doi: S0006-3223(08)00403-4 [pii] 10.1016/j.biopsych.2008.03.032

Soares, J.C., & Mann, J.J. (1997). The functional neuroanatomy of mood disorders. *Journal of Psychiatric Research, 31*(4), 393-432.

Sobin, C., & Sackeim, H.A. (1997). Psychomotor symptoms of depression. *The American Journal of Psychiatry, 154*(1), 4.

Spinhoven, P., Bockting, C.L., Kremers, I.P., Schene, A.H., & Williams, J.M.G. (2007). The endorsement of dysfunctional attitudes is associated with an impaired retrieval of specific autobiographical memories in response to matching cues. *Memory, 15*(3), 324-338.

Sumner, J.A. (2012). The mechanisms underlying overgeneral autobiographical memory: An evaluative review of evidence for the CaR-FA-X model. *Clinical Psychology Review, 32*(1), 34-48.

Sumner, J.A., Griffith, J.W., & Mineka, S. (2011). Examining the mechanisms of overgeneral autobiographical memory: Capture and rumination, and impaired executive control. *Memory, 19*(2), 169-183.

Sutherland, K., & Bryant, R.A. (2007). Rumination and overgeneral autobiographical memory. *Behaviour Research and Therapy, 45*(10), 2407-2416.

T O'Brien, J., Erkinjuntti, T., Reisberg, B., Roman, G., Sawada, T., Pantoni, L., . . . Gorelick, P.B. (2003). Vascular cognitive impairment. *The Lancet Neurology, 2*(2), 89-98.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research, 27*(3), 247-259.

Verbruggen, F., & Logan, G.D. (2008). Response inhibition in the stop-signal paradigm. *Trends in Cognitive Sciences, 12*(11), 418-424.

Von Hippel, W., Vasey, M.W., Gonda, T., & Stern, T. (2008). Executive function deficits, rumination and late-onset depressive symptoms in older adults. *Cognitive Therapy and Research, 32*(4), 474-487.

Welsh, K.A., Breitner, J.C.S., & Magruder-Habib, K.M. (1993). Detection of dementia in the elderly using telephone screening of cognitive status. *Neuropsychiatry Neuropsychol Behav Neurol, 6*(2), 103-110.

Williams, J.M.G. (1999). Depression and the specificity of autobiographical memory. In D. C. Ruben (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 244-267): Cambridge University Press.

Williams, J.M.G., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin, 133*(1), 122.

Williams, J.M.G., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology, 95*(2), 144.

Winthorpe, C., & Rabbitt, P. (1988). Working memory capacity, IQ, age, and the ability to recount autobiographical events. *Practical Aspects of Memory: Current Research and Issues, 2*, 175-179.

Yochim, B., Mast, B.T., & Lichtenberg, P.A. (2003). Cerebrovascular risk factors and depressed mood in inner city older adults. *Clinical Psychologist, 7*(1), 11-20. doi: 10.1080/13284200410001707443