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# COGNITION, REPETITIVE THOUGHT, AND SYSTEMIC INFLAMMATION IN THE MIDLIFE IN THE UNITED STATES STUDY

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# COGNITION, REPETITIVE THOUGHT, AND SYSTEMIC INFLAMMATION IN THE MIDLIFE IN THE UNITED STATES STUDY

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THESIS

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A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science
in the College of Arts and Sciences
at the University of Kentucky

By

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ABSTRACT OF THESIS

COGNITION, REPETITIVE THOUGHT, AND SYSTEMIC INFLAMMATION IN THE MIDLIFE IN THE UNITED STATES STUDY

Segerstrom et al. (2017) found that more repetitive thought (RT) was related to lower interleukin-6 (IL-6), in older adults at average IQ. This study aimed to replicate and extend this finding in midlife adults, with a daily measure of RT, and additional inflammatory biomarkers. 153 participants were drawn from the Midlife in the United States (MIDUS) Refresher project; ages 25-70 ($M$ = 45.07, $SD$ = 10.96), 50.3% female, and 83% Caucasian. Cognition was assessed via the Brief Test of Adult Cognition by Telephone, biological data via fasted blood draw, and RT data were collected as part of the National Study of Daily Experiences daily diary. Total RT (amount one engages in RT) and RT valence (positive vs. negative thought content) were analyzed. As IQ increased, more positive RT was associated with lover levels of IL-6 and CRP after adjusting for age, BMI, and statin use ($\beta$ = -.161, $p$ = .029; $\beta$ = -.240, $p$ = .002). Results did not replicate Segerstrom et al. (2017) but suggested that crystallized intelligence and RT total reflect a cognitive system different than that of fluid intelligence, executive functioning, and RT valence. Future studies should continue to investigate effects of RT on health outcomes.

KEYWORDS: repetitive thought, intelligence, IL-6, inflammation, MIDUS, cognition

Elana M. Gloger

March 29th, 2019

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# CHAPTER ONE: INTRODUCTION

Poor cognitive abilities may contribute to higher risk of smoking, being obese, higher blood pressure, higher risk for psychiatric disorders, less success in managing health behaviors, and higher levels of mortality in adults aged 60 years or older (Batty, Deary, & Macintyre, 2007; Batty, Deary, Schoon, & Gale, 2007a; Deary, Weiss, & Batty, 2010; Sachs et al., 2011). Cognition and intelligence may also contribute to higher levels of systemic inflammation, which can influence the development of chronic diseases and increase one's risk for stroke, heart attack, and mortality (Franceschi et al., 2000; Harris et al., 1999; Newman et al., 2009; Segerstrom, Reed, & Scott, 2017).

Segerstrom, Reed, and Scott (2017) investigated the relationship between intelligence and systemic inflammation, measured using interleukin-6 (IL-6), a pro-inflammatory cytokine. Specifically, they explored the role that repetitive thought (RT) may have in this established relationship. The 120-participant, longitudinal study of older adults ($M_{age}$ = 74 years, $M_{IQ}$ = 113), initially found that higher IQ predicted lower levels of IL-6 and that the total amount that one engages in RT (total RT) mediated this relationship. However, they also found that the amount one engaged in RT did not predict systemic inflammation in individuals with high IQ, but that more engagement in RT did predict lower levels of systemic inflammation in individuals with an average IQ; thus, IQ also moderated the relationship between total RT and systemic inflammation.

A replication of this finding could corroborate the results and expand upon what was found using a larger, more diverse sample with additional markers of systemic inflammation. Segerstrom and colleagues (2017) studied an aging population with a high

mean IQ (M = 113); a replication study focusing on a larger age range and closer-to-average IQ would increase generalizability.

In addition, Segerstrom and colleagues (2017) measured RT at the trait level, which provided a limited picture of day-to-day RT and its potential effects. Although collecting data on RT at the trait level is convenient, there are risks associated with measuring any construct retrospectively, including the biases that may skew one's memory and reporting of such events or feelings (Brown & Harris, 1978; Thompson, Skowronski, Larsen, & Betz, 2013). Additionally, there is evidence of lack of correspondence between retrospective and daily diary data collection. Stone and colleagues (1998) found that 30% of participants were not able to accurately recall what they had endorsed previously on ecological momentary assessments of coping. A daily measure of RT would provide a different, and potentially more accurate, representation of daily RT for each participant.

Lastly, Segerstrom and colleagues (2017) operationalized systemic inflammation as serum IL-6. Additional pro-inflammatory biomarkers would provide a more comprehensive understanding regarding the role of IQ and RT on systemic inflammation.

The aim of this study was to replicate the findings of Segerstrom and colleagues (2017), that IQ moderates the relationship between RT and systemic inflammation, in a sample of larger age range, closer-to-average IQ range, and with additional pro-inflammatory biomarkers: C-reactive protein (CRP) and tumor necrosis factor alpha (TNF-$\alpha$).

**Systemic Inflammation and Cognition**

Inflammation can be an acute and beneficial immune response to sites of injury, invasion, or infection (Segerstrom & Miller, 2004). However, under situations of stress, aging, or illness, systemic levels of inflammation can increase, leading to possible tissue damage, degeneration, and dysfunction of the immune system (Franceschi et al., 2000; Franceschi & Campisi, 2014; Segerstrom & Miller, 2004). Individuals whose bodies remain at low-grade, chronic states of inflammation may be at higher risk for negative health consequences such as schizophrenia, Parkinson's disease, Alzheimer's disease, and multiple sclerosis, and older individuals may be at a higher risk of morbidity and mortality (Franceschi et al., 2000; Stolp & Dziegielewska, 2009).

IL-6, CRP, and TNF-α are commonly studied inflammatory biomarkers because they are easy to detect, easy to study, and sufficiently studied in prior literature (Franceschi & Campisi, 2014; Michaud et al., 2013). IL-6, a primarily pro-inflammatory cytokine involved in specific and nonspecific immune responses, can be induced by TNF-α, a pro-inflammatory cytokine also found in the central nervous system that works to induce other cytokines to sites of injury or infection (Ershler & Keller, 2000; see reviews by Hopkins & Rothwell, 1995; Rothwell & Hopkins, 1995; Yudkin et al., 2000). IL-6 and TNF-α can regulate the synthesis of CRP, part of the innate immune system involved in cell death and inducing cytokines to sites of injury and infection (Gabay & Kushner, 1999; see review by Yudkin et al., 2000).

Systemic inflammation may be predicted and affected by cognitive deficits and decline. Decline of global cognition and executive function were related to elevated levels of IL-6 and CRP (IL-6, r = 0.56; CRP, $r = 0.53$, and IL-6, $r = 0.54$; CRP, $r = 0.54$,

respectively), even after adjusting for socioeconomic status, depression, and other clinical conditions (Kuo et al., 2005; Schram et al., 2007; Tegeler et al., 2016). In 500 middle-aged adults, worse short-term memory, verbal and mathematical reasoning, vocabulary, and verbal fluency were related to higher IL-6; after controlling for age, gender, race, and education, poorer performance on auditory recognition tasks ($\beta = 0.12$, $r = 0.13$) and working memory ($\beta = 0.13$, $r = 0.14$) remained significantly related to elevated levels of IL-6 (Marsland et al., 2006). A similar relationship was found for CRP: decline in vocabulary with age, poorer performance on tasks of short-term memory, verbal fluency, verbal and mathematical reasoning, and vocabulary were related to elevated levels of CRP in older males, with the latter two remaining significant in the adjusted model (*OR* = 1.38 and *OR* = 1.52) (Gimeno, Marmot, & Singh-Manoux, 2008). In a longitudinal study with 1,352 participants, poorer visual organization ($\beta = -0.06$) and Boston Naming Test performance ($\beta = -0.02$) (Jefferson et al., 2011) was related to higher CRP. Further, poorer reading performance after 6.3 years ($\beta = -0.68$), as well as visual organization ($\beta = -0.11$), visual attention and task switching ($\beta = 0.04$), and similarities ($\beta = -0.08$) were related to higher levels of TNF-α at baseline (Jefferson et al., 2011). Increased rate of cognitive decline in individuals with Alzheimer's disease and in healthy adults may also be associated with higher levels of TNF-α (Bruunsgaard et al., 1999; Holmes et al., 2009). Studying cognition and immune function across the lifespan is important for a comprehensive understanding of how the mind effects the body (Batty, Deary, & Macintyre, 2007; Batty, Deary, Schoon, Gale, 2007; Calvin, Batty, Lowe, & Deary, 2011).

**Repetitive Thought**

RT is defined as the "process of thinking attentively, repetitively, or frequently about oneself and one's world" (Segerstrom, Stanton, Alden, & Shortridge, 2003, p. 909). Discrete forms of RT include worry, rumination, and depressive rumination, as well as reflecting, processing, and planning. Across forms, RT qualities are subsumed under three orthogonal dimensions: Valence (positive vs. negative content), Purpose (searching, uncertain, or questioning vs. solving, certain, or affirming), and Total RT (the total amount of RT across all forms) (Segerstrom et al., 2003, 2010).

Among older adults, total RT has been found to be associated with higher IQ ($r$ = 0.26) and more perceived growth following trauma (Segerstrom, Roach, Evans, Schipper, & Darville, 2010; Segerstrom, Eisenlohr-Moul, Evans, & Ram, 2015). Although total RT was previously found to mediate the relationship between IQ and systemic inflammation, RT valence and RT purpose did not (Segerstrom et al., 2017). Further, in subsequent analyses, total RT significantly interacted with IQ, but RT valence and RT purpose did not.

Despite these null findings, there is evidence that RT valence may have some health consequences. Negative RT has associated with maladaptive health outcomes, including higher depression and anxiety, poorer subjective health, lower number of natural killer cells, a higher cortisol awakening response, higher risk for coronary heart disease and angina pectoris, and higher resting blood pressure (Basevitz, Pushkar, Chaikelson, Conway, & Dalton, 2008; Chambers & Davidson, 2000; Harrington & Blankenship, 2002; Kubzansky et al., 1997; Segerstrom, Glover, Craske, Fahey, 1999; Segerstrom, Solomon, Kemeny, & Fahey, 1998; Segerstrom, Tsao, Alden, & Craske,

2000). Alternatively, some individuals engage in positive RT through which they can work through, make sense of, and integrate the experience into their beliefs about the world (Watkins, 2008). Positive RT has not been investigated as extensively as negative RT, but there is evidence to suggest that it may lead to more adaptive health outcomes. Positive RT has associated with more posttraumatic growth, as have cognitive rehearsal and processing 3 months later in cancer patients (Calhoun et al., 2000; Salsman, Segerstrom, Brechting, Carlson, & Andrykowski, 2009). Trait-level reflection has also predicted lower levels of IL-6 (Woody, Figueroa, Benencia, & Zoccola, 2016). Although RT valence did not moderate the relationship between IQ and systemic inflammation or interact with IQ in the study by Segerstrom and colleagues (2017), these findings suggest that further exploration of RT valence effects would be meaningful.

**The Present Study**

The purpose of this study was to replicate and extend the findings of Segerstrom and colleagues (2017). The first aim of this study was to test if the interaction between total RT and cognition (measured as IQ via the North American Adult Reading Test in Segerstrom et al., 2017 and as cognition via the Brief Test of Adult Cognition in the present study), predicts systemic inflammation, thus indicating that cognition moderates the RT-systemic inflammation relationship. The second aim included exploring whether RT valence interacts with cognition, indicating that cognition moderates the RT-systemic inflammation relationship. Lastly, any relationships that did not depend on cognition were tested in mediational models to see if cognition exists as a mechanism by which the relationship between RT and systemic inflammation exists. The study tested the following hypotheses:

1. Higher levels of cognition will be related to lower levels of an inflammatory marker composite. Bonferroni-adjusted post-hoc analyses tested correlations with each inflammatory marker.
2. Individuals who engage in more total RT at lower levels of cognition will have lower levels of systemic inflammation, but lower levels of systemic inflammation are expected for individuals at higher levels of cognition regardless of the amount they engage in RT; thus, the relationship between RT and systemic inflammation will be moderated by cognition. Secondary, exploratory analyses included testing the same hypothesis with RT valence in place of total RT, such that individuals who engage in more positive RT at lower levels of cognition will have lower levels of systemic inflammation, but that those at higher levels of cognition will have lower levels of inflammation regardless of their RT valence. Bonferroni-adjusted post-hoc analyses were performed to investigate each inflammatory marker individually.
3. If hypothesis 2 is not supported, total RT and RT valence were tested in a mediation model. In that case, the following was expected based on previous findings of Segerstrom and colleagues (2017): Individuals with higher levels of cognition will report more total RT and/or more positively valenced RT, which will be associated with lower systemic inflammation. Bonferroni-adjusted post-hoc analyses were performed to investigate each inflammatory marker individually.

# CHAPTER TWO: METHODS

## Participants

The participants for the proposed study were drawn from de-identified participants of the Midlife in the United States (MIDUS) Refresher Project, collected between 2011 and 2014, which studies health and well-being in middle-aged adults. The MIDUS project was approved by the University of Wisconsin Institutional Review Board and follows national, local, and global regulations (Ryff et al., 2011-2014). Participants were selected by random digit dialing to landlines and cellphones to individuals who were noninstitutionalized, English-speaking adults in the United States (Ryff et al., 2011-2014).

Of the possible 3,577 participants who completed the initial MIDUS telephone interview and mail-in questionnaire, 225 participants completed the cognitive task over the phone, had IL-6, CRP, and TNF-α collected via blood draw, and completed the daily diary study, where they indicated at least one positive and one negative event during the 8-day diary. In total, 40 participants were excluded based on the following criteria: current smokers of cigarettes, cigars, pipes, use of chewing tobacco or snuff; current chemotherapy or radiation treatment; current pregnancy; a diagnosis of tuberculosis, thyroid disease, AIDS/HIV, or lupus/autoimmune disease; or use of opioids. Additionally, 32 participants were excluded for use of the following drugs: immunosuppressant, systemic steroids, cytotoxic drugs, TNF-α blockers, or use of more than two of the following: α or β blockers or ACE inhibitors, hormone replacement, thyroid supplements, or antidepressants, anxiolytic, hypnotics, or antipsychotics. These exclusion criteria matched that of the original study (Segerstrom et al., 2017).

The final sample consisted of 153 adults, ranging in age from 25-70 years old ($M$ = 45.07, $SD$ = 10.96). The participants were 50.3% female, 71.2% married, and 83% White/Caucasian. See Table 1 for full report of descriptive statistics.

A power analysis was conducted using G*Power 3.1 to determine the minimum effect size that may be found given a sample size of N = 153, $\alpha$-error probability = 0.05, and 80% power. The final sample would be sufficient to achieve 80% power to detect a small-to-moderate effect size of $\eta^2 = 0.049$. Segerstrom and colleagues (2017) found a similar effect size, $\eta^2 = 0.056$, in their moderation model that found that the interaction between IQ and total RT predicted IL-6; thus, the current sample should be able to sufficiently replicate the findings of the previous study.

Table 1. Descriptive Statistics

| | Mean (SD) | Minimum | Maximum | Skewness |
|---|---|---|---|---|
| Gender (%) | | | | |
| Female | 50.3 | - | - | - |
| Male | 49.7 | | | |
| Race (%) | | - | - | - |
| White | 83 | | | |
| Black/African American | 6.5 | | | |
| Native American/Alaska Native | 1.3 | | | |
| Asian | 1.3 | | | |
| Other | 7.8 | | | |
| Age | 45.07 (10.96) | 25 | 70 | .161 |
| Body Mass Index | 28.83 (7.57) | 19.41 | 77.58 | 2.363 |
| Marital Status (%) | | | | |
| Married | 71.2 | | | |
| Separated | 1.3 | - | - | - |
| Divorced | 10.5 | | | |
| Widowed | 3.3 | | | |
| Never Married | 13.7 | | | |
| Biomarker Data Collection Site (%) | | | | |
| UCLA | 34 | | | |
| UW | 31.4 | - | - | - |
| Georgetown | 34.6 | | | |

**Measures**

Measures were administered as part of a larger battery associated with the MIDUS Refresher Project.

*Demographics:* Participants provided demographic information during an initial 45-minute telephone interview and a 108-page mail questionnaire. Data were collected November 2011 – September 2014. Relevant demographic information includes age, gender, race/ethnicity, BMI, and marital status.

*Cognition:* Cognition was assessed for 20 minutes over the phone with the Brief Test of Adult Cognition by Telephone (BTACT) following the completion of the initial telephone interview and mail questionnaire. Cognition data were collected between February 2012 – September 2014 (Lachman, 2011-2014). The BTACT includes seven facets: Word List Recall, Digit Span Backward, Category Fluency, Red/Green Task, Number Series, Backward Counting, and Short-Delay Word List Recall (Ryff & Lachman, 2011-2014; Lachman, Agrigoroaei, Tun, & Weaver, 2014). In prior study of 84 healthy adults ranging from 23 to 80 years old, the BTACT had high internal consistency ($\alpha = 0.82$). A factor analysis of the seven facets of the measure found that five of the seven tasks loaded onto executive functioning; factor loadings ranged from 0.30 to 0.88 (Lachman, Agrigoroaei, Tun, & Weaver, 2014). For this reason, this measure is described as capturing "cognition" rather than "IQ" for this present study. The MIDUS Refresher project found an internal consistency of $\alpha = 0.71$ in the sample that completed the cognitive assessment over the phone (Lachman, 2011-2014).

*IL-6, CRP, TNF-$\alpha$:* Biomarker data were collected during a 24-hour hospital stay at one of three sites (UCLA, University of Wisconsin, and Georgetown University)

between October 2012 and August 2016. Participants were eligible to participate in the biomarker project following completion of the initial telephone interview and mail questionnaire (Weinstein, Ryff, & Seeman, 2012-2016). IL-6, CRP and TNF-α were assessed via fasted blood draws on Day 2 of the 24-hour hospital stay. IL-6 was measured using the Quantikine High-sensitivity ELISA kit (assay range: 0.156-10 pg/mL; inter-assay CV: 15.66%; intra-assay CV: 3.73%) at MIDUS BioCore Laboratory (University of Wisconsin, Madison, WI). TNF-α was measured by immunoelectrochemiluminescence using a V-plex Custom Human Cytokine Kit (assay range: 0.69-248 pg/mL; inter-assay CV: 7%; intra-assay CV: 3.19%) at MIDUS BioCore Laboratory (University of Wisconsin, Madison, WI). CRP was initially measured in plasma by a BNII nephelometer (assay range: 0.164-800 μg/mL; inter-assay CV: 1.08 - 4.3%; intra-assay CV: 2.3 - 4.4%). Samples with very low levels of this biomarker were re-assayed using a high sensitivity assay (immunoelectrochemiluminescence). In the beginning of 2016, CRP plasma assays were all performed using the high-sensitivity assay. However, due to "technical difficulties" of assaying plasma in the immunoelectrochemiluminescence kits, CRP was eventually assayed in serum (assay range: 0.014-216 μg/mL; inter-assay CV: 4.72 – 5.16%; intra-assay CV: 2.2 – 4.1%; Blood, Urine, and Saliva Data Documentation, p. C5). Corrections and re-assay of this data were applied at the MIDUS BioCore Laboratory (University of Wisconsin, Madison, WI), where: CRP = 0.4906 * (serum MSD value) + 0.1743.

*Repetitive Thought:* Repetitive thought data were collected as part of the MIDUS Refresher Daily Diary Project: National Study of Daily Experiences (NSDE) daily diary over the phone. The NSDE consisted of an 8-day study of self-reported daily experiences

and their effect on daily living, and 80.2% of daily diary participants completed all eight days. Data were collected October 2012 - November 2014. Participants were eligible to participate in the daily diary following completion of the initial telephone interview and mail questionnaire (Ryff & Almeida, 2012-2014). RT was assessed using the following questions and their follow-ups. First, participants either responded "yes" or "no" to the subsequent questions:

Table 2. Negatively and positively valenced questions

| **Negatively-Valenced Questions:** | **Positively-Valenced Questions:** |
|---|---|
| Did you have an argument or a disagreement today? | Did you have a positive interaction with someone today? |
| Did you avoid a disagreement today? | Did you have a positive experience at work? |
| Did anything happen at work or school? | Did you have a positive experience at home? |
| Did anything happen at home? | Did anything happen to a friend that was positive for you? |
| Did any discrimination happen to you? | Did anything else positive happen? |
| Did anything happen to a friend that stressed you? | |
| Did anything else [negative] happen to you? | |

The follow-up questions were not the same for the positively or negatively questions. If the participant responded "yes" to a positively-valenced questions, they were prompted with a follow-up question. For negative events, participants were asked two questions during each daily diary questionnaire. The following questions were used to quantify positive and negative RT:

Table 3. Negatively and positively valenced follow-up questions

| Negatively-Valenced Question Follow-up: | Positively-Valenced Question Follow-up: |
|---|---|
| How often have you thought about personal problems/ concerns? | How much have you thought about this event? |
| How often have you thought about situations that upset you? | |

The negatively valenced questions were measured on a scale of 0 = "None of the time" to 4 = "All of the time," and were strongly correlated ($r = .699$, $p < .001$). The positively valenced questions were measured on a scale of 0 = "Not at all" to 3 = "A lot". The mean of the values across the week for each participant was used to define positive and negative RT. These values were standardized, and total RT was calculated as a sum of the standardized mean values of negative RT and positive RT per person across all diaries. RT valence was calculated as the difference of the standardized mean values of negative RT and positive RT.

**Data Analyses**

1. The composite score from the BTACT was correlated with an inflammation composite comprised of the mean of each participant's standardized values of IL-6, CRP, and TNF-α.
2. The systemic inflammation composite was individually regressed on the composite score from the BTACT (cognition) and total RT, centering each around their sample mean to ensure that zero was an interpretable value. Next, the systemic inflammation composite was regressed onto cognition and total RT in one model. Last, the hypothesis that cognition moderates the relationship between total RT and systemic inflammation was investigated by regressing the systemic inflammation composite variable on the interaction between cognition and total RT. Additionally, the secondary hypothesis that

cognition moderates the relationship between RT valence and systemic inflammation was investigated by regressing the systemic inflammation composite on the interaction between cognition and RT valence. Models were adjusted with centered age and BMI values, and statin use. Post-hoc analyses to investigate each biomarker individually were conducted by regressing each individual standardized inflammatory biomarker on the interaction between cognition and total RT and cognition and RT valence in three separate models. Lastly, the Bonferroni correction was implemented to control for Type I error.

3. If cognition was found to not moderate the relationship between total RT or RT valence and systemic inflammation, total RT or RT valence were tested as mediators between cognition and systemic inflammation. Multiple regression analyses were performed using the composite score from the BTACT, the systemic inflammation composite, and the total RT or RT valence variables. Mediation was determined by the Sobel test and conducted through Preacher's PROCESS in SPSS (Preacher & Hayes, 2004; Sobel, 1982). Post-hoc analyses to investigate each inflammatory biomarker individually was conducted by regressing each individual standardized biomarker on the mediational model of cognition and total RT and/or cognition and RT valence in two separate models. The Bonferroni correction was implemented to control for Type I error.

CHAPTER THREE: RESULTS

## Descriptive Statistics

Table 4 contains the descriptive statistics and correlations among study variables, with RT variables averaged within participants. Compared with the larger MIDUS Refresher sample, this study sample had a higher cognition ($M$ = .509). Of the 153 participants in the present study, 43 had at average or below-MIDUS-average cognition (M < 0.0). Compared to the overall sample from MIDUS that completed the daily diaries, the average amount of negative RT for this study sample was lower ($M$ = -.199), but the average amount of positive RT was higher ($M$ = .073); both were significantly different compared to the overall MIDUS sample ($t$ = -3.201, $p$ = .002). Although IL-6 and CRP were correlated ($r$ = .633, $p$ < .01), TNF-α was not significantly related to either biomarker ($r$ = .138 and $r$ = .141, respectively). Therefore, the Systemic Inflammation Composite was tested to satisfy planned analyses, but univariate analyses were also necessary.

Table 4. Descriptive Statistics and Correlations of Study Variables (N = 153)

| | Mean (SD) | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| 1. Cognition | .509 (.94) | -.213** | -.039 | -.137 | -.125 | .071 | .022 | -.052 | -.161* |
| 2. Age | 45.07 (10.96) | | -.089 | -.016 | -.074 | -.051 | .037 | .106 | .268** |
| 3. RT Total | -.126 (1.0) | | | .006 | - | - | -.046 | -.083 | -.069 |
| 4. RT Valence | -.273 (1.1) | | | | - | - | -.015 | .027 | .042 |
| 5. Neg RT | -.120 (.74) | | | | | -.029 | -.042 | -.038 | -.018 |
| 6. Pos RT | .073 (.73) | | | | | | -.021 | -.078 | -.078 |
| Inflammation Composite | | | | | | | | | |
| 7. TNF-α[a] | .292 (.14) | | | | | | | .138 | .141 |
| 8. CRP[a] | -.060 (.48) | | | | | | | | .633** |
| 9. IL-6[a] | .143 (.32) | | | | | | | | |

Note. [a] = $\log_{10}$ transformed values. * $p$ < .05, ** $p$ <.01

## Hypothesis 1

Cognition was not significantly related to the systemic inflammation composite ($r$ = -0.087, $p$ = 0.285). Higher cognition was significantly related to lower levels of IL-6 ($r$

= -0.161, $p$ =.046), but this relationship did not withstand Bonferroni correction ($\alpha$ = .05/4, or 0.0125). Higher IQ was not significantly related to levels of TNF-$\alpha$ or CRP ($r$ = .022, $p$ = .784; $r$ = -0.052, $p$ = .522).

**Hypothesis 2**

Neither interaction between cognition and Total RT or cognition and RT Valence significantly predicted systemic inflammation, as a composite, in the unadjusted model ($\beta$ = -0.001, SE = .064, $\Delta R^2$ = .000; $\beta$ = -0.154, SE = .052, $\Delta R^2$ = .022). Table 5 contains the results of the adjusted model, controlling for age, BMI, and statin use. Collectively, age, BMI, and statin use accounted for 25.2% of the variance in systemic inflammation (adjusted $R^2$ = .252, $F_{change}$ = 18.114, $p$ < .001). In the adjusted models, the interaction between cognition and RT Valence significantly accounted for 4.8% of the variance in predicting systemic inflammation ($\beta$ = -0.230, $\Delta R^2$ = .048, $F_{change}$ = 10.241, $p$ = .002), but the interaction between cognition and Total RT did not ($\beta$ = .070, $\Delta R^2$ = .005, $F_{change}$ = .964, $p$ = .328).

Table 5. Adjusted Multiple Regression for Models Predicting Systemic Inflammation Composite Variable

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| | B(SE) | β | B(SE) | β | B(SE) | β | B(SE) | β |
| Total RT | | | | | | | | |
| Age | .013(.005) | .188* | .013(.005) | .192* | .012(.057) | .186* | .012(.005) | .186* |
| BMI | .045(.007) | **.468** | .045(.007) | **.470** | .045(.007) | **.468** | .046(.007) | **.478** |
| Statin Use | -.284 (.152) | -.139 | -.286(.152) | -.140 | -.278(.153) | -.136 | -.294(.154) | -.144 |
| IQ | | | .014 (.057) | .018 | .012(.057) | .015 | .016(.057) | .020 |
| Total | | | | | -.030(.051) | -.042 | -.030(.051) | -.042 |
| IQxTotal | | | | | | | .056(.057) | .070 |
| ΔR2 | **.267** | | .000 | | .002 | | .005 | |
| F Change | **18.114** | | .061 | | .340 | | .964 | |
| RT Valence | | | | | | | | |
| Age | .013(.005) | .188* | .013(.005) | .192* | .013(.005) | .190* | .015(.005) | **.221** |
| BMI | .045(.007) | **.468** | .045(.007) | **.470** | .046(.007) | **.477** | .048(.007) | **.498** |
| Statin Use | -.284 (.152) | -.139 | -.286(.152) | -.140 | -.295(.153) | -.144 | -.297(.148) | **-.146** |
| IQ | | | .014 (.057) | .018 | .009(.057) | .011 | .038(.056) | .049 |
| Valence | | | | | -.037(.050) | -.053 | -.071(.050) | -.102 |
| IQxValence | | | | | | | -.144(.045) | **-.230** |
| $\Delta R^2$ | **.267** | | .000 | | .003 | | **.048** | |
| F Change | **18.114** | | .061 | | .531 | | **10.241** | |

*Note.* Bolded results, $p < .01$; * $p < .05$

Post-hoc analyses were conducted to investigate whether cognition moderated the relationship between the RT variables and the individual inflammatory biomarkers. In the unadjusted models, the interaction between Total RT and cognition did not significantly predict levels of TNF-α (β = .013), IL-6 (β = -0.019), or CRP (β = .004). Further, the interaction between RT Valence and cognition did not significantly predict TNF-α (β = -0.078) or IL-6 (β = -0.082). However, for those with higher levels of cognition, more positive RT was related to lower levels of CRP (β = -0.179, $\Delta R^2 = .03$, $F_{change} = 4.636$, $p = .033$). In the adjusted models, age, BMI, and statin use accounted for 4.3% of the variance in TNF-α (adjusted $R^2 = .043$, $F_{change} = 3.254$, $p = .023$), 25.1% of the variance in IL-6 (adjusted $R^2 = .251$, $F_{change} = 17.984$, $p < .001$), and 17.5% of the variance in CRP (adjusted $R^2 = .175$, $F_{change} = 11.743$, $p < .001$). The interaction between cognition and Total RT did not significantly predict levels of TNF-α (β = .055, $\Delta R^2 = .003$, $F_{change}$ =

.453), IL-6 ($\beta = .035$, $\Delta R^2 = .001$, $F_{change} = .234$), or CRP ($\beta = .065$, $\Delta R^2 = .004$, $F_{change} = .747$). However, those with higher levels of cognition, and who engaged in more positive RT, had significantly lower levels of systemic IL-6 ($\beta = -0.161$, $\Delta R^2 = .024$, $F_{change} = 4.867$, $p = .029$) and CRP ($\beta = -0.240$, $\Delta R^2 = .052$, $F_{change} = 10.097$, $p = .002$) but not TNF-$\alpha$ ($\beta = -0.103$, $\Delta R^2 = .010$, $F_{change} = 1.531$, $p = .218$). See Appendix 1 for a full report of the adjusted models.

In the fully adjusted model, for individuals with higher than average cognition (1 SD above the mean), more positive RT was significantly associated with lower systemic inflammation ($\beta = -0.31$, $p = .004$). For individuals with lower than average cognition (1 SD below the mean), more positive RT was not associated with differences in systemic inflammation ($\beta = .106$, $p = .21$). Similar, significant simple main effects were obtained for CRP ($\beta = -0.306$, $p = .007$ vs. $\beta = .128$, $p = .16$; see Figure 1); similar but not statistically significant simple main effects were obtained for IL-6 ($\beta = -.210$, $p = .054$ vs. $\beta = 0.081$, $p = .35$; see Figure 2). As expected, there were not significant simple main effects of RT valence at either level of IQ on TNF-$\alpha$ ($\beta = -0.164$, $p = .188$ vs. $\beta = .023$, $p = .819$).

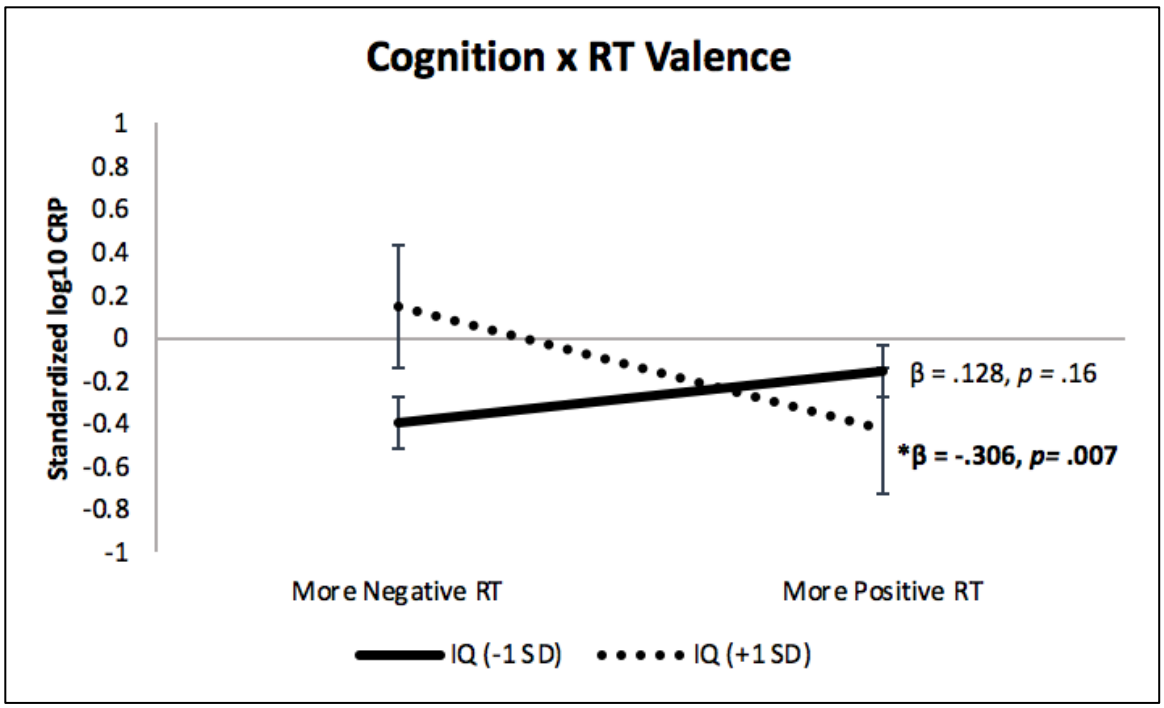


Figure 1. Simple Main Effect of CRP. Standardized $\log_{10}$ CRP for individuals 1 SD below and 1 SD above the cognition mean as predicted by more negative RT (-1 SD RT valence) and high (+1 SD RT valence). Model estimates are shown with their standard errors. CRP = C-reactive protein; RT = repetitive thought.

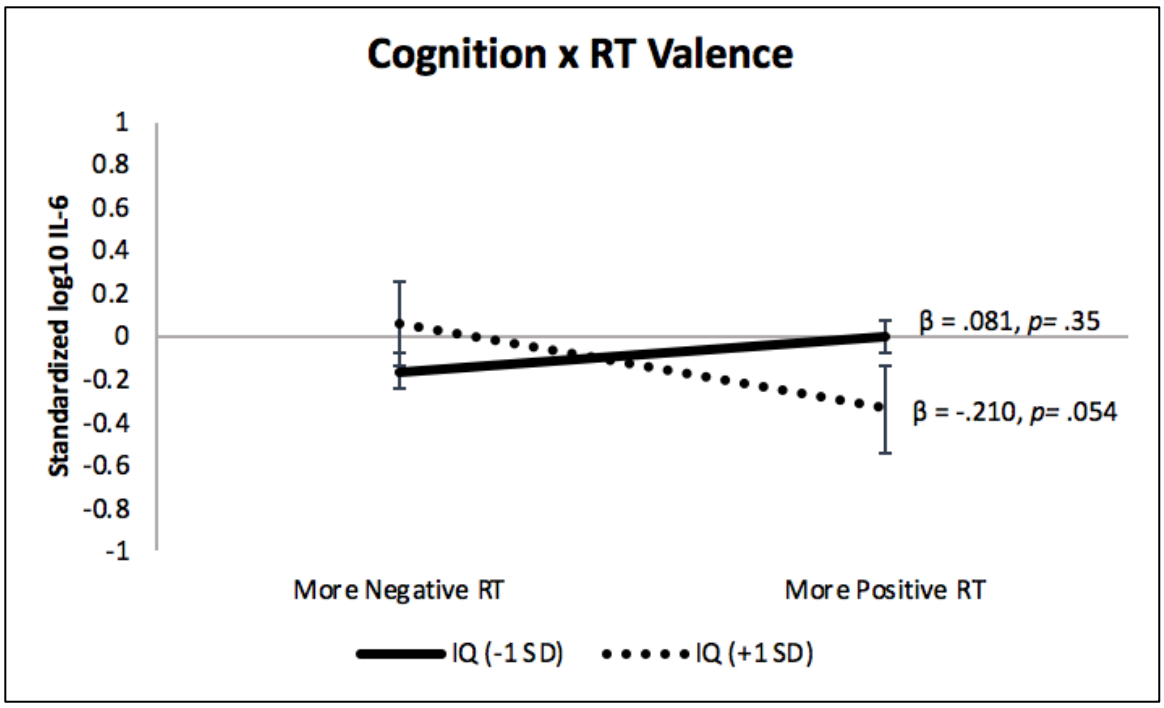


Figure 2. Simple Main Effect of IL-6. Standardized $\log_{10}$ IL-6 for individuals 1 SD below and 1 SD above the cognition mean as predicted by more negative RT (-1 SD RT valence) and high (+1 SD RT valence). Model estimates are shown with their standard errors. CRP = Interleukin-6; RT = repetitive thought.

**Hypothesis 3**

Total RT was tested as a mediator of the relationship between cognition and the systemic inflammation composite as well as each individual inflammatory biomarker because prior analyses revealed that these outcome variables did not depend on the interaction between cognition and total RT. Barron and Kenny's (1996) criteria were used to determine if mediation occurred. However, in both the non-adjusted and adjusted models, cognition was not correlated with total RT ($r$ = -0.039, $p$ = .629; $r$ = .147, $p$ =

.516), therefore, criteria for mediation were not met for models including cognition, total RT and the systemic inflammation composite, TNF-α, IL-6, or CRP.

Additionally, RT valence was tested as a mediator of the relationship between cognition and TNF-α, however, in both the unadjusted and adjusted models, cognition was not related to RT valence and therefore criteria for mediation were not met ($r$ = .137, $p$ = .091; $r$ = .211, $p$ = .148)

# CHAPTER FOUR: DISCUSSION

Driven by the established relationship between cognition and systemic inflammation, this study aimed to replicate previous findings by Segerstrom, Reed, and Scott (2017) in a larger, and more diverse sample of adults. Additionally, this studied aimed to extend the previous findings by looking at an additional facet of RT, valence, and additional biomarkers, CRP and TNF-α. Consistent with Segerstrom and colleagues (2017), individuals with higher cognition had lower levels of IL-6 but not the other biomarkers or the systemic inflammation composite. Further, although cognition consistently moderated the relationship between RT valence and systemic inflammation, it did not moderate effects of RT total and systemic inflammation. There was a significant simple main effect of cognition suggesting that individuals with higher levels of cognition, and whose thought content is more positive, have lower levels of biomarkers that are common prognostic indicators for chronic, systemic inflammation and, in later life, inflammaging.

Unlike the previous study, total RT did not interact with cognition in a way that significantly predicted systemic inflammation. However, the previous findings were extended in that IL-6 and CRP were predicted by the interaction between cognition and RT valence, even after controlling for age, BMI, and statin use. There was minimal overlap in the age range of this study (range= 25-70) compared to the previous study (range = 60-93), suggesting that RT may serve different purposes at different stages in life; in older adults, the facet of RT important for better health outcomes, may not be whether or not an individual has more positive thoughts, but instead that engage in more thinking overall. Additionally, the discrepancy in findings may be due to

measuring/capturing IQ and cognition with different measures, which can be a common problem in replication work (Open Science Collaboration, 2015).

The BTACT includes measures that load heavily onto executive function (EF), including Digit Span Backward, Category Fluency, Red/Green Task, Number Series, Backward Counting (Ryff & Lachman, 2011-2014; Lachman, Agrigoroaei, Tun, & Weaver, 2014), suggesting that the BTACT may capture executive functioning and fluid intelligence more precisely than generalized intelligence or IQ. Previous literature suggests that fluid intelligence is closely related to EF and that poorer EF may be related to any individual's propensity to engage in more negative RT (Blair & Spreen, 1989; Segerstrom et al., 2010). Alternatively, Segerstrom and colleagues (2017) used the North American Adult Reading Test to capture IQ, which reflects crystallized intelligence, rather than fluid intelligence or EF. It may be that two cognitive processing are functioning simultaneously: valence of thought content may be interacting with EF and fluid intelligence versus total RT interacting with crystallized intelligence to separately effect circulating levels of systemic inflammation. However, correlations in this study between cognition and RT valence were small and not statistically significant ($r$ = -0.137, $p$ = .091).

Segerstrom and colleagues (2017) suggested that older adults with an IQ = 103, may benefit from engaging in more RT to protect themselves from future health outcomes related to higher systemic inflammation. Alternatively, this study found a simple main effect of cognition that suggests that individuals with a higher cognitive capacity (+1 SD) had lower levels of systemic inflammation if they engaged in more positive RT. The findings from both studies suggest there are benefits of more positive

RT for individuals functioning at a higher level of cognition and more total RT for individuals functioning at an average level of cognition (based on the IQ mean in the previous sample). This may be related to other positive health behaviors and subsequent outcomes experienced by individuals with higher levels of cognition or because individuals with higher levels of cognition have a better capacity with which to self-regulate themselves (Hofmann, Schmeichel, & Baddeley, 2012; Scheier & Carver, 1987). Treatment or simple self-regulatory skills involving cognitive restructuring or "thinking positively" may be beneficial for individuals with a higher than average level of cognition in helping to work against inflammaging before its onset. On the other hand, these findings may not be generalizable to the half of the general population who function at average to lower-than-average levels of cognition. Future research should investigate the role the RT may have in effecting levels of systemic inflammation, and thus aging and other health outcomes, as it relates to lower levels of cognitive functioning.

This study was not without limitations. Whereas Segerstrom and colleagues (2017) collected data longitudinally, the MIDUS dataset provides single timepoint collections of all survey, cognitive, and biomarker data. Additional data points could have provided more reliable measures of biomarkers and cognition over time. Further, a more precise measurement of IQ through MIDUS would have provided a closer comparison between results. Although the NSDE asked "How much have you thought about this event" following each question about positive events, this was not mirrored following questions about negative events. A more direct comparison between reactions to positive and negative events may have allowed for RT to be captured more precisely. Lastly, despite a possible N = 3,577, there were only 153 adults who completed the

MIDUS survey, cognitive test, National Study of Daily Experiences, and physiological data collection that met our inclusion criteria. A larger N would have allowed for findings with more power.

Future research in this area should investigate the relationship between EF, fluid intelligence, and RT valence to better understand how one's mental abilities affect one's propensity to think positively about their world and potentially experience better future health effects. Additionally, findings in this area could help influence future treatment or skills for those who worry or ruminate to the point of dysfunction or impairment. Lastly, the third facet of RT, purpose, should be investigated to assess the role it may play in protecting against poor future health outcomes.

Overall, this study provides evidence to suggest that a more positively-focused content of RT in middle age may lead to lower levels of systemic inflammation in individuals with a higher level of cognition. In turn, this effect may help protect against future negative effects that chronic, systemic inflammation has on the aging immune system and overall functioning.

Appendix. Adjusted Multiple Regression for Moderation Models Predicting Individual Biomarker Variables

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| **TNF-α** | B(SE) | B | B(SE) | β | B(SE) | β | B(SE) | β |
| Total RT | | | | | | | | |
| Age | .008(.008) | .087 | .009(.008) | .100 | .009(.008) | .098 | .009(.008) | .097 |
| BMI | .022(.011) | .164* | .023(.011) | .171* | .023(.011) | .171* | .024(.011) | .178* |
| Statin Use | -.549(.234) | -.197* | -.556(.235) | -.199* | -.552(.237) | -.198* | -.568(.238) | -.204* |
| Cognition | | | .066(.087) | .062 | .064(.088) | .061 | .069(.088) | .064 |
| Total | | | | | -.016(.079) | -.016 | -.016(.079) | -.016 |
| CogxTotal | | | | | | | .059(.088) | .055 |
| ΔR2 | .061 | | .004 | | .000 | | .003 | |
| F Change | 3.254* | | .564 | | .040 | | .453 | |
| RT Valence | | | | | | | | |
| Age | .008(.008) | .087 | .009(.008) | .100 | .009(.008) | .098 | .010(.008) | .112 |
| BMI | .022(.011) | .164* | .023(.011) | .171* | .024(.011) | .178* | .025(.011) | .187* |
| Statin Use | -.549(.234) | -.197* | -.556(.235) | -.199* | -.567(.236) | -.203* | -.569(.236) | -.204* |
| Cognition | | | .066(.087) | .062 | .059(.088) | .056 | .077(.089) | .073 |
| Valence | | | | | -.046(.077) | -.048 | -.067(.079) | -.071 |
| CogxValence | | | | | | | -.089(.072) | -.103 |
| $\Delta R^2$ | .061 | | .004 | | .002 | | .010 | |
| F Change | 3.254* | | .564 | | .351 | | 1.531 | |
| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
| **IL-6** | B(SE) | β | B(SE) | β | B(SE) | β | B(SE) | β |
| Total RT | | | | | | | | |
| Age | .022(.007) | **.240** | .021(.007) | **.229** | .021(.007) | **.225** | .021(.007) | **.225** |
| BMI | .058(.009) | **.442** | .057(.009) | **.435** | .057(.009) | **.434** | .058(.010) | **.439** |
| Statin Use | -.120(.207) | -.043 | -.115(.208) | -.041 | -.108(.209) | -.039 | -.119(.211) | -.042 |
| Cognition | | | -.053(.077) | -.050 | -.055(.078) | -.052 | -.053(.078) | -.049 |
| Total | | | | | -.026(.070) | -.026 | -.026(.070) | -.026 |
| CogxTotal | | | | | | | .038(.078) | .035 |
| ΔR2 | .266 | | .002 | | .001 | | .001 | |
| F Change | **17.984** | | .476 | | .135 | | .234 | |
| RT Valence | | | | | | | | |
| Age | .022(.007) | **.240** | .021(.007) | **.229** | .021(.007) | **.228** | .023(.007) | **.250** |
| BMI | .058(.009) | **.442** | .057(.009) | **.435** | .058(.010) | **.439** | .060(.009) | **.453** |
| Statin Use | -.120(.207) | -.043 | -.115 | .208 | -.122(.209) | -.044 | -.124(.206) | -.044 |
| Cognition | | | -.053(.077) | -.050 | -.057(.078) | -.054 | -.029(.078) | -.027 |
| Valence | | | | | -.028(.068) | -.030 | -.061(.069) | -.065 |
| CogxValence | | | | | | | -.138(.063) | -.161* |
| $\Delta R^2$ | **.266** | | .002 | | .001 | | .024 | |
| F Change | **17.984** | | .476 | | .170 | | 4.867* | |
| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
| **CRP** | B(SE) | β | B(SE) | β | B(SE) | β | B(SE) | β |
| Total RT | | | | | | | | |
| Age | .008(.007) | .087 | .008(.007) | .093 | .008(.007) | .086 | .008(.007) | .086 |
| BMI | .056(.010) | **.423** | .056(.010) | **.426** | .056(.010) | **.424** | .057(.010) | **.433** |
| Statin Use | -.184(.218) | -.066 | -.187(.218) | -.067 | -.175(.220) | -.063 | -.194(.221) | -.070 |
| Cognition | | | .030(.081) | .028 | .026(.082) | .024 | .031(.082) | .029 |
| Total | | | | | -.048(.073) | -.049 | -.048(.073) | -.049 |
| CogxTotal | | | | | | | .070(.082) | .065 |
| ΔR2 | .191 | | .001 | | .002 | | .004 | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| F Change | **11.743** | | .132 | | .428 | | .747 | |
| RT Valence | | | | | | | | |
| Age | .008(.007) | .087 | .008(.007) | .093 | .008(.007) | .092 | .011(.007) | .124 |
| BMI | .056(.010) | **.423** | .056(.010) | .426 | .057(.010) | **.431** | .060(.010) | **.453** |
| Statin Use | -.184(.218) | -.066 | -.187(.218) | -.067 | -.196(.220) | -.070 | -.199(.213) | -.071 |
| Cognition | | | .030(.081) | .028 | .024(.082) | .023 | .067(.081) | .062 |
| Valence | | | | | -.035(.072) | -.037 | -.084(.072) | -.089 |
| CogxValence | | | | | | | -.206(.065) | **-.240** |
| $\Delta R^2$ | .191 | | .001 | | .001 | | .052 | |
| F Change | **11.743** | | .132 | | .243 | | **10.097** | |

*Note.* Bolded results, $p < .01$; * $p < .05$; Cog = Cognition

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VITA

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## EDUCATION

2017-present **University of Kentucky**, Lexington, KY
Concentration: Health Psychology
Mentor: Suzanne C. Segerstrom, PhD, MPH

2013-2017 **Ohio University**, Athens, OH, Cum Laude
Bachelor of Arts in Psychology with Honors, May 2017
Mentor: Julie Suhr, PhD
Bachelor of Science in Biological Sciences Pre-Professional, May 2017
Minor in Spanish

## RESEARCH EXPERIENCE

**University of Kentucky,** Lexington, KY

2017- 2019 Psychoneuroimmunology Research Lab
Advisor: Suzanne C. Segerstrom, PhD, MPH

2018-2019 Behavioral and Community Based Research Shared Resource Facility (BCBR-SRF), Markey Cancer Center
Advisor: Jamie Studts, PhD & Robin Vanderpool, DrPH, CHES

**Ohio University,** Athens, OH

2014-2017 BRAIN Lab
Advisor: Julie Suhr, PhD

2015-2016 Laboratory of Zincology and Neuroscience
Advisor: Yang Li, MD, PhD

## CLINICAL EXPERIENCE

**Jesse G. Harris Psychological Services Center**, Lexington, KY

2018- Present Graduate Student Therapist & Firefighter Assessments
Supervisor: Mary Beth McGavran, PhD

**Orofacial Pain Clinic**, Kentucky Clinic, Lexington, KY

2018-2019 Practicum Placed Graduate Student
Supervisors: Charles Carlson, PhD, Hayley Meadows, MS, Laura Nagy, MS

**University of Kentucky**, Lexington, KY

2017-2018 Personality and Intelligence Assessments
Supervisors: Gregory Smith, PhD, David Berry, PhD

## TEACHING EXPERIENCE

**University of Kentucky**, Lexington, KY

2018 Teaching Assistant, PSY 215
Supervisor: Andrea Friedrich, PhD

2017 Teaching Assistant, PSY 100
Supervisor: Jonathon Golding, PhD

**Ohio University,** Athens, OH

2015-2016 Undergraduate TA, Ohio University, Athens, OH
Supervisors: Susan Tice-Alicke, PhD & Ann LaComb, MA

## HONORS AND AWARDS

2017 Psychology Outstanding Graduating Senior Award, Ohio University
2017 First Place in Psychology, Ohio University Student Research and Creative Activity Expo
2016-2017 Gaige Paulson Endowed Scholarship, Psychology Department, Ohio University
2016-2017 Merit Grant, Jewish Family Service Association of Cleveland
2016-2017 Faber-Rosen Scholarship, Jewish Family Service Association of Cleveland
2016-2017 Ruth Mathewson Scholarship Fund, Biological Sciences Department, Ohio University
2016-2017 Dean's Scholarship, Ohio University
2016-2017 President, Psi Chi Chapter of Ohio University
2016 Psi Chi Membership Scholarship, Ohio University
2016 Phi Beta Kappa, National Honors Society
2016 First Place in Psychology, Ohio University Student Research and Creative Activity Expo
2016 Psi Chi, The International Honors Society in Psychology
2015-2016 Psychology Research and Teaching Endowment Scholarship, Ohio University
2013-2016 Deans List, College of Arts and Sciences
2013 Alpha Lambda Delta, National Honors Society
2013 Ohio University College of Arts and Sciences Scholar

## PROFESSIONAL DEVELOPMENT AND SERVICE

2019 RISE Award reviewer, APA Division 38
2018-2019 Society for Health Psychology, Campus Representative

2018-present Emerging Leaders Special Interest Group, American Psychosomatic Society
2017-present Advocacy Club, University of Kentucky, Lexington, KY

## RESEARCH PUBLICATIONS

**Gloger, E.**, & Segerstrom, S.C. (under review). Intelligence, repetitive thought, and systemic inflammation in the Midlife of the United States Study.

**Gloger, E.,** & Suhr, J. (under review). Mental health, cognitive, and functional consequences of poor sleep in college students.

Segerstrom, S.C., Hardy, J.K., &, **Gloger, E.** (in preparation). Exposure and reactivity to repetitive thought and the relationship between neuroticism and depressive symptoms.

## RESEARCH PRESENTATIONS

**Gloger, E.**, Segerstrom, S.C. (submitted). *Intelligence, repetitive thought, and systemic inflammation in the Midlife in the United States study*. Symposium submitted to the Association for Behavioral and Cognitive Therapies, 53rd Annual Convention, November, 2019, Atlanta, Georgia.

**Gloger, E.**, Segerstrom, S.C. (August, 2019). *Intelligence, repetitive thought, and systemic inflammation in the Midlife in the United States study*. To be presented at the American Psychological Association 2019 Annual Meeting, Division 38, Chicago, Illinois.

**Gloger, E.**, Segerstrom, S.C. (March, 2019). *Intelligence, repetitive thought, and systemic inflammation in the Midlife in the United States study*. Poster presented at the American Psychosomatic Society 77th Annual Meeting, Vancouver, British Columbia.

**Gloger, E.,** Suhr, J., Smith, J., Callahan, L., Sammler, L. (2017, October). *Sluggish Cognitive Tempo is related to sleep difficulties.* Poster presented at the National Academy of Neuropsychology annual meeting, Boston, Massachusetts.

Smith, J., Suhr, J., **Gloger, E.**, Callahan, L., Sammler, L. (2017, October). *The relation of Sluggish Cognitive Tempo (SCT) to objective indicators of processing speed and working memory.* Poster presented at the National Academy of Neuropsychology annual meeting, Boston, Massachusetts.

Callahan, L., Suhr, J., **Gloger, E.**, Smith, J., Sammler, L. (2017, October). *Is Sluggish Cognitive Tempo related to depression, anxiety, and stress?* Poster presented at the National Academy of Neuropsychology annual meeting, Boston, Massachusetts.

**Gloger, E.** (2017, April). *Is Sluggish Cognitive Tempo related to sleep quality?* Poster presented at the Ohio University Research and Creative Activity Expo, Athens, OH.

Suhr, J., Kinzer, A., & **Gloger, E.** (2016, July). *Does subjective cognitive decline accurately reflect cognitive functioning?* Poster presented at the International Neuropsychological Society mid-year meeting, London, England.

**Gloger, E.,** & Kinzer, A. (2016, April). *The relationship between subjective cognitive decline, objective cognitive impairment, and dementia worry in older adults.* Poster presented at the Ohio University Research and Creative Activity Expo, Athens, OH.