SCHREYER HONORS COLLEGE

DEPARTMENT OF BIOBEHAVIORAL HEALTH

EXAMINING THE RELATIONSHIP BETWEEN EARLY LIFE SES AND ADULT COGNITION: THE IMPORTANCE OF MATERNAL WARMTH AND INFLAMMATION

JOCELYN DIALECTOS
SPRING 2020

A thesis
submitted in partial fulfillment
of the requirements
for a baccalaureate degree
in Biobehavioral Health
with honors in Biobehavioral Health

Reviewed and approved* by the following:

Kyle Murdock
Assistant Professor of Biobehavioral Health
Thesis Supervisor

Helen Kamens
Assistant Professor of Biobehavioral Health
Honors Adviser

* Electronic approvals are on file.

ABSTRACT

Adult health can be shaped by the environment people experience as children. One example of this relationship is the association between early life socioeconomic status (SES) and adult cognition, with low childhood SES relating to worsened adult cognitive function. As low childhood SES also has been associated with high adult inflammatory levels, inflammation may mediate the relationship between early life SES and adult cognition. However, not all people of low childhood SES develop elevated inflammatory levels, and maternal warmth may act as a buffer against high inflammation. This study aimed to evaluate the relationship between childhood SES and adult cognition through inflammation, as well as to examine if there is a protective effect of maternal care. Data was collected through self-report questionnaires, a blood draw, and neuropsychological exams, which measured parental SES during childhood, perceived maternal warmth, inflammatory cytokines (interleukin-6, interleukin-1ß, and interferon gamma), and executive functioning (inhibition, updating/monitoring, and cognitive flexibility). Males demonstrated significantly higher inflammation than females ($p = 0.049$), but there were no other significant relationships found in this study. Future research needs to include a larger, more diverse sample, examine a wider range of cytokines and cognitive measures, and use more objective measurements of early life SES and maternal care in order to better understand these relationships and improve health outcomes.

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ACKNOWLEDGEMENTS

I would like to thank Dr. Murdock for giving me the opportunity to work in his lab, taking the time out of his busy schedule to help me write this thesis, and being my mentor throughout this entire experience. Thank you for all of your invaluable help and patience over these past two years. This thesis would not have been possible without you.

Thank you also to Dr. Kamens, who assisted with the editing process and took the time to answer my many questions. I appreciate the help and direction you gave me.

Lastly, I would like to thank my grandfather, Dr. Stam Zervanos, for exposing me to the research world and getting me involved in the sciences from a young age. Thank you for always being such a great role model to me.

Chapter 1

Introduction

The environment in which children are raised has many implications for their future health outcomes. In addition to a lessened overall life expectancy, having a disadvantaged socioeconomic status (SES) during childhood has been associated with increased adult rates of diabetes, coronary heart disease, and other chronic diseases that can limit daily activity (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010). Other potential aspects of health that may be related to low childhood SES include worsened cognitive function and heightened inflammatory levels (Beck et al., 2018; Carroll, Cohen, & Marsland, 2011). However, the complete effects of early life SES on later health still remain unknown. While research repeatedly has demonstrated these health consequences, not all individuals from low SES backgrounds display poor cognitive performance and high inflammation (Kaplan et al., 2001; Pollitt et al., 2007), demonstrating the importance of further understanding these relationships.

Inflammation is closely tied to immune system function. There are two types of immunity that work in conjunction to respond to foreign bodies: the innate immune system and the adaptive immune system. Innate immunity consists of natural barriers and non-specific defense mechanisms, including skin, mucous membranes, and acidic conditions in the stomach (Parkin & Cohen, 2001). As part of the innate response to pathogens, immune cells release chemicals, called cytokines, that can have either pro-inflammatory effects or anti-inflammatory effects, depending on the type. Cytokines released by leukocyte immune cells are termed interleukins, and cytokines made by certain T cells and monocyte immune cells to target viruses are called

interferons (Parkin & Cohen, 2001). Pro-inflammatory cytokines, such as interleukin-6 (IL-6), interleukin-1ß (IL1B), interferon gamma (IFN-γ), and interleukin-8 (IL-8), activate a heightened inflammatory response and can be measured to determine levels of inflammation in the body. Adaptive immunity, however, involves a more specific approach to attacking pathogens. This type of immunity uses T and B cells to activate the destruction of pathogens and stimulate antibody production. The created antibodies are unique to each different pathogen, resulting in a targeted defense against the specific invader (Parkin & Cohen, 2001). The term, inflammation, will refer to pro-inflammatory cytokines throughout the rest of this paper.

Cognition can be examined using many different variables. Executive function, which refers to the thought processes that control goal-oriented behaviors, attention, self-control, and other indicators of intellect, can be measured through inhibition, updating/monitoring, and cognitive flexibility (Anderson, Anderson, Northam, Jacobs, & Mikiewicz, 2002). Inhibition is the process of purposely overriding natural or automatic thoughts, while updating/monitoring is the ability to integrate new information and replace outdated information (Miyake et al., 2000). Cognitive flexibility concerns how well people can shift their attention between mental sets in order to adapt to new behavioral conditions (Dajani & Uddin, 2015). Other markers of cognition include abstract reasoning, verbal fluency, and visual-spatial skills. Abstract reasoning is the ability to draw logical inferences (Markovits, Thompson, & Brisson, 2015), and verbal fluency measures verbal skills and language retrieval (Shao, Janse, Visser, & Meyer, 2014). According to Mathewson (1999), visual-spatial skills refer to the coordination of vision with input from surrounding objects. Episodic memory is another aspect of cognition that can be measured, and this type of memory is used to remember personal experiences and past events (Tulving, 2002). Additionally, motor function and general cognition can be used as indicators of cognitive

function. These concepts refer, respectively, to the ability to carry out planned actions and the state of a person's overall mental capacity (Rosenbaum et al., 2002; Huntley, Gould, Liu, Smith, & Howard, 2015).

The association between early life SES and adult cognition is well established in previous literature. Adults perform worse on neuropsychological tests when they come from disadvantaged socioeconomic positions (Kaplan et al., 2001). One study, which examined cognitive ability based on executive function, verbal fluency, and visual-spatial skills, found that these measures of cognition were worse in participants from low childhood SES (Beck et al., 2018). A possible explanation for this relationship is that parents may be more likely to engage their children in intellectual activities when they do not have to worry about financial problems (Evans, 2004). Exposure to cognitive stimulation can promote increased dendritic arborization, proper development of the cortex, and greater synapse communication in the brain, all of which are associated with better cognitive function (Turrell et al., 2002). Szanton, Thorpe, and Whitfield (2010) additionally found that though economic hardship at any point in life was related to worsened cognition, having a low income level throughout childhood was associated with the greatest cognitive impairments in African American adults. General cognitive status was worse in participants that experienced financial burden during childhood, suggesting a relationship between early life SES and overall cognition (Szanton et al., 2010). Moreover, this relationship shows a graded pattern, so cognitive performance weakens with each decrease in childhood SES (Kaplan et al., 2001). Collectively, these studies demonstrate an association between low early life SES and poor performance on both general and specific measures of adult cognitive ability. This worsened cognitive performance, which includes deficits in executive functioning, verbal aptitude, memory, overall cognitive status, and other measures of cognition

(Burton, Strauss, Hultsch, & Hunter, 2006), can hinder the ability to solve everyday problems and complete daily tasks (Owsley, Sloane, McGwin Jr, & Ball, 2002; Burton et al., 2006).

Further research demonstrates another relationship between SES and inflammation, as having a disadvantaged socioeconomic position early in life is related to producing higher levels of inflammatory markers in adulthood. For example, Tyrka, Parade, Valentine, Eslinger, and Seifer (2015) found that adverse socioeconomic conditions in early life were associated with higher levels of salivary IL1B. Low SES at any age, including low status during childhood, also has been associated with increased levels of IL-6 and C-reactive protein (CRP), another marker of inflammation (Pollitt et al., 2007; Muscatell, Brosso, & Humphreys, 2018; Carroll et al., 2011). In addition, Chen, Fisher, Bacharier, and Strunk (2003) found that adolescents of low SES displayed greater levels of IFN-γ than those from higher socioeconomic backgrounds. Early life status, in particular, can predict adult inflammatory levels, and this relationship is independent of adult SES and other demographic variables (Carroll et al., 2011). One proposed mechanism for this association is that adverse socioeconomic conditions may alter the functioning of the hypothalamic-pituitary-adrenal (HPA) axis, impacting the stress response. Through the overactivation of the HPA system, individuals become exposed to excess cortisol, which can cause their immune cells to lose sensitivity to glucocorticoid signals (Carroll et al., 2011). Because glucocorticoids inhibit cytokine secretion, this insensitivity to cortisol is related to subsequent increases in adult levels of inflammatory markers, such as CRP and IL-6 (Barnes, 1998; Carroll et al., 2011). Another possible explanation for this relationship is that many of the problems disproportionately seen in low SES populations also have been found to heighten these inflammatory markers. Smoking, physical inactivity, exposure to chronic stress, insufficient social support, and obesity are associated with high levels of CRP and IL-6 (Gruenewald, Cohen,

Matthews, Tracy, & Seeman, 2009). Based on this information, underprivileged early life environments are believed to relate to heightened adult inflammatory markers. This elevated inflammation, in turn, may promote poor health by putting adults at an increased risk of atherosclerosis, stroke, vascular dementia, and other cardiovascular problems (Sartori, Vance, Slater, & Crowe, 2012).

Although the association between childhood SES and inflammation has been supported by multiple studies, not all individuals exhibit this pattern. There are some protective factors that can mitigate the negative effects of a low SES background. Warm parenting, for example, which is characterized by being supportive, responsive, and nurturing, is a known buffer for the relationship between early life SES and adult health (Murdock et al., 2018). A study on the impact of the childhood environment found that children who were exposed to stressful experiences were less likely to develop the adverse health consequences of their disadvantaged backgrounds when their mothers expressed a more positive, responsive affect in parent-child interactions (Evans, Kim, Ting, Tesher, & Shannis, 2007). In another study, which examined the protective role of maternal warmth against adult inflammatory levels, the researchers found that low SES children who experienced supportive maternal caregiving had reduced stimulated cytokine production of IL-6 in adulthood (Chen, Miller, Kobor, & Cole, 2011). Other inflammatory markers also were lower in adults who received maternal warmth throughout their socioeconomically disadvantaged childhood, suggesting that a supportive family environment may buffer the relationship between low childhood SES and heightened pro-inflammatory signaling later in life (Chen et al., 2011). Additionally, having parents who do not offer warmth and support throughout childhood is associated with an oversensitivity to stress in adulthood (Fagundes, Bennett, Derry, & Kiecolt-Glaser, 2011). This heightened stress response, along with

the lack of supportive early relationships, is related to greater levels of pro-inflammatory cytokine production (Fagundes et al., 2011). Thus, warm caregiving may modulate the relationship between childhood SES and adult inflammation, with high parental support relating to lower pro-inflammatory signaling.

In addition to its association with early life SES, inflammation also is related to adult cognitive ability. Multiple studies have demonstrated a relationship between heightened inflammation and impaired adult cognition, using both general and specific measures of cognitive function. According to Teunissen and colleagues (2003), elevated CRP was associated with weaker performance on perceptual interference tasks, even after controlling for confounding variables. Increased levels of CRP were related to higher scores on the interference test, which is indicative of impaired cognition (Teunissen et al., 2003). One study demonstrated an association between elevated IL-8 and deficits in motor function and memory (Baune et al., 2008), and another study showed that greater levels of IL-6 were associated with worsened executive function and impaired verbal ability (Marsland et al., 2015). Additionally, Yaffe and associates (2003) found that cognitive decline across a two-year span was most frequent in participants with the highest levels of CRP and IL-6. The results of their study also demonstrated that having heightened CRP and IL-6 was associated with poorer overall cognitive status (Yaffe et al., 2003). Research on this relationship suggests that chronic exposure to inflammatory cytokines may result in damage to brain tissue, including neuronal deterioration, inhibited neuron production, and atherosclerotic plaque formation (Sartori et al., 2012). Furthermore, Tyrka and colleagues (2015) found that heightened levels of IL1B related to learning impairments and other adverse changes to brain function. Though the normal aging process involves an increase in both inflammation and cognitive decline, high levels of inflammatory markers during adulthood may

play a role in worsening cognition beyond what is typical. This deterioration could involve exacerbated problems with memory, attention, and emotional regulation (Sartori et al., 2012). In concordance with these studies, heightened inflammation in adulthood is related to reductions in cognitive performance and elevations in cognitive disruption.

Circulating inflammation, which represents basal or systemic inflammation within the body, presents a major limitation in the study of inflammation. Levels of circulating cytokines often are close to or below the assay's threshold for detection, especially among healthy individuals (Steptoe, Hamer, & Chida, 2007; Monastero & Pentyala, 2017; Myśliwska, Bryl, Foerster, & Myśliwski, 1998). Additionally, cytokine levels and plasma blood volume both may fluctuate throughout the day, providing another limitation to the study of inflammation (Steptoe et al., 2007). Analyzing cytokine production after ex vivo stimulation can address such limitations (Korenromp et al., 2011; Mommersteeg, Vermetten, Kavelaars, Geuze, & Heijnen, 2008; Lieberman, Pitha, Shin, & Shin, 1989). The process of measuring stimulated cytokines attempts to mirror what is happening inside the body when immune cells are confronted with infection. For example, the current study tested monocyte and T cell inflammatory response via exposure to lipopolysaccharide (LPS) in a sample of healthy, middle-aged adults. LPS, which is a molecule present in the outer membranes of many Gram-negative bacteria, initiates an inflammatory response to levels that are readily detectable (Raetz & Whitfield, 2002). Not only are there methodological benefits to this method, but initial evidence suggests that stimulated inflammation may be more strongly associated with cognition than circulating inflammation (Magaki, Mueller, Dickson, & Kirsch, 2007).

Based on the literature reviewed above, we formulated three hypotheses. Our first hypothesis was that childhood SES would be positively associated with adult cognition.

Secondly, childhood SES would be negatively associated with inflammation among people who received high, but not low, maternal warmth. Our last hypothesis was that inflammation would explain the association between childhood SES and adult cognition.

Chapter 2

Methods

The current study included a total of 55 participants between 48 and 70 years of age (mean = 59.93 years, SD = 6.30 years). The sample consisted of 12 (21.8%) male participants and 43 (78.2%) female participants. Body mass index (BMI) ranged from 20.23 to 41.09 (mean = 26.84, SD = 4.81). Participants underwent a screening process before selection, which excluded anyone who was pregnant or nursing, was working a night shift, or was routinely using nonsteroidal anti-inflammatory (NSAID) medications. Members of the State College community and surrounding areas were recruited through local newspaper ads and flyers, which were displayed in local libraries and other public buildings. Information about the study also was posted on an online website that describes current studies in the area. For the morning of each lab visit, participants were instructed to avoid strenuous exercise and to refrain from consuming caffeine and foods high in fat. All visits started at approximately 8 am. Participants answered self-report questionnaires, had their blood drawn, and completed neuropsychological tasks of executive function. Written informed consent was obtained at the beginning of the visits. This study was approved by The Pennsylvania State University Institutional Review Board.

Measures

SES

Childhood SES was determined using the USA ladder from the MacArthur Scale of Subjective Social Status (Adler, Epel, Castellazzo, & Ickovics, 2000). This scale was chosen to evaluate early life SES because of its high reliability, as well as its significant correlation to

objective measures of educational attainment, income level, and occupational status (Operario, Adler, & Williams, 2004; Goodman et al., 2001). Through this scale, participants were asked to rank their parents' social statuses during their childhood period by drawing an "X" on an image of a ladder. The top of the ladder represented the highest level of educational attainment, income, and employment, while the bottom of the ladder represented the lowest level of educational attainment, income, and employment. Mothers and fathers were ranked on two separate ladders. Those two parental statuses then were averaged and combined into one childhood SES measure. Scores could range from 1 to 9, with 1 being the lowest status and 9 being the highest status.

Inflammation

Inflammation was measured following a blood draw. The pro-inflammatory markers that were evaluated included IL-6, IL1B, and IFN-γ. These inflammatory markers were chosen because they are stress-related cytokines that have been found to relate to childhood SES and adult cognitive function (Marsland et al., 2015; Tyrka et al., 2015; Chen et al., 2003). Whole blood was incubated in a rotational shaker and centrifuged using a force of 1500 x g over 15 minutes to stimulate LPS-induced cytokine production. All supernatants were collected after 4 hours of culture. The blood was diluted with RPMI-1640 at a 1:10 ratio, after which it was stimulated at 37 °C for 4 hours using 1 ng/mL LPS and 5% CO_2. Samples were stored at -80 °C before being analyzed through multiplex (V-PLEX) immunoassays (Meso Scale Diagnostics [MSD], Rockville, MD), which were run according to the Meso Scale Diagnostics protocol (Proinflammatory Panel, 2018). The assays were analyzed using an MSD reader in collaboration with the Biomarker Core Lab at The Pennsylvania State University. To decrease variability, different plates were used for each participant. The mean intra-assay CV values were lower than

4%, and the mean inter-assay CV values were lower than 5%. All cytokine measurements were log-transformed, z-standardized, and combined to create an overall indicator of inflammation. This creation of a cytokine composite was done due to concerns about replicability in past studies that measured individual cytokines separately, as individual analysis increased the risk of type 1 error and measurement variability between assays and plates (Horn et al., 2018; Laman, Kooistra, & Clausen, 2017). The current standard of practice is to reduce the likelihood that these limitations will influence the results by combining cytokines into one score, as has been suggested in a variety of recent studies of inflammation (e.g., Chirinos et al., 2019; Fagundes et al., 2019; Knight et al., 2020).

Maternal warmth

Maternal warmth was measured using the Parental Bonding Instrument (Parker, Tupling, & Brown, 1979). This scale demonstrates high validity and reliability, and it is considered a stable measure of perceived parenting across various emotional states and life experiences (Wilhelm, Niven, Parker, & Hadzi-Pavlovic, 2005). Participants were asked to answer survey questions that evaluated the amount of care they perceived from their mothers in their first 16 years of life. A series of 12 maternal behavior/attitude statements were listed, and participants chose the extent to which those statements applied to their own mothers. Their choices were "Very Like" (1), "Moderately Like" (2), "Moderately Unlike" (3), and "Very Unlike" (4). The numerical values from each response were combined to create a total score for maternal care.

Inhibition

The Delis-Kaplan Executive Function System (D-KEFS) color-word interference test was used to measure inhibition (Delis, Kaplan, & Kramer, 2001). This test is a modified version of the Stroop test, which is commonly used to test inhibition under experimental conditions (Stroop, 1935). The color-word interference test was chosen due to its strong psychometric properties (Latzman & Markon, 2010). During the inhibition condition of this test, participants were shown rows of words that spelled out various colors, but the words were printed in a different color than what they said. Instead of reading the words, participants were asked to say the color of the words as fast as they could. The inhibition/switching condition of the color-word interference test showed participants similar rows of colored words, though this trial also included some words with black box outlines. Participants were asked to read the word if it was surrounded by a black box and to say the color of the word if it was not. This test required participants to switch between stating the color of the words and reading the words. To create a score of inhibition, the total completion time and the total number of errors for both conditions were z-scored and combined. This score then was reverse coded, so higher values were associated with better inhibition. An overall composite was created due to evidence from prior studies that each of the indictors of inhibition selected in the present study are part of a latent construct (Murdock et al., 2016; Murdock, Oddi, & Bridgett, 2013; Latzman & Markon, 2010).

Updating/monitoring

The Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV) digit span test was administered to measure updating/monitoring (Wechsler, 2008). This test is widely used in research as a way to evaluate the ability to update and monitor information in working memory

(Richardson, 2007). Participants underwent three conditions of the digit span test, each time hearing a series of numbers read aloud to them at a one-second interval. Later trials consisted of longer number sequences. Participants first had to repeat the numbers back to a research assistant in the same order that they were spoken. For the second condition, participants were instructed to repeat the number sequence backwards. The third condition required participants to say the number sequence in numerical order from lowest to highest value. An updating/monitoring overall score was created by z-scoring and combining the total number of correct responses and the largest digit span forward. Such indicators have been shown to comprise the latent construct of updating/monitoring in prior work (e.g., Miyake et al., 2000). Higher values indicated better updating/monitoring.

Cognitive flexibility

Cognitive flexibility was measured using the computer-based Wisconsin Card Sorting Test (WCST), which is one of the most common methods of evaluating this branch of executive function (Heaton, 2003; Chan, Shum, Toulopoulou, & Chen, 2008). Participants were shown four stimulus cards that displayed different shapes of varying quantities and colors. They then were tasked with matching other cards to those four stimulus cards, without being taught how to match the patterns. Participants were told whether their choice was correct or incorrect after each matching attempt. However, the correct matching qualification changed with every ten cards, periodically requiring the participants to figure out the new matching system. Three indicators were measured during this test: perseverative responses, perseverative errors, and non-perseverative errors. Perseverative responses occurred when a participant incorrectly followed a previous matching rule, and perseverative errors occurred when a participant incorrectly used a

matching rule from the previous response. All other incorrect responses were considered non-perseverative errors. Similar to the inhibition and updating/monitoring measures, these three indicators were z-scored and combined into a cognitive flexibility score, which was reverse coded to correlate higher values with better cognitive flexibility based on previous research examining such indicators as a latent construct (Murdock et al., 2016; Murdock et al., 2013; Latzman & Markon, 2010).

Covariates

Participant age, sex, and BMI were included in the analyses as covariates due to their potential relationships with SES, inflammation, and cognition (House et al., 1990; Shanahan et al., 2013; Goosby, Cheadle, & McDade, 2016; Murman, 2015). Age and sex were self-reported by the participants, and BMI was calculated by measuring height and weight during the lab visit. Sex was coded as 0 = male and 1 = female.

Statistical Analysis

SPSS statistical software was used to perform the analyses in this study (IBM, 2017). Descriptive statistics were run to expose any outliers and visualize the data distribution. Examination of the descriptive statistics revealed that it was not necessary to remove outliers or to alter the normality of the distribution. The PROCESS macro for SPSS was utilized to simultaneously evaluate the indirect effect of childhood SES on adult cognition via inflammation, as well as the moderating role of maternal warmth on the association between

childhood SES and inflammation. The indirect effect was examined using 5,000 bootstrap samples. An alpha threshold of 0.05 was used to determine statistical significance.

Chapter 3

Results

Table 1 depicts descriptive statistics, and Table 2 illustrates Pearson correlations. Participant sex was significantly associated with the inflammation composite, with male sex corresponding to higher inflammation ($p = 0.049$). No other significant correlations were found.

Table 1. Descriptive statistics for included variables.

Variable	M or N	SD or %
Early life SES	5.23	1.68
Maternal warmth	25.91	9.42
IL1B	2.87	0.28
IL-6	2.71	0.27
IFN-γ	0.91	0.36
Color-word interference – inhibition	54.11	11.30
Color-word interference – inhibition/switching	58.31	12.03
Color-word interference – total errors	2.82	3.67
WCST – perseverative responses	12.47	9.97
WCST – perseverative errors	11.56	8.32
WCST – non-perseverative errors	17.33	16.46
Digit span	28.24	4.80
Largest digit span forward	6.73	1.38
Age	59.93	6.30
Sex		
Male	12	21.8
Female	43	78.2
BMI	26.84	4.81

Note. SES = socioeconomic status; IL1B = interleukin-1ß; IL-6 = interleukin-6; IFN-γ = interferon gamma; WCST = Wisconsin Card Sorting Test; BMI = body mass index

Table 2. Pearson correlations between primary variables.

Variable	1	2	3	4	5	6	7
1. Early life SES	–						
2. Maternal warmth	0.04	–					
3. Inflammation	0.06	0.14	–				
4. Cognitive function	-0.17	-0.06	-0.07	–			
5. Age	0.22	0.09	0.13	-0.10	–		
6. Sex	0.16	-0.13	-0.27*	0.02	-0.20	–	
7. BMI	-0.01	-0.01	0.12	-0.15	0.25	-0.26	–

* $p < 0.05$

As is portrayed in Figure 1, no significant associations were found between early life SES and adult cognitive function (β = -0.44, p = 0.41) and between early life SES and adult inflammation (β = 0.10, p = 0.85). There also was no association between inflammation and cognitive function (β = -0.03, p = 0.83). There was no significant indirect effect of early life SES on adult cognitive function through inflammation (-0.01, SE = 0.33, 95% CI = -0.67 – 0.77). Maternal warmth did not moderate the relationship between early life SES and inflammation (B = -0.01, SE = 0.01, p = 0.97).

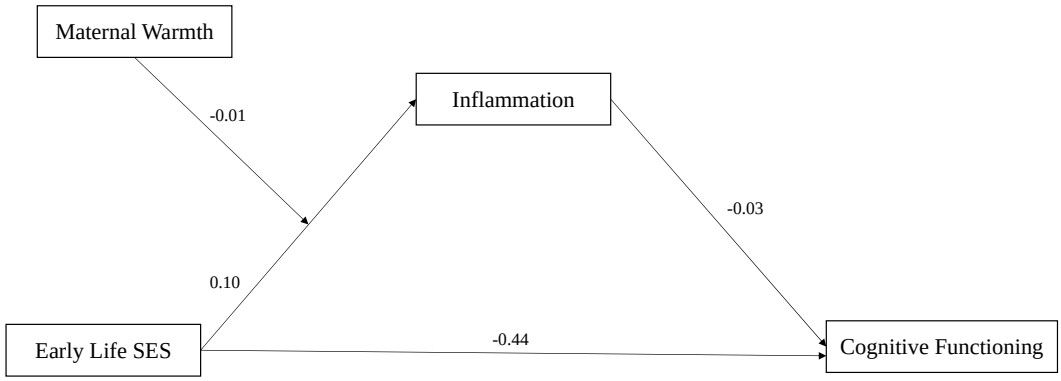

Figure 1. A moderated-mediation model depicting the association between early life SES and adult cognitive functioning via inflammation, with maternal warmth moderating the association between early life SES and inflammation. Standardized regression coefficients are shown for all relationships except that of maternal warmth, which is unstandardized. Age, sex, and BMI were included as control variables and are not depicted in the model.

Chapter 4

Discussion

Support for the three hypotheses proposed in the current study was not identified. One reason for the lack of significant findings could be that the measures of SES and maternal warmth were self-reported by the participants. Self-report measures are based on individual perceptions of events that happened in the past, so participants may not have accurately remembered their childhood environments (Kormos & Gifford, 2014). Additionally, Callahan and Eyberg (2010) found that self-report measures of SES and parental behavior are particularly vulnerable to social desirability bias. Impression management, in which people consciously attempt to make themselves appear better, and self-deception, in which participants unconsciously perceive their past to be more positive than reality, are two such biases that could have influenced the results. The SES and maternal warmth data thus may not have reflected the true situation for each participant, which could have affected the relationships examined in this study.

Another potential reason that the results were insignificant could relate to the specific cytokines tested and the inflammation score used. Unlike Pollitt and colleagues (2007), the current study analyzed IL-6, IL1B, and IFN-γ, and it did not include CRP. Though some studies were able to find significant results using IL1B and IFN-γ, those studies did not include IL-6 in their inflammation composites (Tyrka et al., 2015; Chen et al., 2003). Other previous studies examined IL-6 and CRP, but they did not measure IL1B and IFN-γ as the current study did (Muscatell et al., 2018; Gruenewald et al., 2009; Yaffe et al., 2003). Similarly, Baune and associates (2008) found significant results using IL-8, which was not included in the current study. It is possible that the types of cytokines in the composite analyzed in this study were not

associated with early life SES and adult cognition, but other cytokines may have provided significant findings. Combining the cytokines into one measure also could have affected the results. There may have been significant relationships between inflammation, cognition, and SES if each cytokine had been tested individually, as was done by Gruenewald and team (2009), Yaffe and colleagues (2003), and other prior studies on inflammation. However, testing cytokines individually is accompanied by various issues, including an increased likelihood of type 1 error and problems with replicability (Horn et al., 2018; Laman et al., 2017). The conservative decision was made to combine all measured cytokines into an overall score because the field has moved towards creating an inflammation composite in more recent studies (Tyrka et al., 2015; Chen et al., 2003). Fagundes and colleagues (2019) provide a further discussion of the advantages to forming composite scores.

A third factor that may have contributed to the lack of significant results could be the types of cognitive measures used to examine executive function. This study measured, inhibition, updating/monitoring, and cognitive flexibility to create a cognition score, but other studies have found significant results using different markers of cognitive function. Beck and associates (2018), for example, found that abstract reasoning, verbal fluency, episodic memory, and visual-spatial skills were associated with early life SES. Additionally, Baune and associates (2008) found that independent measures of memory and motor function were significantly related to inflammation, and Burton and team (2006) measured verbal skills and general cognition in their study. Had the present study employed some of these other measures of cognition, there may have been significant results. However, the current study tested measures of executive function because of their significant association with SES and inflammation in previous research (Beck et al., 2018; Marsland et al., 2015). Another reason executive function was chosen for this study is

because it has been shown to have a strong relevance to stress regulation, suggesting it could be implicated in SES, inflammation, and other stress-related factors (Bridgett, Oddi, Laake, Murdock, & Bachmann, 2013). Furthermore, inhibition, updating/monitoring, and cognitive flexibility were combined into an executive function composite due to evidence that these variables comprise a latent construct commonly referred to as executive functioning (Miyake et al., 2000). Given support for a latent construct of executive functioning and efforts to reduce the likelihood of type 1 error, it was decided a priori to utilize an overall indicator in the present study.

Limitations

A significant limitation of the present study is that the participants largely self-reported childhood SES within the middle class range. One way to increase the likelihood of significant effects being identified is by recruiting a larger sample that includes a wider SES range. For example, Beck and associates (2018) studied a large sample of various socioeconomic backgrounds in their research, and they identified a significant relationship between childhood SES and adult cognitive function. Additionally, Pollitt and team (2007) saw a significant association between SES and inflammation using a socioeconomically diverse sample. As a result, recruiting more participants – and, specifically, more individuals of low SES – could help to increase the likelihood of identifying significant effects when using the methodological strategies employed in the present study.

There were other limitations to this study that warrant consideration. In addition to having a small sample size that was not particularly low SES, the participants were mostly

middle-aged and healthy white adults. Because of the potentially unrepresentative sample, the results may be limited in that they are applicable only to the specific group that was studied. A lower SES and less healthy sample may be more likely to yield significant results. Indeed, individuals who have experienced chronic stress during their lives and those who are less healthy demonstrate enhanced inflammation in comparison to less stressed and healthier populations (Cohen et al., 2012; Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Spyridaki, Avgoustinaki, & Margioris, 2016). Over time, high levels of inflammation are thought to impact cognitive functioning (Lin et al., 2018). The sample in the present study may not have experienced the levels of chronic stress and inflammation that are necessary to impact cognitive functioning. As a result, recruiting a sample of lower SES individuals, people who experienced chronic stress, and individuals who are less healthy may lead to the identification of significant associations between childhood SES, inflammation, and cognitive functioning as was hypothesized.

Conclusion and Future Directions

We hypothesized that early life SES would be positively associated with adult cognition, that early life SES would be negatively associated with inflammation among people who received high maternal warmth, and that inflammation would mediate the association between early life SES and adult cognition. Support for those hypotheses was not identified. Therefore, we suggest a number of future directions that may be beneficial for future studies. The ethical and statistical concerns associated with analyzing measures separately should be discussed with experts in the field, and a consensus should be made regarding individual analyses. In addition, future research should use a larger, more diverse sample to further examine the influences of IL-

6, IL1B, and IFN-γ on cognitive function. To delineate which groups are protected by parental support and which groups are not, a better understanding of maternal warmth as a buffer against high inflammation in low SES children is required, as well. This research should use more objective measures of early life SES and maternal warmth to prevent any self-report bias, which may be accomplished by observing families and following up with the children longitudinally. Such research is needed because SES, inflammation, and cognition have been associated with serious health problems in previous studies. Having more extensive knowledge on these relationships will help to prevent health consequences and improve health outcomes.

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Academic Vita

Jocelyn Dialectos

Education

The Pennsylvania State University / University Park, PA / August 2016 – May 2020
College of Health and Human Development
Schreyer Honors College
Bachelor of Science
Major in Biobehavioral Health

Honors and Awards

Alumni Recognition for Student Excellence Award / Spring 2020
Rachael E. Abraham Award / Fall 2019 – Spring 2020
Evan Pugh Scholars Award for Seniors / Spring 2019
Evan Pugh Scholars Award for Juniors / Spring 2018
Bayard D. and Ethel M. Kunkle Scholarship / Fall 2018 – Spring 2019
Golden Key International Honour Society / Fall 2019
Alpha Epsilon Delta National Health Preprofessional Honors Society / Spring 2019
Sigma Alpha Pi National Society of Leadership and Success / Spring 2018
Phi Eta Sigma Honors Fraternity / Spring 2017
Dean's List / Fall 2016 – Spring 2020

Research Experience

Research Assistant, The DRIVES Lab / The Pennsylvania State University / Fall 2018 – Spring 2020
Examined the bidirectionality between cognition, stress, and biological markers of health
Research Intern, The Edward Hand Medical Museum / Summer 2019
Examined the history of diabetes diagnosis and treatment in Lancaster, PA
Research Associate, The Reading Hospital – Tower Health / Summer 2017
Examined the efficacy of the warm handoff approach in treating opioid addiction

Academic Leadership

Biobehavioral Health Society Executive Board / Spring 2020
Human Development and Family Studies Teaching Assistant / Spring 2018
Biology Lecture Assistant / Fall 2017, Fall 2018

Publications

Dialectos, J. M. (in press). The History of Diabetes Diagnosis and Treatment in Lancaster. *Journal of Lancaster General Hospital*.