Fall 12-2022

Socioeconomic Status And Cognitive Function: What Is The Role Of Social Stress?

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SOCIOECONOMIC STATUS AND COGNITIVE FUNCTION:

WHAT IS THE ROLE OF SOCIAL STRESS?

by

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A Thesis Submitted in Partial Fulfillment
of the Requirements for a Degree with Honors
(Psychology)

The Honors College

University of Maine

December 2022

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ABSTRACT

Higher socioeconomic status (SES), as indicated by income, educational attainment, and/or occupational class, has been consistently related to higher late-life cognition; however, why and how these variables are related remain unclear. As low-SES individuals experience more stressful life events and perceive these events to be more stressful than their higher-SES counterparts, patterns of stress exposures and reactivity may in part explain this gradient. Thus, the goal of this study was to characterize the relationship between educational attainment and global cognition in a sample of older adults and determine whether social stress - a composite of family stress, spouse/partner stress, and perceived discrimination - mediates this association. Sex differences in these relationships were also evaluated. Data from 1173 respondents in the Midlife in the United States (MIDUS) study were analyzed via bivariate correlation, independent samples t-test, and simple mediation analyses. Independent of age, race, chronic conditions, and depressed affect, higher education was found to predict higher scores on the MIDUS Brief Test of Adult Cognition (BTACT); the strength of this relationship did not differ significantly by sex. While male and female respondents reported similar levels of overall social stress, female respondents reported greater family and spouse/partner stress. Surprisingly, no evidence for a mediating effect of social stress was found. A discussion of theoretical and methodological explanations of this null finding, as well as future directions, is provided.

ACKNOWLEDGEMENTS

Special thanks go to Dr. Fayeza Ahmed, my mentor and advisor for this project. Her unfaltering support, constructive suggestions, and commitment to my education have motivated me throughout the thesis process and taught me research skills that I will value for a lifetime. I am also grateful for my committee members, who graciously offered their time and consideration throughout the course of this project. Lastly, thank you to the University of Maine Honors College for funding this research.

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INTRODUCTION

By 2050, the population of Americans over 65 years of age is expected to reach 85 million (Rajan et al., 2021). According to the Alzheimer's Association, the percentage of individuals with Alzheimer's disease (AD) more than doubles from ages 65-74 to 75-84 (5.3%→13.8%), and more than 34% of people over 85 years of age are living with AD (2021). With a growing and aging population, the United States is likely to see an increase in the incidence and prevalence of AD that may place undue burden on public services. For this reason, understanding factors that contribute to successful aging - especially in underprivileged populations - should be of great concern to researchers and policy makers alike. In particular, the complex effects of socioeconomic status (SES) on cognitive function require further exploration. The present study was designed to evaluate the hypothesis that SES exerts its effects on late-life cognition via social stress exposure. In order to assess these interrelationships, it was first necessary to investigate the research that is available in terms of SES inequalities in health and chronic stress effects on cognition. This review begins with a formal definition and discussion of approaches to conceptualizing and measuring SES. Various mechanisms proposed to explain the SES-cognition gradient are briefly reviewed, followed by descriptions of social stress, the stress response, and the related concept of allostatic load. Finally, evidence for stress-SES and stress-cognition relationships is presented.

Socioeconomic Status

Socioeconomic status (SES) is a complex construct purported to reflect one's social position in their community or country (Krieger et al., 1997). Researchers typically operationalize this variable with a single measure of income level, years of education, or occupational class. Moderate correlations among these indicators suggest that, although related, they likely represent distinct information about social position (Matthews & Gallo, 2011). As socioeconomic factors also play an important role in the level and change of cognitive functioning in older adults (Lee et al., 2003), the following section discusses education, income, and occupation as they relate to cognitive function.

Education

Education is by far the most widely-used SES indicator, often standing in as a proxy for income or overall SES (Braveman et al., 2005). This variable is typically operationalized as years completed or credentials of formal schooling; its popularity in the social science and epidemiology literature likely reflects its ease of measurement, stability over adulthood, and applicability to unemployed and retired individuals. In the brain health literature, studies consistently find a strong, positive association between education and cognitive function in older adults (e.g., Cadar et al., 2018). This association tends to be weaker in racial/ethnic minority populations, possibly due to differences in duration and quality of education (Manly et al., 2002). Despite the strong effects of education on baseline cognition, mixed results have been reported with respect to its association with cognitive decline. In a sample of women aged 70-79 with more than high school education, Lee et al. (2003) found that each higher level of education was associated with better cognitive function and less cognitive decline. Interestingly, they found no effect of household income or husband's educational attainment on cognitive function or decline. Zahodne and colleagues (2011), on the other hand, utilized data from the Victoria Longitudinal Study and found no association between education and rate of change for any cognitive domain. Similarly, Langa et al. (2008) compared the 1993 and 2002 cohorts from the Health and Retirement Study (HRS) and found increased rates of cognitive morbidity among those with higher levels of education. These findings lend support to the idea that education has direct effects on brain development and function, especially through the development of cognitive reserve (CR).

Indeed, researchers often view education as a CR proxy. Higher levels of education are thought to increase one's "brain reserve" through increased overall brain size, regional brain size, or greater dendritic branching (Stern, 2009). In this way, the brains of the more educated are able to sustain greater damage before reaching a threshold of clinically significant cognitive impairment. However, when this threshold is crossed, more highly-educated individuals have sustained more advanced neuropathology and thus experience more rapid cognitive decline and greater risk of mortality. In addition to this direct effect of education on cognition, a number of indirect pathways have also been proposed. Education has been found to relate to "mental stimulation" in adults' social and professional lives (e.g. Cagney & Lauderdale, 2002), and it may be that this sustained cognitive activity preserves cognitive function into late life. Greater education is also associated with improved health behaviors (e.g. avoiding smoking and excessive alcohol consumption, having an active lifestyle, etc.; Langa et al., 2008). In this way, education promotes general and health-related knowledge, literacy, problem-solving skills, prestige, influence over others and one's own life, and other characteristics (Braveman et al., 2005) that allow individuals to live more "brain healthy" lifestyles. Finally, those with more formal education tend to have better test-taking abilities, so this relationship may be artifactual (Schmand et al., 1995).

The use of education as an SES indicator is not without its limitations. Especially with research concerning older adults, cohort and period effects may be relevant. For example, relative earning potentials of educational credentials such as a high school diploma may differ markedly for those earned in 1960 versus 1990. Similarly, educational level in the United States has consistently risen in successive cohorts (National Center for Education Statistics, 2019), leading to a growing homogeneity within younger cohorts that may decrease this indicator's sensitivity for evaluating social inequalities in aging. As mentioned above, even within a cohort, education level does not have universal meaning. Some have argued that, because education is typically completed decades before dementia onset, other SES indicators may more accurately describe older adults' current resources (Cadar et al., 2018). However, this variable's stability also represents an important strength: because education level cannot decrease as one ages, use of this variable partially rules out "social selection" hypotheses. This is exceedingly important in cross-sectional studies, which cannot definitively rule out the possibility that lower cognitive function causes individuals to drift downward in the social hierarchy (as opposed to lower SES preceding cognitive impairment).

Income

Especially in the United States, SES tends to be equated with more economic factors, namely income. Although this complex variable includes wage earnings, dividends, child support, alimony, and pensions, most studies include a single question about current annual household income. In older adults, income tends to be positively associated with global cognition (Lyu & Burr, 2016; Koster et al., 2005; Singh-Manoux et al., 2005). Marden et al. (2017) found that, while high school completion had the largest effect on memory, high late-life income had the largest benefit for slowing memory decline over 10 years. Evans et al. (1997), on the other hand, found only weak associations with income and cognitive decline on a test of immediate memory in a community-based sample of individuals over the age of 65. Similar to Lee et al. (2003), they found no significant association between income and decline in their test of general cognitive function.

As with education, the sensitivity of income as a predictor of cognitive function is time- and cohort-dependent. For example, great disparities in income before retirement may not be apparent in income after retirement. Similarly, homemakers may report no personal income despite the possibility of a high household income through their spouses. For these reasons, some authors (e.g. Cagney & Lauderdale, 2002) have suggested that measures of total accumulated economic resources or “wealth” are better suited to assess SES in older adults. Regardless of the measure used, however, such economic indicators are more prone to response bias than education or occupational class.

Occupation

"Occupational class" is an SES indicator most frequently used in European public health surveillance and research (Krieger et al., 1997). In this approach, occupations are categorized hierarchically according to their associated prestige, skills, social influence, and/or power. More crude measures divide occupation into manual and non-manual labor. Evans et al. (1997) coded occupation in terms of perceived prestige, with participants assigning a set of occupations with scores out of 100. These authors found that each 5-point difference in prestige score (approximately the difference between the scores for a taxi driver and a roofer) was associated with a 20% decrease in risk of probable AD relative to all other persons and a 25% decrease in risk relative to those with no evidence of disease. Occupation is also considered to be a marker of CR. Consistent with the CR hypothesis, Künzi et al. (2021) found significantly higher perspective memory performance among adults with higher-ranking occupations.

The ordering of occupations into a scale is complex, highly subjective, and may not suit women's occupations as appropriately as they do men's. Similarly, occupational categorizations may not fully capture disparities in working conditions across different racial/ethnic groups. Black workers, for example, are more likely than their white counterparts in the same occupations to be exposed to carcinogens and other pathogenic conditions at work, and also to be paid less, even after taking into account job experience and education (Seabury et al., 2017; Bloome, 2014).

Mechanisms

Several psychosocial and biobehavioral mechanisms have been proposed to explain the consistent finding that SES, as indicated by education, income, and/or occupation, is strongly linked to cognitive function. As Adler and Snibbe (2003) note, no single mechanism completely accounts for the SES-cognition gradient; rather, it is a combination of social, environmental, and individual factors that collectively influence the aging process. The following section outlines the most empirically-supported candidate mechanisms for this relationship; although feedback loops and interactions among mediator domains are likely at play, a discussion of these is beyond the scope of this review. I begin with a discussion of health-related mediators, move to the various environmental hazards that are related to low SES, and end with brief descriptions of psychological dispositions that may play a role in the SES-cognition gradient.

Health-related. Access to primary care is exceptionally important when it comes to preventing or delaying cognitive impairment (Mullins et al., 2021). Regular visits to a primary care provider facilitate the recognition of cognitive changes over time and allow for more effective management of risk factors for dementia. Research on the relationship between cardiovascular health and dementia, for example, suggests that healthcare utilization may mitigate cognitive decline through effective management of hypertension, diabetes, and other vascular risk factors (Walker et al., 2017). Primary care physicians also encourage such behaviors as healthy dieting, smoking cessation, and regular exercise, and this may further mitigate risk factors for dementia. However, access to and utilization of healthcare services is not equally distributed across the American population; lower SES is associated with reduced access to care, poorer health outcomes, and increased mortality and morbidity as individuals age (Bassuk et al., 2002; Stringhini et al., 2018). In fact, in a cross-sectional study of nearly 50000 non-institutionalized older adults, costs were cited as a major reason for not obtaining needed care (Okoro et al., 2002). In this way, variation in healthy aging based on SES may be attributed to differential healthcare access: wealthier older adults have better access to care, and access to care may be associated with better health outcomes, both cognitive and physical. Indeed, Mullins and colleagues (2021) found that adults living in areas with low primary care supply were 25% more likely to develop cognitive impairment and that those without a regular source of medical care were 14% more likely to do so. In addition, access to care may affect one's SES through a downward trajectory, where poverty reduces access to healthcare, which leads to increased morbidity and poverty and further reductions in access to care.

Likely contributing to these findings are individual lifestyle factors and health behaviors. Those with less education and lower income are more likely to perform such risky health behaviors as smoking, having an unhealthy diet, and engaging in low levels of physical activity (Stringhini et al., 2010; Lantz et al., 1998). In turn, lower SES adults face a greater overall disease burden, including obesity, heart disease, stroke, dental disease, cancer, diabetes, and others (National Center for Health Statistics, 2021). Because SES is strongly associated with the occurrence of other common chronic diseases and with increased mortality, it is possible that the link to AD reflects a more general association of poor health with lower SES rather than a disease-specific mechanism. On the other hand, it may be that higher-SES individuals tend to possess the resources (material and psychological) that enable them to lead more "brain healthy" lives. For example, a higher income may allow individuals to better afford fresh fruits and vegetables while avoiding processed meat and refined grains. Additionally, knowledge about the nutritional value of such a diet may be conferred by higher education.

Environmental Exposures. Education and income also affect people's exposure to physical, chemical, biological, and environmental hazards. Lower SES individuals generally inhabit environments with higher levels of toxins, pathogens, and noise, and have jobs that subject them to more physical risks (Blane et al., 2013; Thoits, 2010; Tyrell et al., 2013). At the neighborhood level, residential density and crowding, inadequate housing, poor sanitation, noise, and fear of crime are all more characteristic of lower-SES environments. However, important aspects of the relationship between various lifetime exposures and late-life cognition are not well understood because of their confounding nature, especially with race/ethnicity. For example, Black workers are more likely than their white counterparts in the same occupations to be exposed to carcinogens and other pathogenic conditions at work, and also to be paid less, even after taking into account job experience and education (Evans & Kantrowitz, 2002). These exposures may in turn affect the progression of neuropathology via risk factor-related mechanisms, as described above. A complete discussion of specific pollutants, toxins, and other chemicals is beyond the scope of this review.

Psychological Dispositions. Another prominent explanation of the SES-cognition gradient is that cognitive-emotional factors and disorders play a role. Low-SES environments may kindle disproportionate levels of negative emotions and attitudes, and these variables may have deleterious effects on brain health. Similarly, positive psychosocial factors such as a sense of mastery or control over important aspects of life, self-esteem, optimism, and/or social support and engagement may buffer against AD progression directly or via modification of related risk factors. For example, greater social engagement in older adults is associated with better performance in several domains of cognitive function including memory (Ertel et al., 2008), verbal fluency (Bosma, et al., 2002), executive function (Bosma, et al., 2002), and processing speed (Hultsch, et al., 1999). Research shows that individuals with low SES typically report lower levels of such "resilient resources," (Gallo & Matthews 2003), suggesting that these variables may indeed serve as one link in the chain connecting low SES with dementia.

Stress and Allostatic Load

A final mechanism - and the topic of this research - is stress exposure and reactivity. The following sections define stress in terms of time scale, type, and physiology, including related sex differences. I then move to a discussion of allostatic load and its relationship with SES and cognition.

General Definitions

Stress is a universal human experience. Just as our ancestors faced certain hazards and threats while exercising a limited set of survival skills, modern humans are surrounded with stressors in a world that permits limited coping. It is noteworthy in the context of this review that not all stressful events are considered adverse. For example, exercise, dietary energy restriction, and environmental enrichment are mild stressors that have beneficial effects on cognitive function and may potentially increase one's resistance to further stress (Stranahan and Mattson, 2008; van Praag, 2009). However, adverse stressors have received significantly more research attention because of their strong and consistent relationship with negative health outcomes and are thus the focus of this study.

"Social stress" emerges when one's relationships, esteem, or sense of belonging within a given social context is threatened (Juth & Dickerson, 2013; unless otherwise noted, "stress" shall hereafter refer to social stress). In the field of health psychology, the term "stressor" refers to any event or situation that triggers coping adjustments collectively referred to as the "stress response." Additionally, "stress" is the process by which an individual appraises and responds to threat and "distress" encompasses the negative emotions and cognitions brought about by stress (Straub, 2017).

In the late 1960s, physician John Mason set about describing the psychological characteristics that would make any condition stressful. By summarizing the results of studies measuring the circulating levels of stress hormones before and after individuals were exposed to various situations deemed to be stressful (e.g. parachute jumping), Mason (1968) arrived at three psychological determinants that would induce a stress response in any individual exposed to them: novelty, unpredictability, and/or lack of control. More recently, the presence of social evaluative threat has been added as a fourth characteristic (Dickerson & Kemeny, 2002). Overall, these criteria suggest that distress arises less from events themselves and more from how individuals evaluate them. Indeed, a significant stressor for one person may elicit little or no distress in another.

Timescales

Stressors generally take place along the following timescales: chronic stressors, life events, daily events/hassles, and acute stress (Crosswell & Lockwood, 2020). For brevity, the two broad categories of “acute” and “chronic” shall be discussed. Acute stress refers to any transient exposure (on the order of minutes or hours) to a challenging event or circumstance (Straub, 2017). Sometimes referred to as “significant life events,” common examples include divorce and job loss. Chronic stress, on the other hand, refers to stress that lasts for a long time (days, weeks or months), either because it occurs repeatedly or continuously, or because it poses threats that are not easily adapted to or overcome (Lantz et al., 2005). It also refers to background or ambient stress due to circumstances surrounding living or working environments and to acute stressors that have effects that persist well beyond the initial event. It is important to note that naturalistic experiences of stress rarely fall neatly into one category. For example, death of a loved one is often considered a significant life event but may also be considered a chronic stressor, such as if the family member was sick for years or months before their death. Common areas in which chronic stress arises, including within families, between partners, at work, and societally (in the form of discrimination) shall be discussed next.

Domains

Family Stress. Features of the home environment are an important source of stressors. Most research on family dynamics as a source of stress focuses on caregiving responsibilities; in this context, balancing work and parenting, managing time, and maintaining financial stability all have the potential to elicit a stress response. Those older adults who care for grandchildren may face similar stressors in addition to any stress associated with changes in their relationship with their own children. Some research suggests that older adults' family stress may be more likely to take the form of negative relationships or death in the family (Widmer et al., 2017). Additionally, at least 10% of adults age 65 and older experience some form of mistreatment in a given year, including caregiver neglect, financial fraud and exploitation, and psychological abuse (Rosay & Mulford, 2017).

Marital/Partner Stress. The loss of a spouse due to death or divorce is common among older adults. Late-life intimate partner violence (IPV, also referred to as abuse in later life (ALL)) is another chronic, stress-inducing experience. Although current statistics specific to the prevalence and incidence of late-life IPV are difficult to ascertain, there is evidence to suggest that around 10% of the older adult population experiences some type of violence (Roberto et al., 2013). Such statistics do not encompass disagreements, minor arguments, or microaggressions, which are also likely to contribute to one's stress in a relationship.

Job Stress. An extensive amount of research has been devoted to examining the causes and consequences of job-related stress. In many work environments, social-evaluative threat is a frequent and explicit experience, and the need to feel accepted, liked, and included by others is associated with increased physiological arousal in the workplace (Smith, Birmingham, & Uchino, 2012). Prolonged job stress may lead to burnout, defined as a combination of exhaustion, cynicism, and decreased personal accomplishment in the workplace (Maslach et al., 2001). Especially susceptible to burnout are individuals whose occupation involves responsibility for other people (e.g. healthcare workers; Straub, 2017).

In terms of older adults, approximately 7% of the current labor force is made up of those aged 65 and above, and this value is expected to rise to 9.5% by 2030 (Bureau of Labor Statistics [BLS], 2021). Older adults may remain in the workforce entirely by choice, but it is not uncommon for them to continue working because they cannot afford to live off of social security or retirement benefits (Sewdas et al., 2017). Older workers may face different types of stressors at work, such as physical strength limitations and health concerns, gaps related to using new technology, and the engagement in work.

Discrimination. "Discrimination" refers to any behavior rooted in prejuidical attitudes or beliefs about an individual or group due to certain characteristics (e.g. gender, age, race; Williams et al., 2019). Perceptions of discrimination - subjective assessments of differential treatment based on one's devalued identity - are chronically stressful experiences that have been shown to heighten negative reactions to other daily stressors (Bolger & Zuckerman, 1995). Using data from the Midlife in the United States

Milwaukee sample, Doyle and Molix (2014) found that, among African Americans, perceived discrimination was related to greater relationship strain with family, friends and spouses. Although the majority of literature linking discrimination and health focuses on racial discrimination, exposure to any form of unfair treatment is a strong predictor of poor psychological and physical health (Lewis et al., 2015).

<u>Stress Response</u>

In general, the term “stress response” refers to the psychological, behavioral, cognitive, and physiological reactions related to stress exposure (Straub, 2017). Psychological stress responses include specific emotions triggered by the stressor, as well as efforts to regulate that emotion (Gross, 2002). Behavioral responses include coping behaviors such as smoking or seeking social support, and cognitive responses include appraisals of the exposure (e.g. as a threat versus challenge and whether one will be able to engage in successful coping; Blascovich and Mendes, 2010). Physiologically, changes in immune, autonomic, and neuroendocrine systems tend to accompany stress exposure. As neuroendocrine markers are considered primary mediators of the processes that foster cardiovascular and metabolic dysfunction and ultimately raise risk for morbidity, mortality, and functional decline (McEwen & Seeman, 1999), further details about the physiology of stress follow (see Epel et al., 2018 for details related to other domains of the stress response).

Physiology. Two physiological systems have been extensively described and reviewed in the literature as they relate to the stress response: the sympathetic nervous system (SNS, also referred to as the sympathetic-adrenal-medullary (SAM) axis) and the hypothalamic-pituitary-adrenocortical (HPA) axis. The stress response begins with the release of certain catecholamines (i.e. epinephrine and norepinephrine) into the bloodstream. Commonly referred to as the "fight or flight" response (Selye, 1976), this wave brings about increased heart rate, respiration rate, and blood pressure while suppressing non-essential functions such as digestion (McEwen & Seeman, 1999). Epinephrine also triggers the release of glucose and fats from storage sites, flooding the body with nutrients that allow it to fight the threat off or flee to safety (Straub, 2017). Once the threat is over, the parasympathetic nervous system shifts the body's priorities to "rest and digest," where heart and respiration rates decline and digestion resumes (Sapolsky, 2004).

In a second, slower, HPA response wave, corticotropin-releasing hormone (CRH) is released from the hypothalamus into the bloodstream. CRH then stimulates the anterior pituitary gland to release adrenocorticotrophin hormone (ACTH), signaling the adrenal cortex to secrete glucocorticoid hormones into the bloodstream. The main glucocorticoid in humans is cortisol, which reduces inflammation, promotes healing, and helps mobilize the body's energy sources (Straub, 2017). The HPA axis is governed by a closed-loop negative feedback system that is essential for the termination of the stress response; cortisol acts on the hypothalamus and pituitary to inhibit production of CRH and ACTH (Habib et al., 2001). Cortisol secretion is so closely linked to stress that health psychologists frequently use levels in blood or saliva to index stress exposure (Straub, 2017).

Sex & Gender Differences

While "sex" refers to the genetic and physiological differences between males and females, the term "gender" denotes the experience of being a woman or man and the traditionally differentiated social roles they accomplish (e.g., homemakers vs. resource providers; Deaux, 1985; Eagly & Wood, 2012).

Perceived Stress. Studies consistently reveal that women experience more chronic stressors than men (Lazarus and Folkman, 1984; Cohen & Janicki-Deverts, 2012). Numerous explanations for this finding have been proposed, many of which relate to socialization through gender roles. For example, women may chronically perform more "emotional labor" (e.g. "surface acting," or forcing emotions that are not wholly felt), which is associated with insomnia and family conflict (Wagner et al., 2014). Additionally, individuals in positions of power experience a greater sense of control, which leads to less perceived stress, whereas lower-status individuals experience more stress and use less efficient coping strategies (Thoits, 1991; Bakker and Demerouti, 2007). Indeed, women are six times more likely to occupy clerical positions (BLS, 2021) and thus experience the stress associated with a lack of control. Finally, growing evidence suggests that women and men are stressed by different types of situations. Men are more likely to report that finances and work-related events are sources of stress, whereas women are more likely to cite family and health-related events (Matud, 2004; McDonough & Walters, 2001).

Behavioral Response. There is some evidence to suggest that behavioral responses to stress differ between men and women. Taylor and colleagues (2000) hold that men tend to either fight or flee, whereas women tend to engage in a so-called "tend-and-befriend" stress response. Here, tending refers to nurturing activities that are adaptive when offspring are nearby, while befriending refers to the construction and maintenance of social relationships to alleviate stress. Despite these behavioral differences, men and women report approximately equal success in their attempts to manage stress (30 percent and 35 percent, respectively; American Psychological Association [APA], 2010).

Physiological Response. Findings regarding sex differences in daily cortisol profiles are mixed, with some studies reporting an increased response to waking among women and other studies showing no such sex differences (see review by Clow et al., 2004). Authors finding sex differences generally report very small effect sizes, with sex explaining only 1-3% of variability in the cortisol awakening response (Wüst et al., 2000; Pruessner et al., 1997). In terms of cortisol response to challenge, older women have been shown to mount a three-fold stronger response (Otte et al., 2004). However, this effect is mediated by stressor type: men show greater cortisol responses to mathematical and verbal challenge whereas women show greater cortisol responses to social rejection (Stroud et al., 2002).

Allostatic Load

As we have seen, stress exposure elicits a cascade of cognitive, affective, and biological changes that allow individuals to adapt to and overcome challenges. While functional in the short term, repeated activation of these systems may increase one's allostatic load, the cumulative wear and tear on the body and brain caused by frequent or prolonged physiological adaptation (McEwen, 1999). Allostatic load can increase when (a) stressors occur frequently; (b) inactivation is ineffective or (c) delayed; or (d) adaptation cannot be achieved. Allostatic load is generally operationalized by a summary index reflecting the number of extreme values on markers of neuroendocrine, cardiovascular, and metabolic functioning. For our purposes, allostatic load shall reflect the burden of chronic stress.

Relationship with Cognition. Epidemiological studies confirm that both experiencing a greater number of stressful events and reporting high perceived stress over long periods of time are associated with worse mental and physical health (Epel et al., 2018). The association between greater stressor exposure and increased disease risk has been replicated with many different types of stressor exposures (e.g. discrimination, caregiving, work stress) and a range of aging-related health outcomes (e.g. cardiovascular disease, metabolic syndrome, mortality). Specifically, it has been established that chronic stress has cumulative negative effects on the brain (Lupien et al., 2018) and cognition (Marshall et al., 2015a).

There are a number of mechanisms by which chronically high cortisol levels negatively affect the brain. According to the glucocorticoid cascade hypothesis (Sapolsky et al., 1986), cumulative exposure to glucocorticoids causes degenerative changes in the hippocampus, the brain region that normally inhibits glucocorticoid release (Jacobson and Sapolsky, 1991; Otte et al., 2005). These alterations impair the ability to terminate glucocorticoid secretion at the end of stress, resulting in increased exposure to glucocorticoids and an ever-decreasing ability of the HPA axis to recover from challenge (Sapolsky et al., 1986). Most support for this hypothesis comes from rodent studies, although human studies have shown an inverse association between hippocampal volume and cortisol levels (Lupien et al., 1999). As the hippocampus is essential for episodic memory (Bird and Burgess, 2008), hippocampal atrophy has direct consequences for cognition. Indeed, high cortisol levels in the elderly have been associated with an increased risk for dementia and Alzheimer's disease (AD) (Lupien et al., 1999; Ouanes & Popp, 2019; Ennis et al., 2017). A complete discussion of pathways through which cortisol exerts its effects on cognition is beyond the scope of this review (see Rothman & Mattson, 2010).

Similar conclusions have been drawn in terms of behavioral stress measures. For example, Aggarwal et al. (2014) examined perceived chronic stress and changes in cognitive function in over 6,000 community-based adults aged 65 years and up. They found that, as levels of chronic stress increased, cognitive function declined. Tun and colleagues (2013) found that higher levels of reported social stress were associated with slower processing speeds. In terms of specific cognitive domains, a number of cross-sectional studies have found an inverse association between the amount of cumulative stress and working memory performance among healthy older adults (Dickinson et al., 2011; Tschanz et al., 2012). This relationship has also been extended to executive function, such that greater cumulative experienced stress predicts worse inhibitory control in attentional and sensorimotor domains (Marshall et al., 2015).

Relationship with SES. Although all humans experience some amount of stress, stress exposures are not distributed evenly across social groups. Women, young adults, members of racial-ethnic minority groups, divorced and widowed persons, and poor and working class individuals report greater chronic stress and cumulative stress exposure across their lives (Thoits, 2010; Turner et al., 1995). The chronic experience of low SES could involve a number of stress-eliciting circumstances, including enduring financial hardships, insecurity about the future, and the feelings of marginalization or social exclusion that arise from social, occupational, and/or material disadvantage. Additionally, an individual's negative perception of their relative standing in a social hierarchy might affect their pattern of emotional, behavioral, and physiological reactivity to and recovery from stressors (Rahal et al., 2020); this in turn may affect one's risk for negative health outcomes.

A majority of studies show that low-SES individuals tend to score higher on measures of traumatic and life events, chronic stress, perceived stress, and daily hassles (Gallo et al., 2005; Hatch & Dohrenwend, 2007). When stressful events do happen, individuals who are socioeconomically disadvantaged tend to perceive these events as more stressful (McLeod & Kessler, 1990; Gallo & Matthews, 2003). At the same time, individuals with low SES have less access to social and material resources that would help mitigate the effects of these stressors (Adler & Stewart, 2010). As such, SES demonstrates an inverse association with indicators of psychological distress, such as anxiety and depression symptoms, hostile cognition, and angry emotion (Turner et al., 1995).

Importance, Aims, & Hypotheses

In modern society, stress is ubiquitous. Societal upheaval brought about by the coronavirus pandemic and political unrest have resulted in significant increases in reports of stress (APA, 2020), making it more important than ever to understand relationships between stress and health outcomes. Specifically, the association between social stress and cognitive function in older adults has received relatively little attention. The primary aim of this research was to investigate the interrelationships between SES, social stress, and global cognition among older adults using data from the Midlife in the United States study. Sex differences in these relationships were investigated as a sub-aim. The following questions and hypotheses guided this research:

1. What is the relationship between SES and late-life cognition?
    a. SES is directly related to late-life global cognition, such that higher SES predicts better performance on tests of global cognition.
2. Does social stress mediate the relationship between SES and global cognition?
    a. Social stress will partially mediate the relationship between SES and global cognition.
3. What type(s) of social stress do male and female respondents report the highest levels of?
    a. Females will report higher levels of family and marital/partner stress. Perceived discrimination will be equal for both groups.

4. Does social stress account for a greater proportion of variance in global cognition in male or female respondents?
    a. Social stress will be more strongly related to cognitive function among female respondents.

METHODS

Midlife in the United States (MIDUS) Study

This research made use of cognitive, biological, demographic, and psychosocial data collected in the Midlife in the United States (MIDUS) study. Initiated in 1995 (MIDUS I) by the MacArthur Foundation Research Network on Successful Midlife Development, MIDUS is a national probability sample of non-institutionalized, English-speaking respondents selected from households with a telephone. The original study designed to investigate the influence of psychological and social factors on health as people age, and longitudinal follow-ups were launched in 2004 (MIDUS II) and 2013 (MIDUS III). Measures of cognition and perceived stress were collected in MIDUS II only, so all variables have been drawn from MIDUS II.

Participants

Respondents in the MIDUS II core sample (n = 4512) were aged 35 to 86; as this research is concerned with late-life cognitive functioning, only data from respondents aged 65 and older (n = 1173) were analyzed.

Measures & Covariates

Global Cognition. Cognitive function in MIDUS II was assessed using the Brief Test of Adult Cognition by Telephone (BTACT; Lachman et al., 2014). The

BTACT was designed to enable assessment of global cognition in large community-based samples and to identify non-pathological variation in cognitive function. It includes assessments of episodic memory (EM) and executive function (EF). EM is quantified with immediate and delayed recall trials from the Rey Auditory Verbal Learning Test (RAVLT; Schmidt, 1996). EF is quantified with the Verbal Category Fluency Test (Borkowski et al., 1967), Digit Span Backward Test (Wechsler, 1997), Number Series (Salthouse & Prill, 1987), 30 Seconds and Counting Task (30-SACT; Lachman et al., 2014), and the Stop and Go Switch Task (SGST; Lachman & Tun, 2008). Composite scores for EM, EF, and global cognition were computed as mean z-scores based on the means and standard deviations of the respective tests.

Social Stress. A chronic social stress composite was created as the mean of scores on four indices of negative social interaction: family stress, partner/spouse stress, and perceived discrimination. Six family and partner/spouse items were answered on a 4-point scale ranging from one ("Often") to four ("Never"). Perceived discrimination was evaluated as an 11-item questionnaire with numerical responses corresponding to the estimated number of times a given experience occurred. See Appendix A for specific details about these constructs.

Covariates. Demographic variables used in the study were sex (male = 1; female = 2), age (continuous in years), and race/ethnicity. Participants self-reported their race, with the following response options: White, Black and/or African American, Native American or Aleutian Islander, Asian or Pacific Islander, multiracial, or Other. Because of the low number of individuals who identified as a race other than White, race was dichotomized into "non-Hispanic White" (=1) and "Other" (=2). Education was used as a proxy for SES. During telephone interviews, respondents reported the highest grade of school or year of college completed. The twelve response categories ranged from no schooling to completion of a professional degree. Education was treated as a continuous variable in analyses. Depressed affect was measured as a continuous variable based on seven yes-or-no items, and chronic conditions were dichotomized into present (=1) and absent (= 0).

Statistical Analysis

Descriptive statistics were calculated for all of the variables used in the study. To identify significant associations between variables of interest, bivariate Pearson correlations were computed for the relationships between SES, social stress, global cognition, and covariates. Sex differences in stress reporting were evaluated with independent samples t-test. To test the hypothesis that social stress partially mediates the relationship between SES and late-life cognition, mediation analyses were conducted using the SPSS PROCESS macro (Hayes, 2013) with age, sex, race, chronic conditions, and depressed affect entered as covariates. Finally, the hypothesized model was then run with data from only male and only female respondents. Confidence intervals were compared to determine significant differences in regression weights.

RESULTS

Demographic Information

Table 1
Descriptive Statistics for Predictor and Covariates

	Frequency	Minimum	Maximum	Mean	Std. Deviation
Age (years)		65	84	72.15	5.184
Sex	1173				
Male	513				
Female	660				
Race					
Non-Hispanic White	1042				
Other	75				
Highest level of education completed		1	12	6.66	2.570
Chronic Conditions		0	1	.89	.309
Present (1 or more)	1048				
Absent	125				
Depressed Affect (continuous)		0	7	.28	1.206

Data from a total of 1173 MIDUS 2 respondents aged 65 and older were used in this analysis. Descriptive statistics for demographic information and covariates are presented in Table 1. Respondents had a mean (SD) age of 72.15 (5.18) years; 513 were male (43.7%) and 660 were female (56.3%), and 1042 (88.8%) self-identified as non-Hispanic White. Educational level ranged from no school to the attainment of an advanced degree; mean education level was 6.66, corresponding to some college with no degree. Although the SES variable was intended to represent both education and income, the latter variable was dropped from analyses. Appendix B provides descriptive statistics for respondents' total income, including wages, pension, social security, and other assets. As most individuals reported a value of 0 for this item, it was deemed unreflective of current SES. Similarly, wake cortisol (nmol/L) was excluded from analyses due to its large standard deviation and tenuous association with stress exposure. Finally, a majority (1048, 89.3%) of respondents reported having at least one chronic condition, and 1056 respondents (90%) reported low levels of depressed affect.

Table 2 displays descriptive statistics by sex for the social stress composite and raw scores on its three indicators: family stress, spouse/partner stress, and perceived descrimination. Job stress was not included in the social stress composite. Scores for this variable were obtained from 335 individuals, constituting just over 28% of the sample (Appendix B). Mean z scores for the social stress composite were -0.029 and 0.018 for male and female respondents, respectively. Responses for the family stress and spouse/partner stress ranged from 1 to 4, with mean (SD) scores of 1.805 (0.533) and 1.873 (0.515) for males and females, respectively. Perceived discrimination scores ranged from 0 to 7 among male respondents, with a mean (SD) of 0.556 (1.169). Female respondents reported 0 to 8 instances of discrimination, with a mean (SD) of 0.478 (1.095).

Table 2

Descriptive Statistics by Sex for Stress Composite and Components

	Sex														
	MALE					FEMALE					Total				
	Mean	N	Std. Deviation	Minimum	Maximum	Mean	N	Std. Deviation	Minimum	Maximum	Mean	N	Std. Deviation	Minimum	Maximum
Social Stress (z score)	-.029	361	.679	-1.62	2.03	.018	297	.677	-1.62	2.47	-.008	658	.678	-1.62	2.47
Family Stress	1.805	457	.533	1	4	1.873	570	.515	1	4	1.843	1027	.524	1	4
Spouse/Partner Stress	2.015	379	.527	1	4	2.107	320	.616	1	4	2.057	699	.571	1	4
Perceived Discrimination	.556	437	1.169	0	7	.478	540	1.095	0	8	.513	977	1.129	0	8

Table 4

Pearson Correlation Coefficients – Demographic Variables with Predictors and Covariates

	Age (years)	Sex	Race	Highest level of education completed	Chronic Condition (present/absent)	Depressed Affect (continuous)	Social Stress	Family Stress	Spouse/Partner Stress	Perceived Discrimination	BTACT Composite (z score)	BTACT Episodic Memory (z score)	BTACT Executive Functioning (z score)
Age (years)	--												
Sex	-.045	--											
Race	-.042	.009	--										
Highest level of education completed	-.048	-.138**	-.051	--									
Chronic Condition (present/absent)	.051	.035	.004	-.072*	--								
Depressed Affect (continuous)	-.055	.094**	-.063*	-.031	.057	--							
Social Stress	-.089*	.035	.094*	.002	.145**	.073	--						
Family Stress	-.124**	.064*	.045	-.021	.119**	.112**	.750**	--					
Spouse/Partner Stress	-.056	.081*	.003	-.022	.060	.065	.717**	.402**	--				
Perceived Discrimination	-.090**	-.034	.149**	.123**	.083**	-.014	.588**	.193**	.105**	--			
BTACT Composite (z score)	-.308**	.034	-.136**	.401**	-.087**	.027	.048	.059	.067	.055	--		
BTACT Episodic Memory (z score)	-.275**	.222**	-.096**	.191**	-.082**	.042	.006	.030	.060	-.009	.679**	--	
BTACT Executive Functioning (z score)	-.253**	-.106**	-.121**	.358**	-.072*	.015	.028	.023	.029	.070*	.886**	.423**	--

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Independent Samples T-Test

Table 3 displays results of mean comparisons between social stress variables for male and female respondents. No sex differences were found for the social stress composite; however, female respondents reported greater family stress (t = -2.041, p < 0.05, CI [-0.132, -0.003]) and spouse/partner stress (t = -2.148, p < 0.05, CI [-0.178, -0.008]).

Table 3
Independent Samples T-Test for Sex Differences in Stress Reporting

Independent Samples Test

			Significance				95% Confidence Interval of the Difference	
	t	df	One-Sided p	Two-Sided p	Mean Difference	Std. Error Difference	Lower	Upper
Social Stress	-.889	656	.187	.375	-.0472	.053	-.151	.057
Family Stress	-2.041	1025	.021	.041	-.0671	.033	-.132	-.003
Spouse/Partner Stress	-2.148	697	.016	.032	-.0929	.043	-.178	-.008
Perceived Discrimination	1.078	975	.141	.281	.0783	.073	-.064	.221

Bivariate Correlations

All variables of interest were examined in relation to each other via bivariate Pearson correlation (Table 4). Age was significantly (p < 0.001) and negatively related to family stress and perceived discrimination, such that older respondents tended to report lower levels of each; similarly, age was negatively associated with the social stress composite (p < 0.05). Sex was inversely related to education, such that females tended to report lower levels of education (p < 0.001). Females were also more likely to report higher levels of depressed affect (p < 0.001). Although sex was not significantly related to the social stress composite, females tended to report greater family and spouse/partner stress ($p < 0.05$). Race shared a significant ($p < 0.05$) inverse relationship with depressed affect; non-White ("Other") individuals generally reported lower depressed affect. Additionally, race was positively related to social stress ($p < 0.05$).

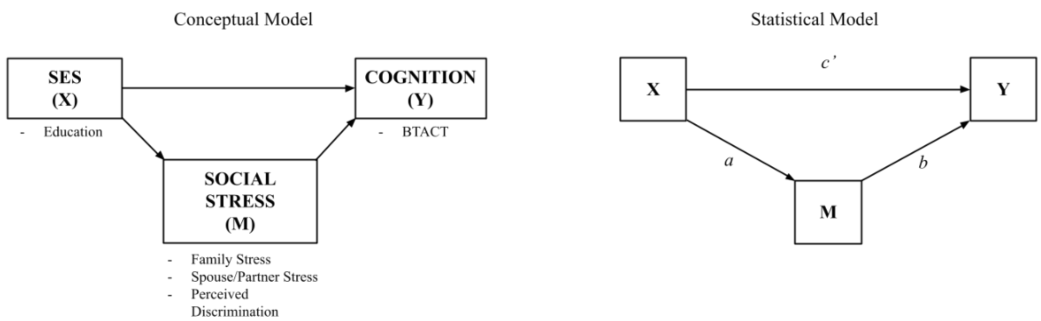
Education was inversely related to chronic conditions ($p < 0.05$) and positively associated with perceived discrimination ($p < 0.001$). The presence of at least one chronic condition was associated with higher overall social stress, family stress, and perceived discrimination ($p < 0.001$). Greater depressed affect predicted greater family stress ($p < 0.001$). Significant, positive interrelationships were also observed between the three indicators of social stress: family stress was related to spouse/partner stress and perceived discrimination ($p < 0.001$); spouse/partner stress to perceived discrimination ($p < 0.001$); and social stress was related to all three indicators ($p < 0.001$).

In terms of cognitive outcome variables, standard scores on BTACT Episodic Memory (EM) and Executive Function (EF) components were significantly ($p < 0.001$) associated with age, race, and sex. Age and race were negatively correlated with global cognition, such that younger age and non-Hispanic White race predicted higher BTACT scores. Sex shared an inverse relationship with EF and positive relationship with EM, such that female sex predicted lower scores on the former and higher scores on the latter. Sex was not significantly related to global cognition. EM and EF components were significantly ($p < 0.001$) and positively correlated with each other and global cognition. None of the cognitive variables were significantly associated with the social stress composite, although perceived discrimination shared a positive relationship with the EF component of global cognition. Education was positively associated with global cognition, EF, and EM ($p < 0.001$).

Mediation Analyses

A simple mediation analysis was conducted to examine the mediating effect of social stress on level of education and cognitive function (Figure 1), with age, sex, race, depressed affect, and chronic conditions as covariates. The PROCESS output for Model 1 can be found in Appendix C and is summarized in Table 5. Model 1 predicted 4.83% of the variance in social stress and 24.87% of the variance in BTACT scores. The total effect of the model ($c = c' + ab$) was found to be significant ($c = 0.1325$, CI [0.1085, 0.1564], $p < 0.001$). Those with higher levels of education tended to report greater social stress ($a = 0.0054$), and those reporting greater social stress earned higher scores on the BTACT ($b = 0.0490$), although neither of these relationships reached statistical significance. Independent of social stress, education level positively influenced global cognition ($c' = 0.1322$, $p < 0.001$). A bootstrap 95% confidence interval for the indirect effect of social stress ($ab = 0.0003$) based on 5,000 bootstrap samples contained zero (CI [-0.011, 0.0022]), indicating that social stress did not act as a mediator of the observed education-cognition gradient.

Figure 1
Conceptual and Statistical Models Used to Assess the Mediating Effect of Social Stress on the Relationship Between Education and Global Cognition



Note. Statistical Model 4; Hayes, 2013. Indirect effect of X on Y through M = *ab*. Direct effect of X on Y = *c'*.

The above model was also run using data from only male respondents (Model 2) and only female respondents (Model 3). Full PROCESS outputs can be found in Appendix C, respectively, and results of these analyses are summarized in Tables 6 and 7. Model 2 predicted 4.68% of the variance in social stress and 26.25% of the variance in BTACT scores. The total effect of Model 2 was found to be significant ($c = 0.13056$, CI [0.0983, 0.1576], $p < 0.001$). Male respondents with higher education tended to report higher levels of social stress ($a = 0.0044$), and those reporting greater social stress earned higher BTACT scores ($b = 0.0651$); however, neither of these relationships reached statistical significance. Independent of social stress, education level positively influenced global cognition ($c' = 0.1277$, $p < 0.001$). A bootstrap 95% confidence interval for the indirect effect of social stress ($ab = 0.0003$) based on 5,000 bootstrap samples contained zero, CI [-0.0019, 0.0035], indicating that social stress did not mediate the relationship between education and cognition observed for male respondents.

The total effect of Model 3 was found to be significant ($c = 0.1377$, CI [0.0967, 0.1787], $p < 0.001$). Model 3 predicted 5.06% of the variance in social stress and 30.36% of the variance in BTACT scores. As with male respondents, females with higher education tended to report higher levels of social stress ($a = 0.0073$), and those reporting greater social stress earned higher BTACT scores ($b = 0.0334$); however, neither of these relationships reached statistical significance. Independent of social stress, education level positively influenced global cognition ($c' = 0.1374$, $p < 0.001$). A bootstrap 95% confidence interval for the indirect effect of social stress ($ab = 0.0002$) based on 5,000 bootstrap samples contained zero, CI [-0.0024, 0.0040], indicating that social stress did not mediate the relationship between education and cognition observed for male respondents.

Estimated direct effects (c') for Models 2 and 3 are shown in Figure 2. In accordance with the visual inspection method described by Cumming and Finch (2005), significant overlap between the two 95% CIs indicates that these two unstandardized regression weights are not significantly different.

Table 5
Summary of Model 1 PROCESS Output

		Outcome								
		M (Social Stress)					Y (Global Cognition)			
Predictor (X)		*β*	SE	*p*	95% CI		*β*	SE	*p*	95% CI
(Education)	*a*	.005	.011	.611	[-.015, .026]	*c'*	.132	.012	< .001	[.108, .156]
M (Social Stress)		—	—	—	—	*b*	2.498	.490	< .001	[1.536, 3.459]
Constant	i_M	.421	.426	.323	[-.415, 1.257]	i_Y	.049	.047	0.296	[-.043, 0.141]
		$R^2 = .048$					$R^2 = 0.285$			
		$F(6, 602) = 5.09, p < .001$					$F(7, 601) = 34.17, p < .001$			

Note. *β* = unstandardized regression coefficient; SE = standard error; CI = confidence interval

Table 6
Summary of Model 2 PROCESS Output

		Outcome								
		M (Social Stress)					Y (Global Cognition)			
Predictor (X)		*β*	SE	*p*	95% CI		*β*	SE	*p*	95% CI
(Education)	*a*	.004	.013	.743	[-.022, .031]	*c'*	.128	.015	< .001	[.098, .157]
M (Social Stress)		—	—	—	—	*b*	.065	.062	.292	[-.056, .187]
Constant	i_M	.681	.550	.217	[-.402, 1.763]	i_Y	2.083	.618	< .001	[.867, 3.300]
		$R^2 = .047$					$R^2 = 0.262$			
		$F(5, 330) = 3.24, p < .05$					$F(6, 329) = 19.51, p < .001$			

Note. *β* = unstandardized regression coefficient; SE = standard error; CI = confidence interval

Table 7

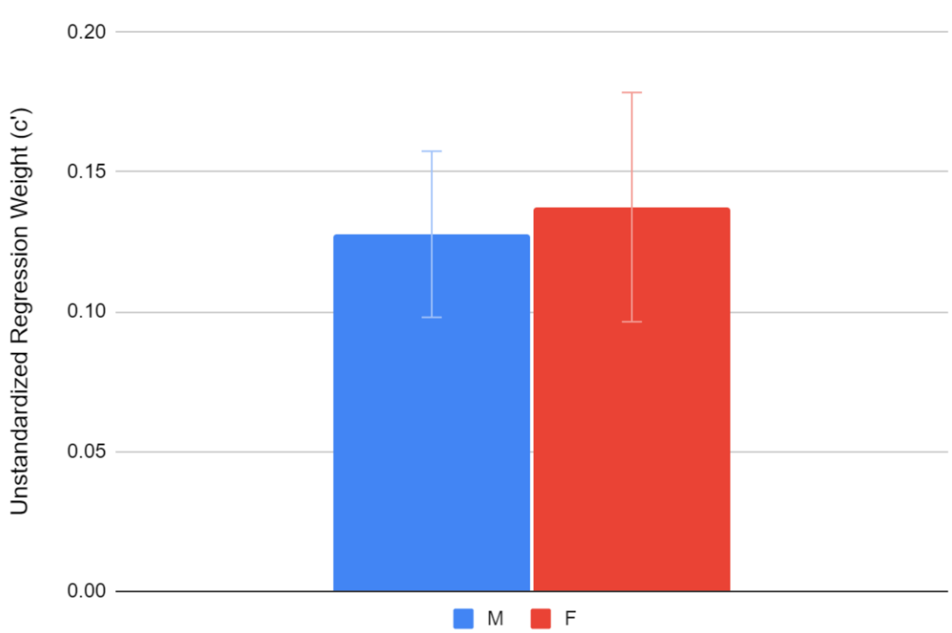
Summary of Model 3 PROCESS Output

		Outcome								
		M (Social Stress)					Y (Global Cognition)			
Predictor (X)		*β*	SE	*p*	95% CI		*β*	SE	*p*	95% CI
(Education)	*a*	.007	.018	.680	[-.028, .042]	*c'*	.137	.021	< .001	[.096, .179]
M (Social Stress)		—	—	—	—	*b*	.065	.062	.292	[-.056, .187]
Constant	i_M	.177	.623	.777	[-1.049, 1.402]	i_Y	3.550	.736	< .001	[2.102, 4.999]
		$R^2 = .051$					$R^2 = 0.262$			
		$F(5, 267) = 2.845$, $p < .05$					$F(6, 266) = 19.324$, $p < .001$			

Note. *β* = unstandardized regression coefficient; SE = standard error; CI = confidence interval

Figure 2

C' Estimates with 95% Confidence Intervals for Male and Female Respondents



DISCUSSION

General Comments

The primary aim of the current study was to investigate the relationships between socioeconomic status (SES, indicated by level of education), social stress, and global cognition in a sample of older adults. Results suggest that, while higher levels of education predict higher global cognition, this SES variable is not related to reported experiences of social stress. Similarly, social stress is not associated with global cognition and does not act as a mediator in the observed SES-cognition relationship.

Important to note is the fact that a single proxy variable, education, was used to indicate SES. Although research suggests that multidimensional SES variables are more sensitive to health disparities (Braveman et al., 2015), other SES indicators were not included in this study due to their limited relevance to older adults. Besides education, income and occupational class are the most frequently used SES proxies. Because a majority of individuals aged 65 and over are retired and report no income from wages, use of these variables would have produced severe underestimates of overall SES. Thus, although an SES composite was intended to be constructed from income and education, it was necessary to exclude income from the analyses.

Cognition

As expected, those with greater education showed higher global cognition, as indicated by higher scores on the MIDUS BTACT (Tables 4 & 5). This result is in line with recent findings on the effects of education on baseline cognition (Livingston et al., 2020). These studies support the theory of cognitive reserve (Stern, 2002) which suggests that educational attainment may supply a set of factors that reduces the age-related changes in the brain and increase brain plasticity (Medaglia et al., 2017). The education-cognition path was found to be significant for both male and female respondents even after controlling for age, race, chronic conditions, and depressed affect (Tables 6 & 7). Visual inspection of the CIs for the corresponding unstandardized regression coefficients (Figure 2) suggests that there is no significant difference in the effect of education on global cognition for male compared to female respondents. This finding is somewhat surprising, given that male respondents tended to report significantly higher educational attainment than females (Table 4) and that Alzheimer's disease and related dementias appear to disproportionately affect females (Podcasy & Epperson, 2016). It appears that this null finding was driven by opposing correlations between sex and the components of the global cognition composite. Consistent with past work (e.g. Herlitz & Rehnman, 2008), female respondents performed better on the EM tasks (Table 4). However, male respondents outperformed females on the EF tasks (Table 4); this result contradicts findings reported elsewhere (e.g. Parsons et al., 2007; Jockwitz et al., 2021), although the literature is mixed (for a review, see Grissom & Reyes, 2019).

Social Stress

While the characteristic features of lower SES, including lower income, education, or occupational status are widely thought to be associated with increased stressor exposure (e.g. Turner et al., 1995), this study found no such relationship between education and overall social stress (Table 4). It is likely that, although overall exposure to stressors remains consistent across the lifespan (Ryff, 1989), the nature and quality of stressors to which people are exposed changes with age. In this way, the measures of social stress used in this study may not have quantified the most salient distressing experiences for older adults. Indeed, Pearlin and Skaff (1996) have found that there are at least two kinds of eventful stressors whose incidence is more common with advancing age: declining functional ability (causing an increase in "daily hassles") and death of loved ones. Thus, the frequent finding that SES is inversely associated with exposure to stressful events in younger samples may reflect the fact that questionnaires used in these studies oversample events most likely to occur in earlier in adulthood (e.g. marriage, divorce, having children, changing or losing a job, etc.).

Greater education was, however, predictive of higher scores on the perceived discrimination component of social stress (Table 4). Although most research on SES-stress relationships focuses on racial discrimination, authors have generally found a similar positive association between education and perceived discrimination (e.g. Kessler et al., 1999). This might reflect a greater tendency for well-educated people to attribute failures to discrimination rather than personal inadequacies, or it may be that higher education leads to greater interaction with individuals belonging to social groups other than one's own and this, in turn, results in increased discrimination exposure. Support for this latter rationale comes from previous research demonstrating that well-educated African Americans are more likely than their less-educated counterparts to experience discrimination (Sigelman & Welch, 1991). Although virtually all studies report positive associations between perceived discrimination and depressive symptomatology (e.g. Dailey et al., 2010), no such relationship was found in this study (Table 4), possibly due to the racial homogeneity of the present sample (Table 1).

Results of this study also suggest that there are no sex differences in overall exposure to social stress (Table 2). As predicted, however, female respondents reported significantly higher levels of family stress and spouse/partner stress (Table 2). Other research has found similar results, with females reporting greater exposure to certain stressors (housing problems, loss of confidant, crises, problems getting along with individuals in their proximal network, and illness of individuals in their distal network), and males reporting higher rates of other stressors (job loss, legal problems, robbery, and work problems; Kendler et al., 2001). There is considerable evidence suggesting that socialization through gender roles and traits and their effect on appraisals of stressors (as threats or challenges) mediates this relationship (for a review, see Mayor, 2015). As females have been shown to experience more distress in the face of a stressor (Lazarus & Folkman, 1984), this may explain this study's finding that female respondents reported higher levels of depressed affect (Table 4).

In contrast with the bulk of the literature on allostatic load and cognitive function, the present study found no relationship between social stress and global cognition (Table 5). This is perhaps due to methodological differences between the present study and other work with self-reported experiences of stress. Stress is most often quantified as the total number of significant life events (SLEs) experienced over a given period of time (usually the previous year), as with the Social Readjustment Rating Scale (SRRS, Appendix D; Holmes & Rahe, 1967). In contrast, items on the family stress and spouse/partner stress components of the social stress composite analyzed in the present study asked respondents to indicate "how much" or "how often" a given experience occurred using general categories (e.g. "A lot," "Often," "Sometimes," etc., Appendix A). These experiences, such as family members making "too many demands" of the respondent, likely do not activate the same cognitive or physiological stress responses as SLEs and might therefore be better characterized not as significant stressors but rather "daily hassles." As such, the scales used in this study reflect the extent to which respondents were chronically affected by given hassles and not their total exposure to significant stressors over the past 12 months. Other authors have found that higher SLE totals coincide with reduced working memory performance among healthy elderly participants (Tschanz et al., 2012; Marshall et al., 2015b). Such a relationship has not been found among young individuals, highlighting the possibility of a cumulative impact of stress that emerges late in life and causes cognitive impairments among the elderly. Thus, although chronic stress has been shown to have more deleterious effects on physical and mental health than acute stress (Epel et al., 2018), it appears as though the total number of SLEs encountered over one's lifetime is more predictive of late life cognitive function than the daily hassles quantified in the present study.

Limitations

Although this study was able to take advantage of the multidimensional nature of MIDUS data, it was limited in some respects. Due to the cross-sectional nature of this work, conclusions about causality cannot be drawn. However, because education is generally completed earlier in one's lifetime and remains stable over time, reverse causation is unlikely. Had a positive relationship been observed between social stress and cognition, it is possible that lower cognitive function temporally preceded and/or caused increased strain on respondents' relationships. It is also important to acknowledge the possible cohort effects that can exist in cross-sectional studies. Group-related differences in the present study may reflect generational differences relevant to SES, namely, access to and quality of education.

As with all self-report data, the influence of reporting bias cannot be ignored. Respondents may have reported social stress when none actually occurred or overrepresented their number of experiences with a given stressful situation. Furthermore, even though this study examined experiences across family, romantic relationships, and society, these three domains do not represent the full range of places and circumstances where social stress can be experienced. As mentioned above, such domains may not adequately capture those stressful experiences which older adults are more likely to encounter and be distressed by.

Additionally, due to the racial homogeneity of the present sample (Table 1), results of this study may not be generalizable to marginalized racial/ethnic populations. Intersectionality literature suggests that at a minimum, age, race, gender, and SES jointly determine the experience of an individual and further determine how they are perceived and treated by others (Harari & Lee, 2021). It is therefore likely that relationships between SES, social stress, and cognition differ in magnitude and mechanism among non-White individuals. For example, Turner and colleagues (2017) showed that while higher education was associated with fewer chronic disorders and lower allostatic load among Whites, there was no association between education and these health outcomes among Blacks. Thus, higher SES may have different benefits for different groups.

In terms of cognition, the BTACT's brevity and lack of content depth may have contributed to this study's null findings. For example, the BTACT Word List Recall test may be a better measure of verbal attention span than the memory processes it purportedly reflects. This test involves administration of only one learning trial, compared to other gold standard memory tests that assess learning over multiple exposures. As the present sample consisted entirely of non-demented older adults, differences in cognitive function between individuals are likely to be much less apparent from examination of such gross scores as total recall. Instead, detection of these subtleties requires a much more sensitive assessment capable of providing information on response consistency, semantic clustering, primacy and recency effects, and other response characteristics related to the subtle pre-clinical changes associated with dementia. Similarly, cognitive testing by phone is limited to auditory stimuli and tasks, whereas traditional (in-person), comprehensive neuropsychological assessment can also assess cognitive skills (including spatial skills) using visual or tactile modalities. Finally, in telephone assessment, distractions to the participants, variations in the quality of the phone connection, and other technical problems must be considered especially for the timed tasks, as they can be a source of measurement error. On the whole, then, the BTACT may not be sensitive enough to detect the subtle differences in late-life cognition that are of interest in non-demented populations.

Future Directions

Limitations notwithstanding, this study adds to the literature by calling attention to the need for more relevant SES indicators for older adults. Additionally, there is a need for researchers to adopt a life-course perspective of stress and acknowledge that experiences of social stress may differ in quality in an age-dependent manner. To meet these needs, future research should examine alternative SES indicators (e.g. community-level SES, retrospective reports of income, etc.) and their utility in predicting cognitive function and decline among older adults. Furthermore, more research is required to understand the effects of stressor severity (i.e. daily hassles vs. SLEs) on cognition and the mechanisms by which they operate. Better characterizing these relationships will facilitate the identification of individuals that are at greater risk for cognitive decline and allow for the development of early intervention strategies.

CONCLUSION

Overall, this study provides further evidence for a positive association between SES, indexed by education, and global cognition in older adults. Although no sex differences were found with respect to this relationship or self-reported levels of overall social stress, as predicted, female respondents indicated significantly higher levels of spouse/partner and family stress. No evidence was found to support the original claim that social stress mediates the education-cognition gradient, though this may be a function of the BTACT's brevity and relative inability to detect subtle differences in cognitive function. These results highlight the importance of sensitive neuropsychological measures and call for a more nuanced treatment of SES and social stress variables, especially in older adults and marginalized racial/ethnic populations.

REFERENCES

2021 Alzheimer's disease facts and figures. (2021). *Alzheimer's & dementia: the journal of the Alzheimer's Association*, 17(3), 327-406. https://doi.org/10.1002/alz.12328

Adler, N. E., & Snibbe, A. C. (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. Current Directions in Psychological Science: A Journal of the American Psychological Society, 12(4), 119-123. https://doi.org/10.1111/1467-8721.01245

Adler, N. E., & Stewart, J. (2010). Health disparities across the lifespan: meaning, methods, and mechanisms. *Annals of the New York Academy of Sciences*, 1186, 5-23. https://doi.org/10.1111/j.1749-6632.2009.05337.x

Aggarwal, N. T., Wilson, R. S., Beck, T. L., Rajan, K. B., Mendes de Leon, C. F., Evans, D. A., & Everson-Rose, S. A. (2014). Perceived stress and change in cognitive function among adults 65 years and older. *Psychosomatic medicine*, 76(1), 80-85. https://doi.org/10.1097/PSY.0000000000000016

American Psychological Association (2010). *Stress in America Findings*. APA. https://www.apa.org/news/press/releases/stress/2010/national-report.pdf

American Psychological Association (2020). *Stress in America 2020: A Mental Health Crisis*. APA. https://www.apa.org/news/press/releases/stress/2020/sia-mental-health-crisis.pdf

Assari S. (2020). Social Epidemiology of Perceived Discrimination in the United States: Role of Race, Educational Attainment, and Income. *International journal of epidemiologic research*, 7(3), 136-141. https://doi.org/10.34172/ijer.2020.24

Bakker, A. B., & Demerouti, E. (2007). The Job Demands-Resources model: State of the art. *Journal of Managerial Psychology*, 22(3), 309-328. https://doi.org/10.1108/02683940710733115

Bassuk, S. S., Berkman, L. F., & Amick, B. C., 3rd (2002). Socioeconomic status and mortality among the elderly: findings from four US communities. *American journal of epidemiology*, 155(6), 520-533. https://doi.org/10.1093/aje/155.6.520

Bird, C. M., & Burgess, N. (2008). The hippocampus and memory: insights from spatial processing. *Nature Reviews Neuroscience*, 9(3), 182-194. https://doi.org/10.1038/nrn2335

Blane, D. & Kelly-Irving, Michelle & d'Errico, Angelo & Bartley, Mel & Montgomery, S.. (2013). Social-biological transitions: how does the social become biological?. *Longitud Life Course Studies*. 4(2). 136-146.

Blascovich, J. and Mendes, W.B. (2010). Social Psychophysiology and Embodiment. In *Handbook of Social Psychology* (eds S.T. Fiske, D.T. Gilbert and G. Lindzey). https://doi.org/10.1002/9780470561119.socpsy001006

Bloome, D. (2014). Racial Inequality Trends and the Intergenerational Persistence of Income and Family Structure. *American sociological review*, 79(6), 1196-1225. https://doi.org/10.1177/0003122414554947

Bolger, N., & Zuckerman, A. (1995). A framework for studying personality in the stress process. *Journal of personality and social psychology*, 69(5), 890-902. https://doi.org/10.1037//0022-3514.69.5.890

Bosma, H., van Boxtel, M. P., Ponds, R. W., Jelicic, M., Houx, P., Metsemakers, J., & Jolles, J. (2002). Engaged lifestyle and cognitive function in middle and old-aged, non-demented persons: a reciprocal association?. *Zeitschrift fur Gerontologie und Geriatrie*, 35(6), 575-581. https://doi.org/10.1007/s00391-002-0080-y

Braveman, P. A., Cubbin, C., Egerter, S., Chideya, S., Marchi, K. S., Metzler, M., & Posner, S. (2005). Socioeconomic status in health research: one size does not fit all. *JAMA*, *294*(22), 2879-2888. https://doi.org/10.1001/jama.294.22.2879

Bureau of Labor Statistics. (2021, September 8). *Civilian labor force, by age, sex, race, and ethnicity* [Data Set]. Employment Projections. U.S. Department of Labor. https://www.bls.gov/emp/tables/civilian-labor-force-summary.htm

Cadar, D., Lassale, C., Davies, H., Llewellyn, D.J., Batty, G.D., & Steptoe, A. (2018). Individual and Area-Based Socioeconomic Factors Associated With Dementia Incidence in England: Evidence From a 12-Year Follow-up in the English Longitudinal Study of Ageing. *JAMA Psychiatry*, 75(7):723. https://doi.org/10.1001/jamapsychiatry.2018.1012

Cagney, K. A., & Lauderdale, D. S. (2002). Education, wealth, and cognitive function in later life. *The journals of gerontology. Series B, Psychological sciences and social sciences*, *57*(2), P163-P172. https://doi.org/10.1093/geronb/57.2.p163

Clow, A., Thorn, L., Evans, P., & Hucklebridge, F. (2004). The awakening cortisol response: methodological issues and significance. *Stress* (Amsterdam, Netherlands), 7(1), 29-37. https://doi.org/10.1080/10253890410001667205

Cohen, S., Doyle, W. J., & Baum, A. (2006). Socioeconomic status is associated with stress hormones. *Psychosomatic medicine*, 68(3), 414-420. https://doi.org/10.1097/01.psy.0000221236.37158.b9

Cohen, S. and Janicki-Deverts, D. (2012). Who's Stressed? Distributions of Psychological Stress in the United States in Probability Samples from 1983, 2006, and 2009. *Journal of Applied Social Psychology*, 42: 1320-1334. https://doi.org/10.1111/j.1559-1816.2012.00900.x

Crosswell, A. D., & Lockwood, K. G. (2020). Best practices for stress measurement: How to measure psychological stress in health research. *Health psychology open*, 7(2), 2055102920933072. https://doi.org/10.1177/2055102920933072

Dailey, A. B., Kasl, S. V., Holford, T. R., Lewis, T. T., & Jones, B. A. (2010). Neighborhood- and individual-level socioeconomic variation in perceptions of racial discrimination. *Ethnicity & health*, 15(2), 145-163. https://doi.org/10.1080/13557851003592561

Deaux, K. (1985). Sex and gender. *Annual Review of Psychology*, 36, 49-81. https://doi.org/10.1146/annurev.ps.36.020185.000405

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychological bulletin*, 130(3), 355-391. https://doi.org/10.1037/0033-2909.130.3.355

Dickinson, W. J., Potter, G. G., Hybels, C. F., McQuoid, D. R., & Steffens, D. C. (2011). Change in stress and social support as predictors of cognitive decline in older adults with and without depression. *International journal of geriatric psychiatry*, 26(12), 1267-1274. https://doi.org/10.1002/gps.2676

Doyle, D. M., & Molix, L. (2014). Perceived discrimination as a stressor for close relationships: identifying psychological and physiological pathways. *Journal of behavioral medicine*, 37(6), 1134-1144. https://doi.org/10.1007/s10865-014-9563-8

Ennis, G. E., An, Y., Resnick, S. M., Ferrucci, L., O'Brien, R. J., & Moffat, S. D. (2017). Long-term cortisol measures predict Alzheimer disease risk. *Neurology*, 88(4), 371-378. https://doi.org/10.1212/WNL.0000000000003537

Epel, E. S., Crosswell, A. D., Mayer, S. E., Prather, A. A., Slavich, G. M., Puterman, E., & Mendes, W. B. (2018). More than a feeling: A unified view of stress measurement for population science. *Frontiers in neuroendocrinology*, 49, 146-169. https://doi.org/10.1016/j.yfrne.2018.03.001

Ertel, K. A., Glymour, M. M., & Berkman, L. F. (2008). Effects of social integration on preserving memory function in a nationally representative US elderly population. *American journal of public health*, 98(7), 1215-1220. https://doi.org/10.2105/AJPH.2007.113654

Evans, D. A., Hebert, L. E., Beckett, L. A., Scherr, P. A., Albert, M. S., Chown, M. J., Pilgrim, D. M., & Taylor, J. O. (1997). Education and other measures of socioeconomic status and risk of incident alzheimer disease in a defined population of older persons. *Archives of Neurology (Chicago)*, 54(11), 1399-1405. https://doi.org/10.1001/archneur.1997.00550230066019

Evans, G. W., & Kantrowitz, E. (2002). Socioeconomic status and health: the potential role of environmental risk exposure. *Annual review of public health*, 23, 303-331. https://doi.org/10.1146/annurev.publhealth.23.112001.112349

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): facts and future directions. *International journal of psychophysiology :*

official journal of the International Organization of Psychophysiology, 72(1), 67-73. https://doi.org/10.1016/j.ijpsycho.2008.03.014

Gallo, L. C., Bogart, L. M., Vranceanu, A. M., & Matthews, K. A. (2005). Socioeconomic status, resources, psychological experiences, and emotional responses: a test of the reserve capacity model. *Journal of personality and social psychology*, 88(2), 386-399. https://doi.org/10.1037/0022-3514.88.2.386

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: do negative emotions play a role? *Psychological bulletin,* 129(1), 10-51. https://doi.org/10.1037/0033-2909.129.1.10

Grissom, N. M., & Reyes, T. M. (2019). Let's call the whole thing off: evaluating gender and sex differences in executive function. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, *44*(1), 86–96. https://doi.org/10.1038/s41386-018-0179-5

Gross J. J. (2002). Emotion regulation: affective, cognitive, and social consequences. *Psychophysiology*, 39(3), 281-291. https://doi.org/10.1017/s0048577201393198

Habib, K. E., Gold, P. W., & Chrousos, G. P. (2001). Neuroendocrinology of stress. *Endocrinology and metabolism clinics of North America*, 30(3), 695-viii. https://doi.org/10.1016/s0889-8529(05)70208-5

Harari, L., & Lee, C. (2021). Intersectionality in quantitative health disparities research: A systematic review of challenges and limitations in empirical studies. *Social science & medicine* (1982), 277, 113876. https://doi.org/10.1016/j.socscimed.2021.113876

Harris, A., Ursin, H., Murison, R., & Eriksen, H. R. (2007). Coffee, stress and cortisol in nursing staff. *Psychoneuroendocrinology*, 32(4), 322-330. https://doi.org/10.1016/j.psyneuen.2007.01.003

Hatch, S. L., & Dohrenwend, B. P. (2007). Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. *American journal of community psychology*, 40(3-4), 313-332. https://doi.org/10.1007/s10464-007-9134-z

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

Herlitz, A., & Rehnman, J. (2008). Sex Differences in Episodic Memory. *Current Directions in Psychological Science*, 17(1), 52-56. https://doi.org/10.1111/j.1467-8721.2008.00547.x

Holmes, T. H., & Rahe, R. H. (1967). The Social Readjustment Rating Scale. *Journal of Psychosomatic Research*, 11(2), 213-218. https://doi.org/10.1016/0022-3999(67)90010-4

Hultsch, D. F., Hertzog, C., Small, B. J., & Dixon, R. A. (1999). Use it or lose it: engaged lifestyle as a buffer of cognitive decline in aging?. *Psychology and aging*, 14(2), 245-263. https://doi.org/10.1037//0882-7974.14.2.245

Jacobson, L., & Sapolsky, R. (1991). The role of the hippocampus in feedback regulation of the hypothalamic-pituitary-adrenocortical axis. *Endocrine reviews*, 12(2), 118-134. https://doi.org/10.1210/edrv-12-2-118

Jockwitz, C., Wiersch, L., Stumme, J., & Caspers, S. (2021). Cognitive profiles in older males and females. *Scientific reports*, 11(1), 6524. https://doi.org/10.1038/s41598-021-84134-8

Juth, V., Dickerson, S. (2013). Social Stress. In: Gellman, M.D., Turner, J.R. (eds) *Encyclopedia of Behavioral Medicine*. Springer, New York, NY. https://doi.org/10.1007/978-1-4419-1005-9_283

Kendler, K. S., Thornton, L. M., & Prescott, C. A. (2001). Gender differences in the rates of exposure to stressful life events and sensitivity to their depressogenic effects. *The American journal of psychiatry*, 158(4), 587-593. https://doi.org/10.1176/appi.ajp.158.4.587

Kessler, R. C., Mickelson, K. D., & Williams, D. R. (1999). The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. *Journal of health and social behavior*, 40(3), 208-230. https://doi.org/10.2307/2676349

Koster, A., Penninx, B. W., Bosma, H., Kempen, G. I., Newman, A. B., Rubin, S. M., Satterfield, S., Atkinson, H. H., Ayonayon, H. N., Rosano, C., Yaffe, K., Harris, T. B., Rooks, R. N., Van Eijk, J. T., & Kritchevsky, S. B. (2005). Socioeconomic differences in cognitive decline and the role of biomedical factors. *Annals of epidemiology*, 15(8), 564-571. https://doi.org/10.1016/j.annepidem.2005.02.008

Krieger, N., Williams, D. R., & Moss, N. E. (1997). Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annual review of public health*, *18*, 341-378. https://doi.org/10.1146/annurev.publhealth.18.1.341

Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M., & Steptoe, A. (2004). Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology*, 29(4), 516-528. https://doi.org/10.1016/s0306-4530(03)00072-6

Künzi, M., Joly-Burra, E., Zuber, S., Haas, M., Tinello, D., Da Silva Coelho, C., Hering, A., Ihle, A., Laera, G., Mikneviciute, G., Stringhini, S., ganski, B., Kliegel, M., & Ballhausen, N. (2021). The relationship between life course socioeconomic conditions and objective and subjective memory in older age. *Brain Sciences*, 11(1), 61. https://doi.org/10.3390/brainsci11010061

Langa, K. M., Larson, E. B., Karlawish, J. H., Cutler, D. M., Kabeto, M. U., Kim, S. Y., & Rosen, A. B. (2008). Trends in the prevalence and mortality of cognitive impairment in the United States: is there evidence of a compression of cognitive morbidity?. *Alzheimer's & dementia : the journal of the Alzheimer's Association*, *4*(2), 134-144. https://doi.org/10.1016/j.jalz.2008.01.001

Lantz, P. M., House, J. S., Lepkowski, J. M., Williams, D. R., Mero, R. P., & Chen, J. (1998). Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. *JAMA*, 279(21), 1703-1708. https://doi.org/10.1001/jama.279.21.1703

Lantz, P. M., House, J. S., Mero, R. P., & Williams, D. R. (2005). Stress, life events, and socioeconomic disparities in health: results from the Americans' Changing Lives Study. *Journal of health and social behavior*, 46(3), 274-288. https://doi.org/10.1177/002214650504600305

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Springer.

Lee, S., Kawachi, I., Berkman, L. F., & Grodstein, F. (2003). Education, other socioeconomic indicators, and cognitive function. *American journal of epidemiology*, 157(8), 712-720. https://doi.org/10.1093/aje/kwg042

Lewis, T. T., Cogburn, C. D., & Williams, D. R. (2015). Self-reported experiences of discrimination and health: scientific advances, ongoing controversies, and emerging issues. *Annual review of clinical psychology*, 11, 407-440. https://doi.org/10.1146/annurev-clinpsy-032814-112728

Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., Brayne, C., Burns, A., Cohen-Mansfield, J., Cooper, C., Costafreda, S. G., Dias, A., Fox, N., Gitlin, L. N., Howard, R., Kales, H. C., Kivimäki, M., Larson, E. B., Ogunniyi, A., Orgeta, V., … Mukadam, N. (2020). Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *Lancet (London, England)*, 396(10248), 413-446. https://doi.org/10.1016/S0140-6736(20)30367-6

Luo, Y., & Waite, L. J. (2005). The impact of childhood and adult SES on physical, mental, and cognitive well-being in later life. *The journals of gerontology. Series B, Psychological sciences and social sciences*, *60*(2), S93-S101. https://doi.org/10.1093/geronb/60.2.s93

Lupien, S. J., Juster, R. P., Raymond, C., & Marin, M. F. (2018). The effects of chronic stress on the human brain: From neurotoxicity, to vulnerability, to opportunity. *Frontiers in neuroendocrinology*, 49, 91-105. https://doi.org/10.1016/j.yfrne.2018.02.001

Lupien, S. J., Nair, N. P., Brière, S., Maheu, F., Tu, M. T., Lemay, M., McEwen, B. S., & Meaney, M. J. (1999). Increased cortisol levels and impaired cognition in human aging: implication for depression and dementia in later life. *Reviews in the neurosciences*, 10(2), 117-139. https://doi.org/10.1515/revneuro.1999.10.2.117

Lyu, J., & Burr, J. A. (2016). Socioeconomic status across the life course and cognitive function among older adults: An examination of the latency, pathways, and accumulation hypotheses. *Journal of Aging and Health*, 28(1), 40-67. https://doi.org/10.1177/0898264315585504

Manly, J. J., Jacobs, D. M., Touradji, P., Small, S. A., & Stern, Y. (2002). Reading level attenuates differences in neuropsychological test performance between African American and White elders. *Journal of the International Neuropsychological Society : JINS*, *8*(3), 341-348. https://doi.org/10.1017/s1355617702813157

Marden, J. R., Tchetgen Tchetgen, E. J., Kawachi, I., & Glymour, M. M. (2017). Contribution of Socioeconomic Status at 3 Life-Course Periods to Late-Life Memory Function and Decline: Early and Late Predictors of Dementia Risk. American journal of epidemiology, 186(7), 805-814. https://doi.org/10.1093/aje/kwx155

Marmot, M. G., & Shipley, M. J. (1996). Do socioeconomic differences in mortality persist after retirement? 25 year follow up of civil servants from the first Whitehall study. *BMJ* (Clinical research ed.), 313(7066), 1177-1180. https://doi.org/10.1136/bmj.313.7066.1177

Marmot, M. G., Shipley, M. J., & Rose, G. (1984). Inequalities in death--specific explanations of a general pattern?. *Lancet* (London, England), 1(8384), 1003-1006. https://doi.org/10.1016/s0140-6736(84)92337-7

Marshall, A. C., Cooper, N. R., & Geeraert, N. (2015). Experienced stress produces inhibitory deficits in old adults' Flanker task performance: First evidence for lifetime stress effects beyond memory. *Biological psychology*, 113, 1-11. https://doi.org/10.1016/j.biopsycho.2015.10.008

Marshall, A. C., Cooper, N. R., Segrave, R., & Geeraert, N. (2015). The effects of long-term stress exposure on aging cognition: A behavioral and EEG investigation. *Neurobiology of Aging*, 36(6), 2136-2144. https://doi.org/10.1016/j.neurobiolaging.2015.02.026

Maslach, C., Schaufeli, W. B., & Leiter, M. P. (2001). Job burnout. *Annual review of psychology*, 52, 397-422. https://doi.org/10.1146/annurev.psych.52.1.397

Mason J. W. (1968). A review of psychoendocrine research on the pituitary-adrenal cortical system. *Psychosomatic medicine*, 30(5), 576-607.

Matthews, K. A., & Gallo, L. C. (2011). Psychological perspectives on pathways linking socioeconomic status and physical health. *Annual review of psychology*, 62, 501-530. https://doi.org/10.1146/annurev.psych.031809.130711

Matud, M. P. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences*, 37(7), 1401-1415. https://doi.org/10.1016/j.paid.2004.01.010

Mayor, E. (2015). Gender roles and traits in stress and health. *Frontiers in psychology*, 6, 779. https://doi.org/10.3389/fpsyg.2015.00779

McDonough, P., & Walters, V. (2001). Gender and health: Reassessing patterns and explanations. *Social Science & Medicine*, 52(4), 547-559. https://doi.org/10.1016/S0277-9536(00)00159-3

McEwen, B. S., Nasca, C., & Gray, J. D. (2016). Stress Effects on Neuronal Structure: Hippocampus, Amygdala, and Prefrontal Cortex. *Neuropsychopharmacology*, 41(1), 3-23. https://doi.org/10.1038/npp.2015.171

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 896, 30-47. https://doi.org/10.1111/j.1749-6632.1999.tb08103.x

McLeod, J. D., & Kessler, R. C. (1990). Socioeconomic status differences in vulnerability to undesirable life events. *Journal of Health and Social Behavior*, 31(2), 162-172. https://doi.org/10.2307/2137170

Medaglia, J. D., Pasqualetti, F., Hamilton, R. H., Thompson-Schill, S. L., & Bassett, D. S. (2017). Brain and cognitive reserve: Translation via network control theory. *Neuroscience and biobehavioral reviews*, 75, 53-64. https://doi.org/10.1016/j.neubiorev.2017.01.016

Mullins, M. A., Bynum, J., Judd, S. E., & Clarke, P. J. (2021). Access to primary care and cognitive impairment: results from a national community study of aging Americans. *BMC geriatrics*, 21(1), 580. https://doi.org/10.1186/s12877-021-02545-8

National Center for Health Statistics. (2021). *Health, United States, 2019*. U.S. Centers for Disease Control and Prevention.

National Center for Education Statistics. (2019). *Status and Trends in the Education of Racial and Ethnic Groups 2018*. U.S. Department of Education.

Okoro, C. A., Strine, T. W., Young, S. L., Balluz, L. S., & Mokdad, A. H. (2005). Access to health care among older adults and receipt of preventive services. Results from the Behavioral Risk Factor Surveillance System, 2002. *Preventive medicine*, 40(3), 337-343. https://doi.org/10.1016/j.ypmed.2004.06.009

Otte, Christian & Hart, Tae & Neylan, TC & Marmar, Charles & Yaffe, K & Mohr, David. (2004). A meta-analysis of cortisol response to challenge in human aging: importance of gender. *Experimental and Clinical Endocrinology & Diabetes*, 112. https://doi.org/10.1055/s-2004-832897

Ouanes, S., & Popp, J. (2019). High Cortisol and the Risk of Dementia and Alzheimer's Disease: A Review of the Literature. *Frontiers in aging neuroscience*, 11, 43. https://doi.org/10.3389/fnagi.2019.00043

Parsons, T. D., Rizzo, A. R., van der Zaag, C., McGee, J. S., & Buckwalter, J. G. (2005). Gender Differences and Cognition Among Older Adults. *Aging, Neuropsychology, and Cognition*, 12(1), 78-88. https://doi.org/10.1080/13825580590925125

Pearlin, L. I., & Skaff, M. M. (1996). Stress and the life course: A paradigmatic alliance. *The Gerontologist*, 36(2), 239-247. https://doi.org/10.1093/geront/36.2.239

Podcasy, J. L., & Epperson, C. N. (2016). Considering sex and gender in Alzheimer disease and other dementias. *Dialogues in clinical neuroscience*, 18(4), 437-446. https://doi.org/10.31887/DCNS.2016.18.4/cepperson

Pruessner, J. C., Wolf, O. T., Hellhammer, D. H., Buske-Kirschbaum, A., von Auer, K., Jobst, S., Kaspers, F., & Kirschbaum, C. (1997). Free cortisol levels after awakening: a reliable biological marker for the assessment of adrenocortical activity. *Life sciences*, 61(26), 2539-2549. https://doi.org/10.1016/s0024-3205(97)01008-4

Rahal, D., Chiang, J. J., Bower, J. E., Irwin, M. R., Venkatraman, J., & Fuligni, A. J. (2020). Subjective social status and stress responsivity in late adolescence. *Stress* (Amsterdam, Netherlands), 23(1), 50-59. https://doi.org/10.1080/10253890.2019.1626369

Rajan, K. B., Weuve, J., Barnes, L. L., McAninch, E. A., Wilson, R. S., & Evans, D. A. (2021). Population estimate of people with clinical Alzheimer's disease and mild cognitive impairment in the United States (2020-2060). *Alzheimer's & dementia : the journal of the Alzheimer's Association*, 17(12), 1966-1975. https://doi.org/10.1002/alz.12362

Roberto, K. A., McCann, B. R., & Brossoie, N. (2013). Intimate partner violence in late life: an analysis of national news reports. *Journal of elder abuse & neglect*, 25(3), 230-241. https://doi.org/10.1080/08946566.2012.751825

Rosay, A. B., PhD, & Mulford, C. F., PhD (2017). Prevalence estimates and correlates of elder abuse in the United States: The National Intimate Partner and Sexual Violence Survey. *Journal of elder abuse & neglect*, 29(1), 1-14. https://doi.org/10.1080/08946566.2016.1249817

Rothman, S. M., & Mattson, M. P. (2010). Adverse stress, hippocampal networks, and Alzheimer's disease. *Neuromolecular medicine*, 12(1), 56-70. https://doi.org/10.1007/s12017-009-8107-9

Sapolsky, R. M. (2004). *Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping*. Holt paperbacks.

Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1986). The neuroendocrinology of stress and aging: the glucocorticoid cascade hypothesis. *Endocrine reviews*, 7(3), 284-301. https://doi.org/10.1210/edrv-7-3-284

Schaaf, M. J., De Kloet, E. R., & Vreugdenhil, E. (2000). Corticosterone effects on BDNF expression in the hippocampus. Implications for memory formation. *Stress* (Amsterdam, Netherlands), 3(3), 201-208. https://doi.org/10.3109/10253890009001124

Schlotz, W., Hellhammer, J., Schulz, P., & Stone, A. A. (2004). Perceived work overload and chronic worrying predict weekend-weekday differences in the cortisol awakening response. *Psychosomatic medicine*, 66(2), 207-214. https://doi.org/10.1097/01.psy.0000116715.78238.56

Schmand, B., Lindeboom, J., Hooijer, C., & Jonker, C. (1995). Relation between education and dementia: the role of test bias revisited. *Journal of neurology, neurosurgery, and psychiatry*, 59(2), 170-174. https://doi.org/10.1136/jnnp.59.2.170

Seabury, S. A., Terp, S., & Boden, L. I. (2017). Racial And Ethnic Differences In The Frequency Of Workplace Injuries And Prevalence Of Work-Related Disability. *Health affairs* (Project Hope), 36(2), 266-273. https://doi.org/10.1377/hlthaff.2016.1185

Selye, H. (1976). *Stress in health and disease.* Butterworth-Heinemann.

Sewdas, R., de Wind, A., van der Zwaan, L., van der Borg, W. E., Steenbeek, R., van der Beek, A. J., & Boot, C. (2017). Why older workers work beyond the retirement age: a qualitative study. *BMC public health*, 17(1), 672. https://doi.org/10.1186/s12889-017-4675-z

Singh-Manoux, A., Richards, M., & Marmot, M. (2005). Socioeconomic position across the lifecourse: How does it relate to cognitive function in mid-life? *Annals of Epidemiology,* 15(8), 572-578. https://doi.org/10.1016/j.annepidem.2004.10.007

Smith, T. W., Birmingham, W., & Uchino, B. N. (2012). Evaluative threat and ambulatory blood pressure: cardiovascular effects of social stress in daily experience. *Health psychology : official journal of the Division of Health Psychology, American Psychological Association*, 31(6), 763-766. https://doi.org/10.1037/a0026947

Stern, Y. (2009). Cognitive reserve. *Neuropsychologia*, *47*(10), 2015-2028. https://doi.org/10.1016/j.neuropsychologia.2009.03.004

Steptoe, A., Cropley, M., Griffith, J., & Kirschbaum, C. (2000). Job strain and anger expression predict early morning elevations in salivary cortisol. *Psychosomatic Medicine*, 62(2), 286-292. https://doi.org/10.1097/00006842-200003000-00022

Steptoe, A., Kunz-Ebrecht, S., Owen, N., Feldman, P. J., Willemsen, G., Kirschbaum, C., & Marmot, M. (2003). Socioeconomic status and stress-related biological responses over the working day. *Psychosomatic medicine*, 65(3), 461-470. https://doi.org/10.1097/01.psy.0000035717.78650.a1

Stranahan, A. M., & Mattson, M. P. (2008). Impact of energy intake and expenditure on neuronal plasticity. *Neuromolecular medicine*, 10(4), 209-218. https://doi.org/10.1007/s12017-008-8043-0

Straub, R.O. (2017). *Health Psychology*. Worth.

Stringhini, S., Carmeli, C., Jokela, M., Avendaño, M., McCrory, C., d'Errico, A., Bochud, M., Barros, H., Costa, G., Chadeau-Hyam, M., Delpierre, C., Gandini, M., Fraga, S., Goldberg, M., Giles, G. G., Lassale, C., Kenny, R. A., Kelly-Irving, M., Paccaud, F., Layte, R., … LIFEPATH Consortium (2018). Socioeconomic status, non-communicable disease risk factors, and walking speed in older adults: multi-cohort population based study. *BMJ* (Clinical research ed.), 360, k1046. https://doi.org/10.1136/bmj.k1046

Stringhini, S., Sabia, S., Shipley, M., Brunner, E., Nabi, H., Kivimaki, M., & Singh-Manoux, A. (2010). Association of socioeconomic position with health behaviors and mortality. *JAMA*, 303(12), 1159-1166. https://doi.org/10.1001/jama.2010.297

Stroud, L. R., Salovey, P., & Epel, E. S. (2002). Sex differences in stress responses: social rejection versus achievement stress. *Biological psychiatry*, 52(4), 318-327. https://doi.org/10.1016/s0006-3223(02)01333-1

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: tend-and-befriend, not fight-or-flight. *Psychological review*, 107(3), 411-429. https://doi.org/10.1037/0033-295x.107.3.411

Thoits, P. A. (2010). Stress and health: Major findings and policy implications. *Journal of Health and Social Behavior*, 51, S41-S53. https://doi.org/10.1177/0022146510383499

Thorn, L., Hucklebridge, F., Evans, P., & Clow, A. (2006). Suspected non-adherence and weekend versus week day differences in the awakening cortisol response. *Psychoneuroendocrinology*, 31(8), 1009-1018. https://doi.org/10.1016/j.psyneuen.2006.05.012

Ryff, C. D. (1989). Beyond Ponce de Leon and life satisfaction: New directions in quest of successful ageing. *International Journal of Behavioral Development*, 12(1), 35-55. https://doi.org/10.1177/016502548901200102

Ryff, Carol D., Almeida, David M., Ayanian, John Z., Carr, Deborah S., Cleary, Paul D., Coe, Christopher, … Williams, David R. Midlife in the United States (MIDUS 2), 2004-2006. Inter-university Consortium for Political and Social Research [distributor], 2021-09-15. https://doi.org/10.3886/ICPSR04652.v8

Tschanz, J. T., Pfister, R., Wanzek, J., Corcoran, C., Smith, K., Tschanz, B. T., Steffens, D. C., Østbye, T., Welsh-Bohmer, K. A., & Norton, M. C. (2013). Stressful life events and cognitive decline in late life: moderation by education and age. The Cache County Study. *International journal of geriatric psychiatry*, 28(8), 821-830. https://doi.org/10.1002/gps.3888

Tun, P. A., Miller-Martinez, D., Lachman, M. E., & Seeman, T. (2013). Social strain and executive function across the lifespan: the dark (and light) sides of social engagement. *Aging, neuropsychology and cognition*, 20(3), 320-338. https://doi.org/10.1080/13825585.2012.707173

Turner, R. J., & Avison, W. R. (2003). Status variations in stress exposure: implications for the interpretation of research on race, socioeconomic status, and gender. *Journal of health and social behavior*, 44(4), 488-505. https://doi.org/10.2307/1519795

Turner, R. J., Brown, T. N., & Hale, W. B. (2017). Race, Socioeconomic Position, and Physical Health: A Descriptive Analysis. *Journal of health and social behavior*, 58(1), 23-36. https://doi.org/10.1177/0022146516687008

Turner, R. J., Wheaton, B., & Lloyd, D. A. (1995). The epidemiology of social stress. *American Sociological Review*, 60(1), 104-125. https://doi.org/10.2307/2096348

Tyrrell, J., Melzer, D., Henley, W., Galloway, T. S., & Osborne, N. J. (2013). Associations between socioeconomic status and environmental toxicant concentrations in adults in the USA: NHANES 2001-2010. *Environment international*, 59, 328-335. https://doi.org/10.1016/j.envint.2013.06.017

U.S. Bureau of Labor Statistics. (2021). *Labor Force Statistics from the Current Population Survey.* U.S. Department of Labor. https://www.bls.gov/cps/cpsaat11.htm

van Praag, H. (2009). Exercise and the brain: something to chew on. *Trends in neurosciences*, 32(5), 283-290. https://doi.org/10.1016/j.tins.2008.12.007

Wagner, D.T., Barnes, C.M. and Scott, B.A. (2014). Driving it Home: How Workplace Emotional Labor Harms Employee Home Life. *Personnel Psychology*, 67: 487-516. https://doi.org/10.1111/peps.12044

Walker, K. A., Power, M. C., & Gottesman, R. F. (2017). Defining the Relationship Between Hypertension, Cognitive Decline, and Dementia: A Review. *Current hypertension reports*, 19(3), 24. https://doi.org/10.1007/s11906-017-0724-3

Widmer, E. D., Girardin, M., & Ludwig, C. (2018). Conflict Structures in Family Networks of Older Adults and Their Relationship With Health-Related Quality of Life. *Journal of family issues*, 39(6), 1573-1597. https://doi.org/10.1177/0192513X17714507

Wilhelm, I., Born, J., Kudielka, B. M., Schlotz, W., & Wüst, S. (2007). Is the cortisol awakening rise a response to awakening?. *Psychoneuroendocrinology*, 32(4), 358-366. https://doi.org/10.1016/j.psyneuen.2007.01.008

Williams, D. R., Lawrence, J. A., Davis, B. A., & Vu, C. (2019). Understanding how discrimination can affect health. *Health services research*, 54 Suppl 2(Suppl 2), 1374-1388. https://doi.org/10.1111/1475-6773.13222

Wright, C. E., & Steptoe, A. (2005). Subjective socioeconomic position, gender and cortisol responses to waking in an elderly population. *Psychoneuroendocrinology*, 30(6), 582-590. https://doi.org/10.1016/j.psyneuen.2005.01.007

Wüst, S., Wolf, J., Hellhammer, D. H., Federenko, I., Schommer, N., & Kirschbaum, C. (2000). The cortisol awakening response - normal values and confounds. Noise & health, 2(7), 79-88.

Zahodne, L. B., Glymour, M. M., Sparks, C., Bontempo, D., Dixon, R. A., MacDonald, S. W., & Manly, J. J. (2011). Education does not slow cognitive decline with aging: 12-year evidence from the victoria longitudinal study. *Journal of the International Neuropsychological Society: JINS*, *17*(6), 1039-1046. https://doi.org/10.1017/S1355617711001044

APPENDICES

APPENDIX A: PSYCHOSOCIAL CONSTRUCTS

(Selected from Ryff et al., 2004)

Family Stress

Items: 4 items - Self-Administered Questionnaire, Section J, Question 2 (g – j):

g. "Not including your spouse or partner, how often do members of your family make too

many demands on you?"

h. "How often do they criticize you?"

i. "How often do they let you down when you are counting on them?"

j. "How often do they get on your nerves?"

Coding:

1 = Often

2 = Sometimes

3 = Rarely

4 = Never

Scaling: Scales were constructed by calculating the mean of the values of the items in each scale. Items were reverse-coded so that high scores reflect higher standing in the scale.

Missing Values: Scales were computed for cases having valid values for at least one item on the particular scale. Scores were not calculated for cases with no valid item on the scales, and coded as "8" for "NOT CALCULATED (Due to missing data)."

Spouse/Partner Stress

Items: 6 items -Self-Administered Questionnaire, Section L, Question 11 (g – l):

g. "How often does your spouse or partner make too many demands on you?"

h. "How often does he or she argue with you?"

i. "How often does he or she make you feel tense?"

j. "How often does he or she criticize you?"

k. "How often does he or she let you down when you are counting on him or her?"

l. "How often does he or she get on your nerves?"

Coding:

1 = Often

2 = Sometimes

3 = Rarely

4 = Never

Scaling: Scales were constructed by calculating the mean of the values of the items in each scale. Items were reverse-coded so that high scores reflect higher standing in the scale.

Missing Values: The scales were computed for cases having valid values for at least one item on the particular scale. Scores were not calculated for cases with no valid item on the scales, and coded as "8" for "NOT CALCULATED (Due to missing data)."

Perceived Discrimination

Items: 11 items - Self-Administered Questionnaire, Section P, Question 1 (a - k):

"How many times in your life have you been discriminated against in each of the following ways because of such things as your race, ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics?"

a. "You were discouraged by a teacher or advisor from seeking higher education."

b. "You were denied a scholarship."

c. "You were not hired for a job."

d. "You were not given a promotion."

e. "You were fired."

f. "You were prevented from renting or buying a home in the neighborhood you wanted."

g. "You were prevented from remaining in a neighborhood because neighbors made life so uncomfortable."

h. "You were hassled by the police."

i. "You were denied a bank loan."

j. "You were denied or provided inferior medical care."

k. "You were denied or provided inferior service by a plumber, care mechanic, or other service provider."

Coding: Each item is answered by frequency (# of times) of its happening.

Scaling: The scale was constructed by taking the number of "1 or higher" responses to the items.

Missing Values: The scales were computed for cases having valid values for at least one item on the scale. Scores were not calculated for cases with no valid item on the scales, and coded as "99998" for "NOT CALCULATED (Due to missing data)."

Job Stress

Items: 6 items - Self-Administered Questionnaires, Section F; Question 31 (a – f):

a. "How often do you think you are unfairly given the jobs that no one else wanted to do?"

b. "How often are you watched more closely than other workers?"

c. "How often does your supervisor or boss use ethnic, racial, or sexual slurs or jokes?"

d. "How often do your coworkers use ethnic, racial, or sexual slurs or jokes?"

e. "How often do you feel that you are ignored or not taken seriously by your boss?"

f. "How often has a co-worker with less experience and qualifications gotten promoted before you?"

Coding:

1 = Once a week or more

2 = A few times a month

3 = A few times a year

4 = Less than once a year

5 = Never

Scaling: The scale was constructed by calculating the sum of the values of the items. Items were reverse-coded so that high scores reflect higher standing in the scale. For an item with a missing value, the mean value of completed items was imputed.

Missing Values: The scale was computed for cases that had valid values for at least three items on the scale. The scale score was not calculated for cases with fewer than three valid items on the scales, and coded as “98” for “NOT CALCULATED (Due to missing data).”

APPENDIX B: DESCRIPTIVE STATISTICS FOR UNUSED VARIABLES

	N	Minimum	Maximum	Mean	Std. Deviation
Household total income from wage, pension, social security, and other sources (USD)	976	0	300000	42582.4	43816.5
Job Stress	335	6	24	8.1	3.3
Wake cortisol (nmol/L)	4271	0	999998	569897.5	495138.3

APPENDIX C: FULL PROCESS OUTPUTS

Model 1

Run MATRIX procedure:

```
***************** PROCESS Procedure for SPSS Version 4.0 *****************

        Written by Andrew F. Hayes, Ph.D.       www.afhayes.com
   Documentation available in Hayes (2022). www.guilford.com/p/hayes3

**************************************************************************
Model  : 4
   Y  : BTACT_Z
   X  : ED
   M  : STRESS

Covariates:
 AGE      SEX      RACE     CHRONCON DEPAF

Sample
Size:  609

**************************************************************************
OUTCOME VARIABLE:
 STRESS

Model Summary
          R       R-sq        MSE          F        df1        df2          p
      .2198      .0483      .4282     5.0932     6.0000   602.0000      .0000

Model
              coeff         se          t          p       LLCI       ULCI
constant      .4208      .4255      .9890      .3231     -.4148     1.2565
ED            .0054      .0106      .5085      .6113     -.0154      .0262
AGE          -.0121      .0054    -2.2248      .0265     -.0228     -.0014
SEX           .0191      .0545      .3511      .7257     -.0879      .1262
RACE          .0704      .0265     2.6608      .0080      .0184      .1224
CHRONCON      .3040      .0815     3.7284      .0002      .1439      .4641
DEPAF         .0376      .0218     1.7265      .0848     -.0052      .0804

**************************************************************************
OUTCOME VARIABLE:
 BTACT_Z

Model Summary
          R       R-sq        MSE          F        df1        df2          p
      .5336      .2847      .5662    34.1699     7.0000   601.0000      .0000

Model
              coeff         se          t          p       LLCI       ULCI
constant     2.4975      .4897     5.1006      .0000     1.5359     3.4592
ED            .1322      .0122    10.8491      .0000      .1083      .1561
STRESS        .0490      .0469     1.0459      .2960     -.0430      .1411
AGE          -.0549      .0063    -8.7361      .0000     -.0673     -.0426
```

SEX	.1824	.0627	2.9094	.0038	.0593	.3056
RACE	-.1251	.0306	-4.0875	.0000	-.1852	-.0650
CHRONCON	-.1409	.0948	-1.4858	.1378	-.3271	.0453
DEPAF	.0018	.0251	.0725	.9422	-.0475	.0511

************************** TOTAL EFFECT MODEL ****************************
OUTCOME VARIABLE:
BTACT_Z

Model Summary

R	R-sq	MSE	F	df1	df2	p
.5323	.2834	.5663	39.6764	6.0000	602.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	2.5182	.4893	5.1465	.0000	1.5572	3.4791
ED	.1325	.0122	10.8722	.0000	.1085	.1564
AGE	-.0555	.0063	-8.8661	.0000	-.0678	-.0432
SEX	.1834	.0627	2.9245	.0036	.0602	.3065
RACE	-.1216	.0304	-3.9978	.0001	-.1814	-.0619
CHRONCON	-.1260	.0937	-1.3439	.1795	-.3101	.0581
DEPAF	.0037	.0250	.1463	.8837	-.0455	.0528

************** TOTAL, DIRECT, AND INDIRECT EFFECTS OF X ON Y **************

Total effect of X on Y

Effect	se	t	p	LLCI	ULCI
.1325	.0122	10.8722	.0000	.1085	.1564

Direct effect of X on Y

Effect	se	t	p	LLCI	ULCI
.1322	.0122	10.8491	.0000	.1083	.1561

Indirect effect(s) of X on Y:

	Effect	BootSE	BootLLCI	BootULCI
STRESS	.0003	.0008	-.0011	.0022

*********************** ANALYSIS NOTES AND ERRORS ************************

Level of confidence for all confidence intervals in output:
95.0000

Number of bootstrap samples for percentile bootstrap confidence intervals:
5000

------ END MATRIX -----

Model 2

Run MATRIX procedure:

***************** PROCESS Procedure for SPSS Version 4.0 *****************

Written by Andrew F. Hayes, Ph.D.   www.afhayes.com
Documentation available in Hayes (2022). www.guilford.com/p/hayes3

****
Model : 4
Y : BTACT_Z
X : ED
M : STRESS

Covariates:
AGE   RACE   CHRONCON DEPAF

Sample
Size: 336

****
OUTCOME VARIABLE:
STRESS

Model Summary

R	R-sq	MSE	F	df1	df2	p
.2164	.0468	.4341	3.2429	5.0000	330.0000	.0071

Model

	coeff	se	t	p	LLCI	ULCI
constant	.6806	.5504	1.2365	.2171	-.4022	1.7633
ED	.0044	.0134	.3287	.7426	-.0220	.0309
AGE	-.0148	.0073	-2.0313	.0430	-.0291	-.0005
RACE	.0647	.0332	1.9509	.0519	-.0005	.1299
CHRONCON	.2678	.1080	2.4801	.0136	.0554	.4802
DEPAF	.0323	.0398	.8118	.4175	-.0460	.1107

****
OUTCOME VARIABLE:
BTACT_Z

Model Summary

R	R-sq	MSE	F	df1	df2	p
.5124	.2625	.5455	19.5213	6.0000	329.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	2.0830	.6184	3.3683	.0008	.8665	3.2996
ED	.1277	.0151	8.4692	.0000	.0980	.1574
STRESS	.0651	.0617	1.0546	.2924	-.0563	.1865
AGE	-.0468	.0082	-5.6956	.0000	-.0630	-.0307
RACE	-.1012	.0374	-2.7073	.0071	-.1748	-.0277
CHRONCON	-.1195	.1222	-.9778	.3289	-.3598	.1209
DEPAF	-.0026	.0447	-.0575	.9542	-.0905	.0854

************************** TOTAL EFFECT MODEL ****************************
OUTCOME VARIABLE:
BTACT_Z

Model Summary

R	R-sq	MSE	F	df1	df2	p
.5100	.2600	.5457	23.1952	5.0000	330.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	2.1273	.6171	3.4473	.0006	.9134	3.3413
ED	.1280	.0151	8.4882	.0000	.0983	.1576
AGE	-.0478	.0082	-5.8480	.0000	-.0639	-.0317
RACE	-.0970	.0372	-2.6091	.0095	-.1701	-.0239
CHRONCON	-.1020	.1211	-.8428	.4000	-.3402	.1361
DEPAF	-.0005	.0447	-.0104	.9917	-.0883	.0874

************** TOTAL, DIRECT, AND INDIRECT EFFECTS OF X ON Y **************

Total effect of X on Y

Effect	se	t	p	LLCI	ULCI
.1280	.0151	8.4882	.0000	.0983	.1576

Direct effect of X on Y

Effect	se	t	p	LLCI	ULCI
.1277	.0151	8.4692	.0000	.0980	.1574

Indirect effect(s) of X on Y:

	Effect	BootSE	BootLLCI	BootULCI
STRESS	.0003	.0013	-.0019	.0035

********************** ANALYSIS NOTES AND ERRORS ***********************

Level of confidence for all confidence intervals in output:
95.0000

Number of bootstrap samples for percentile bootstrap confidence intervals:
5000

------ END MATRIX -----

Model 3

Run MATRIX procedure:

```
***************** PROCESS Procedure for SPSS Version 4.0 *****************

          Written by Andrew F. Hayes, Ph.D.       www.afhayes.com
    Documentation available in Hayes (2022). www.guilford.com/p/hayes3

**************************************************************************
Model  : 4
    Y  : BTACT_Z
    X  : ED
    M  : STRESS

Covariates:
 AGE      RACE     CHRONCON DEPAF

Sample
Size:  273

**************************************************************************
OUTCOME VARIABLE:
 STRESS

Model Summary
          R       R-sq        MSE          F        df1        df2          p
      .2249      .0506      .4280     2.8449     5.0000   267.0000      .0160

Model
              coeff         se          t          p       LLCI       ULCI
constant      .1766      .6225      .2836      .7769    -1.0490     1.4021
ED            .0073      .0176      .4130      .6799     -.0275      .0420
AGE          -.0090      .0084    -1.0787      .2817     -.0255      .0074
RACE          .0791      .0447     1.7714      .0776     -.0088      .1671
CHRONCON      .3481      .1265     2.7509      .0064      .0990      .5973
DEPAF         .0404      .0262     1.5446      .1236     -.0111      .0920

**************************************************************************
OUTCOME VARIABLE:
 BTACT_Z

Model Summary
          R       R-sq        MSE          F        df1        df2          p
      .5510      .3036      .5977    19.3243     6.0000   266.0000      .0000

Model
              coeff         se          t          p       LLCI       ULCI
constant     3.5501      .7357     4.8258      .0000     2.1017     4.9986
ED            .1374      .0209     6.5900      .0000      .0964      .1785
STRESS        .0334      .0723      .4625      .6441     -.1089      .1758
AGE          -.0642      .0099    -6.4842      .0000     -.0837     -.0447
RACE         -.1629      .0531    -3.0676      .0024     -.2675     -.0583
CHRONCON     -.1609      .1516    -1.0613      .2895     -.4595      .1376
DEPAF         .0031      .0311      .0991      .9211     -.0581      .0643

Test(s) of X by M interaction:
          F        df1        df2          p
      .1416     1.0000   265.0000      .7070
```

************************** TOTAL EFFECT MODEL ****************************
OUTCOME VARIABLE:
BTACT_Z

Model Summary

R	R-sq	MSE	F	df1	df2	p
.5505	.3030	.5960	23.2147	5.0000	267.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	3.5560	.7345	4.8417	.0000	2.1100	5.0021
ED	.1377	.0208	6.6135	.0000	.0967	.1787
AGE	-.0645	.0099	-6.5385	.0000	-.0839	-.0451
RACE	-.1603	.0527	-3.0399	.0026	-.2641	-.0565
CHRONCON	-.1493	.1493	-.9998	.3183	-.4433	.1447
DEPAF	.0044	.0309	.1435	.8860	-.0564	.0652

************** TOTAL, DIRECT, AND INDIRECT EFFECTS OF X ON Y **************

Total effect of X on Y

Effect	se	t	p	LLCI	ULCI
.1377	.0208	6.6135	.0000	.0967	.1787

Direct effect of X on Y

Effect	se	t	p	LLCI	ULCI
.1374	.0209	6.5900	.0000	.0964	.1785

Indirect effect(s) of X on Y:

	Effect	BootSE	BootLLCI	BootULCI
STRESS	.0002	.0015	-.0024	.0040

*********************** ANALYSIS NOTES AND ERRORS ************************

Level of confidence for all confidence intervals in output:
95.0000

Number of bootstrap samples for percentile bootstrap confidence intervals:
5000

------ END MATRIX -----

APPENDIX D: SOCIAL READJUSTMENT RATING SCALE (SRRS)

Directions: If an event mentioned has occurred in the past year, or is expected in the near future, copy the number in the score column. If the event has occurred or is expected to occur more than once, multiple this number by the frequency of the event.

Item	**Impact Score**	**My Score**
Death of a spouse	100	
Divorce	76	
Marital Separation	65	
Jail Term	63	
Death of close family member	63	
Personal injury or illness	53	
Marriage	50	
Fired at work	47	
Marital reconciliation	45	
Retirement	45	
Change in health of family member	44	
Pregnancy	40	
Sex difficulties	39	
Business readjustment	39	
Gain of a new family member	39	
Change in financial state	38	
Death of a close friend	37	
Change to a different line of work	36	
Change in number of arguments w/ spouse	35	
Mortgage over $20,000	31	
Foreclosure of mortgage or loan	30	
Change in responsibilities at work	29	
Son or daughter leaving home	29	
Trouble with in-laws	29	
Outstanding personal achievement	28	
Spouse begins or stops work	26	
Begin or end school	26	
Change in living conditions	25	
Revisions of personal habits	24	
Trouble with boss	23	
Change in work hours or conditions	20	
Change in residence	20	

Item	**Impact Score**	**My Score**
Change in schools	20	
Change in recreations	19	
Change in church activities	19	
Change in social activities	19	
Mortgage or loan less than $20,000	17	
Change in sleeping habits	16	
Change in eating habits	15	
Change in number of family get-togethers	15	
Vacation	13	
Christmas approaching	12	
Minor violation of the law	11	
Total Score:		

AUTHOR'S BIOGRAPHY

Zoe Maria Prats was born in Danville, Pennsylvania on April 26, 2001. They were raised in York, Pennsylvania and graduated from York Suburban High School in 2019. Majoring in Psychology, Zoe has minors in Neuroscience and Chemistry. They are a member of the University Orchestra, various chamber music ensembles, and have been a Maine Learning Assistant (MLA) in the Chemistry department for 2 years. They have received the A. Douglas Glanville Award, a Center for Undergraduate Research (CUGR) grant, and an Honors College Thesis Fellowship. Upon graduation, Zoe plans to attend the University of Mississippi as a Mississippi Teaching Corps (MTC) fellow, where they hope to become certified to teach high school Chemistry.