An Examination of Worry as a Mediator of the Effect of Stress on Somatic Health and Cognition

by

Tina Quade
Bachelor of Science (Honours), University of Victoria, 2012

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Supervisory Committee

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Abstract

Background: Previous research has demonstrated that chronic stress negatively impacts cognition and overall health. Perseverative cognitions such as worry can hold the physiological response of a stressor in the body (Brosschot, Gerin, & Thayer, 2006). The current study consists of two components: 1) a conceptual replication examining whether worry mediates the effect of stress on somatic health and stress and 2) extension of the model with cognition as the outcome.

Methods: This study used data from the second wave of the Midlife in the United States data collection: Project 1 (cross-sectional), Project 2 (daily diary), and Project 3 (cognition). Doing so approximated the time-series requirement of a mediation model and enabled access to the variables of interest. Mediation models were run via PROCESS software with covariates adjusted for at each path.

Results: Controlling for age, gender, education, household income, and chronic health conditions, the mediation models revealed mediation of the effect of stress severity on somatic health by worry frequency, duration, and self-identification.

Conclusions: Worry may be the process through which the physiological response to stress is prolonged thereby increasing the prevalence of effects on somatic health and cognition. By understanding the nuances of how stress impacts somatic health and cognition, prevention and intervention strategies can be implemented to reduce potential long-term outcomes.

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An Examination of Worry as a Mediator of the Effect of Stress on Somatic Health and Cognition

Introduction

Individual differences in the short- and long-term impact of stress on cognitive function and physical health suggest that additional factors, such as the accumulation of biological and environmental effects, are influencing this relationship (MacDonald, DeCarlo, & Dixon, 2011; Schwabe, Joels, Roozendaal, Wolf, & Oitzl, 2012). Exploring the impact of perseverative cognitions, a key property of repetitive thought patterns such as worry and rumination, may offer some insight into these individual differences (Watkins, 2008). Perseverative cognitions are repeated or chronic thought patterns of bringing a psychological stressor to mind, whether future –oriented such as worry, past-oriented such as rumination, or both (Brosschot, Gerin, & Thayer, 2006). This resurfacing of a stressor may provide a mechanism in which prolonged physiological activation leads to 'wear and tear' and changes in performance on attention-demanding cognitive tasks (Brosschot, 2010; Brosschot, Gerin, & Thayer, 2006; Sliwinski, Smyth, Hofer, & Stawski, 2006; Verkuil, Brosschot, Gebhardt, Thayer, 2010).

It appears that no study has examined whether perseverative cognitions mediate the relationship between stress and cognition. Investigating the role of perseverative cognitions in this relationship would enhance the research in this area and provide greater understanding of sources of individual differences in stress reactivity and their long-term impact on physical health and cognitive function. The focus of this thesis is two-fold. First, this research aims to replicate previous research that demonstrates worry as a mediator of the effect of stress on somatic health. Second, this research aims to empirically examine whether this mediation model can be extended to further explain the relationship between stress and cognition. If the analysis

herein suggests worry as a mediator in stress relationships then individual differences in stress reactivity may be in part due to perseverative cognitions. By understanding the feedback loop between perseverative cognitions, stress, and cognitive performance and somatic health, targeted interventions can be developed to reduce the impact of chronic stress on overall health.

In order to provide context to this analysis, this thesis includes a brief background of stress and cortisol, one of the key stress hormones. Second, there is a discussion of how stress is measured using physiological and psychological assessments. Third, how stress and perseverative cognitions impact somatic health is reviewed. Fourth, the impact of stress and cognitive interference on cognition is discussed. Together these sections provide a foundational level of understanding of how stress impacts health and cognition and how perseverative cognitions may work to hold the body's response to a stressor long after the stressor has concluded. The stress-cognition and stress-somatic health relationships will inform the hypotheses declared in this thesis. Finally, this thesis reports the results of a novel secondary analysis examining whether worry, a form of perseverative cognition, mediates the relationship between stress and cognition and stress and somatic health.

1.1 Understanding Stress

In this modern age we continue to recruit our primal brain to sift through how to respond to an oncoming stressor; however, what we are responding to and how often has changed. Stressors can be divided into two categories: 1) absolute or real threats that elicit adaptive responses essential for survival or wellbeing (i.e., everyone interprets as stressful); and 2) relative or implied threats with a response ranging from mild to pronounced (i.e., stressors only some individuals would interpret as stressful) (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). Instead of stressors being primarily life or death decisions to flee or fight a predator (absolute

stressor), humans navigate more relative stressors such as traffic jams and time constraints (Link between stress, n.d.). This modern shift away from primarily absolute stressors puts strain on the body since the stress response is an adaptive mechanism built to keep humans alive during short-term stress situations (Link between stress, n.d.). In addition to the absolute-relative categorization, stressors can also be divided based on whether they are internal (endogenous) or external (exogenous) to the person experiencing the stress or whether they are physical or psychological stressors (Stressor, n.d.; University of Maryland Medical Center, 2013). An internal stressor could be inflammation, worry, or rumination whereas an external origin could be experiencing physical violence, being cut with a knife, or breaking a bone. Comparatively, a physical stressor can span inflammation, pain, and hot/cold temperature whereas a psychological stressor can include anything psychologically interpreted as negative or threatening (Stressor, n.d.). The potential for an event to trigger as a stressor is ubiquitous, requiring one or more of the following four components: novelty, unpredictability, threat to the ego and sense of low control (From stress, 2014; Mason, 1968). Similarly, Dickerson and Kemeny (2004) state that uncontrollability and social-evaluative elements are the key triggers. Ultimately this continuous stress response can become maladaptive as the physical and psychological systems receive little to no reprieve between stressors.

Stress is a term commonly held responsible for health outcomes such as depression, heart attack, disrupted immune system, and gastrointestinal disease (Schlotz, 2013; Tovian et al., n.d.). When initially introduced by Hans Selye in 1956 it was described as a ‘speedometer of life’ that represents, at one time, the total wear and tear in the body (Selye, 1956, p. 276). This wear and tear is triggered by a stressor, which is something that leads to the release of stress hormones (Stressor, n.d.). In this view, Selye proposed that the experience of stress was the body’s general

responses of adapting to demands for change (Selye, 1956). This process, known as the General Adaptation Syndrome, includes three phases:

1. The “alarm phase” where defense systems are triggered.
2. The “stage of resistance” where alarm stage processes are countered and the body tries to regain homeostasis.
3. The “stage of exhaustion: where one or more organs are exhausted. Symptoms of disease or dysfunction present themselves (Everly & Lating, 2013, pp. 39-40).

Essentially, stress is a disruption to the homeostasis or equilibrium of the body and stressors are the “disturbing forces” (Johnson, Kamilaris, Chrousos, & Gold, 1992, p. 115). Adaptive responses work to redirect behaviour and energy so that the body can regain homeostasis (Johnson et al., 1992).

Popular culture views the term “stress” as purely a negative experience. This end of the stress spectrum, known as distress or negative stress, is when demands are perceived to be outside one’s coping skills, decreasing performance over the short- or long-term (Mills, Reiss, & Dombeck, 2008). Conversely eustress, otherwise known as positive stress, can be motivating if the experience is within our coping abilities (What is stress, n.d.). In fact, in eustress situations a stressor, sometimes labelled as a challenge, becomes arousing and leads to increases in performance (What is stress, n.d.). Examples of eustress include winning a race or beginning a new relationship where there is a tight line walked between excitement and ambiguity (Mills et al., 2008). This is the paradox of stress. On one hand humans thrive on it to motivate and excite, while on the other hand too much stress can lead to function declines, exhaustion, and ill health (What is stress, n.d.). A common theme when discussing stressors is that the process of categorizing a stressor as threatening or not and labeling a stressor as positive or negative is

highly influenced by the individual's perception of a stressor (What is stress?, n.d.). This process is known as the cognitive-affective domain of cognitive appraisal and affective integration (Everly & Lating, 2013). Cognitive appraisal is the cognitive interpretation of what is happening in our surroundings and affective integration is the feelings that we attach to the interpretation (Everly & Lating, 2013). If the individual exposed to the stressor doesn't label the experience as negative or threatening then they can have a completely different interpretation and physiological experience of what is happening compared to another person. This reveals one of the challenges in defining stress: our perception of whether a stressor is good (eustress) or bad (distress) involves a combination of how the situation is appraised, the past experiences brought to the situation, and the coping skills available to the individual. Essentially something is categorized as a stressor when it is cognitively interpreted as such and when there is an emotional reaction attached to the stimuli (Everly & Lating, 2013).

1.2 Stress Reactivity

Defined as "the capacity or tendency to respond to a stressor" (Schlotz, 2013, p. 1891), stress reactivity occurs across stress response domains (i.e. physiology, behaviour, subjective experience, cognitive function) and is considered a vulnerability factor for diseases such as cardiovascular disease, depression, and anxiety. Stress reactivity can be explored as general stress reactivity, or as individual system responses such as cardiovascular stress reactivity or endocrine stress reactivity (Schlotz, 2013). Individual differences in stress reactivity can be explained by individual response specificity (IRS) and stimulus response specificity (SRS) (Schlotz, 2013). Individual response specificity addresses differences in patterns of responses, such as low increases of cortisol paired with high blood pressure, whereas SRS addresses response patterns related to stressors, such as activation of the HPA axis by socially threatening

stressors (Schlotz, 2013). Testing stress reactivity in a laboratory setting offers better standardization and control over confounds (Schlotz, 2013). Comparatively, ambulatory methods of assessment offer better ecological validity through assessment of stress reactivity in daily life, which "…is associated with sex, age, ethnicity, personality factors, pre-existing disease, and the presence or absence of chronic stress" (Schlotz, 2013, p. 1892)

1.2.1 Physiological response to stress. On a physiological level the stress response begins in the amygdala where images and sounds are scanned for danger (Understanding, 2011). If a sensory nerve cell detects a threat then it triggers a cascade of nerve signals and hormones that elicit the fight or flight response (How cells communicate, n.d.). As a result, the amygdala communicates with the hypothalamus, sending signals to the sympathetic nervous system (SNS), which mobilizes energy and triggers the 'fight or flight' response (Thayer & Brosschot, 2005). Specifically, after the sensory nerve cells detect a threat, the hypothalamus sends a signal to the pituitary gland, which releases chemical messengers that trigger the adrenal glands to release cortisol, a liposoluble glucocorticoid (How cells communicate, n.d.; Marin et al., 2011). With receptors throughout almost all somatic cells (i.e., body and brain), cortisol is an end product of hypothalamic-pituitary-adrenal (HPA) axis activation where its role is to optimize adaptability and performance in changing environments and to increase energy in the fight-or-flight response (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Marin et al., 2011; Yeager, Pioli, & Guyre, 2011). Cortisol does this by increasing blood glucose levels and the brain's ability to use it along with suppressing the digestive and reproductive systems (Everly & Lating, 2013; Stress: Coping, n.d.; Stress management, 2013). In addition to the release of cortisol by the HPA axis, epinephrine (adrenaline) and norepinephrine (noradrenaline) are released by the adrenals in the sympathetic-adrenal-medullary (SAM) system, which increase heart rate, blood pressure and

energy, as well as decrease blood flow to organs (e.g., kidneys, digestive functions, and skin) (Everly & Lating, 2013; How cells communicate, n.d.; Stress: Coping, n.d.; Stress management, 2013). Once the danger has ceased, the hypothalamus signals the parasympathetic nervous system (PNS), which ceases the fight-or-flight response and switches the body over to rest-and-digest (Thayer & Brosschot, 2005).

Regardless of whether the perception of an immediate threat such as hunger, isolation, or danger is real, the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA) axis are activated, thereby generating energy for the fight-or-flight response (Sapolsky, Romero, & Munck, 2000; University of Maryland Medical Center, 2013). This process manifests as muscle tension, rapid breathing, heavy breathing, increased heart rate and blood pressure, decreased digestion, diarrhoea, constipation, and increased liver glucose production (Tovian et al., n.d.). This oscillation between energy mobilization and restoration is important in being a stable, adaptable, and healthy individual (Thayer & Brosschot, 2005). However, if the sympathetic nervous system becomes hyperactive then large energy demands ensue and, over time, result in symptoms such as emotional distress, gastrointestinal distress, muscular tension such as headaches and back, or jaw pain (Thayer & Brosschot, 2005; University of Maryland Medical Center, 2013). These symptoms are most commonly experienced in response to acute stressors and tend to be short-term, leading to the least burden from which the body needs to recover (Stress, n.d.). Comparatively, chronic stress involves a constant suppression of the fight or flight response, which can manifest as outcomes across multiple body systems (Stress, n.d.). Chronic stress can lead to tension or migraine headaches from chronic muscle tension, risk for hypertension, heart attack, or stroke, diabetes, ulcers, premenstrual syndrome, and impotence as well as altered testosterone, sperm production, and

menstrual cycles, worsened menopausal symptoms, and decreased sexual desire in women (Tovian et al., n.d.). Taking license from research in allostasis and allostatic load, there are four kinds of experiences that can result in physiological overburden: repeated novel events, an inability to adapt to a repeated stressor, an extended duration of response, and an inadequate response. Repeated novel events implies a constant exposure to new experiences, which can elevate stress mediators over time (McEwen & Seeman, 1999, 2009). A physical stress mediator is a biomarker such as cortisol, epinephrine (adrenaline), or norepinephrine (noradrenaline), which indicates the body's physiological response to a stressor (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). The inability to adapt to a repeated stressor occurs when the body is unable to adapt to a repeated influx of stress mediators thereby leading to an overabundance of stress mediators (McEwen & Seeman, 1999, 2009). An extended duration of response occurs when the body is unable to shut off a hormonal response, which leads to the body's lengthened exposure to the stress response (McEwen & Seeman, 1999, 2009). Conversely, an inadequate response can occur when the body doesn't response sufficiently to a stressor (McEwen & Seeman, 1999, 2009). For example, in response to a stressor an individual may experience higher levels of inflammatory cytokines as a result of insufficient release of glucocorticoids, the class of hormones to which cortisol belongs (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; McEwen & Seeman, 1999, 2009). Examples of stressors that lead to chronic stress include interpersonal conflicts and work-related pressures (University of Maryland Medical Center, 2013).

1.2.1.1 Impact of cortisol on function and health. Excess cortisol secretion from chronic stress can impact function and health at the primary, secondary, and tertiary level. At the primary effect level, gene expression is regulated by cortisol through DNA interactions and protein-to-protein transcription regulation (McEwen & Seeman, 2009). For example, research has related

shortened telomeres with chronic stress, leading to accelerated aging and increased risk of disease (Epel et al., 2004). Elevated cortisol can lead to cellular events such as inflammatory responses and glucose regulation, which can manifest into physiological damage known as secondary outcomes (Sapolsky, Romero, & Munck, 2000). If the body frequently undergoes the stress response then the hypothalamic-pituitary-adrenocortical (HPA) axis may become over-activated, thereby risking sustained dysregulation of cortisol levels and high levels of excitatory amino acid neurotransmitters (McEwen & Seeman, 1999). This process can lead to glucocorticoid receptor resistance (GCR), which disrupts the HPA axis response that controls inflammation and increases the risk of succumbing to viruses such as the common cold (Cohen et al., 2012). Secondary outcomes that can manifest over time as a cumulative outcome of primary effects include sub-clinical levels of metabolic syndromes such as elevated HDL cholesterol, high waist girth, hyperglycemia, formation of insulin resistance and elevated blood pressure (Juster et al., 2010; Takamiya et al., 2004). Over time these secondary outcomes can transition into tertiary outcomes, which are the diseases or disorders that are the cumulative result of allostatic load (Korte, Koolhaas, Wingfield, & McEwen, 2005; Lupien et al., 2007; McEwen, 1998, as cited in Juster et al., 2010; McEwen & Seeman, 2009). For example, pro-inflammatory cytokines, such as IL-6, that are triggered in an inflammatory response increase the risk of a dysfunctional immune system and the development of chronic conditions such as cardiovascular disease, type II diabetes, and functional decline (Kiecolt-Glaser et al., 2003; Lupien et al., 2007; McEwen & Seeman, 1999, 2009; Sapolsky, Romero, & Munck, 2000).

Reduced excitability and neuronal atrophy may occur, particularly in the hippocampus. This can lead to sympathetic-adrenal-medullary (SAM) system and HPA axis overstimulation and can alter the interpretation of situations such that an individual is more like to perceive or

anticipate more stressors (Juster et al., 2010; McEwen & Seeman, 1999). As a result, a feedback loop can develop where neuronal atrophy leads to an altered evaluation of stressors and over-activity of the HPA axis, which then increases cortisol and increases the risk of further neuronal atrophy (Miller, Chen, & Zhou, 2007). Hypersecretion of cortisol likely leads to an altered evaluation of stressors; the loss of hippocampal neurons leads to desensitization of circulating cortisol leading to an underestimation of cortisol levels (Sapolsky, Krey, & McEwen, 1986). In addition to an altered perception of stress, neuronal atrophy in the hippocampus can lead to cognitive deficits in recall and memory formation (Sapolsky, Romero, & Munck, 2000).

1.2.2 Psychological responses to stressors. Stress appraisal, or how an individual appraises the significance of a stressor, is one of the keys to how and whether a stressor will impact an individual's physical and psychological health. Individual differences in the brain's perception of a stressor and the subsequent physiological responses account for part of the occurrence of stress-related disease (Juster et al., 2010; McEwen, 1998). Perceived stress is positively associated with HPA axis activity and dysregulated cortisol, with chronic stress exposure resulting in changes in cortisol fluctuation and increased cortisol volume per day (Miller, Chen, & Zhou, 2007). Collectively, these outcomes can result in altered interpretation of stressors (negative appraisal) and lead to a positive feedback loop where HPA axis overstimulation perpetuates more neuronal atrophy and changes in stress appraisal (Juster et al., 2010; Miller et al., 2007). In addition to HPA axis dysregulation, uncertainty and threat triggers the sympathetic nervous system's (SNS) fight-or-flight response, which down regulates the prefrontal cortex (Thayer & Brosschot, 2005). This process has been adaptive in genuine situations of threat and uncertainty, but can be problematic in modern life where the prefrontal cortex is essential in inhibiting ongoing SNS mobilization in response to constant stressors as

well as "…cognitive functions such as working memory, sustained attention, behavioural inhibition, and general mental flexibility" (Thayer & Brosschot, 2005, p. 1055). An individual's perception of a stressor as being positive, negative, or neutral is influenced by a participant's values, beliefs, commitments, and expectations (Lazarus & DeLongis, 1983). In respect to the physiological impact of a stressor the most important factor may be the management of a stressor rather than the actual physiological arousal experienced by the individual (Lazarus, 1996). This is because effective coping (management) can ease the stress response and, conversely, ineffective coping can exacerbate the possibly harmful effects of stress (Lazarus, 1996).

1.3 Stress Across the Lifespan

Stress impacts cognition regardless of when it is experienced during the lifespan. Exposure during early childhood increases stress reactivity and cognitive deficits in adulthood (Heim & Nemeroff, 2001; Lupien, McEwen, Gunnar, & Heim, 2009). Stress experienced in utero has been linked to increased HPA axis basal activity throughout childhood along with neurological, cognitive, and behavioural disturbances such as attention deficit hyperactivity disorder, sleep disturbances, unsociable and inconsiderate behaviour, and some psychiatric disorders (i.e. depressive symptoms, mood and anxiety disorders, drug abuse) (Hedges & Woon, 2011; Heim & Nemeroff, 2001; Lupien, McEwen, Gunnar, & Heim, 2009). This occurs regardless of whether the cortisol originates via exogenous glucocorticoids consumed by the mother (e.g. ingesting cortisol to control inflammation) or maternal stress or anxiety. These effects of stress in utero may be moderated by the quality of postnatal care (Lupien, McEwen, Gunnar, & Heim, 2009). A child's HPA axis activity level is influenced by the mother's psychological state, with sensitive parenting showing lower HPA axis activity or a shorter length of activation. Once born, severe deprivation, neglect, or abuse in care is associated with

hypocortisolism (low cortisol output), likely as a result of the down-regulation of the HPA axis. Similar to in utero studies, this may be reversible through sensitive and supportive care (Hedges & Woon, 2011; Heim & Nemeroff, 2011; Lupien, McEwen, Gunnar, & Heim, 2009).

Depression and anxiety prevalence increases in adolescence, which coincides with evidence of increased glucocorticoid receptor mRNA levels (i.e., gene expression of receptors for cortisol) in the prefrontal cortex during this period in comparison to other stages of the lifespan (Lupien, McEwen, Gunnar, & Heim, 2009). The frontal cortex may be specifically vulnerable to stress due to evidence of altered grey matter volume and neuronal integrity as well as a small anterior cingulate cortex in those exposed to adversity since early life (Cohen et al., 2006; Lupien, McEwen, Gunnar, & Heim, 2009). The differences in brain structures between those who experience early life stress and those who do not are not limited to psychopathologies like PTSD; even moderate levels of early life stress and trauma appear to lead to brain alterations (Cohen et al., 2006). However, low cortisol levels may be a risk factor for developing PTSD in response to adulthood trauma (Lupien, McEwen, Gunnar, & Heim, 2009). Studies demonstrating HPA axis hyperactivity and smaller hippocampal volume in adulthood has been linked to childhood trauma or abuse, even in instances of adult PTSD or depression (Gilbertson et al., 2002; Heim et al., 2000; Vythilingam et al., 2002).

Major life events, which are often tandem with adulthood, can be key sources of stress as individuals age (Almeida & Horn, 2004). Whether based on age-related expectations (e.g., getting married, moving out of their parents' house), or biological changes (e.g., menopause, child-bearing), age- and gender-based differences in stress have often pointed to life events (Almeida & Horn, 2004). These differences include the primary domains in which stressors occur, the stress severity ratings, and the perceived risk of the stressor(s) (i.e. financial risk or

how others feel about the individual). The following are insights based on research using the Daily Inventory of Stressful Events (DISE), which employs investigator-rated severity (objective) and participant self-rating (subjective) (Almeida & Horn, 2004). Using three domains – interpersonal tensions, network stressors, and overloads – the results indicated that younger and middle-aged adults had a higher proportion of overloads in comparison to the older adult group, which is likely due to a strong career focus during these stages of life. Comparatively, older adults experienced more network stressors and stressors involving another person, in particular spouses. In terms of gender, women's frequent reports were overload, network, and child-related stressors whereas men had more stressors with a co-worker. Overall the gender and age differences are similar to other research where younger adults (35 – 45 years old) reported more financial, work, home, personal life, and family and friend hassles in comparison to older adults (65 – 74 years old) and women reported more environmental and social issue hassles (Folkman, Lazarus, Pimley, Novacek, 1987). When comparing stress severity ratings, subjective ratings were medium and objective ratings were low severity, with the highest ratings amongst younger and middle-aged participants (Almeida & Horn, 2004). Age differences were not found in the objective stress severity ratings. This discrepancy may be due to life experience and coping methods that may have influenced the participants' subjective experience of the stressor in question (Almeida & Horn, 2004). For example, older adults may employ more emotion-focus coping such as positive reappraisal and emotional distancing, which may truncate the stress process (Folkman, Lazarus, Pimley, Novacek, 1987). Age- and gender-related differences were also found in the perceived risk of a stressor. Overall, younger and middle-aged adults reported higher perceived risk in the way others felt about them, whereas men reported greater financial risk and women reported greater risk in what others think about them (Almeida & Horn, 2004).

1.4 Measuring Stress

Researchers need to consider several factors when deciding how to measure stress. Primarily, a researcher decides whether they will examine physical stress mediators, such as cortisol, epinephrine (adrenaline), or norepinephrine (noradrenaline), or the psychological aspects of stress. The psychological measures of stress address the perception and appraisal of a stressor. These variables are often self-report, but may also include the perspective of a third party. For example, in MIDUS II – Project 2 the stress severity rating is determined by the interviewer. The choice of which method or methods to use is based on a number of factors such as time, resources, and ethics.

1.4.1 Physiological measures of stress. There are several ways to approach the physiological measurement of stress. Catecholamines such as epinephrine and norepinephrine can be measured via blood analysis or can be inferred by using proxies of sympathetic activation (e.g., blood pressure, vagal tone) (How to measure, 2007). Cortisol secretion can be measured by using blood, urine, saliva or hair analysis, as well as electrochemical immunosensing. The cortisol can either be produced by the body (endogenous) or can be introduced to the body via injection or consumption for therapy or research purposes (exogenous) (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). Of primary consideration when measuring cortisol are the challenges in obtaining an accurate measurement. Special considerations need to be taken in comparing cortisol measures from the same time of day, eliminating food or beverage consumption that stimulates the nervous system, and ensuring participant compliance to the strict sampling protocols (primarily due to diurnal fluctuation considerations) (How to measure, 2007).

1.4.1.1 Blood analysis. Cortisol blood analysis is infrequently used in research due to the expense (e.g., medical staff, specialized equipment), biohazard regulations, and ethics for

participant burden. Critics of blood analysis argue that the procedure leads to confounded cortisol readings due to spikes in cortisol levels from the anticipation of the blood draw (Levine, Zagoory-Sharon, Feldman, Lewis, & Weller, 2007).

1.4.1.2 Urinary analysis. A 24-hour urinary analysis can used to analyze cortisol secreted in the urine. This process requires collection of all urine excreted in a 24-hour period using a special bag or container (Dugdale, 2013). Test results can be impacted by dehydration, certain medications, x-ray exams using dyes within three days of the urine test, vagina fluid in the sample, emotional stress, heavy exercise and a urinary tract infection (Dugdale, 2013). Since cortisol levels have a diurnal rhythm, several collections of 24-hour urinary samples may be required to capture an accurate reading (Wisse, 2013).

1.4.1.3 Saliva analysis. Saliva analysis is the most frequently used method for measuring cortisol because it consists of non-invasive collection, protocols that can be executed in the participant's home, and relatively inexpensive assay costs (How to measure, 2007). In fact, recent technological advances have led to the development of an external device that can attach to smartphones, effectively creating a real-time, inexpensive, disposable cortisol assay (Ehrenkranz, Polson, & Espiritu, 2014; Tew, 2014).

However, there are several concerns in respect to saliva analyses including the immunoassays used, the competition with cortisone, and the reliance on participant-driven sample collection. Assay results can vary from one laboratory to another due to differences in the method for quantifying cortisol based on its reaction with an antibody (Darwish, 2006). This makes cross comparison between studies challenging because the analyses may have used different antibodies to bind to the cortisol (Miller, Plessow, Rauh, Groschl, & Krischbaum, 2013). Another problem in saliva analysis is the competition between cortisol and cortisone in

binding with the antibody. Whereas cortisol is the target hormone (i.e., the hormone of measurement interest), cortisone is an abundant hormone that is cross-reactive with cortisol. Cortisone's longer half-life means that as the temporal distance between cortisol release and saliva collection increases, the likelihood of a higher cortisone concentration also increases. This can skew the assay results by a factor of three or more (Miller et al., 2013). Reliance on participant-driven saliva collection in a natural environment is also problematic. Even when following a prescribed sampling protocol, Kudielka, Broderick, and Kirschbaum (2003) demonstrated that in their experiment 26% of participants neglected to follow the protocol at least once and 21% at least twice. This can lead to issues in the validity of participant-driven saliva collection because of potential non-compliance in the predefined sampling protocol. Additionally, sampling issues may arise in researcher-driven saliva collection due to individual differences in participants' diurnal cortisol cycles. For example, some participants may have fairly flat cortisol cycles, which leads to difficulty in analysing their results unless individual baseline diurnal cortisol levels are predetermined (Out, Granger, Sephton, & Segerstrom, 2013; Stone et al., 2001). Even if all participants have typical diurnal cortisol cycles, the cost of researcher-driven saliva collection can be prohibitive and burdensome to participants, especially if multiple samples are required.

1.4.1.4 Hair analysis. Hair analysis enables researchers to bypass common issues related to blood or saliva cortisol sampling, including low variance in diurnal rhythm, non-compliance to sampling protocol, artificial cortisol surge due to testing conditions, and competition with cortisone in antibody-binding. Much like the rings of a tree truck, hair grows at a predictable rate of 1cm per month; therefore, researchers use the hair shaft to look at past deposits of cortisol with the centimeter closest to the scalp representing the last month (Kaushik, Vasudev, Arya,

Pusha, & Bhansali, 2014). This method enables researchers to answer questions related to long-term exposure to stress (Kaushik et al., 2014; Sauve, Koren, Walsh, Tokmakejian, & van Uum, 2007). However, this method has several challenges, including finding enough participants who are willing to provide a pencil-thick swatch of hair cut from the root and dealing with issues related to variance in cortisol readings in artificially coloured hair (Sauve et al., 2007).

1.4.1.5 Electrochemical immunosensing. New technologies such as electrochemical immunosensing are being developed in a bid to overcome the challenges of other physiological measures of cortisol. For example, standard immunoassays require antibody or antigen labels to bind to the antigen in order to demonstrate its detection, in this case cortisol (Kaushik, Vasudev, Arya, Pusha, & Bhansali, 2014). Electrochemical immunosensing is a label-free method that measures cortisol through changes in electrical properties of a conductive microelectrode (Kaushik et al., 2014). This technique enables a high degree of precision (reduction in error) through its procedure automation and has the potential to be integrated into microchips and wearable technologies (Kaushik et al., 2014).

Physiological measures of stress provide an understanding of the body's cascade of stress mediators in response to a stressor. However, these measurement methods are often costly, invasive, and stress inducing, thereby potentially confounding the assay. An alternative is to use psychological measures of stress, which can be collected in a variety of settings at very little cost to the researcher.

1.4.2 Psychological measures of stress. In contrast to physiological measures, psychological measures of stress rely on the individual's interpretation or rating of a stressor. Benefits of this method include non-invasiveness, low-cost, and measurement of subjective experience of a stressor while maintaining a close approximation of the cortisol response (Miller,

Chen, & Zhou, 2007).

1.4.2.1 Daily Inventory of Stressful Events (DISE). The Daily Inventory of Stressful Events (DISE) is an investigator-based measurement approach that objectively classifies stressors by their content, severity, and threat appraisal using daily telephone interviews (Almeida, Wethington, & Kessler, 2002). Trained coders use structured and semi-structured questions to obtain a comprehensive overview of stressors. A more precise evaluation is achieved through open-ended questions and reducing biased responses by determining whether a negative rating for the day is due to an objective stressor, mood disturbance, or ill health (Almeida et al., 2002). Past research found that on average participants' stressors were subjectively rated as medium severity, but were rated as low severity by objective coders (Almeida & Horn, 2004). A limitation of the DISE is whether the trained coders' objective rating is more representative of the stressor experienced by the participant since it does not account for subjective experience and related influences such as coping mechanisms, emotion regulation, and perceived control.

1.4.2.2 Perceived Stress Scale (PSS). As the most widely used measure of global stress, the Perceived Stress Scale (PSS) is a subjective measure that asks participants to rate the severity of stress in response to 14 positive and negative questions (Barbosa-Leiker et al., 2013; Cohen, Kamarck, & Mermelstein, 1983). Such questions include, "In the last month, how often have you been upset because of something that happened unexpectedly?" and "In the last month, how often have you felt confident about your ability to handle your personal problems?" (Cohen et al., 1983; p. 394). Responses are collected using a Likert scale: 0 never, 1 almost never, 2 sometimes, 3 fairly often, and 4 very often (Cohen et al., 1983; p. 394). Intending to measure two constructs - stress and emotions or feelings that counter stress - the PSS provides comparison of

stress perception between individuals (Barbosa-Leiker et al., 2013; Golden-Kreutz, Browne, Frierson, & Andersen, 2004). By asking questions that query perceptions that counter stress, such as the perceived ability to handle an irritation, the PSS satisfies arguments supporting the inclusion of positive psychological states in stress research because of their potential role in buffering against negative health effects (Barbosa-Leiker et al., 2013; Folkman, 1997, 2008; Folkman & Moskowitz, 2000).

1.5 Impact of Stress and Perseverative Cognitions on Somatic Health

Exploring the ability of perseverative cognitions to prolong physiological activation of a stressor offers insight into individual differences in the effect of stress on somatic health and cognitive function (Brosschot, 2010; Brosschot, Gerin, & Thayer, 2006; O'Connor, Walker, Hendrickx, Talbot, & Schaefer, 2013; Schwabe, Joels, Roozendaal, Wolf, & Oitzl, 2012). Perseverative cognition is an over-arching term that references several repetitive thought constructs such as worry, rumination, and anticipated stressors (Brosschot, Gerin, & Thayer, 2006). The consistent feature that ties these constructs together under the umbrella of perseverative cognitions is the "…repeated or chronic activation of the cognitive representation of one or more psychological stressors" (Brosschot, Gerin, & Thayer, 2006, p. 114). Repetitive thought is the act of repeatedly bringing to mind a stressor whether from the past, or in the future (Brosschot, Gerin, & Thayer, 2006). Repetitive thoughts can hold the physiological activation in the body along with impeding the emotional and cognitive process of a situation (Gianferante, et al., 2014). In regards to physiological activation, research has demonstrated that worry and rumination are mediators for slow cortisol recovery after a negative mood induction (speech stressor) and an emotional stressor (harassment) (Brosschot, Verkuil, & Thayer, 2010). Examples of activation include increased awakening cortisol and indicators of an altered

autonomic nervous system (ANS; decreased parasympathetic activity and increased sympathetic nervous system via high blood pressure) (Querstret & Cropley, 2013; Zygmunt & Stanczyk, 2010). If prolonged, these activations can lead to cardiovascular and autoimmune disease among other chronic health conditions (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Malik et al., 1996). Proposed as the "perseverative cognition hypothesis", perseverative cognitions are mechanism through which the activation of stress is prolonged and thus facilitates the impact of stress on health (Brosschot, Gerin, & Thayer, 2006). In the context of this study, the mediation model is the process through which worry impacts the body such that worry mediates the effect of stress on somatic health (Brosschot, Pieper, & Thayer, 2005).

1.5.1 A primer on worry. Of particular interest in the context of this study is the impact of worry on somatic health and cognition. Closely related to the fear process, worry attempts to actively problem solve an uncertain future outcome that has at least one possible negative outcome that violates one or more goals of the individual (Brosschot, Gerin, & Thayer, 2006; Tallis & Eysenck, 1994). These negative affect-laden thoughts are often unproductive or counterproductive, prolonging or magnifying negative affect (Brosschot, Gerin, & Thayer, 2006; Davey, 1994, as cited in Brosschot, Gerin, & Thayer, 2006).

It is noteworthy to mention that worry, not anxiety, is the focus of this research. Brosschot and colleagues (2006) note that worry plays a role in anxiety disorders, such as generalized anxiety disorder. Anxiety is more closely related to fear and is defined as "…an unpleasant state characterized by affective, cognitive, and physiological elements such as fear, worry, apprehension and tension" (Hughes, 2011). However, worry demonstrates stronger links to cognitive intrusion hence its inclusion with other repetitive thought processes that underlie perseverative cognitions (Zebb & Beck, 1998).

In terms of individual differences of worrying, one way people differ in how much they worry is their level of linking, or how strongly they believe their lower level goals must be obtained in order to achieve their higher-level goals (Verkuil, Brosschot, Gebhardt, & Korrelboom, 2015). In a clinical study of work stress, high linkers who suffered from work stress reported almost twice as much worry as the healthy comparison group with goal linking more strongly predicting worry frequency and duration than trait worry (Verkuil, Brosschot, Gebhardt, & Korrelboom, 2015).

Prolonged physiological activation resulting from worry is theorized to be due to the following three-part function of worry:

1. Threat appraisal of personal cost, imminence, likelihood and estimated self-efficacy.
2. Physiological and psychological worry activation via identification of a threat (alarm), entry of threat into awareness as a reminder of being unresolved (prompt), and anticipation of dangers (preparation).
3. Maintenance of chronic worry via catastrophising and missed attempts to problem solve (Brosschot, Gerin, & Thayer, 2006; Tallis & Eysenck, 1994).

If control, be it illusionary or actual, is present, then an individual may not progress past the first stage of threat appraisal due to belief in their ability to cope or exert control in the situation (Bandura, 1990; Miller, 1979; Tallis & Eysenck, 1994). However, the degree of threat appraised is impacted by the frequency and total number of intrusive thoughts (Tallis & Eysenck, 1994). During the second stage, if an individual has not yet responded to a threat then mental representations such as thoughts and images will alter to maintain a level of novelty, since this increases the likelihood of capturing the individual's attention and act on the threat (Tallis & Eysenck, 1994). In the third stage, the threat rarely materializes, thereby maintaining the threat

preparation and worry process and the fight-or-flight activation (Brosschot, Gerin, & Thayer, 2006; Tallis & Eysenck, 1994). The process of worrying may increase the likelihood of reaching the third stage of maintained, or chronic, worry. This is because problem solving is often interrupted by worry activation, negative future mental modeling, and ineffective coping choices (Tallis & Eysenck, 1994). This process of worry, threat appraisal, activation, and maintenance mirrors Mason's (1968) requirements for an event to trigger as a stressor: novelty, unpredictability, threat to the ego and low sense of control.

There are several reasons why an individual worries, including goal commitment, coping strategy, and biological vulnerability (Verkuil, Brosschot, Gebhardt, & Thayer, 2010). The duration of perseverative cognitions triggered by threats to goal attainment is based on the individual's commitment to achieving the goal, which is a combination of the importance or value of the goal and the ability of the individual to cope with negative outcome expectancies (hopelessness) or no outcome expectancies (helplessness) (Verkuil, et al., 2010; Ursin & Eriksen, 2004). An individual may also have motivation to use worry as a coping mechanism either as a problem solving strategy or as cognitive avoidance (Verkuil et al., 2010). In terms of problem solving, worry is abstract, which makes concrete action unlikely, and high worriers are not likely to implement their solutions (Borcovec, Ray, & Stöber, 1998; Verkuil et al., 2010). Worry can also be used as a cognitive avoidance technique to limit emotional exposure to threatening information (Verkuil et al., 2010). Although tempting, this technique negatively reinforces worry as a coping mechanism (Verkuil et al., 2010). It interferes with the emotional processing of the information, the integration of information incompatible with the fear, and the subsequent formation of new memory that evokes emotional change (Foa & Kozak, 1986; Verkuil, Brosschot, Gebhardt, & Thayer, 2010). Worry may also be the result of a biological

susceptibility, such as low prefrontal inhibition experienced in chronic stress situations (Verkuil et al., 2010). Linked to low heart rate variability (HRV), worry is characterized by low PNS activation (hypoactivity) where the stimulating SNS dominates and the heart rate changes more slowly in response to a changing environment (Malik et al., 1996; Thayer & Brosschot, 2005; Verkuil et al., 2010). If chronic, excessive, and uncontrollable, worry can be intolerable and can result in diagnosis of generalized anxiety disorder (GAD) (Borkovek et al., 1998).

1.5.2 How perseverative cognitions relate to somatic health. The resurfacing of a stressor may provide a mechanism in which prolonged physiological activation leads to 'wear and tear' on the body and altered performance on attention-demanding cognitive tasks (Brosschot, Gerin, & Thayer, 2006; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Sliwinski, Smyth, Hofer, & Stawski, 2006; Verkuil, Brosschot, Gebhardt, Thayer, 2010). Such wear and tear can manifest as low heart rate variability, slow blood pressure recovery, increased resting blood pressure level, and low natural killer cells (Brosschot, Pieper, & Thayer, 2005). Even changes in immune response can occur as a result of a predictable and fleeting occurrence such as exam stress experienced by university students (Kiecolt-Glaser et al., 2002).

Repressing emotions or being unaware of psychological conflicts can transform into somatic symptoms, a process known as somatization (Rief, 2013). Somatic symptoms are bodily symptoms such as pain, fatigue, insomnia, dizziness, and heart palpitations, which are separate from cognitive, emotional, and other types of symptoms (Kroenke, 2003). Individual differences can occur in severity, persistence, degree of impairment, distress level, and financial costs (direct and indirect) of somatic complaints (Kroenke, 2003). According to Kroenke (2003), approximately 80% of the general population has one or more symptoms per month with around 20-25% developing chronic or recurrent symptoms. Somatosensary amplification, or intensifying

the perception of symptoms, occurs when attention is focused on bodily sensations, health worries, and catastrophizing of the sensations (Rief, 2013). Regardless of whether a stressor is episodic acute or chronic, the total load of the stress response on the human body is most important. If the body is unsuccessful in coping with the stressor then continued activation may result in psychological friction (e.g. aggression, irritability, impatience), physical tension (e.g. migraines and chest pains), immune system compromise (e.g. wound healing, inflammation), disease or symptom exacerbation (e.g. cardiovascular disease, autoimmune disease, hypertension; diabetes mellitus), or affective disorders (e.g. major depression) (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Linden, Earle, Gerin, & Christenfeld, 1997; Stewart & France, 2001; Stewart, Janicki, & Kamarch, 2006; Stress, n.d.; Thayer & Brosschot, 2005).

A daily diary study of 69 teachers from Dutch primary and secondary schools illustrates the impact of worry on the stress-somatic health relationship (Verkuil, Brosschot, Meerman, and Thayer, 2012). Participants were randomly prompted five times a day for six days to report their somatic symptoms (Subjective Health Complaints inventory), worry episodes, duration of worry episode(s), intensity of worry episode(s), number of stressful events, and negative affect on a mobile handheld device (Verkuil et al., 2012). In addition to the daily diary measures, participants filled out baseline questionnaires about somatic complaints, trait worry, daily hassles for the previous two months, and trait negative affect (Verkuil et al., 2012). The results indicated that worry intensity mediated the effect of stressful events on somatic health complaints (Verkuil et al., 2012). These results were independent of daily negative affect and various biobehavioural variables (i.e. age, gender, education, alcohol, caffeine, alcohol, physical effort, trait worry, daily problems, sleep quality the previous night, and previous subjective health complaints) (Verkuil et al., 2012). The importance of including perseverative cognitions in research of health and

cognitive function is summed by Borkovec, Ray, and Stöber (1998, p. 562): "What we think affects how we feel, what we feel affects how we think, how we think and feel affect how we behave, how we behave affects how we feel, etc." Repetitive thoughts have an intimate connection to our physiological experiences and our cognitive functioning.

1.6 Impact of Stress and Cognitive Interference on Cognition

A recent publication discusses the source of cognitive decline as a multitude of biological and physical health influences on the body over time (MacDonald, DeCarlo, & Dixon, 2011). As research continues to address various influences on cognition, lifestyle factors such as stress are increasingly being considered as a key influence. In this vein, it is important to understand how stress and cognitive interference, a form of repetitive thought, can impact cognitive function.

1.6.1 Excess cortisol impacts cognitive function. As previously discussed, when receiving input about a threat, whether real or perceived, a cascade of activation between the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenocortical (HPA) axis releases catecholamines, including epinephrine and norepinephrine, and glucocorticoids (GCs), including cortisol, to stimulate energy for the flight-or-fight response (Sapolsky, Romero, & Munck, 2000). Of particular interest is where the GCs bind in the brain. During circadian troughs (night time phase) the GCs primarily bind to mineralcorticoid receptors (MR or Type 1) in the limbic system including the hippocampus, parahippocampal gyrus, entorhinal, and insural cortices (Lupien et al., 2005). However, during circadian peaks or stressful events these receptors become saturated so the excess distributes onto glucocorticoid receptors (GRs or Type II) into the hippocampus and cortical structure, especially the prefrontal cortex (Lupien et al, 2005). Therefore, in times of stress and daytime arousal the GCs saturate throughout the limbic system

(both MRs and GRs) and the prefrontal cortex (GRs). Understanding this differential binding affinity is insightful since both MRs and GRs are found in the hippocampus - a brain structure that can experience reduced volume, dendritic atrophy, and neuronal loss when exposed to chronically elevated glucocorticoid levels (Marin et al., 2011).

These stress responses can be protective in the short-term (i.e. fight or flight response) and damaging in the long-term (i.e. potential neurological damage) (McEwen & Seeman, 1999). Glucocorticoids (GCs), such as cortisol, impact cognition quickly during stress by reducing explicit memory at the time of incident, and slowly over time through risk of neuronal damage and/or atrophy (Seeman et al., 1997). Long-term outcomes include reduced excitability and neuronal damage and atrophy, especially in the hippocampus, resulting from an overactive HPA axis and over-stimulation of excitatory amino acid neurotransmitters (Lupien et al., 2005; McEwen & Seeman, 1999). Collectively, these outcomes can result in altered interpretation of stressors (negative appraisal) and lead to a positive feedback loop where HPA-axis overstimulation perpetuates more neuronal atrophy and changes in stress appraisal (Juster et al., 2010; Miller, Chen, & Zhou, 2007). In a population-based study of stress and cognition in older adults (65 years and older), lower cognitive function and accelerated cognitive decline were associated with higher levels or intensity of perceived stress (Aggarwal et al., 2014). These results were independent of personality or other measures of psychosocial functioning including depression, neuroticism, social engagement, and social network (Aggarwal et al., 2014). If enough hippocampal neuronal atrophy occurs then cognitive deficits may develop, impeding information recall and new memory formation (Galotti, Fernandes, Fugelsang, & Stolz, 2010; Lupien et al., 1998; Seeman et al., 1997). Specifically, GCs impact the hippocampus in the short- and long-term: quickly during stress, which reversibly affects explicit memory, and slowly over

time, which increases the risk of neuronal damage and/or atrophy (Seeman et al., 1997). The connection between elevated GC levels, impaired memory function, and hippocampal neuronal atrophy are summed in the 'glucocorticoid cascade hypothesis' (Sapolsky, Krey, & McEwean, 1986). This hypothesis unveiled key insights into GCs impairment of the hippocampus. Of particular interest to this thesis are:

- The hippocampus' ability to survive insults such as stroke or seizure is impaired by GCs.
- The hippocampal neurons are specifically vulnerable to the damaging effects of GCs. Even though other brain structures have MR receptor sites (e.g. pituitary, hypothalamus, prefrontal cortex) they do not appear to lose MR receptors.
- The likelihood of hippocampal damage resulting from an insult (e.g. stroke or seizure) is increased by a history of elevated GC levels before and after the incident.
- GCs present as a rapid and persistent danger to the hippocampus. The ability of GCs to impair exists even at non-elevated levels in the aging hippocampus (Sapolsky et al., 1986).

Altered cortisol output is also found within pathological aging. Researchers found a stepwise association between cortisol secretion and cognition; normal elderly controls secreted the least amount of cortisol, those with Alzheimer's disease had the highest levels, and those with mild cognitive impairment had levels in between the two (Arsenault-Lapierre, Chertkow, & Lupien, 2010).

Research has continuously demonstrated the negative impact of chronic stress on cognitive function, especially forms of cognition such as long-term memory and new memory formation. However, it is not just the experience of repeated or continuous stressors that can

impact cognition. The next section discusses how cognitive interference is similar to perseverative cognitions and how it, too, negatively impacts cognitive function

1.6.2 Cognitive interference and cognition. Cognitive interference (CI) is described as negative off-task, unwanted, or intruding thoughts or dialogue that are brief, sudden and unexpected and distract the individual from the current task (Clark, 2004 as cited in Stawski, Mogle, & Sliwinski, 2011; Coy, O'Brien, Tabaczynski, Northern, & Carels, 2011; Mikulincer, Babkoff, Caspy, & Weiss, 1990; Sarason, Pierce, & Sarason, 1996). Higher levels of cognitive interference are found in individuals who are stressed, manifesting in cognitions of "…stressful experiences, general worries, self and context…" (Stawski, Mogle, Sliwinski, 2013, p. 169). From a cognition perspective, the presence of a stressor triggers a call to action to do something to resolve the stress (Sarason, 1984). This response can elicit task-relevant thoughts, such as the case where an individual is presented with a self-selected challenge (e.g. finishing a triathlon), or task-irrelevant intrusive thoughts, such as when a challenge is imposed by the situational demands or constraints (e.g. taking on responsibilities because of others' inaction) (Sarason, 1984). These task-irrelevant thoughts, known as cognitive interference, may impact cognitive performance. They do this by acting as a cognitive overload where the exposure to and suppression of intrusive thoughts compete for attentional resources while processing information that requires controlled attention (Kahneman, 1973; Stawski, Mogle, & Sliwinski, 2013; Sliwinski, Smyth, Hofer, & Stawksi, 2006). To counter the overload, cognitive restructuring in anxious individuals can be used to strengthen task-oriented thinking rather than self-preoccupied thinking, as task-oriented thoughts are more adaptive as they are able to set aside irrelevant thoughts and focus on the task at hand (Meichenbaum & Butler, 1980).

The characteristics of cognitive interference are similar to perseverative cognitions, as are some of the ways it impacts cognition (Stawski, Mogle, & Sliwinski, 2011). Such research includes demonstration that intrusive thoughts mediate the relationship between neuroticism and cognitive function (Munoz, Sliwinski, Smyth, Almeida, & King, 2013). Neuroticism impairs the ability to cope with stress and is characterized by greater reactivity and a disposition to experience negative emotional states (Mohiyeddini, Bauer, & Semple, 2015; Munoz, Sliwinski, Smyth, Almeida, & King, 2013). In summary, cognitive interference can act similarly to perseverative cognitions in their repetitive and intrusive nature. Therefore, it is conceivable that perseverative cognitions such as worry may impact cognition in ways similar to cognitive interference. The intention of this brief background of stress, somatic health and cognition has been to provide context for the forthcoming analysis. The ubiquitous impact of stress within the body and mind has been discussed, as has its specific impacts via cortisol on somatic health and cognition. The concept of perseverative cognitions was discussed and they were identified as a key component of prolonged physiological activation of a stressor. Furthermore, cognitive interference was argued to be akin to perseverative cognitions, thereby bridging the possibility for perseverative cognitions to impact not just somatic health, but also cognitive function.

The purpose of this study is to evaluate a model by Brosschot, Gerin, and Thayer (2006) that proposed the mediation of worry on the relationship between stress and somatic health. Using publically accessible data from the Midlife in the United States (MIDUS) national longitudinal study of health and well-being, this study seeks to replicate that model and extend it to cognition to determine if worry also moderates the relationship between stress and cognition. As such, this study seeks to evaluate the degree to which perseverative cognition mediates the

relationship between stress and somatic health and whether perseverative cognition mediates the relationship between stress and cognition?

Method

2.1 Participants and Procedures

2.1.1 Midlife in the United States (MIDUS). This study conducted analyses using publically accessible data from the Midlife in the United States (MIDUS) data collection, a nationally representative longitudinal study of health and well-being. The MIDUS study currently consists of two waves of data collection (MIDUS I and MIDUS II). As discussed in sections 2.1.1.1, 2.1.1.2, and 2.1.1.3, each wave consists of individual projects that focus on specific topics. Each project pulls from the primary pool of participants from MIDUS I – Project 1 (the first project of MIDUS) with additional groups of participants added, such as the Milwaukee group and the twin study. For the purpose of the questions in this research the following data sets were used: MIDUS II – Project 1, MIDUS II – Project 2, and MIDUS II – Project 3.

2.1.1.1 MIDUS II – Project 1. MIDUS II – Project 1 (n = 4963) is from the second wave of Project 1, which looks at midlife development and age-related differences in physical and mental health and social responsibility by including behavioural, social, and psychological factors. Participants in this study completed a telephone interview approximately 30 minutes long and two 55-page self-administered questionnaires (SAQs) between January 2004 and September 2006. A total of 81% of participants who completed the telephone interview also completed both SAQs.

2.1.1.2 MIDUS II – Project 2 (National Study of Daily Experiences; NSDE). MIDUS II – Project 2 (n = 2022) includes participants who completed the first wave of Project 2 (n = 794), an expanded wave that completed MIDUS II - Project 1 but not MIDUS I - Project 2 (n = 1048),

and a group from Milwaukee who competed the baseline MIDUS Milwaukee study (n = 180). The purpose of this study was to examine day-to-day changes in physical and emotional reactivity to stressors and how sociodemographics, health, personality, and genetics influence these daily patterns.

2.1.1.3 MIDUS II – Project 3. The purpose of MIDUS II – Project 3 (n = 4512) was to explore the relationship between cognition and overall mental and physical health. Specifically, this project set out to:

1) Develop a nationally representative sample of midlife cognition to explore both the character and range of function; and

2) Investigate the relationship between cognitive function and biopsychosocial factors such as socio-economic status (SES), health status, and stressful life events.

Data was collected using the Brief Test of Adult Cognition by Telephone (BTACT), a telephone interview that delivers a comprehensive cognitive battery including measures of speed and reaction time. Cognitive assessments include word list recall (Rey Auditory-Verbal Learning Test), digits backward (WAIS III), category fluency (Drachman & Leavitt, 1972), red/green test, number series (Salthouse & Prill, 1987), backward counting, and short-delay word recall. The response rate for this test battery was over 86% of the MIDUS II participant pool.

3.1 Measures

All three MIDUS II projects used in this analysis contain a large number of measures, tests, and individual questions pertaining to areas such as physical and mental health, social responsibility, socioeconomic status, personality, genetics influence and cognition. A subset of these questions has been selected for this present study.

3.1.1 Stress severity. Stress severity rating from MIDUS II – Project 2 was used to measure participant stress. This stress severity rating was calculated by asking participants the questions Daily Inventory of Stressful Events with a follow up question of how stressful each question was for them (see below for detail). The stress severity rating ($M = 1.93$, $SD = 0.67$) was selected over total stressors ($M = 0.71$, $SD = 0.48$) due to a greater amount of variance between participants and to align with previous research using the same stress measure (Daily Inventory of Stressful Events, DISE) (Almeida, Neupert, Banks, & Serido, 2005; Almeida, Stawski, Cichy, 2011; Sliwinski, Almeida, Smyth, & Stawski, 2009). Although laboratory methods of assessment have better standardization, ambulatory methods of assessing stress reactivity offer better ecological validity through assessment of stress reactivity in daily life (Schlotz, 2013). Therefore, MIDUS II – Project 2 offers better ecological validity in the assessment of stress reactivity than common laboratory settings because it employs daily questioning in the natural environment of the participant. According to Schlotz (2013, p. 1892), "…stress reactivity is associated with sex, age, ethnicity, personality factors, pre-existing disease, and the presence or absence of chronic stress". The within-person average of stress severity ratings across all completed days was computed and labeled as mean daily stress severity. The mean daily stress severity was based on answers to the following questions:

- "Did you have an argument or disagreement with anyone since (this time/we spoke) yesterday?"
- "Since (this time/we spoke) yesterday, did anything happen that you could have argued about but you decided to let pass in order to avoid a disagreement?"
- "Since (this time/we spoke) yesterday, did anything happen at work or school (other than what you already mentioned) that most people would consider stressful?"

- "Since (this time/we spoke) yesterday, did anything happen at home (other than what you already mentioned that most people would consider stressful?"
- "Many people experience discrimination on the basis of such things as race, sex, or age. Did anything like this happen to you since (this time/we spoke) yesterday?
- "Since (this time/we spoke) yesterday, did anything happen to a close friend or relative (other than what you're already mentioned) that turned out to be stressful for you?"
- "Did anything else happen to you since (this time/we spoke) yesterday that people would consider stressful?"

Each question was followed up with the rating of "How stressful was this for you?"

3.1.2 Worry variables. Considering the availability of questions in the data sets, worry was chosen to represent the concept of perseverative cognition. In the absence of a standardized measure, individual worry variables were chosen from MIDUS II – Project 1 that provide assessment of both frequency and duration of worry relative to the individual and to others as well as self-identification as a worrier. The worry variables were recoded so that high ratings such as "more", "a lot more", "every day", "all day long", and "a lot" corresponded with high instead of low numerical values. The following is a list of the worry variables and the recoding process:

- Frequency of worrying: "Thinking about the PAST 12 MONTHS, did you worry: every day, just about every day, most days, about half the days, or less than half the days?"
- As a follow up to the previous question - how long worry lasts on days you worry: "On days you worry, does the worry usually last all day long, most of the day, about half the day, or less than half the day?"

- Worrying describes you how well: “Please indicate how well each of the following describes you: worrying.”

3.1.3 Somatic health measures. Somatic health was identified by two different composite measurements: somatic amplification and the number of physical symptoms. The somatic amplification composite score is a mean score of five items assessed in the Self-Administered Questionnaire. These five questions from MIDUS II – Project 1 ask the following questions: ‘I hate to be too hot or too cold’, ‘I am quick to sense hunger contractions in my stomach’, ‘sudden loud noises really bother me’, ‘I am often aware of various things happening in my body’ and ‘I have a low tolerance for pain’. Response options consisted of 1 = not at all true, 2 = a little bit true, 3 = moderately true, 4 = extremely true. The questions were developed based on research by Barskey, Goodson, Lane, and Cleary (1988) that demonstrated self-reported somatosensory amplification as distinct from depression, anxiety, and hostility in predicting symptomology, overall discomfort, and disability.

The physical symptoms composite from MIDUS II – Project 2 was comprised of a variety of questions regarding physiological and psychological health symptoms. These 28 symptoms include: headache, backache, muscle soreness, fatigue, joint pain, muscle weakness, cough, sore throat, fever, chills, other cold or flu symptoms, nausea, allergies, diarrhea, constipation, poor appetite, other stomach problems, chest pain, dizziness, shortness of breath, menstrual related symptoms, hot flashes or flushes, any other physical symptoms, skin related symptoms, eye related symptoms, ear related symptoms, teeth related symptoms, leg or foot related symptoms. This composite score is similar to the Somatic Health Complaints inventory used in previous research on the effects of stress and worry on somatic health (Eriksen, Ihlebæk, & Ursin, 1999; Verkuil, Brosschot, Meerman, & Thayer, 2012). The variable used in this

analysis represented the median number of physical symptoms per day across the eight consecutive days of the study.

3.1.4 Cognition measures. Cognitive measures were included from the MIDUS II – Project 3 data collection, which tested participants using the Brief Test of Adult Cognition by Telephone (BTACT). The BTACT is a telephone assessment method that includes six testing subsets indexing key domains of cognitive function (Lachman, Agrigoroaei, Tun, & Weaver, 2014). Exploratory and confirmatory factor analysis revealed two factors interpreted as executive function and episodic memory (Lachman et al., 2014).

3.1.4.1 Executive function. As a top-down process, executive function is when information is modified from automatic processes such as sensory input, memories, habituated skills, and motor memory (Miller & Wallis, 2009). Executive function is used when an input needs to be modified to achieve a goal and manifests intentional behaviour, self-awareness, planning, response inhibition, response monitoring and attention during dual tasks (Yogev-Seligmann, Hausdorff, & Giladi, 2008). This domain of cognition was assessed using a composite score comprised of z-scores for digits backward, category fluency, number series, backward counting, and task-switching.

3.1.4.1.1 Digits backward. A digits backward task from WAIS III (Weschler, 1997) required participants to immediately repeat a string of numbers backwards to the researcher (Tun & Lachman, 2009). Participants were given two iterations of successively longer strings of numbers beginning with two numbers and ending with eight numbers.

3.1.4.1.2 Category fluency. Category fluency, adapted from Drachman and Leavitt (1972), asked participants to list as many items as possible within 60 seconds that relate to a category, in this case "animals" (Tun & Lachman, 2009).

3.1.4.1.3 Number series. Using Salthouse and Prill's (1987) number series task, participants were asked to provide the next number in a string of increasing or decreasing numbers (Tun & Lachman, 2009). Participants were provided with the first five numbers in the series and were asked to provide the next value. A total of 2.5 minutes was allotted to this task.

3.1.4.1.4 Backward counting. Participants were given 30 seconds to count backwards as fast as possible from 100 (Tun and Lachman, 2009).

3.1.4.1.5 Task switching. Participants were first instructed to respond "STOP" when the interviewer said "RED" and "GO" when the interviewer said "GREEN". After 20 trials the participant was instructed to do the opposite: say "GO" when the interviewer said "RED" and "STOP" when the interviewer said "GREEN".

3.1.4.2 Episodic memory. Episodic memory is a past-oriented memory system, built atop other systems such as semantic memory, which enables an individual to consciously bring to mind past experiences (Tulving, 2002). Developing late and deteriorating early, episodic memory coexists with the concept of self, autonoetic awareness (i.e. awareness of subjective time when an event occurred), and subjectively sensed time (2002).

3.1.4.2.1 Word list recall – immediate and delayed. For immediate recall, participants were verbally provided with a list of 15 words and then asked to immediately repeat as many words as possible in whatever order they chose within 90 seconds. Delayed recall followed approximately 10.75 minutes later after digits backward, category fluency, red/green test,

number series, and backward counting tasks. Participants were given 40 seconds to recall as many words as possible in any order from the original list.

3.1.5 Covariates. Age, gender, socioeconomic status (i.e. education and household income), and conditions were included as covariates. Gender was coded as a dichotomous variable with male as the reference group (i.e. male = 0, female = 1).

3.1.5.1 Socioeconomic Status. Research has demonstrated a positive correlation between socioeconomic status and cognition, such that lower socioeconomic status, including education and household income, is associated with cognitive decline (Tschanz et al., 2013) and poorer working memory (Evans and Schamberg, 2009) and executive function (Zhang, Gale, Erickson, Brown, Woody, & Hedges, 2015).

3.1.5.1.1 Education. MIDUS 2 – Project 1 participant education level was categorized in the following manner: 1 = some grade school to some high school, 2 = GED (General Education Diploma) to graduated high school, 3 = some college (no bachelors degree), and 4 = graduated college to doctorate or professional degree. Inclusion of education as a covariate was based on research demonstrating a positive association between education and cognition, including global cognition, and episodic memory (Jefferson et al., 2011). Research by Tschanz and colleagues (2013) also demonstrated education to be a moderator for the relationship between stressful life events and cognitive decline in late life such that those with less education and more stressful life events experienced faster cognitive decline in late life.

3.1.5.1.2 Household income. MIDUS II – Project 1 household total income, including wage, pension, social security income, and government assistance from the participant and a spouse (if applicable) was controlled for based on research demonstrating an association between

current economic status on cognitive function (Zhang, Gale, Erickson, Brown, Woody, & Hedges, 2015).

3.1.5.2 Chronic health condition. The number of diagnosed chronic health conditions (CHCs) was included to determine if the relationship between stress and cognition or somatic health remained even after controlling a potential source of chronic stress (i.e. if stressor severity, or worry, or the mediation is significant even in populations without diagnosed CHCs). Chronic health conditions (over the past 12 months) were defined in the MIDUS II – Project 1 data set as the following: asthma/bronchitis/emphysema, Tuberculosis, other lung problems, joint/bone diseases, sciatica/lumbago/backache, persistent skin trouble, thyroid disease, hay fever, stomach troubles, urinary/bladder problems, constipation, gall bladder trouble, persistent foot troubles, varicose veins, AIDS/HIV, lupus/autoimmune disorder, persistent gum/mouth troubles, persistent teeth troubles, high blood pressure/hypertension, anxiety/depression, alcohol/drug problem, migraine headaches, chronic sleep problems, diabetes/high blood sugar, neurological disorder, stroke, ulcer, hernia, piles/haemorrhoids, and swallowing problems.

4.1 Analysis Strategy and Process

To replicate previous work by Brosschot and colleagues, mediation analysis was used to determine if stress impacts somatic complaints or cognition via worry (see Equation 1). Mediation asks the question of how the predictor X influences the outcome Y by evaluating the influence of additional variables on the outcome, thereby going beyond establishing a relationship (i.e. correlation) by focusing on understanding the process (Hayes, 2012). See Equations 1 to 3 for the mediation calculation when including a covariate (MacKinnon, Kisbu-Sakarya, & Gottschall, 2013). Note that this study included four covariates. Figure 1 illustrates

the models that were tested. For clarity, the visual representation of the model excludes the covariates. For Equation 1, *i* is the intercept, *c* is the coefficient that demonstrates how strongly X predicts Y, X is the predictor, *d* is the covariate coefficient that shows the relation of the covariate to Y while controlling for other independent variables, and C is the covariate (MacKinnon et al., 2013). In Equation 2, the mediator is included as an additional predictor of Y where the coefficient *d'* corresponds to the relation of the covariate to Y (MacKinnon et al., 2013). In Equation 3, *a* is the coefficient that relates X to the mediator and the *f* coefficient relates the covariate to the mediator while controlling for X (MacKinnon et al., 2013). It is important to note that in this model all covariates are regressed on the outcome at every pathway.

$$Y = i_1 + cX + dC + e_1 \quad (1)$$

$$Y = i_2 + c'X + bM + d'C + e_2 \quad (2)$$

$$M = i_3 + aX + fC + e_3 \quad (3)$$

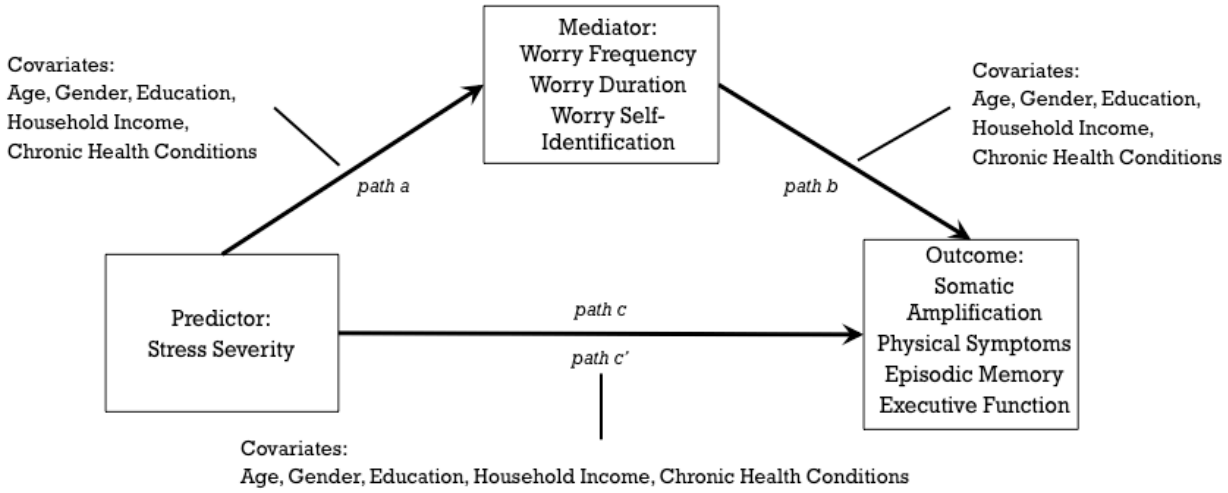

Figure 1. Detailed representation of the mediation models.

The analysis was run in SPSS 23.0 using the PROCESS computational tool, which allows for statistical adjustment of covariates, for all mediation analyses (Hayes, 2012; Hayes, 2016). A

total of 12 models were run using SPSS 23.0: the mediation of worry on the relationship between stress and somatic amplification, and the mediation of worry on the relationship between stress and cognition (see Figure 2).

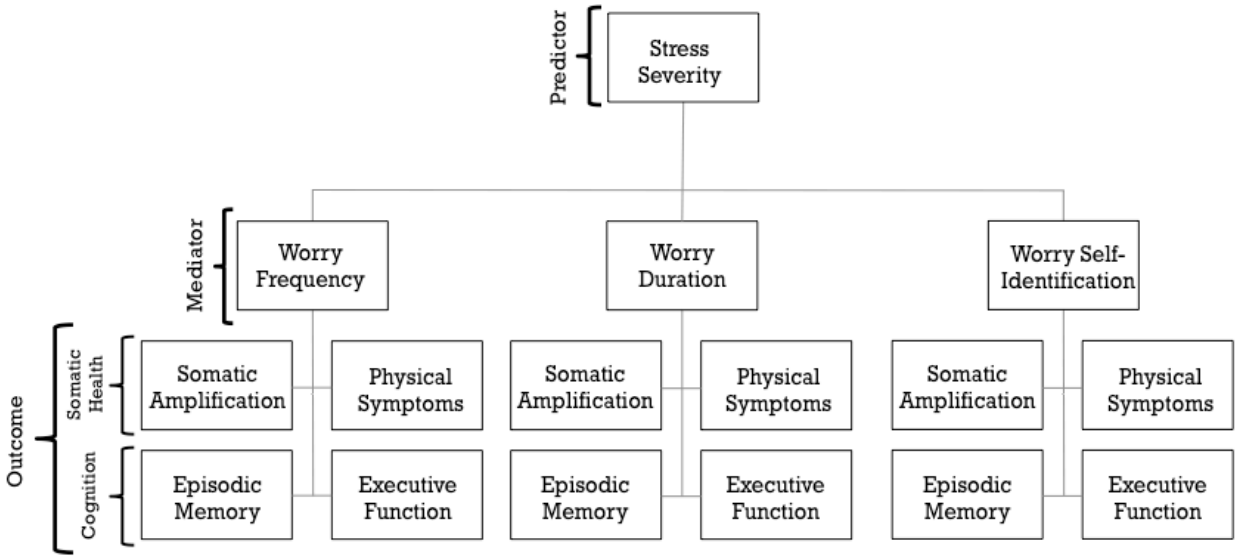

Figure 2. Visual representation of the 12 mediation models examined in this study.

Each model was run while adjusting for covariates (age, gender, education, household income, number of chronic health conditions). The significance of a mediating effect was evaluated using the test of joint significance (if path a and path b are significant), the Sobel test and bootstrapping (10000 samples) of the indirect effect of X on Y with 95% confidence intervals, which tests whether the indirect effect (path ab, or X → M → Y) is different from zero (Fairchild & MacKinnon, 2009; Hayes, 2013; Kenny, 2015). Note that the Sobel test was included for convention; however, this method requires a normal distribution, which does not fit with the commonly asymmetric product of paths a and b (Hayes, 2009). Kappa squared (κ^2) is commonly used in mediation as a measure of effect size; however, this method does not transfer to models that include covariates and therefore is not provided in the results section (Fairchild & MacKinnon, 2009; Hayes, n.d; Preacher & Kelly, 2011). Instead, PROCESS provides the user

with an alternative index of the variance explained by the indirect effect (P_M, or mediation ratio, or ratio of indirect effect to the total effect; see Equation 4), and the ratio of indirect effect to the direct effect (R_M; see Equation 5) (Hayes, n.d.).

$$P_M = \frac{ab}{c'+ab} \quad (4)$$

$$R_M = \frac{ab}{c'} \quad (5)$$

However, these methods do have their drawbacks. For example, when looking at the P_M it is important to ensure that $n > 500$ and to take into consideration the context of the effect size when interpreting the impact; a large effect size in a small population may be less meaningful then a smaller effect size in a larger sample (Preacher & Kelly, 2011). R_M does not provide any new information, but rather bundles the P_M information into a new expression: P_M expresses the proportion of the total effect that is mediated whereas R_M expresses how much larger the indirect effect is versus the direct effect and whether it is statistically significant via confidence intervals (Preacher & Kelly, 2011). R_M has been found to be unstable with $n < 5000$; since no model in this analysis has $n > 5000$ this value will not be used (Preacher & Kelly, 2011). Hayes (2013) also cautions against the use of P_M and R_M as measures of effect size due to their large sampling variance as witnessed in the bootstrapping standard error. However, since the analyses used in this study have large sample sizes it is permissible to use as long as "…the total effect [path c] is larger than the indirect effect [path c'] and of the same sign" (Hayes, 2013, p. 193). When determining the presence or absence of mediation the bootstrapping of the indirect effect with 95% confidence intervals was the deciding factor. This was based on it being a more valid and powerful method of testing mediation because, unlike the Sobel test, it does not require the

assumption of a normal distribution of the product of path a and b (Hayes, 2009; MacKinnon, Lockwood, & Williams, 2004; Williams & MacKinnon, 2008).

Since the timeline for data collection can be lengthy and expensive, secondary analysis such as that used for this thesis offers an alternative by asking questions of a previously collected data set (Dunn, 2010). Using secondary analysis is a cost- and time-effective method to answer the replication and novel hypotheses herein hence a primary strength of using the MIDUS data sets is the access to multi-disciplinary longitudinal data. Key challenges of using previously collected data are the lack of control in the questions being asked, the measures used, and the protocol for the data collection as well as risk of sampling bias where the study sample characteristics do not reflect the target population (Cheng & Phillips, 2014).

4.1.1 Dataset preparation. Prior to running the analyses the data were merged into one data set, scanned for consistency in response coding, and checked for outliers and the standard assumptions of regression. Variables of interest from MIDUS II – Project 1, MIDUS II – Project 2, and MIDUS II – Project 3 were merged into a single data set. MIDUS II – Project 2 data is a repeated measures design; therefore, prior to merging, the variables (i.e. stress severity and physical symptoms) were isolated into a new data set, restructured from long to wide format, and each transformed into a new variable of the mean value across the study (possible range of 1 – 8 days of data). Once a single data set was created the following variables were recoded for ease of interpretation: gender, education, total household income, number of chronic health conditions, and all worry variables.

Participant gender (B1PRSEX) was recoded (B1PRSEX_R) from 1 = male and 2 = female to 0 = male and 1 = female. Note that although the variable reads as sex, the question asked pertained to gender. The education variable for MIDUS 2 – Project 1 (B1PB33) was

recoded (B1PB33_R) using the same categories from MIDUS 1 – Project 1 (A1PEDUCP). This was done to decrease the number of categories from 12 to four for ease of interpretation. The recoding is documented in Table 1.

Total household income was highly skewed across the models (1.577 – 1.645) due to a group of participants earning $102,500 or more annually. Following the method used by previous research, total household income was recoded into the following quintiles: $0 to < $21,000, $21,000 to < $40,250, $40,250 to < $63,750, $63,750 to < $102,750, $102,750 or greater (Boehm, Chen, Williams, Ryff, Kubzansky, 2015). This reduced the skew (- .283 to - .215). Number of chronic health conditions was highly skewed across the models (1.221 – 1.492) due to a group of participants reporting more than 10 chronic health conditions. These participants (10 or more) were grouped together such that the values included 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 or more. This reduced the skew to .929 to 1.220.

Table 1. Recoding Process for Education Categories

Original Education Coding (B1PB33)	Recoded Education Coding (B1PB33_R)
1 = no school/some grade school (1-6)	1 = some grade school to some high school
2 = eighth grade/junior high school (7-8)	
3 = some high school (9-12, no diploma/no GED)	
4 = GED	2 = GED to graduated high school
5 = graduated from high school	
6 = 1-2 years of college, no degree yet	3 = some college (no bachelor's degree)
7 = 3 or more years of college, no degree yet	

8 = graduated from a two-year college or vocation school	4 = graduated college to doctorate or professional degree
9 = graduated from a four- or five-year college, or bachelor's degree	
10 = some graduate school	
11 = master's degree	
12 = PhD, ED.D., MD, DDS, LLB, LLD, JD, or other professional designation	

The following worry variables for MIDUS 2 – Project 2 were recoded so that high ratings such as "more", "a lot more", "every day", "all day long", and "a lot" corresponded with high instead of low numerical values:

- B1PA84 - Frequency of worrying: "Thinking about the PAST 12 MONTHS, did you worry: every day, just about every day, most days, about half the days, or less than half the days?"

  Original response: 1 = every day, 2 = just about every day, 3 = most days, 4 = about half the days, 5 = less than half the days

  Recoded: 1 = less than half the days, 2 = about half the days, 3 = most days, 4 = just about every day, 5 = every day
- As a follow up to the previous question, B1PA84a - How long worry lasts on days you worry: "On days you worry, does the worry usually last all day long, most of the day, about half the day, or less than half the day?"

  Original response: 1 = all day long, 2 = most of the day, 3 = about half, 4 = less than half the day

Recoded: 1 = less than half the day, 2 = about half, 3 = most of the day, 4 = all day long

- B1SE6H = Worrying describes you how well: "Please indicate how well each of the following describes you: worrying."

  Original response: 1 = a lot, 2 = somewhat, 3 = a little, 4 = not at all

  Recoded: 1 = not at all, 2 = a little, 3 = somewhat, 4 = a lot

The stress severity and physical symptom variables both come from the daily diary MIDUS II – Project 2. In this data set stress severity represented as a mean value for the stress severity questions of each day. In order to merge this variable with the data set a composite variable needed to be created. To do a summation variable across all days was created and descriptive statistics were run to determine if the mean or median should be used. The skew was not strong (.878) so an overall mean stress severity variable across the daily means was created. For physical symptoms the daily value was the total number of physical symptoms reported that day. Daily total physical symptoms as well as cumulative physical symptoms across days were highly skewed (1.357 – 3.203) so the median number of physical symptoms across all days was used in the mediation analyses (von Hippel, 2005).

Each variable was checked for univariate, bivariate, and multivariate outliers and for compliance with the OLS regression assumptions of normality, linearity, and homoscedasticity. Since each model had a unique subsample a dummy variable was created to code all participants with complete or incomplete data for each unique model. Cases were then selected for complete data before outliers and assumptions were checked. This ensured accurate screening since the mediation models use listwise deletion. Univariate outlier checks included the range of values (minimum and maximum) and z-scores. Scatterplot matrixes were used to check for bivariate outliers for each model. Multivariate outliers were checked using Mahalanobis' distance and Q-

Q plots. Participants flagged as outliers were individually examined, but did not appear to be outside of the range of anticipated values. Normality was assessed at the univariate level by histogram, Q-Q plot, Kolmogorov-Smirnov test, skew, kurtosis. There was a positive skew for household income, number of chronic health conditions, and mean physical symptoms, which was not deemed unusual. Education had a slight negative skew for most models indicating that overall the samples had mostly completed high school or gone on to post-secondary school. Most models had a positive skew in how often participants worried. Normality was assessed by normal P-P plot of regression standardized residuals. Linearity was assessed using a bivariate scatterplot matrix for each model where an ellipsoid shape represented a linear relationship. None of the models represented an ellipsoid shape. Homoscedasticity was tested using a residual scatterplot, which plots the regression standardized residual (difference between the actual dependent variable scores and the predicted dependent variable scores on the Y-axis) versus the regression standardized predicted value. None of the models satisfied this assumption. The result is a potentially weakened, but not invalidated, analysis (Tabachnick & Fidell, 2013). In the end, all outliers were retained, as they are important in understanding those experiencing more CHCs, stress severity, and worry and how this impacts the outcomes.

The percentage of missing data varied by the data set and type of measure when looking at all variables used in the models. The largest percentages of missing data were for length of worry (72.0%), household income and number of chronic health conditions (70.8%), mean stress severity (65.8%), and median number of physical symptoms (61.9%). Missing data for all variables is likely due to inapplicability or non-disclosure. In addition to this reasoning, length of worry is likely due to being a follow up question to the other worry variable, how often the participant worries. If the participant did not indicate that they worry often then they may have

indicated that the length of worry was non-applicable. Stress severity and physical symptoms are from the MIDUS II – Project 2 National Study of Daily Experiences. Missing data from these variables may also be due to non-participation in the daily diary study. It is assumed that any missing data from these participants is missing at random.

Results

Each model tested had different subsample demographics in comparison to MIDUS II – Project 1, the largest of the data sets used in this analysis. Table 2, Table 3, and Table 4 present the demographic information for the models tested in comparison to the overall MIDUS II – Project 1 demographics, which closely resemble each other. Overall, the majority of the sample was female (54.9% - 66.5%) with a mean age of 52.58 – 55.91 years and 2.38 – 3.05 chronic health conditions. On average, participants had some college education and a household income of between $40,250 and $63,750. Of importance to note is that previous research that probed the longitudinal retention of participants across MIDUS I to MIDUS II demonstrated a higher retention amongst white females, those who are married, and individuals with more education (Radler and Ryff, 2010). The correlation and results for all 12 mediation models are presented below. Each mediator (three in total) is tested in four different models with predictor as mean stress severity and the outcome as somatic amplification, number of physical symptoms, episodic memory, or executive function. All models were adjusted for the effects of age, gender, level of education attained, total household income, and number of chronic health conditions on the predictor, mediator, and outcome variable. As such, each unstandardized coefficient is independent of these covariate effects.

5.1 Correlations Among All Variables

A correlation matrix of all variables in the models was run prior to the mediation analyses. This matrix demonstrates to what degree the variables are related. If the correlation is very high then it may indicate multi-collinearity or singularity, where the variables are perfectly correlated (Field, 2009). Of particular interest are the correlations between the worry variables,

which are intended to measure some aspect of worry; therefore, the variables should show moderate correlations ($r \sim .30$) (Field, 2009). See Table 5 for the results using listwise deletion.

5.2 Mediating Effect of Worry Frequency

5.2.1 Stress severity and somatic amplification. Using regression analysis to test the hypothesis that worry frequency mediates the effect of stress severity on somatic amplification, the results show that mean stress severity is a significant predictor of worry frequency ($b = .230$, $SE = .045$, $p < .05$) such that for every one unit increase in mean stress severity there is an increase of .230 units in worry frequency. Worry frequency is a significant predictor of somatic amplification ($b = .042$, $SE = .012$, $p < .001$) such that one unit increase in worry frequency leads to .042 units increase in somatic amplification. Both path c' ($b = .077$, $SE = .020$, $p < .001$) and path c ($b = .087$, $SE = .020$, $p < .001$) are significant indicating that in both models stress severity has an influence on somatic amplification. See Figure 3 and Table 6 for details of the results. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = .004, UL = .017), the Sobel test ($Z = 0.01$, $p < .01$) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of indirect to total effect of stress severity on somatic amplification (P_M; LL = .044, UL = .257) adds further evidence of the indirect effect being different from zero since the confidence interval does not cross zero and path c is larger than c' with the same direction of sign (Hayes, 2013). Together these tests show that mediation did occur and that the third variable, frequency of worry, is part of the process in how stress severity impacts somatic amplification.

Table 1. Participant Demographics for Models with Worry Frequency as the Mediator

	MIDUS II – Project 1	S_Amp	P_Sym	EM	EF
	(*n* = 3849) M (SD)	(*n* = 1533) M (SD)	(*n* = 1542) M (SD)	(*n* = 1505) M (SD)	(*n* = 1506) M (SD)
Age	55.98 (12.30)	55.70 (11.94)	55.75 (11.98)	55.83 (11.98)	55.84 (11.99)
Education	3.09 (0.98)	3.18 (0.92)	3.18 (0.92)	3.18 (0.93)	3.18 (0.93)
HH Income	3.15 (1.40)	3.24 (1.36)	3.23 (1.36)	3.23 (1.35)	3.23 (1.35)
CHCs	2.38 (2.26)	2.38 (2.24)	2.39 (2.25)	2.40 (2.25)	2.40 (2.25)
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Gender					
Female	2115 (54.9)	876 (57.1)	882 (57.2)	858 (57.0)	859 (57.0)
Male	1734 (45.1)	657 (42.9)	660 (42.8)	647 (43.0)	647 (43.0)

Note: S_Amp is somatic amplification. P_Sym is median physical symptoms. EM is episodic memory. EF is executive function. HH Income is household income. CHCs is number of chronic health conditions.

Table 2. Participant Demographics for Models with Worry Duration as the Mediator

	MIDUS II – Project 1	S_Amp	P_Sym	EM	EF
	(*n* = 3849) M (SD)	(*n* = 455) M (SD)	(*n* = 459) M (SD)	(*n* = 447) M (SD)	(*n* = 448) M (SD)
Age	55.98 (12.30)	52.58 (10.98)	52.69 (11.13)	52.74 (11.13)	52.78 (11.14)
Education	3.09 (0.98)	3.11 (0.95)	3.11 (0.95)	3.12 (0.95)	3.11 (0.96)
HH Income	3.15 (1.40)	3.25 (1.43)	3.24 (1.43)	3.24 (1.42)	3.24 (1.42)
CHCs	2.38 (2.26)	3.04 (2.62)	3.03 (2.61)	3.05 (2.63)	3.05 (2.63)
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Gender					
Female	2115 (54.9)	301 (66.2)	305 (66.4)	293 (65.5)	294 (65.6)
Male	1734 (45.1)	154 (33.8)	154 (33.6)	154 (34.5)	154 (34.4)

Note: S_Amp is somatic amplification. P_Sym is median physical symptoms. EM is episodic memory. EF is executive function. HH Income is household income. CHCs is number of chronic health conditions.

Table 3. Participant Demographics for Models with Worry Self-Identification as the Mediator

	MIDUS II – Project 1	S_Amp	P_Sym	EM	EF
	(*n* = 3849) M (SD)	(*n* = 1539) M (SD)	(*n* = 1547) M (SD)	(*n* = 1510) M (SD)	(*n* = 1511) M (SD)
Age	55.98 (12.30)	55.79 (11.99)	55.83 (12.02)	55.90 (12.02)	55.91 (12.03)
Education	3.09 (0.98)	3.18 (0.92)	3.18 (0.92)	3.18 (0.92)	3.18 (0.93)
HH Income	3.15 (1.40)	3.23 (1.36)	3.23 (1.36)	3.23 (1.35)	3.23 (1.35)
CHCs	2.38 (2.26)	2.39 (2.25)	2.40 (2.25)	2.40 (2.26)	2.41 (2.26)
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Gender					
Female	2115 (54.9)	881 (57.2)	886 (57.3)	858 (57.0)	863 (57.1)
Male	1734 (45.1)	658 (42.8)	661 (42.7)	647 (43.0)	648 (42.9)

Note: S_Amp refers to somatic amplification. P_Sym refers to median physical symptoms. EM refers to episodic memory. EF refers to executive function. HH Income refers to household income. CHCs refers to number of chronic health conditions.

Table 4. Correlations Among All Variables Used in the Twelve Models (N = 437).

	Age	Gender	Educ	HHI	CHC	SSev	Frequ	Dura	Self_I	S_Amp	P_Sym	EM	EF
Age	1												
Gender	0.018	1											
Educ	-.0178**	-0.140**	1										
HHI	-0.286**	-0.118*	0.333**	1									
CHC	0.242**	0.193**	-0.180**	-0.292**	1								
SSev	-0.029	0.258**	-0.151**	-0.133**	0.209**	1							
Frequ	-0.139**	0.063	0.014	0.016	0.088	0.157**	1						
Dura	-0.009	0.134**	-0.048	-0.136**	0.321**	0.268**	0.201**	1					
Self-I	-0.137**	0.169**	-0.030	0.040	0.153**	0.247**	0.337**	0.223**	1				
S_Amp	0.024	0.211**	-0.033	-0.118*	0.257**	0.171**	0.087	0.208**	0.257**	1			
P_Sym	0.100*	0.112*	-0.168**	-0.192**	0.505**	0.197**	0.084	0.193**	0.110*	0.209**	1		
EM	-0.265**	0.177**	0.166**	0.171**	-0.098*	-0.008	0.101*	-0.087	0.037	-0.001	-0.036	1	
EF	-0.364**	-0.207**	0.393**	0.304**	-0.248**	-.0196**	0.024	-0.180**	-0.009	-0.144**	-0.183**	0.356**	1

Note: HHI is household income. CHCs is number of chronic health conditions. SSev is stress severity. Frequ is worry frequency. Dura is worry duration. Self_I is worry self-identification. S_Amp is somatic amplification. P_Sym is median physical symptoms. EM is episodic memory. EF is executive function.
* $p < .05$, ** $p < .001$

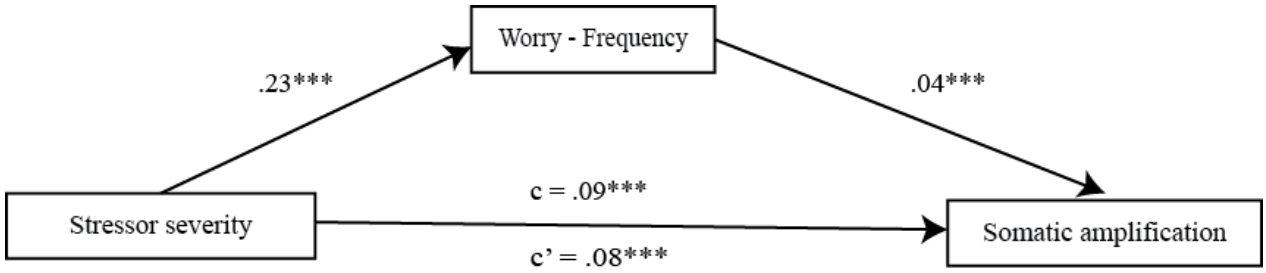

Figure 3. Unstandardized regression coefficients for the relationship between stressor severity and somatic amplification as mediated by worry frequency.
$^{**}p < .01$, $^{***}p < .001$

Table 1. Regression Results for the Mediation of the Effect of Stress Severity on Somatic Amplification by Worry Frequency

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	2.284	.102	< .001	2.084	2.484
SSev	.087	.020	< .001	.047	.127
Age	-.003	.001	< .01	-.005	-.001
Gender	.147	.027	< .001	.095	.200
Educ	-.025	.014	> .05	-.053	.003
HHI	.004	.010	> .05	-.017	.024
CHC	.049	.006	< .001	.037	.060
$R^2_{Y,X}$	.106				
Path a (X → M)					
Intercept	2.108	.226	< .001	1.664	2.551
SSev	.230	.045	< .001	.142	.319
Age	-.020	.003	< .001	-.025	-.015
Gender	.106	.059	> .05	-.011	.222
Educ	-.032	.032	> .05	-.094	.030
HHI	.005	.023	> .05	-.040	.050
CHC	.099	.013	< .001	.073	.125
$R^2_{M,X}$	.105				
Path b (M → Y)					
Intercept	2.196	.104	< .001	1.991	2.400

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Frequ	.042	.012	< .001	.020	.065
SSev	.077	.020	< .001	.037	.117
Age	-.002	.001	> .05	-.004	.0001
Gender	.143	.027	< .001	.091	.195
Educ	-.024	.014	> .05	-.051	.004
HHI	.004	.010	> .05	-.017	.024
CHC	.045	.006	< .001	.033	.056
$R^2_{Y,MX}$	.113				
Path c' (X → Y; a X b)	.077	.020	< .001	.037	.117
Indirect effect of X on Y	.010	.003	-	.004	.017
P_M	.112	.055	-	.044	.257
	Effect	SE	Z	*p*	
Sobel	.010	.003	2.94	< .01	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 2. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Frequ is worry frequency.

5.2.2 Stress severity and physical symptoms. In testing the hypothesis that worry frequency mediates the effect of stress severity on physical symptoms, the results show that mean stress severity is a significant predictor of worry frequency (b = .229, SE = .045, p < .001) with one unit increase in mean stress severity leading to .229 units increase in worry frequency. Worry frequency is a significant predictor of physical symptoms (b = .165, SE = .039, p < .001) with one unit increase in worry frequency resulting in an increase of .165 units in physical symptoms. Both path c' (b = .196, SE = .069, p < .01) and path c (b = .233, SE = .069, p < .001) are significant indicating that in both models stress severity has an influence on somatic amplification. See Figure 4 and Table 7 or analysis details. The significance of the mediation

was tested using bias-corrected bootstrapped confidence interval for the indirect effect (c') using 1o000 samples (LL = .019, UL = .064) and the Sobel test (Z = 0.04, p < .01) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of indirect to total effect of stress severity on physical symptoms (PM; LL = .071, UL = .411) adds further evidence of the indirect effect being different from zero since the confidence intervals do not cross zero and path c is larger than c' with the same direction of sign (Hayes, 2013). Together these tests show that mediation did occur and that the third variable, frequency of worry, is part of the process in how stress severity impacts the number of physical symptoms experienced by an individual.

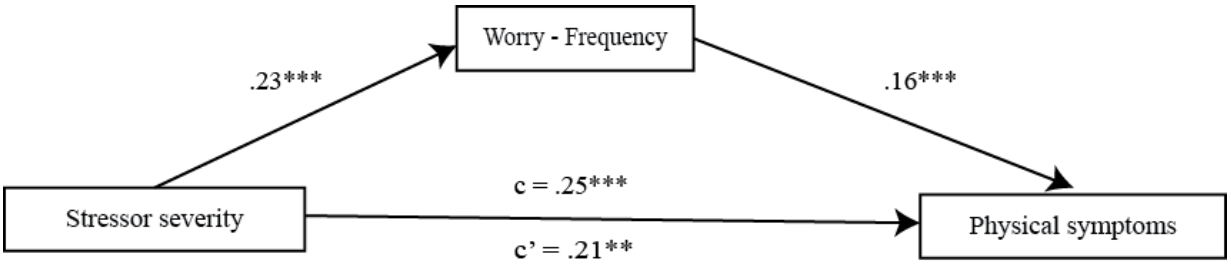

Figure 4. Unstandardized regression coefficients for the relationship between stressor severity and median physical symptoms as mediated by worry frequency.
*$p < .05$, ** $p < .01$, ***$p < .001$

Table 7. Regression Results for the Mediation of the Effect of Stress Severity on Physical Symptoms by Worry Frequency

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	1.481	.345	< .001	.804	2.160
SSev	.233	.069	< .001	.098	.368
Age	-.009	.004	< .05	-.017	-.002
Gender	.078	.091	> .05	-.100	.256
Educ	-.089	.048	> .05	-.184	.006
HHI	-.100	.035	< .01	-.168	-.030
CHC	.372	.020	< .001	.332	.411
$R^2_{Y,X}$	.234				
Path a (X → M)					
Intercept	2.084	.225	< .001	1.643	2.525
SSev	.229	.045	< .001	.141	.317
Age	-.019	.003	< .001	-.024	-.014
Gender	.112	.059	> .05	-.004	.228
Educ	-.032	.032	> .05	-.094	.030
HHI	.005	.023	> .05	-.040	.050
CHC	.096	.013	< .001	.071	.122
$R^2_{M,X}$	.103				
Path b (M → Y)					
Intercept	1.140	.343	< .01	.449	1.832
Frequ	.164	.039	< .001	.087	.240
SSev	.196	.069	< .01	.060	.331
Age	-.006	.004	> .05	-.014	.002
Gender	.059	.090	> .05	-.118	.236
Educ	-.084	.048	> .05	-.178	.011
HHI	-.100	.035	< .01	-.169	-.031
CHC	.356	.020	< .001	.316	.396
$R^2_{Y,MX}$	.242				
Path c' (X → Y; a X b)	.196	.069	< .01	.060	.331

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Indirect effect of X on Y	.038	.011	-	.019	.064
P_M	.161	.107	-	.071	.411
	Effect	SE	Z	*p*	
Sobel	.038	.012	3.208	< .01	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 3. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Frequ is worry frequency.

5.2.3 Stress severity and episodic memory. The mediation analysis rejects the hypothesis that worry frequency mediates the effect of stress severity on episodic memory. The results show that mean stress severity is a significant predictor of worry frequency (b = .233, SE = .046, p < .001), indicating that one unit increase in mean stress severity leads to .233 units increase in frequency worry. Conversely, worry frequency is not a significant predictor of episodic memory (b = -.006, SE = .021, p > .05). Both path c' (b = -.054, SE = .037, p > .05) and path c (b = -.056, SE = .036, p > .05) are non-significant; therefore no mediation has occurred. See Figure 5 and Table 8 for analysis details. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (c') using 10000 samples (LL = -.012, UL = .009) and the Sobel test (Z = -0.001, p > .05) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of indirect to total effect of stress severity on episodic memory (PM; LL = -.457, UL = .730) adds further evidence of the indirect effect not being different from zero since the confidence intervals cross zero and path c is larger than c' with the same direction of sign (Hayes, 2013). Together these tests show

that mean stress severity is related to worry frequency, but is not related to episodic memory, and that mediation did not occur.

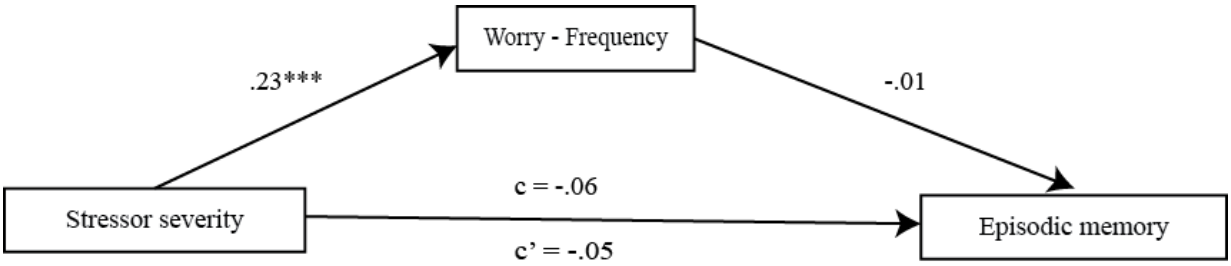

Figure 5. Unstandardized regression coefficients for the relationship between stressor severity and episodic memory with absence of mediation by worry frequency.
$^{**}p < .01$, $^{***}p < .001$

Table 8. Regression Results for the Mediation of the Effect of Stress Severity on Episodic Memory by Worry Frequency

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	.400	.182	< .05	.043	.756
SSev	-.056	.036	> .05	-.127	.016
Age	-.021	.002	< .001	-.025	-.017
Gender	.496	.048	< .001	.402	.590
Educ	.154	.026	< .001	.104	.204
HHI	.061	.019	< .01	.024	.098
CHC	-.013	.011	> .05	-.033	.008
$R^2_{Y,X}$					
Path a (X → M)					
Intercept	2.089	.228	< .001	1.640	2.537
SSev	.233	.046	< .001	.144	.323
Age	-.019	.003	< .001	-.024	-.014
Gender	.100	.060	> .05	-.018	.217
Educ	-.030	.032	> .05	-.093	.003
HHI	.006	.024	> .05	-.040	.052
CHC	.097	.013	< .001	.071	.123

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
$R^2_{M,X}$	.103				
Path b (M → Y)					
Intercept	.411	.187	< .05	.045	.778
Frequ	-.006	.021	> .05	-.046	.035
SSev	-.054	.037	> .05	-.126	.018
Age	-.021	.002	< .001	-.025	-.017
Gender	.497	.048	< .001	.403	.590
Educ	.154	.026	< .001	.104	.204
HHI	.061	.019	< .01	.024	.098
CHC	-.012	.011	> .05	-.033	-.009
$R^2_{Y,MX}$	.181				
Path c' (X → Y; a X b)	-.054	.037	> .05	-.126	.018
Indirect effect of X on Y	-.001	.005	-	-.012	.009
P_M	.024	47.715	-	-.457	.730
	Effect	SE	Z	*p*	
Sobel	-.001	.005	-.269	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 4. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Frequ is worry frequency.

5.2.4 Stress severity and executive function. The mediation analysis rejects the hypothesis that worry frequency mediates the effect of stress severity on executive function. The results show that mean stress severity is a significant predictor of worry frequency (*b* = .235, *SE* = .046, *p* < .001) such that one unit increase in stress severity leads to .235 units increase in worry frequency. Worry frequency is not a significant predictor of executive function (*b* = -.014, *SE* = .018, *p* > .05); therefore no mediation has occurred. Furthermore, both path c' (*b* = -.023,

SE = .032, *p* > .05) and path c (*b* = -.026, *SE* = .036, *p* > .05) are non-significant indicating that in this sample stress severity does not have an influence on executive function. See Figure 6 and Table 9 for analysis details. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (c') using 10000 samples (LL = -.013, UL = .005) and the Sobel test (*Z* = -0.003, *p* > .05) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). Since path c' is larger than path c the P_M is not used to determine whether the indirect effect is different from zero (Hayes, 2013). Together these tests show that mean stress severity is related to worry frequency, but is not related to executive function, and that mediation did not occur.

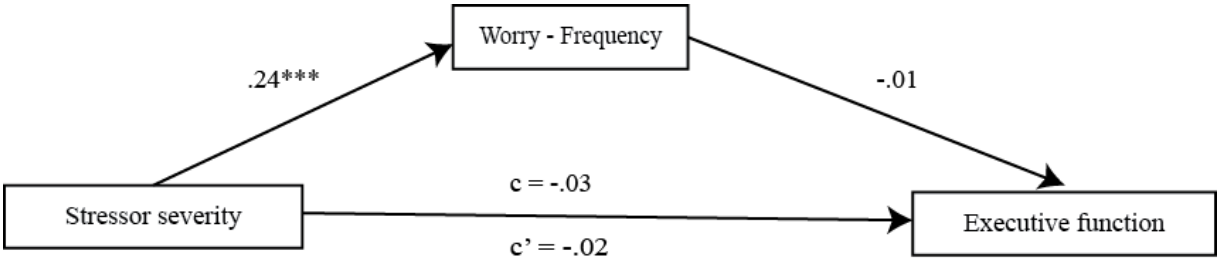

Figure 6. Unstandardized regression coefficients for the relationship between stressor severity and executive function with absence of mediation by worry frequency.
*p < .05, **p < .01, ***p < .001

Table 9. Regression Results for the Mediation of the Effect of Stress Severity on Executive Function by Worry Frequency

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	.606	.159	< .001	.294	.918
SSev	-.026	.032	> .05	-.089	.036
Age	-.025	.002	< .001	-.029	-.022
Gender	-.134	.042	< .01	-.216	-.052
Educ	.280	.022	< .001	.236	.323
HHI	.083	.016	< .001	.051	.116
CHC	-.022	.009	< .05	-.040	-.004
$R^2_{Y,X}$	.307				
Path a (X → M)					
Intercept	2.088	.228	< .001	1.640	2.537
SSev	.235	.046	< .001	.146	.325
Age	-.019	.003	< .001	-.024	-.014
Gender	.100	.060	> .05	-.018	.218
Educ	-.031	.032	> .05	-.094	.032
HHI	.005	.024	> .05	-.041	.052
CHC	.097	.013	< .001	.071	.123
$R^2_{M,X}$	.103				
Path b (M → Y)					
Intercept	.634	.163	< .001	.314	.955
Frequ	-.014	.018	> .05	-.049	.022
SSev	-.023	.032	> .05	-.086	.040
Age	-.026	.002	< .001	-.029	-.022
Gender	-.133	.042	< .01	-.215	-.050
Educ	.279	.022	< .001	.236	.323
HHI	.084	.016	< .001	.051	.116
CHC	-.021	.009	< .05	-.039	-.003
$R^2_{Y,MX}$	.308				
Path c' (X → Y; a X b)	-.023	.032	> .05	-.086	.040

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Indirect effect of X on Y	-.003	.005	-	-.013	.005
P_M	.121	12.519	-	-.240	13.907
	Effect	SE	Z	*p*	
Sobel	-.003	.004	-.737	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 5. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Frequ is worry frequency.

5.3 Mediating effect of worry duration.

5.3.1 Stress severity and somatic amplification. Using regression analysis to test the hypothesis that worry duration mediates the effect of stress severity on somatic amplification, the results show that mean stress severity is a significant predictor of worry duration (b = .281, SE = .068, p < .001) such that for every one unit increase in mean stress severity there is a 0.281 unit increase in worry duration. Worry duration is a significant predictor of somatic amplification (b = .067, SE = .030, p < .05) such that one unit increase in worry duration leads to an increase of .042 units in somatic amplification. Both path c' (b = .077, SE = .020, p < .001) and path c (b = .080, SE = .020, p < .001) are significant indicating that in both models stress severity has an influence on somatic amplification. See Figure 7 and Table 10 for details of the results. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = .002, UL = .046) and the Sobel test (Z = 1.922, p > .05) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of indirect to total effect of stress severity on somatic amplification (P_M; LL = -.010, UL = 1.636) suggests no mediation. However, preference is given

to the more valid and powerful bootstrapped confidence interval of the indirect effect, which does not cross zero, thereby indicating the presence of mediation (Hayes, 2009; Hayes, 2013). Therefore, it is determined that mediation did occur and that the third variable, worry duration, is part of the process in how stress severity impacts somatic amplification.

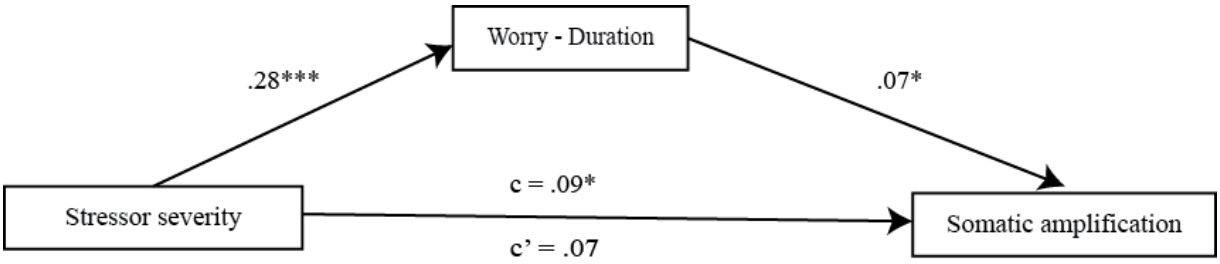

Figure 7. Unstandardized regression coefficients for the relationship between stressor severity and somatic amplification with mediation by worry duration.
*$p < .05$, **$p < .01$, ***$p < .001$

Table 10. Regression Results for the Mediation of the Effect of Stress Severity on Somatic Amplification by Worry Duration

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	2.258	.198	< .001	1.870	2.647
SSev	.087	.043	< .05	.002	.171
Age	-.002	.002	> .05	-.007	.003
Gender	.167	.052	< .01	.065	.270
Educ	.024	.027	> .05	-.029	.078
HHI	-.027	.019	> .05	-.063	.010
CHC	.042	.010	< .001	.022	.061
$R^2_{Y,X}$	.117				
Path a (X → M)					
Intercept	1.111	.312	< .001	.498	1.724
SSev	.281	.068	< .001	.147	.415
Age	-.007	.004	< .05	-.014	-.0001

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Gender	.053	.082	> .05	-.109	.214
Educ	.040	.043	> .05	-.043	.124
HHI	-.041	.029	> .05	-.099	.017
CHC	.096	.016	< .001	.065	.126
$R^2_{M,X}$	.157				
Path b (M → Y)					
Intercept	2.184	.200	< .001	1.792	2.576
Dura	.067	.030	< .05	.008	.125
SSev	.068	.044	> .05	-.018	.154
Age	-.002	.002	> .05	-.006	.003
Gender	.164	.052	< .01	.062	.266
Educ	.022	.027	> .05	-.031	.075
HHI	-.024	.019	> .05	-.061	.012
CHC	.035	.010	< .001	.015	.055
$R^2_{Y,MX}$	.117				
Path c' (X → Y; a X b)	.068	.044	> .05	-.018	.154
Indirect effect of X on Y	.019	.011	-	.002	.046
P_M	.216	6.605	-	-.010	1.636
	Effect	SE	Z	*p*	
Sobel	.019	.010	1.922	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 6. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Dura is worry duration.

5.3.2 Stress severity and physical symptoms. Using regression analysis to test the hypothesis that worry duration mediates the effect of stress severity on physical symptoms, the results show that mean stress severity is a significant predictor of worry duration (b = .274, SE =

.068, p < .001) such that for every one unit increase in mean stress severity there is .274 units increase in worry duration. Worry duration is not a significant predictor of the number physical symptoms (b = .068, SE = .119, p > .05); therefore, mediation is not present. This is confirmed by the bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = -.042 UL = .089) and the Sobel test (Z = .551, p > .05) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of indirect to total effect of stress severity on physical symptoms (PM; LL = -.181, UL = .479) also suggests no mediation. Both path c' (b = .356, SE = .174, p < .05) and path c (b = .375, SE = .171, p < .05) are significant indicating that stress severity exerts an influence on the number of physical symptoms. See Figure 8 and Table 11 details of the results.

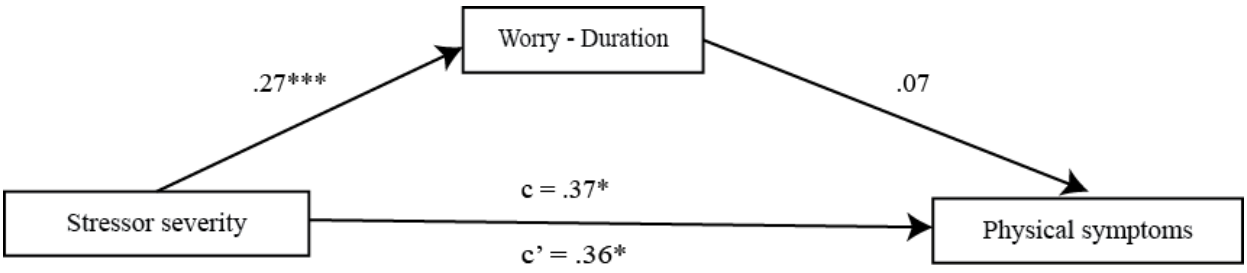

Figure 8. Unstandardized regression coefficients for the relationship between stressor severity and median physical symptoms with absence of mediation by worry duration.
*p < .05, ***p < .001

Table 11. Regression Results for the Mediation of the Effect of Stress Severity on Physical Symptoms by Worry Duration

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	1.532	.779	< .05	.001	3.063
SSev	.375	.171	< .05	.038	.711
Age	-.009	.009	> .05	-.027	.009
Gender	-.039	.206	> .05	-.445	.366
Educ	-.134	.107	> .05	-.344	.076
HHI	-.083	.074	> .05	-.228	.062
CHC	.404	.039	< .001	.327	.480
$R^2_{Y,X}$	.258				
Path a (X → M)					
Intercept	1.109	.309	< .001	.502	1.716
SSev	.274	.068	< .001	.141	.408
Age	-.007	.004	> .05	-.014	.0004
Gender	.056	.082	> .05	-.104	.217
Educ	.039	.042	> .05	-.044	.122
HHI	-.044	.029	> .05	-.101	.014
CHC	.094	.016	< .001	.063	.124
$R^2_{M,X}$	.153				
Path b (M → Y)					
Intercept	1.457	.791	> .05	-.097	3.011
Dura	.068	.119	> .05	-.165	.302
SSev	.356	.174	< .05	.014	.699
Age	-.009	.009	> .05	-.026	.009
Gender	-.043	.207	> .05	-.449	.363
Educ	-.137	.107	> .05	-.347	.074
HHI	-.080	.074	> .05	-.226	.066
CHC	.397	.041	< .001	.317	.477
$R^2_{Y,MX}$	.258				
Path c' (X → Y; a X b)	.356	.174	< .05	.014	.699

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Indirect effect of X on Y	.019	.033	-	-.042	.089
P_M	.050	7.982	-	-.181	.479
	Effect	SE	Z	*p*	
Sobel	.019	.034	.551	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 7. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Dura is worry duration.

5.3.3 Stress severity and episodic memory. Using regression analysis to test the hypothesis that worry duration mediates the effect of stress severity on episodic memory, the results show that mean stress severity is a significant predictor of worry duration (b = .275, SE = .069, p < .001) such that for every one unit increase in mean stress severity there is .275 units increase in worry duration. Worry duration is a significant predictor of episodic memory (b = -.108, SE = .053, p < .05) such that one unit of change in worry duration leads to a decrease of .042 units in episodic memory score. Both path c' (b = -.132, SE = .079, p > .05) and path c (b = -.043, SE = .078, p > .05) are non-significant indicating that in this sample stress severity does not directly influence episodic memory scores. However, this does not mean that the mediation model is a poor fit for the data. Since paths a and b are significant their significance is tested using bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = -.077, UL = .001) and the Sobel test (Z = -1.762, p > .05) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of indirect to total effect of stress severity on episodic memory (P_M; LL = .149, UL = 1905.178) suggests that mediation is present since the confidence intervals do not cross zero (Hayes, 2009;

Hayes, 2013). See Figure 9 and Table 12 for details of the results. Therefore, it is determined that stress severity has an indirect effect on episodic memory through worry duration.

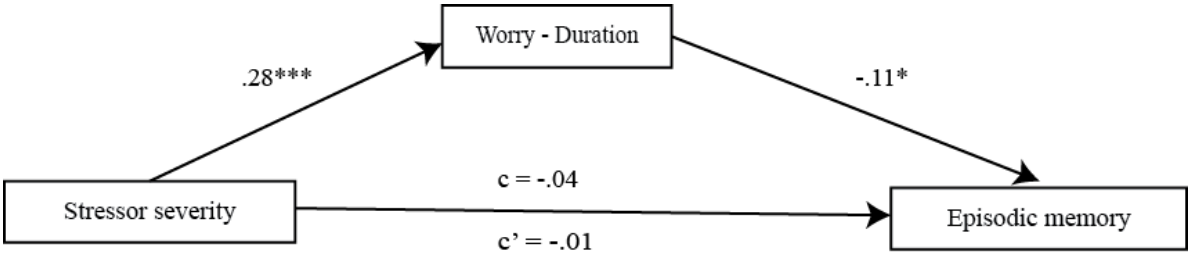

Figure 9. Unstandardized regression coefficients for the relationship between stressor severity and episodic memory with absence of mediation by worry duration.
*$p < .05$, ***$p < .001$

Table 12. Regression Results for the Mediation of the Effect of Stress Severity on Episodic Memory by Worry Duration

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	.544	.352	> .05	-.148	1.236
SSev	-.043	.078	> .05	-.195	.110
Age	-.021	.004	< .001	-.029	-.013
Gender	.436	.093	< .001	.253	.619
Educ	.117	.048	< .05	.022	.211
HHI	.047	.034	> .05	-.019	.113
CHC	-.015	.018	> .05	-.049	.020
$R^2_{Y,X}$	.141				
Path a (X → M)					
Intercept	1.095	.314	< .001	.478	1.711
SSev	.275	.069	< .001	.139	.411
Age	-.006	.004	> .05	-.014	.001
Gender	.063	.083	> .05	-.100	.227
Educ	.037	.043	> .05	-.047	.121
HHI	-.041	.030	> .05	-.100	.018

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
CHC	.092	.016	< .001	.061	.123
$R^2_{M,X}$	.151				
Path b (M → Y)					
Intercept	.662	.356	> .05	-.036	1.361
Dura	-.108	.053	< .05	-.213	-.003
SSev	-.013	.079	> .05	-.168	.141
Age	-.022	.004	< .001	-.030	-.014
Gender	.443	.093	< .001	.260	.625
Educ	.121	.048	< .05	.026	.215
HHI	.043	.034	> .05	-.023	.109
CHC	-.005	.018	> .05	-.041	.031
$R^2_{Y,MX}$	.149				
Path c' (X → Y; a X b)	-.013	.079	> .05	-.168	.141
Indirect effect of X on Y	-.030	.019	-	-.077	-.001
P_M	.693	41.441	-	.149	1905.178
	Effect	SE	Z	*p*	
Sobel	-.030	.017	-1.762	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 8. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Dura is worry duration.

5.3.4 Stress severity and executive function. This model tests the hypothesis that worry duration mediates the effect of stress severity on executive function. The results demonstrate that mean stress severity is a significant predictor of worry duration (b = .269, SE = .069, p < .001) such that for every one-unit increase in mean stress severity there is an increase of .269 units in worry duration. Worry duration is a significant predictor of executive function (b = -.126, SE = .058, p < .01) such that one unit of change in worry duration leads to a decrease of .126 units in

executive function score. Path c is significant (b = -.156, SE = .070, p < .05), but drops to non-significance for path c' (b = -.122, SE = .071, p > .05). These results point to mediation, which is corroborated by the bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = -.078, UL = -.009), the Sobel test (Z = -2.132, p < .05), and the P_M (LL = .032, UL = 1.75) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). Overall these results suggest that worry duration mediates the effect of stress severity on executive function. See Figure 10 and Table 13 for details of the results.

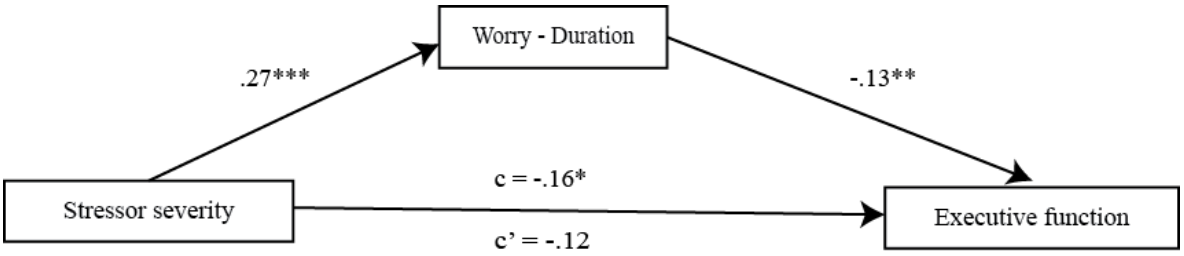

Figure 10. Unstandardized regression coefficients for the relationships between stressor severity and executive function with mediation by worry duration.
*p < .05, **p < .01, ***p < .001

Table 13. Regression Results for the Mediation of the Effect of Stress Severity on Executive Function by Worry Duration

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	.960	.318	< .01	.335	1.586
SSev	-.156	.070	< .05	-.293	-.019
Age	-.025	.004	< .001	-.032	-.018
Gender	-.245	.084	< .01	-.411	-.079
Educ	.279	.043	< .001	.193	.364
HHI	.063	.030	< .05	.003	.122
CHC	-.020	.016	> .05	-.051	.011
$R^2_{Y,X}$	.304				
Path a (X → M)					
Intercept	1.101	.314	< .001	.485	1.718
SSev	.269	.069	< .001	.134	.405
Age	-.007	.004	> .05	-.014	.001
Gender	.063	.083	> .05	-.101	.226
Educ	.040	.043	> .05	-.044	.125
HHI	-.040	.030	> .05	-.099	.019
CHC	.093	.016	< .001	.062	.123
$R^2_{M,X}$	.149				
Path b (M → Y)					
Intercept	1.100	.321	< .001	.469	1.729
Dura	-.126	.048	< .01	-.220	-.032
SSev	-.122	.071	> .05	-.261	.017
Age	-.026	.004	< .001	-.033	-.019
Gender	-.237	.084	< .01	-.402	-.072
Educ	.284	.043	< .001	.199	.368
HHI	.058	.030	> .05	-.002	.117
CHC	-.008	.016	> .05	-.041	.024
$R^2_{Y,MX}$	.315				
Path c' (X → Y; a X b)	-.122	.071	> .05	-.261	.017

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Indirect effect of X on Y	-.034	.017	-	-.078	-.009
P_M	.218	5.066	-	.032	1.725
	Effect	SE	Z	*p*	
Sobel	-.034	.016	-2.132	< .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 9. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Dura is worry duration.

5.4 Mediating effect of worry self-identification.

5.4.1 Stress severity and somatic amplification. Using regression analysis to test the hypothesis that worry self-identification mediates the effect of stress severity on somatic amplification, the results show that mean stress severity is a significant predictor of worry self-identification (b = .214, SE = .034, p < .001) such that for every one unit increase in mean stress severity there is .214 units increase in worry self-identification. Worry self-identification is a significant predictor of somatic amplification (b = .102, SE = .015, p < .001) such that one unit increase in worry duration leads to an increase of .102 units in somatic amplification. Both path c (b = .083, SE = .020, p < .001) and path c' (b = .061, SE = .020, p < .01) are significant indicating that in both models stress severity has an influence on somatic amplification. See Figure 11 and Table 14 for details of the results. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = .014, UL = .033) and the Sobel test (Z = 4.578, p < .001) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). These tests, along with the ratio of the indirect to total effect of stress severity on somatic amplification (P_M;

LL = .146, UL = .556) suggests that mediation is present since the confidence intervals do not cross zero (Hayes, 2009; Hayes, 2013). Therefore, it is determined that mediation did occur and that stress severity impacts somatic amplification through the third variable, worry self-identification.

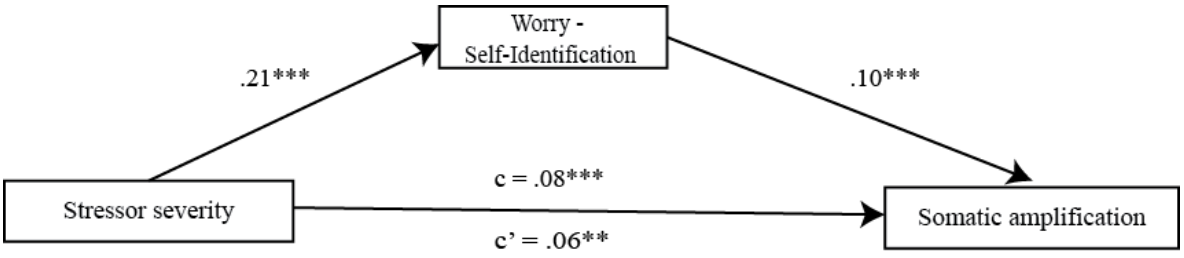

Figure 11. Unstandardized regression coefficients for the relationship between stressor severity and somatic amplification with mediation by worry self-identification.
*$p < .05$, **$p < .01$, ***$p < .001$

Table 14. Regression Results for the Mediation of the Effect of Stress Severity on Somatic Amplification by Worry Self-Identification

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	2.304	.102	< .001	2.105	2.503
SSev	.083	.020	< .001	.043	.123
Age	-.003	.001	< .05	-.005	-.001
Gender	.152	.027	< .001	.100	.205
Educ	-.030	.014	< .05	-.058	-.002
HHI	.003	.010	> .05	-.018	.023
CHC	.049	.006	< .001	.037	.060
$R^2_{Y,X}$	.108				
Path a (X → M)					
Intercept	2.512	.168	< .001	2.183	2.841
SSev	.214	.034	< .001	.148	.280
Age	-.012	.002	< .001	-.016	-.009
Gender	.174	.044	< .001	.088	.261

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Educ	-.092	.024	< .001	-.138	-.046
HHI	.018	.017	> .05	-.015	.052
CHC	.076	.010	< .001	.057	.095
$R^2_{M,X}$	.134				
Path b (M → Y)					
Intercept	2.047	.107	< .001	1.837	2.258
Self_I	.102	.015	< .001	.072	.132
SSev	.061	.020	< .01	.021	.101
Age	-.002	.001	> .05	-.004	.001
Gender	.135	.027	< .001	.083	.187
Educ	-.020	.014	> .05	-.048	.007
HHI	.001	.010	> .05	-.019	.021
CHC	.041	.010	< .001	.029	.053
$R^2_{Y,MX}$	.133				
Path c' (X → Y; a X b)	.061	.020	< .01	.021	.101
Indirect effect of X on Y	.022	.005	-	.014	.033
P_M	.264	.128	-	.146	.556
	Effect	SE	Z	*p*	
Sobel	.022	.005	4.578	< .001	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 10. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Self_I is worry self-identification.

5.4.2 Stress severity and physical symptoms. In testing the hypothesis that worry self-identification mediates the effect of stress severity on physical symptoms, the results show that mean stress severity is a significant predictor of worry self-identification (*b* = .214, *SE* = .034, *p*

< .001) such that for every one unit increase in mean stress severity there is .214 units increase in worry self-identification. Worry self-identification is a significant predictor of the number of physical symptoms (b = .177, SE = .052, p < .001) such that one unit increase in worry duration leads to an increase of .102 units in somatic amplification. Both path c (b = .247, SE = .069, p < .001) and path c' (b = .209, SE = .069, p < .01) are significant indicating that in both models stress severity has an influence on the number of physical symptoms reported. See Figure 12 and Table 15 for details of the results. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = .014, UL = .069) and the Sobel test (Z = 2.966, p < .01) (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). These tests, along with the ratio of the indirect to total effect of stress severity on physical symptoms (P_M; LL = .047, UL = .383) suggests that mediation is present since the confidence intervals do not cross zero (Hayes, 2009; Hayes, 2013). Therefore, it is determined that mediation did occur and that stress severity impacts the number of physical symptoms reported through the third variable, worry self-identification.

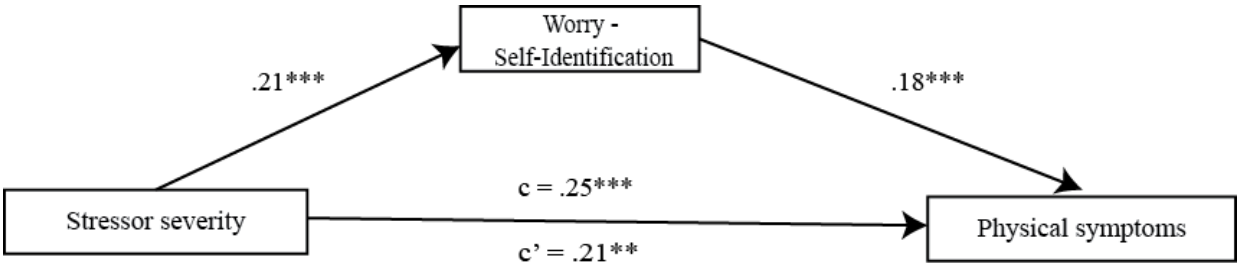

Figure 12. Unstandardized regression coefficients for the relationship between stressor severity and median physical symptoms with absence of mediation by worry self-identification.
*p < .05, **p < .01, ***p < .001

Table 15. Regression Results for the Mediation of the Effect of Stress Severity on Physical Symptoms by Worry Self-Identification

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	1.451	.342	< .001	.779	2.122
SSev	.247	.069	< .001	.112	.382
Age	-.010	.004	< .05	-.017	-.002
Gender	.080	.090	> .05	-.097	.257
Educ	-.092	.048	> .05	-.186	.003
HHI	-.092	.035	< .01	-.161	-.023
CHC	.374	.020	< .001	.334	.413
$R^2_{Y,X}$	.238				
Path a (X → M)					
Intercept	2.513	.167	< .001	2.186	2.840
SSev	.214	.034	< .001	.148	.280
Age	-.012	.002	< .001	-.016	-.009
Gender	.175	.044	< .001	.089	.262
Educ	-.091	.023	< .001	-.137	-.045
HHI	.017	.017	> .05	-.017	.050
CHC	.075	.010	< .001	.056	.094
$R^2_{M,X}$	.134				
Path b (M → Y)					
Intercept	1.007	.365	< .01	.290	1.724
Self_I	.177	.052	< .001	.074	.279
SSev	.209	.069	< .01	.073	.345
Age	-.008	.004	> .05	-.015	.0001
Gender	.049	.090	> .05	-.129	.226
Educ	-.076	.048	> .05	-.170	.019
HHI	-.095	.035	< .01	-.163	-.026
CHC	.360	.020	< .001	.321	.400
$R^2_{Y,MX}$	.244				
Path c' (X → Y; a X b)	.209	.069	< .01	.073	.345

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Indirect effect of X on Y	.038	.014	-	.014	.069
P_M	.153	.190	-	.047	.383
	Effect	SE	Z	*p*	
Sobel	.038	.013	2.966	< .01	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 11. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Self_I is worry self-identification.

5.4.3 Stress severity and episodic memory. The mediation analysis rejects the hypothesis that worry self-identification mediates the effect of stress severity on episodic memory. The results show that mean stress severity is a significant predictor of worry self-identification (b = .220, SE = .034, p < .001), indicating that one unit increase in mean stress severity leads to .220 units increase in frequency worry. Conversely, worry self-identification is not a significant predictor of episodic memory (b = -.027, SE = .028, p > .05). Both path c (b = -.066, SE = .037, p > .05) and path c' (b = -.060, SE = .037, p > .05) are non-significant; therefore no mediation has occurred. See Figure 13 and Table 16 for analysis details. The significance of the mediation was tested using bias-corrected bootstrapped confidence interval for the indirect effect (c') using 10000 samples (LL = -.019, UL = .006) and the Sobel test (Z = -.952, p > .05), which indicate no presence of mediation (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of the indirect to total effect of stress severity on episodic memory (P_M; LL = -.457, UL = .730) confirms these findings. Together these tests show that mean stress severity is related to worry self-identification, but is not related to episodic memory, and that mediation did not occur.

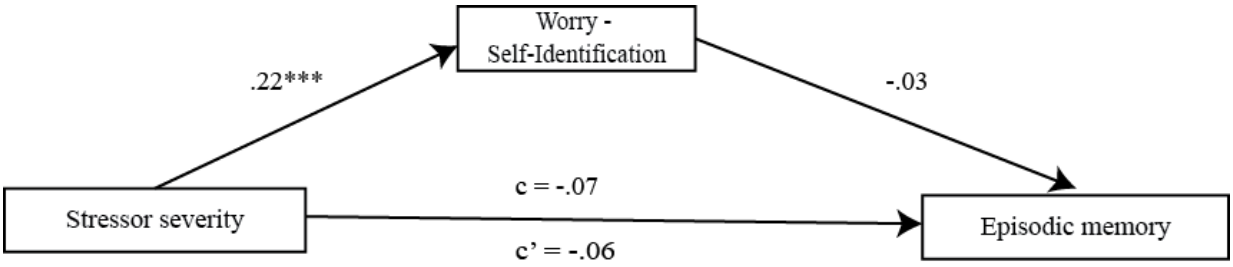

Figure 13. Unstandardized regression coefficients for the relationship between stressor severity and episodic memory with absence of mediation by worry self-identification.
*$p < .05$, ***$p < .001$

Table 16. Regression Results for the Mediation of the Effect of Stress Severity on Episodic Memory by Worry Self-Identification

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	.449	.182	< .05	.093	.806
SSev	-.066	.037	> .05	-.138	.006
Age	-.021	.002	< .001	-.025	-.017
Gender	.502	.048	< .001	.408	.596
Educ	.151	.026	< .001	.101	.201
HHI	.057	.019	< .01	.020	.094
CHC	-.013	.011	> .05	-.034	.008
$R^2_{Y,X}$	.179				
Path a (X → M)					
Intercept	2.452	.169	< .001	2.121	2.783
SSev	.220	.034	< .001	.154	.287
Age	-.012	.002	< .001	-.016	-.008
Gender	.176	.045	< .001	.089	.264
Educ	-.087	.024	< .001	-.134	-.041
HHI	.021	.017	> .05	-.013	.055
CHC	.075	.010	< .001	.056	.094
$R^2_{M,X}$	.134				
Path b (M → Y)					
Intercept	.516	.194	< .01	.135	.897

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Self_I	-.027	.028	> .05	-.082	.028
SSev	-.060	.037	> .05	-.133	.013
Age	-.021	.002	< .001	-.025	-.017
Gender	.507	.048	< .001	.413	.602
Educ	.149	.026	< .001	.099	.199
HHI	.058	.019	< .01	.021	.095
CHC	-.011	.011	> .05	-.032	.010
$R^2_{Y,MX}$	.180				
Path c' (X → Y; a X b)	-.060	.037	> .05	-.133	.013
Indirect effect of X on Y	-.006	.006	-	-.019	.006
P_M	.091	8.436	-	-.217	1.143
	Effect	SE	Z	*p*	
Sobel	-.006	.006	-.952	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 12. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Self_I is worry self-identification.

5.4.4 Stress severity and executive function. The testing of the hypothesis that worry self-identification mediates the effect of stress severity on executive function revealed that mean stress severity is a significant predictor of worry duration (b = .223, SE = .034, p < .001) such that for every one unit increase in mean stress severity there is .223 units increase in worry self-identification. Worry self-identification is a significant predictor of executive function (b = -.048, SE = .024, p < .05) such that one unit of change in worry duration leads to a decrease of .048 units in executive function score. Both path c (b = -.031, SE = .032, p > .05) and path c' (b = -.020, SE = .033, p > .05) are non-significant indicating that in this sample stress severity does not directly influence executive function scores. Mediation significance is tested using bias-corrected

bootstrapped confidence interval for the indirect effect (path c') using 10000 samples (LL = -.023, UL = .001) and the Sobel test (Z = -1.862, p > .05), which indicate that no mediation is occurring (Fairchild & MacKinnon, 2009; Kenny, 2015; Preacher & Hayes, 2004; Preacher & Kelly, 2011). The ratio of the indirect to total effect of stress severity on executive function (P_M; LL = -.324, UL = 46.100), like the indirect effect of X on Y, suggests no mediation since the confidence intervals cross zero (Hayes, 2009; Hayes, 2013). Therefore, even though path a and path b are significant it is concluded that stress severity does not have an indirect effect on executive function through worry self-identification. See Figure 14 and Table 17 for details of the results.

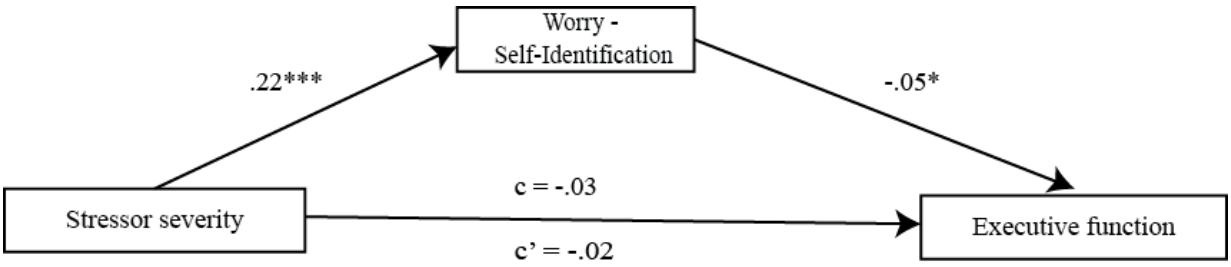

Figure 14. Unstandardized regression coefficients for the relationships between stressor severity and executive function with mediation by worry self-identification.
*p < .05, **p < .01, ***p < .001

Table 17. Regression Results for the Mediation of the Effect of Stress Severity on Executive Function by Worry Self-Identification

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Path c (X → Y)					
Intercept	.644	.159	< .001	.333	.955
SSev	-.031	.032	> .05	-.093	.032
Age	-.025	.002	< .001	-.029	-.022
Gender	-.131	.042	< .01	-.213	-.049
Educ	.275	.022	< .001	.231	.318
HHI	.080	.016	< .001	.048	.112
CHC	-.025	.009	< .01	-.043	-.007
$R^2_{Y,X}$	.303				
Path a (X → M)					
Intercept	2.452	.169	< .001	2.121	2.783
SSev	.223	.034	< .001	.156	.289
Age	-.012	.002	< .001	-.016	-.008
Gender	.177	.045	< .001	.089	.264
Educ	-.089	.024	< .001	-.136	-.043
HHI	.020	.017	> .05	-.014	.055
CHC	.075	.010	< .001	.056	.094
$R^2_{M,X}$	.367				
Path b (M → Y)					
Intercept	.761	.169	< .001	.429	1.092
Self_I	-.048	.024	< .05	-.095	-.0001
SSev	-.020	.032	> .05	-.084	.043
Age	-.026	.002	< .001	-.029	-.022
Gender	-.123	.042	< .01	-.205	-.040
Educ	.270	.022	< .001	.227	.314
HHI	.081	.016	< .001	.049	.113
CHC	-.022	.009	< .05	-.040	-.003
$R^2_{Y,MX}$	.304				
Path c' (X → Y; a X b)	-.020	.032	> .05	-.084	.043

Model	Estimate	SE	*p*	CI (lower)	CI (upper)
Indirect effect of X on Y	-.011	.006	-	-.023	.001
P_M	.345	18.383	-	-.324	46.099
	Effect	SE	Z	*p*	
Sobel	-.011	.006	-1.862	> .05	

Note: Unstandardized regression coefficients for path a, b, c, and c' are illustrated in Figure 13. Proportion of variance in Y explained by X ($R^2_{Y,X}$). Proportion of variance in M explained by X ($R^2_{M,X}$). Proportion of variance in Y explained by X and M ($R^2_{Y,MX}$). The 95% confidence interval is determined by using 1000 bias correct bootstrapping samples. CI (lower) = lower bound of 95% confidence interval; CI (upper) = upper bound. P_M is the ratio of indirect to total effect of X on Y. R_M is the ratio of indirect to direct effect of X on Y. Self_I is worry self-identification.

In all of the models, worry regressed onto mean stress severity (path a) was significant as was the influence of the number of chronic health conditions (CHCs) on worry where one unit increase in the number of chronic health conditions lead to between .075 and .099 units increase in worry frequency, duration, or self-identification. In consideration of variations in sample size and sample characteristics in the previous 12 models all models were re-run after constraining the sample to those with complete data across all variables for all models. This reduced the sample size (n = 437) and created a core sample from which to test each model. In comparison to the sample demographics reported in Tables 2, 3, and 4, the participant demographics for this constrained sample were approximately 3.5 years younger, had approximately .70 more chronic health conditions, and were predominantly female (65.4%) in comparison to the MIDUS II – Project 1 sample (see Table 18). These differences are greater than the comparison between MIDUS II – Project 1 and the individual model samples (see Table 2, Table 3, and Table 4).

Table 18. Participant Demographics for MIDUS II - Project 1 Versus Sample With Complete Data Across All Variables

	MIDUS II – Project 1	Complete Data Across Variables
	(*n* = 3849) M (SD)	(*n* = 437) M (SD)
Age	55.98 (12.30)	52.52 (10.86)
Education	3.09 (0.98)	3.11 (0.96)
HH Income	3.15 (1.40)	3.26 (1.42)
CHCs	2.38 (2.26)	3.03 (2.62)
	n (%)	*n* (%)
Gender		
Female	2115 (54.9)	286 (65.4)
Male	1734 (45.1)	151 (34.6)

All models exhibited the same pattern as the original models in that worry regressed onto mean stress severity (path a) was significant. Additionally all models showed the same pattern in the covariates where the number of chronic health conditions had a significant positive relationship with worry. There were no models mediated by worry frequency, which differs from the previous analyses regressing somatic amplification or median physical symptoms onto mean stress severity. This is likely due to a reduction in power and sample size (n = 1533 and n = 1542, respectively). In the models testing worry duration as a mediator, mediation was found when executive function was regressed onto mean stress severity, but not when episodic memory was regressed onto stress severity as the upper level confidence interval of the indirect effect of X on Y increased from -.001 to .001 thereby crossing zero. When testing worry self-

identification the regression of somatic amplification onto mean stress severity remained significant; however, unlike the original models the regression of median physical symptoms onto mean stress severity was not significant. Similar to the worry frequency models, this is likely due to a reduction in sample size extending to a reduction in power.
For models that were significant but then became non-significant it is likely an issue of sample size and power.

In summary, the mediation models had mixed results. This analysis set out to determine: (1) if worry mediated the relationship between stress severity and somatic health; and (2) if worry mediated the relationship between stress severity and cognition. There was a mediation of the effect of mean stress severity on somatic health (somatic amplification and physical symptoms) by worry frequency and self-identification. Worry duration only mediated the effect of mean stress severity on somatic amplification, not physical symptoms. In terms of cognition, only worry duration was found to mediate the effect of mean stress severity on episodic memory and executive function. See Table 19 for the summary of regression results and Figure 15 for a summary of the mediation results.

Table 19. Summary Regression Results for the Mediation of Stress Severity on Somatic Health and Cognition by Worry

Model		Path c (X → Y)		Path a (X → M)		Path b (M → Y)		Path c' (X → Y; a X b)		Indirect Effect of X on Y		P_M		Sobel (Z)
		b	SE	*b*	SE	*b*	SE	*b*	SE	CI (LL)	CI (UL)	CI (LL)	CI (UL)	
Frequ	S_ Amp	.087***	.020	.230***	.045	.042***	.012	.077***	.02	.004	.017	.044	.257	2.940**
	P_ Sym	.233***	.069	.229***	.045	.164***	.039	.196**	.069	.019	.064	.071	.411	3.208**
	EM	-.056	.036	.233***	.046	-.006	.021	-.054	.037	-.012	.009	-.457	.730	-.269
	EF	-.026	.032	.235***	.046	-.014	.018	-.023	.032	-.013	.005	-.240	13.907	-.737
Dura	S_ Amp	.087*	.043	.281***	.068	.067*	.030	.068	.044	.002	.046	-.010	1.636	1.922
	P_ Sym	.375*	.171	.274***	.068	.068	.119	.356*	.174	-.042	.089	-.181	.479	.551
	EM	-.043	.078	.275***	.069	-.108*	.053	-.013	.079	-.077	-.001	.149	1905.178	-1.762
	EF	-.156*	.070	.269***	.069	-.126**	.048	-.122	.071	-.078	-.009	.032	1.725	-2.312*
Self_I	S_ Amp	.083***	.020	.214***	.034	.102***	.015	.061**	.02	.014	.033	.146	.556	4.578***
	P_ Sym	.247***	.069	.214***	.034	.177***	.052	.209**	.069	.014	.069	.047	.383	2.966**
	EM	-.066	.037	.220***	.034	-.027	.028	-.060	.037	-.019	.006	-.217	1.143	-.952
	EF	-.031	.032	.223***	.034	-.043*	.024	-.020	.032	-.023	.001	-.324	46.099	-1.862

Note: Frequ is worry frequency. Dura is worry duration. Self_I is worry self-identification. S_Amp is somatic amplification. P_Sym is the median number of physical symptoms. EM is episodic memory. EF is executive function.

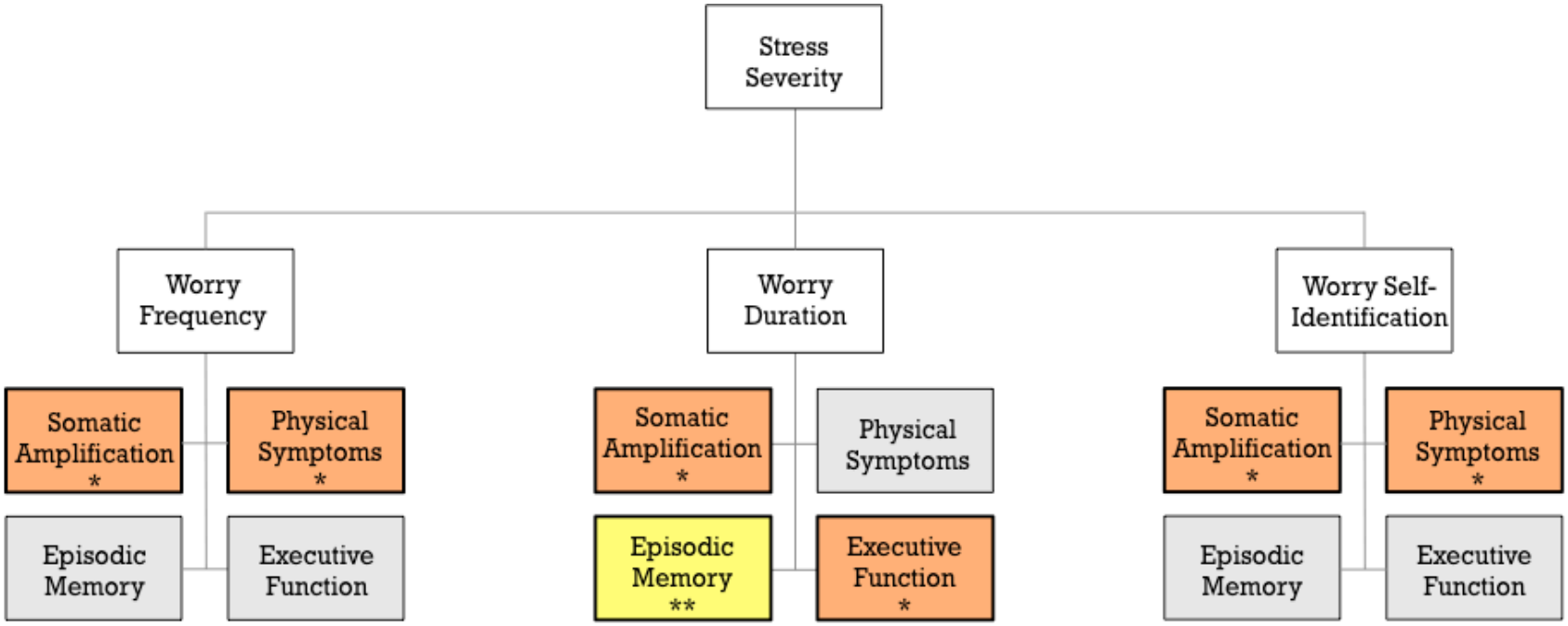

Figure 15. Overview of results for models examining whether worry mediates the effect of stress on somatic health or cognition.

* = mediation occurred, ** = indirect effect, no asterix = no mediation occurred

Discussion

The purpose of this study was to conceptually replicate previous research showing the mediation of the effect of stress on somatic health by worry (Brosschot, Gerin, & Thayer, 2006; Verkuil, Brosschot, Meerman, & Thayer, 2012) and to extend this model to include cognition as a separate outcome. This extension was based on previous research demonstrating the close relationship between perseverative cognitions and cognitive interference (CI) and the impact of CI on cognition (Sarason, 1984; Sliwinski, Smyth, Hofer, & Stawksi, 2006; Stawski, Mogle, Sliwinski, 2013; Stawski, Mogle, & Sliwinski, 2013). This study examined these relationships using mediation analysis on three data sets from the Midlife in the United States national longitudinal studies of health and wellbeing: MIDUS II – Project 1 (cross-sectional), MIDUS II – Project 2 (daily diary), MIDUS II – Project 3 (cognitive). These projects were selected based on satisfying one or more variables of interest for the mediation analyses. Prior to discussing the hypotheses it is important to revisit the correlation matrix in Table 5. Of interest is whether there is a moderate correlation between the worry variables, which would demonstrate that the individual variables are related to each other and thereby capturing a relatively similar construct. Fields (2009) suggests an r-value of around .3 if one intends to perform a factor analysis on a measure with questions trying to measure the same construct. Using this criteria, the correlation between worry frequency and worry self-identification has a moderate correlation as expected with the other two correlations falling just below the criteria (i.e. worry frequency and worry duration, worry duration and worry self-identification). Overall the correlations between worry variables were expected to be slightly higher, which would indicate that they are measuring a similar construct, but each correlation was significant and therefore the analysis proceeded.

The first hypothesis, whether worry mediates the relationship between stress and somatic health, is supported by the results. As reviewed in section 1.5, Brosschot, Gerin, and Thayer (2006) proposed the perseverative cognition hypothesis where perseverative cognitions facilitate the prolonged activation of stress and are the process through which stress effects health. This current study demonstrates that worry frequency, worry duration, and self-identification as a worrier mediate the effect of stress severity on somatic amplification. For models with number of physical symptoms as the outcome, worry frequency and self-identification were the only significant mediators. This pattern of worry and self-identification as a worrier may indicate that the variables are closely related. It is possible that the more frequently an individual worries the more likely they are to identify themselves as a worrier, which may be rooted in the development of a self-narrative that is created as an individual attempts to connect life events (Gergen & Gergen, 1988). This phenomenon of frequency of experience leading to self-labeling or self-identification can be found in bullying literature (Theriot, Dulmus, Sowers, & Johnson, 2005). To our knowledge this is the first study to use a worry self-identification variable as a mediator in the stress-somatic health and stress-cognition relationships. These results are of particular importance as the mediation model presents a good fit for the data outside of the variables and context used in previous research (Brosschot, Gerin, & Thayer, 2006; Verkuil, Brosschot, Meerman, & Thayer, 2012). In addition to worry self-identification, this study introduced somatic amplification, an alternative somatic health measure that focuses on the intensified perception of symptoms that occur when attention is focused on bodily sensations, health worries, and catastrophizing of the sensations (Rief, 2013). Cumulatively, these results suggest that, as a whole, stress affects somatic health through worry and adds to the body of research by extending the model to related, but not identical, variables.

The second hypothesis, whether worry mediates the relationship between stress and cognition, is also supported by the results. Worry duration was consistently the sole significant mediator of models with a cognitive outcome (episodic memory or executive function). Furthermore, the regression of executive function on stress severity (path c) was only significant in the mediation model using worry duration as a mediator. This pattern was present in both the individual samples for each model and the analyses run on complete data across all variables. Several reasons could explain this inconsistency. An explanation for worry duration being the only significant mediator in models with a cognitive outcome could be a similarity between worry duration and prolonged activation of a stressor. It is plausible that longer durations of worry, in contrast to the frequency or self-identification of worry, is more closely related to prolonged activation. However, if this is the case then one would expect worry duration to mediate the relationship between the stress and somatic health variables, but this was not the case in this study. As previously discussed, repetitive thoughts have been found to maintain physiological activation in the body along with impeding cognitive processes (Gianferante et al., 2014). Furthermore, the participants who answered the worry duration question may have inherently different characteristics. It is possible that if tested as a moderated mediation then significant results may have been found for the other worry variables. For example, previous research has demonstrated that negative affect, the experience of negative emotions such as anger, irritation, worry, depression, sadness, and annoyance, may be a unique predictor of cognitive interference within persons and can account for the effect of stress on between person differences in cognitive interference (Stawski, Mogle, Sliwinski, 2011). This study is novel in that, to our knowledge, it is the first to extend the concept of the perseverative cognition hypothesis to outcomes of cognition. In summary, the role of frequency and self-identification in

the stress-somatic health relationship and the role of worry duration in the stress-cognition relationship remained even after restricting the sample to participants with complete data across all variables in the study.

From a broader perspective, the analyses revealed that stress was a significant predictor of every worry variable even after adjusting for the effect of the covariates (i.e. age, gender, education, household income, and number of chronic health conditions). This reinforces the impact that stress has on worry, although it is not clear whether this is a unidirectional or bidirectional relationship. Additionally, for all of the path b analyses, the number of chronic health conditions was a significant predictor of every somatic health and cognitive variable after adjusting for the effects of the covariates. Path a was comprised of somatic health or cognition variables regressed onto worry. This is an important finding as it highlights the unique impact of chronic health conditions on both somatic health variables (i.e. somatic amplification and physical symptoms) and cognition (i.e. episodic memory and executive function).

A key feature of this study is the use of conceptual replication in testing the first hypothesis, which was based on work by Brosschot (Brosschot, Gerin, and Thayer (2006; Brosschot, 2010; Verkuil, Brosschot, Gebhardt, & Thayer, 2010; Verkuil, Brosschot, Meerman, & Thayer, 2012). The goal of a conceptual replication is to test the hypothesis of a previous experiment by maintaining the primary focus of the analysis whilst using different measures and methods (Schmidt, 2009). A combination of reviews (Brosschot, 2010; Brosschot et al., 2006; Verkuil et al., 2010) and an experiment (Verkuil et al., 2012) formed the foundation of this study.

This study is a conceptual replication in that the population, setting, and select measures are different from the studies that form the foundation of the model (Brosschot, Gerin, and

Thayer, 2006; Verkuil, Brosschot, Gebhardt, & Thayer, 2010; Verkuil, Brosschot, Meerman, & Thayer, 2012). The sample for this study is a nationally representative sample of mid-life adults versus a sample of primary and secondary teachers (Verkuil et al., 2012). The measures used in this study were collected via a combination of telephone survey and mailed paper surveys versus a palmtop computer that prompted participants to respond to a set of questions five times a day for six days (2012). We chose stress severity from a daily diary project to represent stress, as it is more indicative of the individual's stress reactivity. This was similar to Verkuil and colleagues (2012) in that it was a daily assessment, but different in that the original study measured total stressful events. The worry variables were single retrospective questions that represented a greater cross-section of the variable aspects frequency, duration, and self-identification. In contrast the original study assessed trait worry using the Penn State Worry Questionnaire and state worry using daily worry episodes and the length of worry episodes at each prompt. The measure used for reporting somatic complaints was similar between our study and the original, with the original using the Subjective Health Complaints (SHC) inventory. The list of physical symptoms used in MIDUS is similar to the SHC inventory, although the MIDUS physical symptom list was broader in that it included teeth-, ear-, eye-, and menstrual-related symptoms as well as muscle soreness, and muscle weakness. In contrast, the SHC inventory had greater overall health breadth in that it included mental health (i.e. anxiety, sadness/depression) and sleep problems. Knowing these similarities and differences is important in determining the extent of conceptual versus direct replication and in developing next steps in replicating the research for greater understanding of the relationships being tested. According to Schmidt (2009) the goal of a conceptual replication is to gain greater understanding of the mechanism being tested and to determine whether the theory is corroborated. Replication is important in order to ensure that

scientists produce dependable and replicable research. This research dependability movement, a relatively recent redirection towards improving the integrity of research, specifically suggests direct replication as one of the key components (Lishner, 2015). Although not a direct replication, this study adds to the cumulative body of research as it provides a conceptual replication design embedded with within-study replication as well as a model extension to test whether the stress-cognition model is also mediated by worry.

Strengths, Limitations, and Future Directions

The foundation of this study consists of both replication and model extension. Replication is a cornerstone of theory development and testing, giving opportunity to increase the confirmatory power of the replication and enabling generalization to a broader application (Schmidt, 2009). Although some declare direct replication as superior in determining result dependability and reducing false-positive results (Lishner, 2015; Simmons, Nelson, & Simonsohn, 2011), this study is strengthened by its use of both model extension and replication within the study by testing of multiple mediation models within the data to determine whether a consistent pattern of results develops (Murayama, Pekrun, & Fielder, 2014). Additionally, in contrast to previous research (Verkuil, Brosschot, Meerman, & Thayer, 2012) this study used stress severity rather than total stressors to ensure sufficient variability within the measure so that a greater range of response options was captured. Stress severity also provides an indication of the intensity of the stressors being experienced, which is linked to the magnitude of the physiological stress response (vom Berg-Maurer, Trivedi, Bollmann, De Marco, Ryu, 2016). Derived from the daily diary study of stressful experiences, this variable also offered greater ecological validity in comparison to laboratory methods as it surveys the participant in their natural environment (Schlotz, 2013).

As discussed in section 1.4, secondary analysis of existing longitudinal data provides a rich source of data that can be used to explain the influence of a predictor over time. The key challenges with this approach are the lack of control in how the data is collected and what questions are asked (Cheng & Phillips, 2014). As a longitudinal study, the MIDUS II pools of participants are subject to attrition. Research by Radler and Ryff (2010) demonstrated that from MIDUS I to MIDUS II the participant demographics were increasingly composed of white

females, those who are married, and individuals with more education. This could impact the results herein as research has demonstrated that low socioeconomic and ethnic minority groups tend to experience more frequent stressful events (Hatch & Dohrenwend, 2007; Keith, 2014; Perry, Harp, & Oser, 2013). Therefore, the results may underrepresent the population-level prevalence of stress severity and its impact on somatic health and cognition. This being understood, the decision to pursue analysis using the MIDUS data sets was made after considering the advantages of using a large population-based longitudinal study with a wide breadth of variables to best answer the hypotheses herein whilst limiting time and expense.

Of additional consideration is that various degrees of retrospective responses may introduce recall bias into the results. Recall bias, or recollection error, occurs when previous states are recollected as better or worse than they actually were (Blome & Augustin, 2015). Additionally, retrospective responses can be biased by the participant's current state or experience (present state effect) or can be reconstructed by comparing their current state with how they think they've changed over a period of time (e.g. improved, deteriorated, unchanged, plateaued after improving) (Blome & Augustin, 2015; Norman, 2003). In this study, the risk of one of these biases varies depending on the nature of the recall asked by the question, with recall ranges of the last 24 hours to the last 12 months. Retrospective responses were provided for all variables since real-time momentary assessment, otherwise known as experience sampling, was not used during the collection of the data. For example, mobile sensing technology can provide a method to collect real-time data, detect changes in the human voice that relate to stress, and gather environmental contextual information that can be used to cue the participant's recall during a follow up survey (Lu, Rabbi, Chittaranjan, Frauendorfer, SchmidMast, Campbell, … Choudhury, 2012; Rahman, Zhang, Voida, & Choudhury, 2014).

Mediation models are dependent on ordered time sequencing between predictor, mediator, and outcome. It is important to note that we are unable to confirm whether the projects used in this analysis adhere to this time sequencing as the implementation dates for each project overlap considerably. Tate's (2015) critique of the misuse of mediation stresses that mediation analyses are time-ordered where the mediator temporally lies between the predictor and the outcome. In this study, somatic amplification scores were drawn from the same project as the worry variables: MIDUS II – Project 1, which ran from 2004 to 2006. Comparatively, physical symptom questions were asked at the same time as stress severity questions: MIDUS II – Project 2, which ran from 2004 – 2009. Similarly, the cognitive variables were drawn from MIDUS II – Project 3, which ran from 2004 to 2006. All of these projects ran on different schedules, therefore it is unclear the sequence in which the questions were asked. As such, responses may be differentially impacted by influences such as life events and changes in health.

In addition to the potential mistiming of the sequence of variables in the mediation models, it is possible that other mediating or moderating variables are missing. For example, there are many moderating factors for stress reactivity, including sex, age, ethnicity, personality, pre-existing disease, and chronic stress (Schlotz, 2013). Aside from absolute stress that is undeniably threatening, such as escaping a burning building, the experience of relative stress is highly individual because the stress response depends on psychological determinants (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). Schlotz (2013) recommends a repeated measure design assessing global and individual system assessment (e.g. cardiovascular, endocrine) using real-time self-reporting methods rather than retrospective accounts.

Understanding the strengths and limitations of this study, there are several recommendations for future research. In their review of the effects of perseverative cognitions,

Brosschot, Gerin, and Thayer (2006) concluded that state worry yielded more consistent results across included studies than did trait worry although there is debate over whether a distinction between state and trait is arbitrary or whether other categories could be more useful (Allen & Potkay, 1981; Fridhandler, 1986; Zuckerman, 1983). In the absence of a standardized measure of worry, this study used three different operationalizations of worry from MIDUS II – Project 1 (frequency, duration, and self-identification), which are most closely categorized as trait worry as they are retrospective single time-point measures. Future research could benefit from the inclusion of both trait scales, such as the Penn-State Worry Questionnaire, and state measurement, such as experience sampling questions of worry frequency and duration (Verkuil, Brosschot, & Thayer, 2007).

Future research may also benefit from considering several other variables, such as the effect of negative affect on somatic health complaints and its relationship to stress and worry and the role of coping, emotion regulation, and control in stress appraisal. As previously discussed, negative affect may be a unique predictor of cognitive interference and has been shown to impact somatic health independently of worry (Stawski, Mogle, Sliwinski, 2011; Verkuil, Brosschot, Meerman, & Thayer, 2012). Furthermore, in a prospective daily diary design (Charles and Almedia, 2006), concurrent state negative affect was related to somatic symptoms and previous-day state negative affect predicted pain and gastrointestinal symptoms, which are included in the physical symptoms reporting in this study. In addition to negative affect, coping, emotion regulation, and control are important variables to consider when measuring the stress response. Commonly considered a moderator of the effect of stress on health, coping requires the management of internal and external demands that strain or exceed an individual's experience or resources (Lazarus & Folkman, 1987). Connected to coping is emotion regulation, a strategy that

monitors, evaluates, and modifies emotional reactions (Thompson, 1991). The most disruptive stressors occur on a daily basis, such as relationship conflicts, home-related responsibilities, and work pressures, enabling them to cumulate over time if allowed to persist (Almeida & Horn, 2004; Neupert et al., 2007). Therefore, emotion regulation is pivotal in reducing distressing feelings originating from the stressor, a process that is connected to higher well-being (Charles & Luong, 2013). Lastly, perceived control is an important component of stress appraisal, coping, and emotion regulation because it is the foundation from which an individual traverses challenges and demands and seeks to maintain balance between gains and losses (Lachman & Prenda-Firth, 2004). Individuals with higher levels of perceived control have greater well-being, motivation to take action in spite of hardship, health promoting behaviours, and functional status and less acute and chronic illness (Lachman & Prenda-Firth, 2004).

In closing, this study found compelling patterns of the mediation of the effect of stress severity on both somatic health and cognition by worry. Of particular importance is that these results were discovered via conceptual replication, included a model extension to cognition as an outcome, and tested the model across multiple variable combinations. As additional waves of data are added to the MIDUS group of projects, future research will benefit from leveraging two waves (change from time one to time two) or more (longitudinal design) to understand between-person (interpersonal) and within-person (intrapersonal) differences as well as the trajectory of change over time (Hofer & Piccinin, 2009). From a health-care provider perspective, understanding the impact of stress on somatic health and cognition and the role worry plays in maintaining the physiological activation of stress is important for assisting patients in optimizing their health and wellbeing. Worry reduction and stress management techniques can be included as part of a cohesive package of care provided to patients, such as those with chronic health

conditions, in a bid to reduce the potential excessive wear and tear on the body and empower patients with self-management strategies.

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