PUT YOUR HEART INTO IT: DOES PHYSIOLOGY FACILITATE COPING WITH RACIAL DISCRIMINATION AND CAN THIS PROCESS REDUCE MENTAL HEALTH SYMPTOMS?

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ABSTRACT

Vanessa V. Volpe: Put Your Heart into it: Does Physiology Facilitate Coping with Racial Discrimination and Can this Process Reduce Mental Health Symptoms?
(Under the direction of Jean-Louis Gariépy and Patrick Curran)

Racial discrimination is a central contributor to racial disparities in mental health, even after controlling for socioeconomic status (Williams & Mohammed, 2009). Furthermore, both self-reports of racial discrimination and mental health symptoms increase during the transition from adolescence to young adulthood for Black college students attending a predominantly White institution. While many Black students draw upon coping strategies to combat the risk that racial discrimination poses to their mental health, the extant literature is unclear regarding which strategies are most optimal. The present study aimed to clarify the coping literature by examining intraindividual estimates of respiratory sinus arrhythmia (RSA) as partial mediators in the relation between coping strategies and mental health symptoms. Black college students completed an online questionnaire (N=205) and a laboratory visit that recorded heart rate in response to an *in vivo* challenge via electrocardiogram (N=115). Using structural equation modeling and time series analysis, results indicated that: 1) more frequent use of John Henryism to cope with racial discrimination was uniquely associated with fewer self-reported symptoms of depression and anxiety in the short-term, above and beyond other coping strategies, and 2) elements of RSA during recovery from the challenge were directly associated with coping strategies and mental health symptoms. Frequency of use of John Henryism may merit further examination as an index of health risk during the transition from adolescence to young adulthood. While partial mediation of hypothesized paths was not supported, this work suggests

fruitful new directions for research on the developmental impact of racial discrimination and coping strategies for Black young adults.

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CHAPTER 1: INTRODUCTION

Please note: This dissertation uses the term "Black" to refer to individuals who self-identify as such, including African American, Caribbean, African, and Bi-/Multi-racial individuals.

In the United States, Black individuals experience poorer health than White individuals in many areas, including early onset of illness, greater severity of disease, and poorer survival outcomes (e.g. Krieger & Sydney, 1996). More specifically in terms of mental health, Black individuals are considered a high-need population (US Dept. of Health and Human Services, 2001), with more persistent mood disorder diagnoses than White individuals (Breslau et al., 2005). In non-clinical populations Black adults are still 20% more likely to report experiencing significant psychological distress than White adults (National Center for Health Statistics, 2012). Experiences of racial discrimination have been identified as a central contributor to these racial disparities in mental health, even after controlling for socioeconomic status (see Williams & Mohammed, 2009, for a review). Racial discrimination is often defined broadly as differential treatment of individuals based on their racial group membership. In the current study, racial discrimination is further operationalized as systematic actions delivered by members of the dominant racial group that have differential and negative impacts on members of non-dominant racial groups (Williams, Neighbors, & Jackson, 2003). While the majority of literature examines the impact of self-reported experiences of racial discrimination on mental health, the current study utilizes an *in vivo* experience of racial discrimination to conduct a microgenetic analysis of processes through which mental health risk may be conferred.

While the experience of racial discrimination may compromise healthy development at any age, the transition from adolescence to young adulthood heightens stress susceptibility for Black individuals as they forge identities and undergo physical, cognitive, and social transitions (e.g. Fisher, Wallace, & Fenton, 2000; Neblett et al., 2008; Seaton, Neblett, Upton, Powell Hammond, & Sellers, 2011; Sellers, Linder, Martin, & Lewis, 2006). During this developmental period, experiences of racial discrimination may exert their pernicious influence by threatening one component of an individual's identity – racial group membership (Arnett, 2000; Shanahan, 2000; Thoits, 1991). Indeed, one of the central tasks of adolescence and young adulthood, scaffolded by increases in abstract reasoning, is identity development (Erikson, 1968) and during this time Black youth are actively exploring their *racial* identity (Spencer, 2006). This identity development is shaped by interpersonal interactions and social comparison processes (Settersten, 1999; 2010) as young adults compare themselves to others to generate meaning about who they are and their significance in larger society. However, experiences of racial discrimination communicate negative messages about an individual as a member of a certain racial group, a membership that is assigned by phenotypic characteristics and not of the individual's own volition. Because a central developmental task of young adulthood is the acquisition of autonomy and agency (Benson & Elder, 2011; Schawartz, Cote, & Arnett, 2005), facing experiences of racial discrimination may threaten psychological well-being. Indeed, distress due to experiencing racial discrimination during this time has been associated with poorer mental health outcomes, including depression, anxiety, and substance abuse (Williams & Mohammed, 2009, for a review).

While there is evidence that experiences of racial discrimination confer mental health risk for Black individuals during their college years, more research that examines acute physiological

responses to racial discrimination is needed. Furthermore, from a clinical perspective, differences in the impact of racial discrimination are likely to be tied to the coping strategies individuals draw upon to recover from these experiences. However, the coping literature remains inconclusive about the benefits of coping strategies for Black young adults in the face of racial discrimination. Therefore, the current study seeks to resolve inconsistencies in the coping literature by examining autonomic regulation following an *in vivo* situation of racial discrimination as a central process through which coping strategies are impactful for mental health. Prior studies have demonstrated that physiological responses to laboratory scenarios evoking racial discrimination are related to reports of psychological distress and health outcomes (e.g. Brody et al., 2014; Neblett & Roberts, 2013), however these studies have either focused on a challenge period rather than a recovery period, focused on older adult populations, and/or have not examined parasympathetic activity. While racial discrimination is not a variable under statistical consideration in my analyses, the current study focuses on individuals' response to one laboratory task that mirrors elements of a racial discrimination experience. The literature on racial discrimination and mental health will be reviewed below to demonstrate the need for further work on the mechanisms by which racial discrimination confers mental health risk. This section is presented to stress the importance of the current study as a first step towards understanding the psychophysiological underpinnings of individuals' complex responses to racial discrimination.

Racial Discrimination and Mental Health

Unfortunately, research suggests that Black individuals commonly experience racial discrimination in the United States. A majority of Black youth self-report experiencing racial discrimination either "recently" (last three months, Prelow, Danoff-Burg, Swenson, & Pulgiano,

2004; last year, Guthrie, Young, Williams, Boyd, & Kintner, 2002) or during their lifetime (Gibbons, Gerrard, Cleveland, Wills, & Brody, 2004). Experiences with racial discrimination have been consistently linked with a host of negative psychosocial outcomes for Black youth over time, including increased internalizing (Seaton, Caldwell, Sellers, & Jackson, 2008) and externalizing (Brody et al., 2006) symptoms, and lower self-esteem (Greene, Pahl, & Way, 2006). Specific to mental health, more frequent experiences of racial discrimination have been associated with increased depression and anxiety symptoms in Black adolescents and young adults (Gaylord-Harden & Cunningham, 2009; Neblett et al., 2008; Prelow, Danoff-Burg, Swenson, & Pulgiano, 2004). Several prominent reviews in both health and psychology fields have systematically documented the negative impact of experiences of racial discrimination on the mental health of Black individuals, taking the position that experiences of racial discrimination are akin to pervasive interpersonal stressors which disrupt cognitive, emotional, and biological systems (Williams & Mohammed, 2009; Williams, Neighbors, & Jackson, 2003; Pascoe & Smart Richman, 2009; Mays, Cochran, & Barnes, 2007; Paradies, 2006). To compound the long-term impact of experiences of racial discrimination on mental health, Black young adults with higher levels of education have been found to be least likely to seek help for mental health challenges (Broman, 2012). Experiencing racial discrimination, combined with limited help seeking behaviors, may confer potent developmental risk for Black young adults (e.g. Neblett & Roberts, 2013), as evidence suggests that the transition from adolescence to young adulthood poses greater threat of facing mental health challenges.

While college is a time of transition for students of all racial/ethnic backgrounds, this transition has the potential to be uniquely stressful for students of color. Black students who attend predominantly White institutions (PWIs) may face negative stereotypes about their racial

group from both students and faculty, contributing to perceptions of the campus as unfriendly to students of color (Ancis, Sedlacek, & Mohr, 2000). Indeed, research has found that Black students at PWIs self-report a lack of support and frequent experiences of racism, discrimination, and alienation (Fisher & Hartmann, 1995; Suarez-Balcazar, Orellana-Damacela, Portillo, Rowan, & Andrews-Guillen, 2003). The experience of stereotype threat and prejudice has also been associated with increased anxiety symptoms in college students of color (Aronson, Fried, & Good, 2002).

Black college women report perceiving more frequent instances of racial discrimination (Biasco, Goodwin, & Vitale, 2001) and increased symptoms of depression and anxiety upon entering college (Karlsen & Nazroo, 2002; Kessler, Mickelson, & Williams, 1999) and they are less likely to seek treatment for such mental health concerns as compared to their White peers (e.g. Eisenberg, Gollust, Golberstein, & Hefer, 2007; Rosenthal & Wilson, 2008). Although less is known about the mental health challenges of Black college males, qualitative work suggests that they see the college environment as hostile and subsequently experience psychological distress (Smith, Allen, & Danley, 2007). For both sexes, the experience of racial discrimination has been found to result in psychological symptoms consistent with experiencing psychological trauma (Pieterse, Carter, Evans, & Walter, 2010). Work on microaggressions, defined as "brief and commonplace daily verbal, behavioral, and environmental indignities, whether intentional or unintentional, that communicate hostile, derogatory, or negative racial slights and insults to the target person or group" (Sue et al. 2007, p. 272), has demonstrated that microaggressions pose mental health risk for Black college students. For example, Blume and colleagues (2012) found that more frequent experiences of microaggressions leads to increased risk of higher anxiety symptoms. Such negative interpersonal experiences on college campuses may contribute to stress

and feelings of isolation, thus ultimately impacting mental health. Taken together, this work suggests that the transition to a PWI seems to confer specific mental health risk for Black young adults due to the increased risk of racial discrimination.

While such findings acknowledge the role of racial discrimination on the mental health of Black individuals and suggest that the college years may be an especially important period of risk, most studies examine *self-reported* experiences of racial discrimination across the lifetime and/or the past year (e.g. Boynton, O'Hara, Covault, Scott, & Tennen, 2014). In recent years the rise of ambulatory technologies and daily diary studies has called for increased attention to the acute impacts of such experiences and their temporal sequelae. While self-reported measures provide insights into chronic stress due to racial discrimination, they may be hampered by threats to validity such as social desirability, memory and latency effects, and/or variability in the experiences they capture. Indeed, many studies that employ self-reported measures of racial discrimination suggest that participants report relatively low levels of racial discrimination, even when qualitative and audit studies suggest the experience of racial discrimination is much more prevalent (Williams & Mohammed, 2009, for a review). Therefore, in order to more precisely measure the processes by which racial discrimination confers mental health risk, the current study utilizes a laboratory paradigm that captures *in vivo* responses to a task that mimics elements of the experience of racial discrimination.

Coping and Mental Health

Individual differences in the impact of racial discrimination on the mental health of Black young adults may be tied to the coping strategies they employ. According to the hallmark transactional theory of stress and coping proposed by Lazarus and Folkman (1984), coping refers to cognitive and behavioral efforts to manage an encounter that is perceived as stressful by an

individual within a given context. In this framework coping processes are initiated jointly by the cognitive, emotional, and stress systems, with implications for long-term mental health. More specifically, in the transactional theory, coping is posited as critical mediator in the relation between stress and mental health outcomes. While Lazarus and Folkman (1984) provided important groundwork for conceptualizing and measuring coping, recent research has focused on the efficacy of specific coping strategies for mental health.

One important distinction in the present coping literature is between "culturally-relevant" coping strategies and "mainstream" coping strategies, though there is much less literature on the former. Coping strategies that are based on an Afrocentric worldview and grounded in the historical, cultural, and philosophical tradition of people of African descent in the United States (e.g. Chambers et al., 1998) are referred to as "culturally-relevant" coping strategies. In contrast, "mainstream" coping strategies are based on a conceptual framework believed to be applicable to all individuals regardless of their racial/ethnic background (e.g. Utsey, Ponterotto, Reynolds, & Cancelli, 2000). For example, communalistic approaches such as collective action or sacrifice may govern culturally-relevant coping efforts, while mainstream coping may instead emphasize individual effort. Both types of strategies have been examined in the literature. After controlling for the use of mainstream coping strategies, distress due to experiences of racial discrimination independently predicts the use of communalistic, spiritual, and emotional debriefing coping strategies (Gaylord-Harden & Cunningham, 2009; Lewis-Coles & Constantine, 2006). Furthermore, Black adolescents and adults have been found to endorse more culturally-relevant coping strategies than mainstream coping strategies. Yet other research reports that mainstream coping strategies also have mental health benefits for Black adults, including greater quality of life (e.g. Utsey, Bolden, Lanier, & Williams, 2007). Taken together this work suggests that both

culturally-relevant and mainstream coping strategies may confer unique mental health benefits. Thus the current proposal examines two mainstream and two culturally-relevant coping strategies.

Active Coping. The first mainstream coping strategy that may impact mental health is active coping. Active coping encompasses a set of strategies (sometimes referred to as "approach-oriented" or "approach" coping strategies) that include active behaviors meant to address the situation at hand. This often includes strategies such as positive cognitive restructuring and problem focused behaviors such as reporting or confronting the source of a perceived injustice. Active coping strategies may be contrasted with avoidant coping strategies, which require fewer cognitive resources and often serve to minimize acute threat to the mistreated individual (Feagin, 1991). Avoidant strategies include behaviors such as self-distraction, denial, and behavioral disengagement.

In adolescent samples, active coping strategies appear effective for mental health. Studies of Black adolescents' coping strategies find that approach coping strategies are related to greater feelings of self-efficacy and less psychological distress while the reverse is true for avoidant coping strategies (e.g. Moos, 2002). Furthermore, adolescents who use avoidant coping are more likely to espouse what Scott and House (2005) call "internalizing and externalizing" coping strategies. These strategies are akin to internalizing and externalizing mental health symptoms, with internalizing symptoms involving negative self-evaluations, helplessness, and social anxiety and externalizing symptoms including yelling and/or cursing to release negative emotions. While both internalizing and externalizing coping are employed with the aim of alleviating negative emotions (Stevenson, Reed, Bodison, & Bishop, 1997) these strategies have been shown to

escalate psychological distress and hostile interactions (Noh, Beiser, Kaspar, Hou, & Reumans, 1999).

Research on the health benefits of active coping for Black *adults* is less clear. For example, studies show that Black adults report significantly more avoidant coping strategies than problem-solving or support-seeking strategies (Plummer & Slane, 1996; Utsey, Ponterotto, Reynolds, & Cancelli, 2000) and this avoidance is related to reductions in self-esteem and life satisfaction (Utsey, Ponterotto, Reynolds, & Cancelli, 2000), and increases in negative emotions (Hyers, 2007) and distress (Smith, Stewart, Myers, & Latu, 2008). However, such results came from cross-sectional studies and therefore causal links between avoidant coping and these psychosocial outcomes could not be evaluated. Other studies report associations in the opposite direction or lack of associations between the use of avoidant coping and mental health. For example, Sanders Thompson (2006) found that greater use of avoidant coping strategies was associated with reductions in mental health symptoms and that the reverse was true for use of approach-oriented coping strategies. One study examined coping strategies in conjunction with racial identity using cluster analysis and found no difference in well-being as a result of different combinations of identity and coping strategies (Forsyth & Carter, 2012). Significant differences in mental health symptoms as a function of cluster *were* observed, such that the use of bargaining and cultural hypervigilance was associated with increased mental health symptoms compared to the use of empowered resistance. Empowered resistance, "the channeling of community and/or legal resources to make those involved accountable for their actions," (p. 130) is easily classifiable as an active coping strategy, which may lend credence to the notion that active coping is best for mental health. However, it was the joint use of active coping in combination with racial identity variables that was associated with fewer mental health symptoms and this

result therefore may not be solely replicated with active coping. Taken together, the conclusion that avoidant coping preserves cognitive resources and is less dangerous in the short-term for many Black individuals awaits longitudinal analysis.

Social Support. The second mainstream coping strategy with implications for mental health is social support, defined as "a social network's provision of psychological and material resources intended to benefit an individual's ability to cope with stress" (Cohen, 2004, p. 676). Brief interventions focused on social belonging, which seek to induce a sense of security and provide encouragement for incoming college students of color, have provided compelling evidence that social support may function as a protective factor in this population. For example, in a randomized control trial conducted with Black undergraduates, a one-time intervention designed to generate feelings of social belonging upon entering college significantly improved GPA, self-reported health, and psychological well-being, and decreased the number of visits to the doctor 3 years following the intervention (Walton & Cohen, 2011).

Yet other work has suggested that seeking social support may facilitate rumination on negative emotions and strain personal relationships, specifically for Black women, thus amplifying risk for poorer mental health outcomes in some cases (Gray & Keith, 2003). To examine this possibility, one study of Black college students tested three competing models of social support: that social support would buffer students from the harmful impacts of racial discrimination, that social support networks would mobilize support for individuals exposed to racial discrimination, and that seeking social support would deteriorate that support for those exposed to racial discrimination. Only the third model was supported, suggesting that Black college students actually perceive lower levels of social support when support is sought following an instance of racial discrimination. The authors surmised that if students are

uncomfortable seeking social support following an instance of racial discrimination and perceive the responses they receive as inadequate, devaluing, or reprimanding, isolation may be their ultimate strategy and the social support may thus be ineffective (Prelow, Mosher, & Bowman, 2006). Alternatively, as experiences of racial discrimination often are a function of larger societal and systemic injustice, a lack of agency may make it difficult for some individuals to recruit or benefit from social support when facing discrimination. Yet other research suggests that the beneficial effects of social support may only be enhanced for individuals who are members of more concealable socially stigmatized groups (Frable, Platt, & Hoey, 1998). Thus, cultural factors such as the social visibility of one's group may also explain conflicting findings regarding the value of social support as a buffer against negative health outcomes.

Spiritual Coping. Spiritual coping is a broad construct which may encompass religious faith and participation in religious groups and belief systems. Dimensions of spiritual coping have often been examined as a culturally-relevant coping strategy for Black women (Broman, 1996; Christian, Al-Mateen, Webb, & Donatelli, 2000; Mattis, 2002). In one qualitative study, Shorter-Gooden (2004) found that many participants "rested on faith" in coping with racial discrimination, which entailed relying on prayer, spiritual beliefs, or their relationship with God. Shorter-Gooden (2004) posited that resting on faith is an internal coping resource that has positive effects on one's self-esteem, but did not test this assertion. However, a related body of literature on religiosity offers clear evidence that the individual and communal aspects of religious involvement confer mental health benefits. For example, one study reported that for Black adolescents living in neighborhoods with limited financial and structural resources, religiosity is an important resilience factor (e.g. Ball, Armistead, & Austin, 2003). Other studies find that attendance at religious services buffers Black adults from the negative emotions

associated with experiences of racial discrimination (Bierman, 2006) and reduces depressive symptoms for Black youth (Van Dyk & Elias, 2007). Conversely, Black adults with *low* levels of religiosity are more likely to experience suicidal ideation following depressive symptoms associated with an instance of racial discrimination (Walker, Salami, Carter, & Flowers, 2014). Such evidence suggests that feeling a connection with a higher being through spirituality, akin to social support, is associated with better mental health (Houltberg, Henry, Merten, & Robinson, 2011). However, in one meta-analysis, some aspects of religious involvement (such as service attendance) appeared to be related with personal growth following a stressful event while other aspects of spiritual coping (such as pleading for direct intercession) were linked to increased mental health symptoms (Ano & Vasconcellas, 2005).

John Henryism. Another culturally-relevant coping strategy is "John Henryism" – defined as a prolonged, high-effort psychological response to stress (James, 1994). This coping strategy is named after the folk legend of John Henry, a Black steel driver who raced to set more railroad tracks than a steam-powered hammer in order to retain his employment. Bennett and colleagues (2004) supported the cultural relevance of this coping style by confirming that it is more prevalent in the Black community than in the White community. It should be noted that similar strategies could certainly be extended to other populations facing adversity, however research on John Henryism in these populations has not been explicitly examined. While John Henryism is a culturally-specific coping strategy for Black individuals, John Henryism is likely not *only* culturally-specific for Black individuals. John Henryism is considered a culturally-relevant coping strategy in the current study due to its origins as a strategy that characterizes the "larger protracted struggle of African-American men and women to free themselves from pervasive and deeply entrenched systems of social and economic oppression" (James, 1994, p.

175). This style is evidenced by a high personal sense of control, determination to succeed, and a preference not to seek help. Individuals who use this strategy tend to actively approach and solve problems through high levels of perceived control and determination. While such culturally-specific active coping may confer more mental health benefits than more passive forms, research indicates that the prolonged high effort response involved in John Henryism is often detrimental for Black adults. Indeed, in the urban legend John Henry was able to win the race but died of overexertion shortly thereafter. In line with allostatic load theories, research on physical health has found that individuals who score high on the John Henryism scale have higher heart rates, are prone to hypertension (Bennett et al., 2004), and are at increased risk of cardiovascular disease (Merritt, Bennett, Williams, Sollers, & Thayer, 2004). However, other research has found no associations or opposite associations between John Henyrism and mental health. For example, John Henryism has been associated with lower levels of depression both in a population of individuals with sickle cell disease (McDougald et al., 2009) as well as in a normative health population (Bronder, Speight, Witherspoon, & Thomas, 2014). It has also been associated with lower psychological distress and substance use (Kiecolt, Hughes, & Keith, 2009). However, the potential benefits of John Henryism as a coping strategy for mental health have not been examined in the context of Black young adults attending a PWI.

In sum, extant literature is unclear regarding the most successful coping strategies for offsetting the impact of experiences of racial discrimination on mental health (Brondolo, Brady Ver Halen, Pencille, Beatty, & Contrada, 2009), and this study aims to fill this gap with specific attention to Black college students. As a starting point for further research, the current study will be guided by the following assumptions that are drawn from the literature. Research has indicated that in the face of stress individuals frequently draw from a hierarchy of coping

strategies across multiple contexts, and that this repertoire is often developed by adolescence (e.g. Frydenberg, 2008; Frydenberg & Lewis, 1994; Lewis & Frydenberg, 2004). Therefore, the current study examines coping strategies as relatively stable individual tendencies towards a particular cognitive/emotional style of managing experiences of racial discrimination. Furthermore, Cairns and Green (1979) argued that the questionnaire method is well suited to measure personal tendencies and dispositions, as it requires subjects to appraise them over time and contexts. Therefore, the current study uses questionnaires rather than observational techniques to measure the strategies individuals use to cope with an experience of racial discrimination. Finally, because coping processes involve cognitive, emotional, *and* biological systems, (see Lazarus & Folkman, 1984), the current proposal will contribute to the literature by using *in vivo* measures of physiological responses to an instance of racial discrimination. More specifically, I posit that autonomic responses, measured as changes in parasympathetic input to the heart, will mediate the relation between coping strategies and mental health in a sample of Black young adults enrolled in a PWI.

The Mediating Role of Physiology

Theoretically, either mediating or moderating pathways for physiology could be proposed in the relation between coping strategies and mental health. However, the following section presents initial evidence from the literature which has informed the decision to explore a mediating pathway in the current study.

Physiological Responses to Racial Discrimination. The Biopsychosocial Model (Clark, Anderson, Clark, & Williams, 1999) posits that, in the face of racial discrimination, both psychological and physiological systems respond to the perception of threat. From this perspective, the experience of racial discrimination constitutes a challenge to homeostasis,

necessitating both a cognitive/emotional response and a physiological response. Indeed, Clark and colleagues (1999) use evidence from early stress research (Burchfield, 1979; Cohen & Lazarus, 1979; Light & Obrist, 1980; Ursin, Baade, & Levine, 1978) to theorize that the magnitude and duration of cardiovascular responses to experiences of racial discrimination are intimately associated with the individual's ability to cope with such experience (p. 812). This model suggests that physiological responses mediate the relation between coping processes and health outcomes such that a cardiovascular response of a large magnitude that persists beyond the stressful experience will pose health risk. A physiological response can be mounted from multiple biological systems, with one of the most rapid and flexible of such systems being the autonomic nervous system. As an arm of the peripheral nervous system, the autonomic nervous system manages the body's responses to internal and external environments. There are two branches of the autonomic nervous system– the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) – which both maintain an active discourse with cognitive and emotional processes via their origins in the brain stem.

The SNS is responsible for the direct mobilization of bodily resources necessary to initiate a response to internal or external demands. This system is best known for mounting a "fight or flight" response (Canon, 1929), enabling the individual to act in the service of defense. Sympathetic activation innervates multiple tissues and organs, performing functions necessary for active engagement with the environment, including: increasing oxygen intake, increasing heart rate and blood flow to skeletal muscles, converting glycogen to glucose in the liver which then increases blood sugar for active use, and inhibiting the digestion processes in which the body engages during resting states. In short, the PNS serves to maintain homeostasis in optimal environmental conditions. Similar to the SNS, the PNS also acts upon tissues and organs

throughout the body, however the function of the PNS is in the service of anabolism. Innervations by the PNS limit oxygen intake, decrease heart rate and blood flow, increase energy storage (as glycogen) in the liver, and promote engagement in digestion processes in the stomach and intestines. In the absence of challenge, such processes allow the body to engage in activities of maintenance and repair. The ability to recruit PNS activity has been postulated as an especially important indicator of stress vulnerability with consequences for long-term health.

PNS activity is commonly measured via the magnitude of heart rate variability as induced by the vagus nerve (Porges, 1992, 1995; Porges & Byrne, 1992). The vagus nerve is the tenth cranial nerve, beginning in the brainstem and connecting with multiple organs. The vagus nerve is bilaterally organized, with one branch originating in the dorsal motor nucleus and the other originating in the nucleus ambiguus. Myelinated vagal pathways from the nucleus ambiguus feed into the sino-atrial node and are thus especially important to consider as they regulate heart rate. This input from the nucleus ambiguus may be quantified by deriving a measurement of respiratory sinus arrhythmia (RSA)- the rhythmic increase and decrease in heart rate associated with respiration. Heart rate increases during respiratory inspiration due to decreased outflow from vagal efferents and decreases during respiratory expiration due to increased outflow from vagal efferents. Due to the vagal impact on heart rate, the amplitude of RSA functions as an index of PNS activity.

According to Polyvagal Theory, the PNS is responsible for regulating homeostatic processes and promoting calm states of social engagement, thus conferring restorative benefits for the body during times where challenge is absent (Porges, 1992, p.499). Therefore, PNS activity may play an especially crucial role following experiences of racial discrimination. For example, the neurovisceral integration model of health disparities (Thayer & Friedman, 2004)

suggests that rumination following an experience of racial discrimination is associated with lower levels of PNS activity for Black individuals. This decreased PNS activity is thereby associated with greater psychological distress. A rapid reinstatement of PNS activity following an experience of racial discrimination allows for a faster recovery of homeostasis, as it lowers SNS input to the viscera whose chronic innervation would otherwise increase risk of poor health in the long-term. As RSA functions as a measure of PNS activity, the current study measures RSA during recovery from an *in vivo* laboratory task that mimics elements of an experience of racial discrimination.

Harrell and colleagues (2003) stressed the need to conduct additional studies utilizing a "moderated psychophysiology" methodological approach. In this approach, intensive physiological measurements are recorded in response to laboratory analogs of racial discrimination, allowing researchers to better understand individual differences in the process by which racial discrimination is linked to mental health outcomes. This approach does not necessitate conducting moderation analyses in statistical terms. The past decade has seen a large increase in the number of studies of racial discrimination that quantify PNS and SNS activity during a challenging laboratory situation. However, the majority of these studies have examined acute reactions to the stressor itself, rather than examining PNS activity during a period of recovery when the individual is allowed to recuperate from the acute stressor. Furthermore, it has been common practice in these studies to capture patterns of RSA change across a laboratory task through averages computed across specified time intervals to estimate change scores. Because the relative input of the SNS and PNS to the heart may vary from second to second, this computational approach may obscure the patterns of individual differences that may be most

critical to the quantification of health risks. Therefore, the current study uses a continuous measure of individual change in RSA variability during the recovery period of a laboratory visit.

RSA is most often measured using change scores, subtracting an aggregate estimate of RSA during basal resting periods from an aggregate of RSA during periods of challenge in laboratory tasks. However, the current study examines patterns of RSA during the recovery period, when the challenge has been removed. As the literature suggests, the primary function of coping strategies is restorative, as they are aimed at reinitiating a calm state through appropriate control of stressful situations. Because this restorative activity has both cognitive/emotional and physiological components, I aim to quantify patterns of RSA during a period that, for different individuals, may or may not signal a capability to reinstate PNS control. Some studies of RSA have also examined interaction terms in order to capture the joint contribution of an aggregate of RSA during a stress task and an aggregate of RSA following a stress task. While this approach affords the researcher additional information about the joint contribution of both response to and recovery from stressor, in studies with smaller samples it may not always be feasible to estimate such interactions. Therefore, given the utility of the current study as a foundation for further examination, I will partial out the magnitude of PNS activity at stressor, thereby accounting for the acute response to challenge while estimating PNS reinstatement during recovery rather than examining interactions.

Coping Strategies and Respiratory Sinus Arrhythmia. According to the allostatic load model (McEwen & Stellar, 1993), pervasive experiences of chronic stressors may "get under the skin," causing a "wear and tear" on bodily systems responsible for the initiation and regulation of responses. Such strenuous use of these systems results in an increased susceptibility to the development of poor health outcomes. Allostatic load typically occurs when the SNS is

chronically activated and PNS activity is dampened such that reinstatement of restorative processes is compromised. In the past ten years, over 58 longitudinal and cross-sectional studies have specifically examined the negative impact of allostatic load on the development of health symptoms (Juster, McEwen, & Lupien, 2009, for a review). Most notably, greater allostatic load has been associated with increased depressive symptoms (e.g. Maloney, Boneva, Nater, & Reeves, 2009), PTSD (e.g. Glover, 2006), and personality types associated with higher levels of anxiety (e.g. Sun, Wang, Zhang, & Li, 2007). Black college students who experience racial discrimination as a chronic and stressful experience may be at heightened risk for developing these symptoms. However, whether racial discrimination is experienced by some individuals as chronic and stressful could depend on the relative efficacy of the coping strategies they employ over time.

Few studies have examined the link between coping strategies and physiology during recovery from stress. Utilizing a questionnaire, one study found that avoidant coping in response to stressful and uncontrollable social situations exacerbates physiological arousal in a sample of White children (e.g. Dufton, Dunn, Slosky, & Compas, 2011). Another experimental study of German adolescents showed that a social anger-inducing laboratory task did not increase heart rate when adolescents used an active coping strategy measured *in vivo*, but the task did produce an increase in heart rate when subjects used rumination instead (Vögele, Sorg, Studtmann, & Weber, 2010). While these studies provide preliminary support for the notion that coping and autonomic responses are linked, they do not specifically concern the Black college student population in the United States, the stressor of racial discrimination, and/or the full range of coping strategies considered in the present study.

Despite limited research, it may be postulated that the coping strategies Black college students employ in the face of racial discrimination should play a critical role in their RSA patterns during recovery from those experiences. Because coping strategies are initiated following an experience of stress, the ability of the PNS to restore homeostatic equilibrium during recovery from an experience of racial discrimination may be the process by which coping strategies exert an influence on mental health. More specifically, if coping strategies that involve an active mobilization of cognitive and emotional resources in response to a stressor, such as active coping and John Henryism, are indeed effective in reducing the risk for mental health symptoms, they should do so by permitting patterns of RSA augmentation post-stressor. Similarly, if social support and spiritual coping strategies do not encourage rumination or promote unhealthy relationships, they should also permit patterns of PNS activity post-stressor because they provide individuals with emotional assurance of the benefits of communing with others. Just as the function of the cognitive/emotional system of coping is to re-establish cognitive equilibrium between self and environment following challenge, so too is the goal of the PNS activity during that same period of time. In sum, while the current coping literature makes it difficult to draw concrete predictions about the exact relations between coping strategies and RSA, strategies should be associated with PNS activity during recovery. Polyvagal Theory (Porges, 1995) asserts that it is specifically in the *absence* of threat that the PNS is enlisted in the service of social engagement and restorative processes. In this way, examining PNS activity during recovery from a racial discrimination experience will illuminate the role of different coping strategies as promoters or inhibitors of restorative processes.

Respiratory Sinus Arrhythmia and Mental Health Symptoms. A large body of literature has examined associations between resting RSA and symptoms of depression and

anxiety. High levels of RSA are often found to be associated with decreased symptoms of depression (De Jonge et al., 2007; Glassman, Bigger, Gaffney, & Van Zyl, 2007; Rottenberg, Wilhelm, Gross, & Gotlib, 2002; Vaccarino et al., 2008) and anxiety (Friedman & Thayer, 1998). Often, such studies are conducted by comparing clinically depressed individuals to controls (Van der Kooy et al., 2006; Licht et al., 2008). However, other studies of non-clinical samples have reported a positive association between RSA and depression (Bosch et al., 2009; Thayer, Smith, Rossy, Sollers, & Friedman, 1998), including a recent study that utilized a sample of middle aged Black adults (Keen, Turner, Mwendwa, Callender, & Campbell, 2015). Yet another study found that higher levels of RSA are associated with increased symptoms of anxiety in a non-clinical sample (Jonsson, 2007). The authors suggested that prior studies were largely conducted on panic disorder, which may be characterized by somatic symptoms related to decreased heart functioning, and therefore the relation between RSA and anxiety may be more convoluted in non-clinical samples. However, all of these studies have examined basal RSA rather than recovery following an instance of racial discrimination, and basal RSA may be indicative of larger individual differences in physiological functioning.

Overview of the Current Study

The current study seeks to examine the impact of Black college students' use of four different coping strategies on their mental health symptoms as mediated by patterns of RSA during recovery from an *in vivo* experience of racial discrimination. Black undergraduate college students aged 18 –32 were recruited on campus to participate in a two-part study. In the first part of this study subjects completed an online survey in which they reported demographic information, coping strategies, and symptoms of depression and anxiety. In the second part of this study subjects were exposed to an *in vivo* experience of racial discrimination in the

laboratory, designed to activate cognitive/emotional systems. Subjects' heart rate was monitored throughout the laboratory visit, including a baseline period, a stress period, and a recovery period. The first set of analyses will examine the relation between coping strategies and mental health symptoms. The second set of analyses seeks to model patterns of change in RSA across the recovery period. The third set of analyses examines the role of patterns of RSA as a mediator in the relation between coping strategies and mental health symptoms.

The current study offers several unique contributions to the literature. First, I propose to draw upon methodological and statistical approaches specifically designed to capture dynamic physiological processes. As emphasized by Porges (1976), instead of relying on population differences between subjects the current study will directly capture intraindividual variability by employing time series analysis. Second, my literature review suggests that the notion that coping with an experience of racial discrimination involves the recruitment of both cognitive and physiological resources, although a sound proposition based on the work of Lazarus and Folkman (1984) and Clark et al. (1999), has never been put to empirical testing. Thus a unique contribution of my dissertation will be to test the empirical validity of this proposition. Given that the focus of the current study is to examine the mediating role of PNS activity in the relation between coping strategies and mental health, the use of an *in vivo* task that measures elements of racial discrimination will better capture these relations than survey methods of racial discrimination experiences. Typically, in prior research coping strategies have been posited as a mediator between self-reported experiences of racial discrimination and health outcomes (e.g. Seaton et al., 2014). Therefore, another unique contribution of the current research is to examine how physiological support for coping strategies affects mental health. Furthermore, the current study adds to the literature by examining multiple coping strategies, both mainstream and

culturally-relevant. Through the following aims, the overall goal of this investigation is to clarify the present inconsistencies in the literature on the utility of specific coping strategies via a systematic investigation of the processes through which different coping strategies may or may not confer mental health risk.

Specific Aim 1*: Examine the association between coping strategies (active coping, social support, spiritual coping, John Henryism) and mental health symptoms (depression, anxiety) in the current sample.* Given the literature reviewed above, there is sufficient evidence to hypothesize that there will be a relation between coping strategies and mental health symptoms. However, there remains insufficient evidence to conclude that any one coping strategy would be more protective against mental health symptoms than others. Furthermore, as prior research has found both positive and negative relations between the coping strategies and mental health outcomes under examination, specific hypotheses regarding the directionality of these relationships cannot be formulated at this time. A unique contribution of the current study, as specified in Aim 3, is to examine the role of physiological activity in these relations.

Specific Aim 2*: Describe patterns of intra-individual variability in RSA during recovery from a racial discrimination laboratory task in order to generate the estimates needed to test Specific Aim 3.* Based on the literature, three overall patterns of intra-individual variability in RSA during recovery are hypothesized: no change in RSA during recovery, reduction in the amplitude of RSA during recovery, and an augmentation of this amplitude during recovery. Reduction in the amplitude of RSA during recovery would reflect a weakened or insufficient ability to recruit PNS influence following the removal of a stressor. Augmentation of the amplitude of RSA during recovery would reflect the ability to adaptively recruit PNS influence in the service of restoring homeostatic balance following the removal of a stressor. No change in

the amplitude of RSA during recovery could be indicative of an absence of stress response from which to recover or a constitutional difficulty to reinstate PNS activity following the removal of a stressor. However, partialing out PNS activity at stress (in Aim 3 below) will allow me to determine whether an absence of change in RSA during recovery reflects a lack of response to the stressor or a constitutional difficulty to reengage this system. These three overall patterns will be appropriately parameterized for each individual participant via time series analysis.

Specific Aim 3*: Examine the role of patterns of RSA change during recovery as mediators in the relation between coping strategies (active coping, social support, spiritual coping, and John Henryism) and mental health symptoms (depression and anxiety).* I examine the hypothesis that benefits to mental health accrued through the instantiation of coping strategies may be obtained through physiological support in the form of PNS reengagement during recovery from an experience of racial discrimination. After completing the time series process for each participant's second-by-second RSA data across the recovery period in Specific Aim 2, I will extract estimates that quantify the patterns described above. More specifically, patterns of RSA change will be quantified by parameters that describe: a) initial level of RSA at the beginning of removal of challenge (i.e. recovery) via an intercept estimate from each person's time series equation, b) magnitude of change in RSA across recovery, via slope estimates for "time" and "time squared" variables to capture a linear change component and anticipated quadratic change component from each person's time series equation and c) the "inertia" of RSA across recovery, that is, the degree to which RSA scores tend to remain unchanged from moment to moment via the autoregressive term estimate from each person's time series equation. Specifically, I expect that fewer symptoms of depression and anxiety will be reported when a disposition to use any of the four coping strategies is facilitated by a

mobilization of PNS activity during the recovery period. Furthermore, I hypothesize that each of the four coping strategies will predict fewer symptoms of depression and anxiety through the magnitude of change in RSA across recovery through a greater and more positive linear magnitude of change in RSA (i.e. augmentation of the amplitude of RSA). A similar mediation effect is expected for those participants who also evidence a more positive quadratic term (i.e. augmentation of the amplitude of RSA). I do not predict that greater "inertia" in RSA scores during recovery will mediate the relation between coping strategies and symptoms of depression and anxiety, because inertia may be associated with either augmentation of or reduction in the amplitude of RSA. It should be noted that, given the inconsistencies in the present coping literature, I hypothesize that the above parameters will be partial rather than full mediators in the relation between coping strategies and mental health symptoms.

CHAPTER 2: METHOD

Participants

The sample is comprised of Black undergraduate students recruited from a large public, historically White university in the southeastern United States. Criteria for participation included: undergraduate student status at the university, 18 years or older, fluency in written and spoken English, and self-identification as Black (including African American, Caribbean, African, and/or Bi-/Multi-racial). Research does suggest that these groups have different cultural considerations pertaining to their experiences of racial discrimination (e.g. Seaton, Caldwell, Sellers, & Jackson, 2008). However, members of these groups were included in order to recruit an adequate number of participants and to develop a sample that is representative of the Black college population from which it is drawn.

Students participated in this study from March 2014 – December 2016. The study consisted of two parts and 205 students completed part 1 while a subset of participants (n=116) completed both parts. The discrepancy between the numbers of participants who completed both parts compared to only the first part is due to study design, as there was no option to complete part 2 during 2014. The following descriptive information pertains to the sub-sample of participants (who completed both parts) which was used for analyses (n=116). Students were recruited via flyers (47.1%), campus organization or class announcements 5.8%), and the Psychology Department's undergraduate participant pool (47.1%). There were no differences on study variables as a result of method of recruitment. Students earned $20 cash or 2.5 participant

pool credits for participating in both parts of the two-part study ($10 or 1 participant pool credit if they only elected to complete the first part of the study).

About 73 percent of the sample was female, a percentage which was slightly higher than the proportion of Black female students enrolled at the university during the fall of 2015 (about 65%). The majority of participants were in-state students (78%), were not transfer students (85%), and had no known cardiovascular health concerns (95%). The age of participants in the sample ranged from 18 to 32 years, with 94% of participants being 22 years old or younger. Participants were well distributed with respect to year in college, with approximately 34% of students in their first year of college, 27% of students in their second year, 14% of students in their third year, and 25% of students in their fourth year or beyond. In terms of ethnicity, the majority of the sample identified as African American (83%). Thirteen percent of the sample identified as bi-/multi-racial, four percent of the sample identified as Afro-Central American/Caribbean, and less than one percent of the sample identified as Native African/ African immigrant. In terms of highest level of parental education, 32.5% of participants reported that at least one of their parents earned a Masters degree of higher, 32.5% reported a Bachelors degree, 17.5% reported an Associates degree or vocational training, 12.5% reported a GED, and 5% reported that their most educated parent did not complete high school.

Measures

Demographic Measures. For the purposes of the current study, demographic measures of interest as covariates include self-reported age, sex, and highest level of parental education.

Physiological Measures. Trained research assistants gathered psychophysiology measures during the laboratory visit. Using Einthoven's triangle method, electrodes were placed directly onto the skin to record each participant's heart rate continuously via electrocardiogram

(ECG). Biopac's MP100 data acquisition system was used to transmit and record ECG signal onto a laboratory computer via the AcqKnowledge program. These methods have been used extensively in psychophysiological research, including research with college students and Black populations (e.g. Neblett & Roberts, 2013).

Coping Strategies. Coping strategies were measured with two scales in order to capture mainstream coping strategies (i.e. those not related to any specific culture or racial/ethnic group) and culturally-relevant coping strategies (i.e. those developed with the unique considerations of a specific racial/ethnic group in mind). The Brief COPE (Carver, 1997) scale was adapted for the purposes of this study by presenting participants with 28 items corresponding to "things you may or may not be doing to deal with racial discrimination" on a scale from 0 (I haven't been doing this at all) to 4 (I've been doing this a lot). For the purposes of this proposal, three coping strategies were measured using the Brief COPE. Following the Brief COPE scale specifications, two questions comprised the active coping subscale (sample Cronbach's α=.76) and two questions comprised the spiritual coping subscale (sample Cronbach's α=.93). Example items include: "I've been concentrating my efforts on doing something about the situation I'm in" (active coping) and "I've been trying to find comfort in my religion or spiritual beliefs" (spiritual coping). Each subscale was calculated by summing two items. The social support subscale was created by using four items – two from the instrumental social support sub-scale (e.g. "I've been getting help and advice from other people") and two from the emotional social support sub-scale (e.g. "I've been getting comfort and understanding from someone"), with a sample Cronbach's α=.92. While the Brief COPE was not originally used with Black samples and has not received much attention in the discipline of psychology, research in public health and medicine has frequently used this scale to assess coping strategies that Black adults may employ in the face of

racial discrimination in investigations that examine the activity of the autonomic nervous system (e.g. Clark, 2003).

The John Henryism Active Coping Scale (JHAC12; James, Strogatz, Wing, & Ramsey, 1987) was used to measure one culturally-relevant coping strategy. John Henryism captures a strong personality predisposition to engage in effortful, active coping with stressors in the environment. The JHAC12 provides participants with 12 items about how they "see themselves", asking them to indicate how true or false each statement is "for them personally" on a scale from 1 (completely false) to 5 (completely true). Example items include "It's not always easy, but I manage to find a way to do the things I really need to get done," "I feel that I am the kind of individual who stands up for what he/she believes in, regardless of the consequences," and "Hard work has really helped me get ahead in life." In the current sample the internal reliability of the scale was good, with Cronbach's α=.81.

Mental Health Measures. Mental health symptoms of depression were measured using a modified Center for Epidemiological Studies Depression Scale (CES-D-12-NLSCY; Poulin, Hand, & Boudreau, 2005). The original CES-D scale was modified to contain fewer items and used simpler, more straightforward language for use with children and adolescents in a large Canadian population health survey. The CESD-12-NLSCY asks participants about how frequently they experienced 12 depressive symptoms in the past seven days on a scale from 0 (rarely or none of the time; 1-2 days) to 3 (most of the time; 5-7 days). Examples of symptoms include "I felt that everything I did was an effort," "I had trouble keeping my mind on what I was doing," and "I had crying spells." An overall score on depressive symptoms was computed by summing the scores on each item. While the CESD-12-NLSCY has not been frequently used with Black college populations it has been used successfully with Black adolescent populations

(e.g. Seaton, Upton, Gilbert, & Volpe, 2014). This scale was selected because its scores on this measure have been demonstrated to inform researchers about the risk of clinical depression and the psychometric investigations of the scale properties confirm cutoffs that are clinically useful. A score of 0-11 suggests minimal depressive symptoms, 12-20 suggests depressive symptoms in the clinical range, and 21-36 suggests very clinically elevated depressive symptoms. Furthermore, the original CES-D measure has been widely used in large-scale national studies of mental health in the United States and does appear to be sensitive to the considerations of several racial/ethnic groups. In the current sample the internal reliability of the scale was good, with Cronbach's α=.82.

Mental health symptoms of anxiety were measured using the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI presents participants with 21 symptoms and asks to what degree they have been bothered by each of the symptoms during the past month on a scale from 0 (not at all) to 3 (severely – it bothered me a lot). Examples of symptoms include "fear of worst happening," "heart pounding/racing," and "fear of losing control." An overall score on anxiety symptoms was computed by summing the scores on each item. The BAI has been found to be a good measure of anxiety with non-clinical college populations (Creamer, Foran, & Bell, 1995) and also provides clinically relevant cutoffs. A score of 0-21 suggest low anxiety, 22-35 moderate anxiety, and a score of 36 or more may be a potential cause for clinical concern. In the current sample the internal reliability of the scale was good, with Cronbach's α=.85. These measures of depression and anxiety are appropriate for Black young adults, with reliability estimates in prior studies ranging from α =.73 to .95.

Procedure

The study consisted of two sessions – an online session (Session 1) and a laboratory visit (Session 2). In Session 1 students complete an online Qualtrics questionnaire including consent, eligibility, and measures listed above. Session 2 was held a minimum of 72 hours following completion of the questionnaire in order to dampen any stress from Session 1. Upon arrival in the laboratory, participants were greeted by a Black research assistant (BRA) and a White research assistant (WRA). Throughout the laboratory task the participant was audiorecorded, unless they declined. The BRA conducted the session with the exception of the stress task, which was conducted by the WRA. After obtaining consent, the BRA secured ECG leads to the participant, instructed the participant to relax for 5 minutes, and exited the room. The BRA's exit began a 5-minute period in which the participant was alone in the room and a record of heart rate for the resting baseline period was obtained. For the purpose of the current study baseline measures are not examined. Next, the WRA entered the room and presented the participant with the stress task, which combined elements of two paradigms well-established in the literature: the Trier Social Stress test (Kirschbaum, Pirke, & Hellhammer, 1993) and vignettes of racial discrimination (Neblett & Roberts, 2013). In this task the WRA told the participant that the task required him/her to imagine himself/herself in a scenario. The WRA told the participant that the WRA would select a random scenario from a box of everyday scenarios students at UNC may experience and read the scenario aloud, after which the participant would be required to respond verbally to the scenario. While the WRA ostensibly draws a scenario from the box at random, all the scenarios are identical. The scenario that the WRA reads is as follows:

> It is the first day of class. The instructor asks you to exchange contact information and get to know the person sitting next to you. The person sitting next to you looks like me. After exchanging contact information, the person sitting next to

> you says, "Oh wow, you got into (college name)! So are you actually really smart or did they just have to let a certain number of Black people in?"

After the scenario is read, the participant responds verbally. While the specific content of their answer is not considered in the context of the current analyses, it is used to determine the RSA value during the stress to use in analyses as a control variable. Following the scenario, the WRA instructed the participant to engage in another 5-minutes of relaxation – the "recovery" period. Following this period, the BRA re-entered the room, debriefed the participant, invited feedback and questions, and provided a list of community organizations and health resources on campus.

CHAPTER 3: DATA ANALYSIS OVERVIEW

Initial Data Preparation and Inspection

In order to estimate RSA from the psychophysiological data collected, each individual's ECG waveform was converted into an IBI (interbeat interval) file, which graphs time in seconds on the X-axis and RR(s) (aka RR interval) on the Y-axis. Greater RR interval means more time between heartbeats, where R is a point (aka the beat) corresponding to the peak of the QRS complex of the ECG wave and RR is the interval between successive Rs (aka beats). The QRS complex is a graphical representation of parts of the ECG waveform that together correspond to the depolarization of the ventricles of the heart – with positive (upright) deflections termed R waves, and negative (inverted) deflections termed Q and S waves. IBI files for each participant were then submitted to RSAseconds (Gates, Gatze-Kopp, Sandsten, & Blandon, 2015), which is a program that was developed to estimate second-by-second RSA using a multiple window technique and spectrogram analysis. This generated a continuous series of approximately 150 RSA timepoints for each participant during recovery, which was then submitted to a time series analysis. RSAseconds was also used to calculate each participant's RSA value immediately following (one second after) delivery of the scenario during the challenge period from each participant's IBI sequence. This value was used as a control variable ("RSA at stress") in the final analysis below.

Specific Aim 1 – Unique Associations between Coping Strategies and Mental Health Symptoms

A regression analysis within a structural equation framework examines Specific Aim 1 – testing the relation between coping strategies and mental health. In this framework, symptoms of depression and anxiety are concurrently regressed on four coping strategies – active coping, social support, spiritual coping, and John Henryism. Covariates include participant sex, age, and parental education. A covariance is modeled between depression and anxiety symptoms as they have often demonstrated comorbidity in research with college populations (Eisenberg, Gollust, Golberstein, & Hefner, 2007). In addition covariances are modeled between each coping strategy as they share the same general construct. Model fit was first assessed. If model fit was adequate regression, parameters were examined to determine if each coping strategy was independently associated with symptoms of depression and/or anxiety.

Specific Aim 2 – Intraindividual Estimates of RSA during Recovery

Time Series Analysis as a Tool for Modeling Intra-individual Variability

Specific Aim 2 aims to arrive at estimates of patterns of RSA during recovery via time series analysis. Specific Aim 2 is focused solely on obtaining these estimates, which will be explicitly modeled within a SEM framework in Specific Aim 3. The statistical approach of time series analysis models a time series, defined as "successive observations of one or more variables obtained on a given experimental unit where observations are dependent upon each other due to an underlying process" (Baltes, Reese, & Nesselroade, 1988, p. 171). This approach is optimal when inferences about the nature of such processes are desired, but the lack of independence among observations prevents use of many conventional statistical techniques (Baltes, Reese, & Nesselroade, 1988). While there are many statistical approaches that appropriately adjust for dependence within repeated measures data (i.e. multilevel modeling, some latent growth curve

modeling approaches), I expect that patterns of RSA change during recovery will be best understood by estimating individual parameters rather than group or sample-level means. Indeed, Porges (1976) called specifically for the use of time series in examining intraindividual variation in physiological processes. While psychologists often examine interindividual (between-person) variation with the assumption that interindividual variation is indicative of phenomena at the intraindividual level, research suggests that this assumption remains largely unsupported (e.g. Molenaar, 2004). Instead, "time-dependent variation within a single individual" is a more appropriate focus for examining questions about the parameters of intraindividual patterns (Molenaar, 2004, p.202). Furthermore, time series analysis is especially appropriate for continuous data numbering in the hundreds where points in time are equally spaced (Glass, Wilson, & Gottman, 1972). This statistical approach is optimal because my second specific aim consists of modeling patterns of intraindividual variation in RSA during recovery.

Introduction to Time Series Analysis

There are multiple components of a time series. Variation within a time series may be divided into four parts – trend, seasonal, cyclical, and irregular components (Persons, 1919). Trend is any systematic change in the level of a series. For example, a long-term increase or decrease in the pattern of data would indicate a trend. Both the direction and slope of a trend may remain constant or change throughout the course of the series and the trend need not be linear. Next, a seasonal component is a repeating pattern of increase and decrease that occurs consistently throughout the time series. Seasonal components are traditionally restricted to patterns of time that repeat over durations related to aspects of a calendar (e.g. months of a year, days of the week). However, with the present RSA data seasonal components of the time series are not anticipated, largely because the durations necessary to quantify such a consistently

repeating component are not intuitively identifiable if they are present at all (i.e. number of seconds that could quantify substantively different "segments" of time during recovery). A cyclical component, similar to a seasonal component, entails a pattern of fluctuation that reoccurs across periods of time. In contrast, the duration of a cyclical component is not fixed, meaning its magnitude can vary over time and may not be attributable to any specific time periods. Therefore, cyclical components may indeed be present in the current time series analysis as one's RSA values may fluctuate across time during recovery but the consistency of this fluctuation may vary across time intervals. Often cyclical components may be visually represented as together with their trend components because they may be embedded in a larger cyclical pattern over a period of time. Finally, irregular components quantify any remaining unsystematic variability in the time series after accounting for the systematic trend, seasonal, and cyclical components. Another important component of a time series is its autocorrelation. The autocorrelation in a time series is the correlation between each observation in the series across time, i.e. the degree to which points in a series are impacted by prior points in that series. Autocorrelations may be of any length, referred to as the lag of the autocorrelation. For example, a lag-1 autocorrelation indicates the correlation between the data point under examination and the point immediately preceding it. Another element of a time series is stationarity, typified by a mean, variance, and covariance that remain constant throughout the series. Stationarity is an important assumption as the values of the time series are only considered accurate estimates of population values when the mean, variance, and covariance remain constant.

Specific Application of Time Series Analysis to Research Aim 2

Time series analyses are employed to give mathematical expression to each individual's time series for RSA during recovery, with the goal of estimating patterned elements in each

individual's time series. Time series analyses (n=116) was conducted in several steps. This process has two goals: 1) to describe dynamic patterns of RSA during recovery for each individual; and 2) to extract model estimates that define these patterns for each participant so that they may be used in further analyses.

Step 1: Fit a separate regression model to each time series that quantifies each participant's RSA pattern during recovery (n=116).

First, each time series was modeled using a regression framework according to the following model: $y_t=b_0+b_1t+\varepsilon_t$, where t is the time variable, b_0 is the level of the series when t=0, b_1 is the amount of change in the series associated with a one-unit increase in time, and ε_t is the random error. If this linear regression model fully accounts for the trend, the residual error series will not contain any remaining trend component. Therefore, the residual error series was examined to determine if a higher-order polynomial term must be included in order to construct a better-fitting model, especially if a quadratic is theoretically anticipated for some participants. If a linear model is found to be insufficient, a quadratic parameter was added to the regression, b_2t_2. After the addition of the quadratic parameter, the residual error series was re-examined to determine if any trend component remained. An examination of R^2 change following each progressive model specification guided the construction of a most optimally fitting model for each time series.

Step 2: It was likely that an autocorrelation will remain despite fitting a quadratic parameter to the data. Therefore, I examined a plot of the strength of the autocorrelation function (ACF) of the residuals for the optimally fitting model from Step 1 for any large autocorrelations across lags. Only five percent of autocorrelations are expected to reach statistical significance if

the residuals are appropriately unsystematic (i.e. irregular, a "white noise series"). The ACF plot charts the $p < .05$ criterion for statistical significance, allowing us to understand the degree to which autocorrelation is present in the series. If the residuals were found to be autocorrelated, analysis proceeded to the next step. If residuals were not autocorrelated, analysis proceeded to Step 4.

Step 3: If the residuals were autocorrelated, ARIMA terms were included. This specific approach was taken because a degree of non-stationarity in each time series was anticipated, due to potential quadratic and other time-varying trends evidenced in prior literature which examines RSA.

Overview of ARIMA models. Components of ARIMA models may be included within an existing regression model to improve its accuracy. The name ARIMA refers to three distinct elements of a class of models whose goal is to explain the autocorrelation in a given time series. The AR(*p*) component models the autoregressive component of the time series with *p* terms, where *p* is the number of preceding observations that exhibit significant autocorrelation. For example, an ARIMA model of type (1, *d, q*) includes one predictor, the observation immediately preceding the current value, and an ARIMA (2, *d, q*) model includes two predictors, the first and the second preceding observations (Jebb, Tay, Wang, & Huang, 2015, p. 16). An order 1 autoregressive component can be expressed mathematically as: $y_t = \Phi_1 (y_{t-1}) + \varepsilon_t$, where Φ_1 is the autoregressive coefficient and y_{t-1} is the immediately preceding observation. This model can be expanded for quadratic patterns by the inclusion of an additional parameter: $\Phi_2 (y_{t-2})$.

The MA(*q*) component models the moving average terms, defined as the influence of "random shocks" which vary across time and interact with such complexity that their behavior is

ostensibly random (McCleary, Hay, Meidinger, & McDowell, 1980, p.40). This component accounts for the notion that random shocks result in autocorrelation because each data point may contain a trace of the lingering effects of prior unobserved shocks. An order-1 moving average component can be expressed mathematically as: $y_t = \theta_1 (\varepsilon_{t-1}) + \varepsilon_t$, where ε_t is the value of the random shock at t, ε_{t-1} is the value of the previous random shock, and θ is the coefficient of the shock. This model can be expanded for quadratic patterns by the inclusion of an additional $\theta_2 (\varepsilon_{t-2})$. Model building strategies (discussed below) were employed to determine if AR terms, MA terms, or both AR and MA terms were needed for model specification. Often, autocorrelation may be explained by either AR or MA terms and such models are often more easily interpretable and parsimonious (Jebb et al., 2015).

Finally, the I(*d*) component does not add predictors to the modeling equation, but rather indicates what operation has been performed on the series in order to render it stationary. While the exact process of differencing for each time series will be discussed more specifically below, in general terms, making the series stationary is often accomplished by taking either the first differences or second differences (i.e. the first differences of the first differences) of the original series. The series must be stationary before any AR or MA terms can be included (Cowpertwait & Metcalfe, 2009).

Step 3a: Determine if the series is stationary. First, a plot of the series was examined for systematic changes in mean level and variance. More formally, an augmented Dickey-Fuller (ADF) test was also conducted to test for stationarity.

Step 3b: Transform the series to stationarity. If the mean, variance, and/or autocorrelation are not constant over time, the series was differenced in order to establish

stationarity. Differencing transforms the values of a series into a series of the differences between observations adjacent in time. Each successive order of differencing should further remove trend and reduce the overall series variance. In practice the order of differencing rarely needs to be greater than two in order to stationarize the series (Jebb et al., 2015, p. 21). As a starting point, first differencing was conducted for each time series with a non-significant ADF test. If the series was still not stationary, second differencing was then conducted and a new ADF test was examined.

Step 3c: Determine how many AR and MA terms will be required to explain the series autocorrelation by inspecting ACF and PACF (partial autocorrelation function) plots of the residual error series. Similar to autocorrelation function plots, PACF plots chart the strength of the partial autocorrelation function (the autocorrelation of each lag after controlling for the autocorrelation due to all preceding lags, McCleary et al., 1980) across different lags in the series and include the $p < .05$ criterion for statistical significance. Patterns in these plots guide the inclusion of AR and MA terms. For example, an autocorrelation that is best explained by AR terms has a steadily decaying ACF and a PACF that drops after p lags, signaling that the series would require p AR terms. An autocorrelation that evidences a drop-off in the ACF after q lags and a gradually decaying PACF is best explained by MA terms, signaling that the series would require q MA terms. Often only one or two AR or MA terms are required per time series (Jebb et al., 2015).

Step 3d: Fit a parsimonious model via the inclusion of ARIMA components as determined above. Times series can be modeled with AR terms, MA terms, or a mixture of AR and MA terms. However, the inclusion of AR terms is often preferred because their interpretation is more straightforward (one effect carrying on through time rather than "shocks").

After fitting the model, if the inclusion of ARIMA terms has been successful in explaining the autocorrelation of a stationary series, the residual error should appear as unsystematic error variance, the series should have a mean of zero, and the series should have some constant variance. Therefore, visual inspection of the ACF and PACF plots helped assess model adequacy. In these plots, all autocorrelations are expected to be zero with 5% expected to be statistically significant due to sampling error (Jebb et al., 2015, p. 18). Furthermore, a Ljung-Box test (Ljung & Box, 1978) was conducted to determine if the model residuals were indistinguishable from a random white noise series. If there was a fair magnitude of remaining autocorrelation, the model was re-specified accordingly.

Step 3e: If autocorrelation remained, multiple models were specified and then compared. The process of fitting ARIMA models is iterative and exploratory, essentially data driven (Jebb et al., 2015). However, following modeling conventions, non-mixed models were ruled out prior to fitting more complex mixed models. Re-specification of the model proceeded from the continued examination of the pattern of residuals in ACF and PACF plots. The search for the best-fitting model was facilitated by model comparison utilizing the AIC, a model fit criterion that incorporates penalties for model complexity. Smaller AIC values indicate a better relative fit of the model to the time series. Models within two AIC points are comparable, a difference of 4-7 points indicates considerable support for the model with the smaller AIC, and a difference of 10 points or more indicates full strong support for the model with a smaller AIC (Jebb et al., 2015, p. 20). Models were appropriately re-specified using this framework until strong support for model fit was established.

Step 4: Once adequate model fit was established, each estimate of the time series was output to a separate data set for the analysis of research aim 3.

Specific Aim 3 – Mediation of the Relation between Coping and Mental Health Symptoms by Estimates of RSA during Recovery

Regression analysis within a structural equation framework examined Specific Aim 3 – examining RSA estimates as mediators in the relation between coping strategies and mental health (see Figure 1 for full model). In this model, symptoms of depression and anxiety were regressed on the estimates of the time series. While these estimates originally included the intercept, slopes, and autoregressive term of the series, the "Results" section below describes how the time series mean, variance, and number of autoregressive and moving average terms were included instead. These estimates were regressed on four coping strategies – active coping, social support, spiritual coping, and John Henryism. Covariates included participant sex, age, and parental education. An additional covariate in this model was RSA value at the stress period, in order to adequately control for the magnitude of PNS activity in the face of the racial discrimination challenge. A covariance was maintained between depression and anxiety symptoms and between each of the coping strategies as noted in Specific Aim 1. Model fit was first assessed. If model fit was adequate, regression parameters were examined to determine the significance and interpretations of each of the regression pathways. Mediation was determined via significance tests of the indirect effects of coping strategies on mental health symptoms.

CHAPTER 4: RESULTS

Descriptive Statistics

Descriptive statistics for study variables are presented in Table 1 (n=116) and Table 2 (n=205). Descriptive statistics and distributions of variables remained consistent across these two samples and therefore the smaller sample (n=116) will be discussed below in order to present a more conservative description of sample characteristics. According to the clinical cutoffs suggested by the depression and anxiety scales, a majority of participants self-reported minimal depressive symptoms (67.2%) and low levels of anxiety (94.8%), as would be expected in a non-clinical sample. However, clinically significant symptoms of depression were reported by approximately one-third of participants, with 27.6% of participants reporting symptoms within the clinical range and 5.2% of participants reporting very clinically elevated symptoms. In contrast, only 4.4% of participants reported symptoms indicative of a moderate level of anxiety, and only .9% of participants reported anxiety symptoms indicative of a potential cause for clinical concern.

Symptoms of depression and anxiety were positively skewed, with more individuals indicating fewer symptoms of depression and anxiety. The distribution of depressive symptoms sufficiently approximated a normal distribution (skewness value of .72) such that a transformation of the variable was not deemed necessary. However, the distribution of anxiety symptoms appeared more starkly positively skewed (skewness value of 1.66) and was therefore submitted to a square root transformation (new skewness value=.16). Each of the mediating

variables approximated a normal distribution with the exception of the RSA variance of the time series. The distribution of RSA variance was noticeably positively skewed (skewness value=1.90), therefore a square root transformation was conducted (new skewness value=.79).

Three count variables also served as mediators (their inclusion is discussed in Specific Aim 3 below); however their distributions were sufficiently normally distributed. The number of autoregressive terms ranged from zero to four, with one participant requiring zero terms, approximately 8% of participants requiring one term, 34% of participants requiring two terms, 46% of participants requiring three terms, and 11% of participants requiring four terms. The orders of differencing ranged from zero to two, with approximately 37% of participants not requiring any differencing of their time series, 24% of participants requiring one order of differencing, and 40% of participants requiring two orders of differencing. The number of moving average terms ranged from zero to four, with 35% of participants requiring zero terms, 33% of participants requiring one term, 21% of participants requiring two terms, 8% of participants requiring three terms, and 2% of participants requiring four terms. This distribution, while skewed, sufficiently approximated a normal distribution (skewness value=.98). Therefore, these variables were not submitted to transformations.

While distributional assumptions are not made regarding predictor variables, it is important to note that the majority of coping measures were approximately normally distributed with the exception of spiritual coping. The distribution of spiritual coping was approximately bimodal, with majorities of individuals indicating that they never used spiritual coping when experiencing racial discrimination or that they use spiritual coping very frequently when experiencing racial discrimination. No potential outliers in any variable were observed via inspection of variable plots.

Intercorrelations between Study Variables

A correlation matrix for study variables is presented in Table 2 (n=116) and Table 4 (n=205). Once again there were few substantive differences in results for these two samples, with the exception of anxiety being associated with John Henryism and the order of differencing being associated with spiritual coping in the larger sample. Therefore, intercorrelations are discussed in terms of the smaller sample (n=116) to provide a more conservative description of intercorrelations amongst study measures as the larger sample included a greater degree of missing data. Intercorrelations indicate significant relations between active coping, social support, and spiritual coping ranging from *r* values of .24 to .51, indicating small to moderate positive correlations. This is expected given that these variables are measured using the same scale. John Henryism was significantly associated with spiritual coping (r=.22, p=.041), but was not significantly associated with active coping or social support. As anticipated, depression and anxiety were significantly positively correlated (r=.52, p<.001), such that more symptoms of depression were associated with more symptoms of anxiety. John Henryism was significantly negatively associated with symptoms of depression (r=-.25, p=.020), indicating that more frequent use of John Henryism was associated with fewer symptoms of depression in the current sample.

As anticipated, the control variable of RSA value at stress was significantly associated with the mean RSA value during recovery (r=.56, p<.001). Similarly, number of autoregressive terms was significantly negatively associated with RSA variance during recovery (r=-.23, p=.015), indicating that as the number of lags that explain a significant amount of unique autocorrelation in the time series increases there is less variability within the time series. This makes intuitive sense, as the number of autoregressive terms is an indication of the degree to

which past RSA values are uniquely related to any given RSA value on a second-by-second basis, suggestive of increased inertia in RSA values across time during recovery. The number of autoregressive terms was also significantly negatively associated with the number of moving average terms included in the time series model (r=-.36, p<.001). This relation can be explained in terms of the functions of autoregressive and moving average terms in a time series model. In the estimated time series models, including an autoregressive term is equivalent to multiplying any given RSA value in the series by a factor of $n-\varphi_1 B$, while including a moving average term is equivalent to multiplying any given residual (error) term in the series by a factor of $n-\theta_1 B$, where n is the number of autoregressive or moving average terms, B is a backshift operator, φ is the autoregressive coefficient, and θ is the moving average coefficient. In this way, the autoregressive and moving average terms serve to negate the influence of one another in the time series model. Order of differencing was also negatively associated with the number of autoregressive terms (r= -.50, p=.013) and the number of moving average terms (r= -.27, p=.03), indicating support for its use as a control variable in subsequent analyses.

Missingness

Of the total number of participants who completed both parts of the study (n=116), there was a degree of missingness on study variables. There was no missing data on the outcome variables of symptoms of anxiety and depression. One participant declined to provide responses to the Brief COPE, which resulted in a sample size of 115 for measures of active coping, social support, and spiritual coping. The measure of John Henryism was added as a study modification approved for use four months after data collection had commenced. Therefore, only 85 participants were able to complete the John Henryism measure by study design. For those variables associated with the time series analysis (number of autoregressive terms, number of

moving average terms, variance of RSA series, and mean of RSA series), two participants have missing data due to computer malfunctions during administration of the protocol that did not enable participants' IBI intervals to be correctly recorded during the recovery period. Furthermore, four participants have missing data on the control variable (RSA value at time of stress) due to similar computer malfunctions which affected recording during the stress period. As the missingness described here is largely due to study design, multiple imputation was not considered a viable option. Furthermore, it is feasible to assume that missingness is sufficiently MCAR (missing completely at random) in the current study.

Table 3 presents the number of participants with data on each variable for the larger sample. The number of participants with missing data on RSA variables remains the same as described above due to study design. Similarly, there was some missing data on John Henryism (n=72) due to its addition as a study modification. Twenty participants were missing data on coping strategies because they declined to complete the Brief COPE measure. There was no missing data on mental health outcomes. The majority of this missingness is due to study design.

Specific Aim 1 – Unique Associations between Coping Strategies and Mental Health Symptoms

Because Specific Aim 1 did not require information from the laboratory visit, a full model utilizing all study participants (n=205) was first run to assess the unique impact of coping strategies on symptoms of depression and anxiety. Using MPlus software, a path analysis (see Figure 2) was implemented in which symptoms of depression and anxiety were regressed on active coping, social support, spiritual coping, and John Henryism. Sex, age, and parental education were exogenous covariates in the model and covariances were estimated between symptoms of depression and anxiety and between coping strategies. Results of this model are

presented in Table 5. This model was a good fit for the data, as indicated by multiple model fit indices (see Table 7), therefore model estimates were examined.

Results indicated that John Henryism was significantly associated with fewer symptoms of depression (β=-.23, *p*=.010) and anxiety (β= -.23, *p*=.010), above and beyond the other coping strategies in the model. More frequent use of spiritual coping was significantly associated with more symptoms of anxiety (β= .16, *p*=.042) above and beyond the other coping strategies in the model. Active coping and social support were not uniquely associated with symptoms of depression or anxiety. Participant sex was significantly associated with the use of spiritual coping, indicating that female students more frequently used spiritual coping in response to instances of racial discrimination (β= -.39 *p*=.012). Significant covariances between depression and anxiety and some coping strategies were also observed (see Figure 3 for significant paths). While this model was a good fit for the data, R^2 estimates indicate that this set of predictors explains just eight percent of the variance in depression and six percent of the variance in anxiety, indicating small effect sizes (Cohen, 1988). This further justifies the inclusion of additional variables such as partial mediators of the relation between coping strategies and mental health symptoms.

The same model (see Figure 2) was also evaluated using our sub-sample of participants who completed the laboratory visit (n=115). Results of this model are presented in Table 6 and Figure 4. This model was a good fit for the data, as indicated by multiple model fit indices (see Table 7), therefore model estimates were examined. Sex remained significantly associated with the use of spiritual coping, indicating that female students more frequently used spiritual coping in response to instances of racial discrimination (β= -.24 *p*=.008). However, John Henryism was no longer significantly associated with symptoms of depression and anxiety in this reduced

sample. While this model was a good fit for the data, R^2 estimates indicate that the amount of variance in symptoms of depression and anxiety explained by this set of predictors is also small (depression, R^2=.05; anxiety R^2=.07). This model also justifies the inclusion of additional variables such as partial mediators of the relation between coping strategies and mental health symptoms.

From these results, it is clear that if we nearly double the number of participants in our sample, our model is a better fit for the data. While the full model sample (n=205) had more power to detect significant associations between coping strategies and symptoms of depression and anxiety, analysis will proceed to subsequent aims. Partial mediation was hypothesized, therefore, while there do not appear to be significant relations to mediate in the smaller sub-sample (n=115), a direct relation between predictors and mediators and/or predictors and outcomes would still provide important information about hypothesized relations. Furthermore, Aim 3 will analyze the full incomplete data set using maximum likelihood estimation, which uses each available case in computing estimates. Maximum likelihood estimation computes separate likelihoods for participants with complete data on all study variables and participants with incomplete data on some study variables. Both likelihoods are maximized within the same equations to find final estimates and standard errors, both of which are considered unbiased estimates of population parameters.

In summary, using data from 205 Black undergraduate students, results indicated that two coping strategies were uniquely associated with symptoms of depression and anxiety. John Henryism was significantly negatively associated with symptoms of both depression and anxiety, such that more frequent use of John Henryism to respond to racial discrimination was associated with fewer symptoms of depression and anxiety. More frequent use of spiritual coping was

significantly associated with more symptoms of anxiety in the present sample. Female students self-reported using spiritual coping significantly more frequently than males in the present sample.

Specific Aim 2 – Intraindividual Estimates of RSA during Recovery

In order to provide an overall picture of RSA change across the laboratory visit, Figure 5 displays spaghetti plots of five randomly selected participants' RSA across baseline, stress, and recovery. Only five participants were selected for graphing in order to clearly visualize individual trajectories. RSA values at stress are indicated in the clear window. Time in seconds is graphed on the horizontal axis, however the duration of the stress task varied from participant to participant, therefore each RSA series was graphed by centering the values at each participant's RSA value at stress in order to visually inspect the impact of the stress task on RSA values. Therefore, it should be noted that all participants did not experience stress at exactly 541 seconds and therefore the horizontal axis should not be interpreted as such. Furthermore, because the duration of the stress task varied from participant to participant, comparable cutoffs for baseline and recovery periods could not be included on the same graph. Details of this plot are provided in order to further facilitate visual inspection. Three participants who demonstrated low to moderate variability in RSA across the laboratory paradigm are presented in the first detail, while participants who demonstrated high variability in RSA across the laboratory paradigm are presented in the second detail. These plots demonstrate a large degree of variability in participants' responses across the laboratory paradigm. They also provide initial support for the effectiveness of the ability of the stress task to produce physiological reactivity as participants experienced a reduction in amplitude of RSA immediately following delivery of the stressor.

Results of each time series analysis performed are presented in Table 6. As an illustration of the process to which each participant's RSA time series was submitted, one case will be discussed in detail (participant 506). This process was completed for 114 participants. Time series analysis was conducted using R software and a number of R packages, including: ts, lmtest, tseries, and forecast.

Data for the participant was first imported into R from a text file containing the participant's second-by-second RSA values (computed from the RSAseconds program). Next, visual inspection of the time series decomposition provided initial exploration of the components of each series (Figure 6). A multiplicative decomposition model was selected because a quadratic trend was hypothesized for RSA values over time. According to this multiplicative decomposition model, each value of the time series is the product of its trend/cyclical, seasonal, and random components, which are often difficult to observe visually without decomposition. It should be noted that seconds were binned in 10 second increments in order to generate this decomposition. Furthermore, in this decomposition it is important to note that the trend and cyclical components of the time series are both contained in the "trend" plot, which is defensible because cyclical components of a time series are not of a fixed duration in the series and therefore are not attributable to any naturally-occurring time points (as a seasonal component would be). Both trend and cyclical components of the time series concern patterns of fluctuation that vary over time. The top section of the figure is a plot of the raw "observed" time series. The trend section of the figure preliminarily indicates that RSA may follow a higher-order trend and perhaps a quadratic will not be sufficient. As seconds were binned in 10 second increments, the seasonal component here is not of interest as it was essentially forced upon the decomposition. Inspection of the raw time series verifies that there is not a meaningful seasonal component to

the series that warrants examination. Finally, the bottom section of the figure indicates irregular variation in the time series, any remaining variation in the series after the above systematic components have been removed.

After inspection of the time series decomposition, standard regression methods were employed to model change in RSA across recovery. This method assessed how the trend in the series could be best described as a function of time. As the time series decomposition indicated a higher-order trend than a quadratic model, a cubic model was first fit to the time series analysis using ordinary least squares estimation, resulting in a statistically significant adjusted R^2 (.22, $p<.001$). In order to confirm that a cubic model was the most optimal for the data, quadratic and linear models were also fit and evaluated (R^2 values presented in Table 8). After fitting a cubic regression model to the time series, a graph of the partial autocorrelation function (PACF) of the residuals of this model was examined to determine if there was any remaining significant autocorrelation. As we can see in the graph of the PACF (Figure 7), three lags exceeded statistical significance, where the null hypothesis indicates that no autocorrelation remained in the series. As significant autocorrelation remained in the time series, ARIMA terms were deemed necessary to adequately characterize the time series.

Before adding any autoregressive (AR) or moving average (MA) terms, the series must be stationary. Transformation to stationarity does serve to de-trend the series, however this step is necessary to arrive at estimates of autoregressive and moving average terms that demonstrate the least bias possible. Therefore, an Augmented Dickey-Fuller Test, which tests the null hypothesis that the series is not stationary, was first performed. Results indicated that the series was not stationary (ADF= -3.42, p=.053), and thus the original series was first differenced and re-examined for stationarity. However, an Augmented Dickey-Fuller Test once again indicated

that the series did not achieve stationarity (*ADF*= -3.13, *p*=.108). Therefore, the first difference of the first difference of the original time series was calculated (i.e., the series was second differenced) and this time series did achieve stationarity (*ADF*=-7.51, *p*<.001). This transformed time series was thus subsequently used to fit models with AR and MA terms.

In order to determine the number of AR and MA terms sufficient for explaining the time series, a baseline model was first fit specifying zero AR and zero MA terms. Plots of the autocorrelation and partial autocorrelation function of the residuals for this model were inspected (see Figure 8). The partial correlations here represent the degree of correlation between RSA at a given second and a one second lag of RSA that is not explained by the correlation between RSA at lags greater than one second. It is the unique correlation at each given lag, the difference between the actual correlation and the expected correlation due to the tendency of that correlation to persist across time. The autocorrelation is the degree to which RSA at a given time is correlated with RSA at the following time point, which carries on through time. This correlation squared is the degree to which RSA at that time point is correlated with two time points in the future. Observing the participant's autocorrelation function plot, it can be seen that significant autocorrelation persists across many time lags, in segments relevant to positive and negative relationships in time. However, the partial autocorrelation function plot indicates that two or three unique autocorrelations should be sufficient to characterize this model, as exemplified by a sharp (and non-significant) drop in the autocorrelation between lags at lag-4.

Therefore, an ARIMA model with two autoregressive terms was first fit to the time series. This model resulted in a model fit value of AIC= -1054.23. A Ljung-Box test was conducted to test the null hypothesis that the model residuals are uncorrelated after the addition of two autoregressive terms. A sufficient model necessitates failing to reject the null hypothesis,

indicating that there is no remaining autocorrelation to characterize in the time series. The Ljung-Box test indicated that significant residual correlation remained (χ^2= 11.56, p <.001) and thus this model was rejected. Next, a model with three autoregressive terms was fit to the time series. This model resulted in a model fit value of AIC= -1067.46. A Ljung-Box test indicated that model residuals were uncorrelated after the addition of three autoregressive terms (χ^2= .41, p =.524), and thus this model was retained for comparison against competing models. As a general rule, when increasing the orders of AR or MA terms in a given model, the AIC value will increase. As the most optimal model will have a non-significant Ljung-Box test and the smallest AIC value possible, the general process for identifying models includes 1) examining the PACF and ACF to determine how many AR and/or MA terms may be necessary, 2) specifying an appropriate number of AR terms or MA terms – as models that contain only AR or MA terms are more parsimonious, and 3) examining the sequence of models one AR or MA term less than any model which passes the Ljung-Box test as a competing model, in order to detect any noticeable differences in the AIC that may indicate that a mixed model (which includes both AR and MA terms) is a better fit.

Thus, a model that included two autoregressive terms and one moving average term was next fit to the time series. This model resulted in an AIC value = -1069.71. Furthermore, the Ljung-Box test indicated that model residuals were uncorrelated after the addition of two autoregressive terms and one moving average term (χ^2= .003, p =.952). In order to confirm that this was the simplest model in which residuals were uncorrelated, a model including one autoregressive term and two moving average terms was fit to the time series (AIC= -1013.35). The Ljung-Box test indicated that model residuals remained correlated (χ^2= 4.27, p =.039) and thus examination of competing models concluded. As the AIC values for the (2, 2, 1) model and

the (3, 2, 0) model were 2.25 values apart, they may be considered comparable (Burnham & Anderson, 2004, p. 271). Therefore, I selected (3, 2, 0) as the optimally fitting ARIMA model, as a model with only autoregressive or moving average terms is inherently more parsimonious. In this model, all autoregressive terms were significantly different from zero ($p<.001$).

In summary, to address Specific Aim 2 each participant's time series was submitted to an iterative data-driven process to arrive at optimal numerical estimates of trajectories of RSA across recovery. First, each time series' trend component was inspected visually using multiplicative time series decomposition in order to gauge the functional form of the trajectory independent from the random and seasonal components. Next, this functional form was fit to each time series using standard regression methods and competing functional forms were inspected. After determining optimal fit of a given functional form, a PACF plot of the residuals for this optimally fitting model was examined to determine if any remaining significant autocorrelation existed in the residuals. If remaining significant autocorrelation existed, in order to arrive at unbiased estimates of regression predictors, autoregressive and moving average terms needed to be added to account for this significant autocorrelation. Before this was accomplished each time series was assessed for stationarity and transformed to stationarity if it did not meet this assumption of ARIMA models. PACF and ACF plots of the stationarized series were then examined to determine if an autoregressive or moving average signature was evidenced in the autocorrelation plot and this information was used to fit an initial model with the appropriate number of terms. Competing ARIMA models were examined for each participant, with AIC values and Ljung-Box test results guiding decision on final model parameterization.

Specific Aim 3 – Mediation of the Relation between Coping and Mental Health Symptoms by Estimates of RSA during Recovery

Contrary to my hypotheses, all time series were not best characterized by a quadratic trend in Specific Aim 2. About 71 percent of participants' times series were best represented as a cubic trend, 25 percent of participants' time series were best represented as a quadratic trend, and 4 percent of participants' time series were best represented as a linear trend. Furthermore, while I anticipated that one or two autoregressive and/or moving average terms would be sufficient to characterize each time series, best fitting models ranged from including 0-4 autoregressive terms and 0-4 moving average terms. About 40 percent of participants' time series needed second-order differencing and 24 percent of participants' time series needed first-order differencing. Given the wider variety of time series results than hypothesized, the original terms included as mediators in Specific Aim 3 needed revision. The magnitude of trend (aka slope) and ARIMA terms could no longer be included in the model for Specific Aim 3 as the number and significance of these terms in the time series model varied widely from person to person. Indeed, when a series was transformed to stationarity, the trend component was subsequently transformed to be held constant across time, thereby removing it as a meaningful characterization of the series in order to arrive at unbiased estimates. Therefore, instead of using the magnitude of AR and MA terms as mediators, the number of AR and MA terms in the selected model was used instead. The order of differencing was also included as a control variable in the model. Due to the variability in differencing required in the present sample, this variable was included to control for the change in the nature of the AR and MA terms in the model as a function of fitting these terms to series with various orders of differencing. The trend components were replaced with the variance of the entire RSA time series, which aimed to capture an overall index of how much variability in RSA existed across the recovery period (i.e. the overall degree of RSA

change from the mean RSA value across time), not unlike how the trend components might characterize the overall magnitude of RSA change across recovery. In addition, the intercept term in a differenced model refers to the series mean. Therefore, instead of using intercept as a mediator, the mean of the RSA series across recovery was used as a mediator. These edits do not substantively alter my theoretical questions.

To evaluate Specific Aim 3, a path analysis model (see Figure 9) was implemented using MPlus software in which mental health symptoms were regressed on time series mediators, mental health symptoms were regressed on coping strategies and the control variable of RSA at stress (the "direct effects"), and time series mediators were regressed on coping strategies and the control variable of RSA at stress. Sex, age, and parental education were exogenous covariates in the model and covariances were estimated between symptoms of depression and anxiety, between coping strategies, and between the number of autoregressive, moving average, and order of differencing terms in the model. Results of this model are presented in Table 9 and significant paths and covariances are represented in Figure 10. This model demonstrated good fit for the data, as indicated by multiple model fit indices (see Table 13), therefore model estimates were examined.

Results did not support the partial mediation of the relationship between coping strategies and mental health symptoms by time series RSA estimates. However, significant direct paths were found. Similar to the results of Specific Aim 1, results of Specific Aim 3 indicated that John Henryism was significantly associated with symptoms of depression (β=-.23, p=.009) and anxiety (β=-.23, p=.009) above and beyond the other coping strategies in the model. Active coping, social support, and spiritual coping were not uniquely associated with symptoms of depression or anxiety in this model. More frequent use of active coping was significantly

associated with greater variance in RSA during recovery (β= .29, *p*=.005). However, more frequent use of social support was significantly associated with less variance in RSA during recovery (β= -.25 *p*=.032). The greater number of autoregressive terms necessary to capture a participant's autocorrelation of RSA across recovery was associated with fewer symptoms of anxiety (β= -.26 *p*=.029). The greater the order of differencing required to transform a participant's time series to stationarity was associated with greater use of spiritual coping (β= .28, *p*=.003). Participant sex remained a significantly associated with use of spiritual coping, indicating that female students more frequently used spiritual coping in response to instances of racial discrimination (β= -.24 *p*=.008). As anticipated, as a control variable RSA at stress was significantly associated with mean RSA during recovery (β= .55 *p*<.001). Significant covariances between depression and anxiety, number of autoregressive and moving average terms and order of differencing in the time series model, and some coping strategies were also observed (see Table 9 and Figure 10 for significant paths). While this model was an adequate fit for the data, R^2 estimates indicate that the amount of variance in symptoms of depression and anxiety explained by this set of predictors was small to moderate (depression, R^2=.11; anxiety R^2=.15).

As noted in the "Descriptive Statistics" section above, the number of autoregressive and moving average terms and the order of differencing were count variables. Upon examination of their distributions, statistical approaches compatible with a Poisson distribution were not deemed necessary. While the distributions of these variables did approximate continuous normal distributions, a sensitivity analysis was conducted to confirm that the estimation of robust standard errors to account for heterogeneity and lack of normality would not substantively alter the results. Results of this model are presented in Table 10. There were no substantive

differences in the results of this analysis using robust standard errors compared to the previous model.

Due to potential power concerns discussed in the results of Specific Aim 1, another model which included only the coping strategy of John Henryism was examined. This is further justified because John Henryism has demonstrated a direct relation with at least one mental health outcome across multiple models. Therefore, in an attempt to reduce estimation burden on the model, a modified path analysis model (see Figure 11) was implemented using MPlus software. This model was identical to the previous model, with the exception that this model did not estimate 1) paths between mental health symptoms and active coping, social support, and spiritual coping, 2) paths between time series mediators and active coping, social support, and spiritual coping, 3) paths between exogenous control variables and active coping, social support, and spiritual coping, and 4) covariances between active coping, social support, spiritual coping, and John Henryism. Results of this model are presented in Table 11 and significant paths and covariances are represented in Figure 11. This model was a good fit for the data, as indicated by multiple model fit indices (see Table 13), therefore model estimates were examined. Model estimates were not substantively different than the previous model which included all coping strategies. For completeness, a sensitivity analysis was also conducted to determine if there were substantive differences between the present model examining only John Henryism and this same model with robust standard errors. No substantive differences in model results were observed (see Table 12).

In summary, there were no substantive differences in model results between the model examining four coping strategies versus the model examining only John Henryism. As both models demonstrated adequate fit to the data, the additional information on multiple coping

strategies afforded by the model which includes all four coping strategies provides good rationale for its interpretation to answer Specific Aim 3. Contrary to my hypothesis, partial mediation was not supported. However, three coping strategies were related to RSA estimates. Active coping was associated with more variance in RSA during recovery, social support was associated with less variance in RSA during recovery, and spiritual coping was associated with greater orders of differencing necessary to capture the RSA time series estimates. The more autoregressive terms required to estimate one's RSA time series was significantly associated with fewer self-reported symptoms of anxiety. Sensitivity analyses were conducted using robust standard errors and results from these models did not differ substantively from prior models.

CHAPTER 5: DISCUSSION

The purpose of the current study was to examine the mediating role of respiratory sinus arrhythmia in the relationship between coping strategies and mental health outcomes in an attempt to clarify the literature on the utility of specific coping strategies in reducing symptoms of depression and anxiety. Results of this study do not provide evidence for partial mediation as hypothesized, however, they shed light on the direct relationships between coping strategies, physiology during recovery, and symptoms of depression and anxiety.

Specific Aim 1 – Unique Associations between Coping Strategies and Mental Health Symptoms

With regards to Specific Aim 1, I hypothesized significant direct associations of symptoms of depression and anxiety with coping strategies. Due to inconsistencies in the literature, directionality of these relationships was not hypothesized. Using a sample of 205 Black undergraduate students, only spiritual coping and John Henryism were uniquely associated with symptoms of depression and anxiety. More frequent use of John Henryism was associated with fewer symptoms of depression and anxiety, a finding that has not been previously reported in the literature on Black young adults. More frequent use of John Henryism may be effective in the reduction of symptoms of anxiety and depression because it inspires increased effort and subsequent feelings of agency in response to a challenge. Self-determination and hope may result from the more frequent use of John Henryism, emotions which have been shown to reduce symptoms of depression and anxiety in college students (Arnau, Rosen, Finch, Rhudy, & Fortunato, 2007). While active coping and John Henryism share similar conceptualizations, John

Henryism may be a more culturally-specific coping strategy with implications for Black youth. As measured in the current study, this construct contains targeted questions about hard work and overcoming obstacles. Indeed, active coping was measured broadly and inquired about "concentrating one's efforts on doing something about the situation I'm in" and "taking action to try to make the situation better," while the John Henryism scale more specifically mentioned success and goal-related behavior such as "working harder" and "staying with it until the job is completely done." Active coping and John Henryism were not significantly correlated in the present sample (see Table 2). Furthermore, when both active coping and John Henryism are in the same model, only John Henryism retains a unique impact on symptoms of depression and anxiety. While the main components of John Henryism include high effort and active engagement in problem-solving that are akin to active coping, John Henryism also includes dimensions of persistence and determination to succeed that may make this high-effort coping strategy notably distinct from the more general construct of active coping. However, these possibilities merit further empirical attention.

More frequent use of spiritual coping was associated with increased symptoms of anxiety. It should be emphasized that use of spiritual coping was measured broadly with two items and did not pinpoint specific spiritual behaviors, emotions, and/or cognitions that may be involved in coping with racial discrimination. Furthermore, the distribution of spiritual coping was roughly bimodal, such that many participants reported never using spiritual coping and many participants reported using spiritual coping very frequently. The literature suggests that, when employed to the extreme, high frequency spiritual coping may be associated with greater symptoms of anxiety, as seen in the present study. For example, pleading for direct intercession has been found to be associated with increased mental health symptoms. As suggested by Ano

and Vasconcellas (2005), elements of spiritual coping in which individuals may be expecting to reap rewards or see the influence of deities in their everyday lives might be closely associated with excessive monitoring of one's environment and potential self-blame, self-doubt, and dependence leading to anxiety when these rewards or influences are not observed. In a similar vein, Johnson and colleagues (2011) have shown that adults with advanced illness who reported more negative past experiences with spirituality and increased concurrent negative feelings about spirituality had elevated symptoms of anxiety (Johnson et al., 2011), further suggesting that some elements of spiritual coping may be linked to increased mental health symptoms.

While some studies report that direct action and social support are effective coping strategies when facing race-related difficulties (e.g. Shorter-Gooden, 2004), other studies do report null findings (e.g. Greer, 2011). Some scholars have suggested that the complexity of this form of coping, which involves both cognitive and emotional appraisals of one's social network and its support structure (Harrell, 2000; Lewis-Coles & Constantine, 2006), makes it a difficult construct to assess reliably. Conversely, perhaps these coping strategies are impactful, but only in conjunction with other personal resources.

Specific Aim 2 – Intraindividual Estimates of RSA during Recovery

Here the aim was to estimate intraindividual change in RSA during recovery using time series analysis to generate optimally-fitting ARIMA models for each participant. Contrary to my hypotheses, most participants' time series did not exhibit the expected quadratic trend. Perhaps a five-minute recovery time allowed participants to experience various changes in mental states with effects on the rapidly responding autonomic nervous system. While five minutes is the standard length of a recovery period in the literature (e.g. Morris-Prather, Harrell, Collins, Leonard, Boss, & Lee, 1996; Neblett & Roberts, 2013), these studies do not examine second-by-

second RSA, but rather average across the five minute recovery period. This may explain the large number of time series that demonstrated cubic trends. As many time series needed to be differenced in order to achieve the stationarity necessary to add autoregressive and moving average terms to account for autocorrelation, many trend components were removed and thus trend components were no longer directly comparable across individuals. Additionally, many autoregressive and moving average terms were necessary to characterize each series, which did not enable direct comparison of the magnitude of model estimates. Instead, number of autoregressive terms, number of moving average terms, mean RSA during recovery, and variance of RSA during recovery were included as mediators in subsequent analyses.

Specific Aim 3 – Mediation of the Relation between Coping and Mental Health Symptoms by Estimates of RSA during Recovery

With regards to Specific Aim 3, I hypothesized that RSA estimates of intraindividual change during recovery would partially mediate the relationship between coping strategies and mental health symptoms. Significant direct relationships were detected between John Henryism and symptoms of depression and anxiety; however these relationships were not partially mediated by any of the RSA variables. While there were significant relationships between active coping and social support and RSA variance, RSA variance was not associated with symptoms of depression or anxiety. A significant association was also observed between spiritual coping and the order of differencing necessary for each time series; however the order of differencing was not significantly associated with mental health symptoms. Similarly, while the number of autoregressive terms in the time series model was significantly associated with a decrease in anxiety symptoms, none of the four coping strategies were significantly associated with the number of autoregressive terms in the time series model. One potential explanation for a lack of significant partial mediation may be the use of depression and anxiety as intrapsychic constructs.

Indeed, a recent study that examined the association between vagal flexibility and perceived stress, anxiety, depression, and loneliness, found a significant association only between vagal flexibility and loneliness (Muhtadie, Akinola, Koslov, & Berry Mendes, 2015). The authors posit that loneliness is an inherently social and interindividual experience, which may therefore have a relation to RSA activity because the PNS is inherently a social engagement system (Porges, 2001) that facilitates social interaction under safe environmental conditions. Yet another explanation for the lack of significant partial mediation may be the treatment of all study variables as relatively stable trait-level characteristics. While there is sufficient evidence, based on the literature, to assume that coping strategies (e.g. Frydenberg, 2008) and RSA variables (e.g. Beauchaine, Neuhas, Brenner, & Gatzke-Kopp, 2008; Sloan, Shapiro, Bagiella, Gorman, & Bigger, 1995) are relatively stable over periods of least several weeks, symptoms of depression and anxiety may not meet this assumption, especially among a sample of Black college students. The current study assumed that mental health symptoms were relatively stable based on prior research on symptoms of depression and anxiety in non-clinical samples, which have found strong positive correlations between mental health symptoms assessed up to one year following an initial assessment (e.g. Beeghly, Olson, Weinberg, Pierre, Downey, & Tronick, 2003; Yaptangco, Crowell, Baucom, Bride, & Hansen, 2015). As some of these studies used different measures of mental health symptoms it may be that these measures are better able to capture trait-level symptomatology. Furthermore, research on the experiences of Black college students at predominantly White institutions suggests that everyday experiences, especially those involving racial discrimination, may substantially alter day-to-day affective states and subsequently impact depressive symptomatology (e.g. Hoggard, Byrd, & Sellers, 2015; Hoggard, Hill, Gray, & Sellers, 2015). Therefore, the present investigation would benefit from multiple

assessments of symptoms over time and thus offer the possibility to disentangle trait- and state-level impacts on symptomology.

While my results did not support partial mediation, significant direct paths between predictors, mediators, and outcomes begin to inform our understanding of the relationships between coping strategies, RSA during recovery, and mental health symptoms. More frequent use of spiritual coping was associated with a higher order of differencing required to stationarize a participant's RSA time series. A higher order of differencing indicates that the raw time series exhibited a long-term trend, lacked a tendency to return to its mean value, and/or had significant remaining autocorrelation across time. Therefore, this finding may indicate that a tendency to use spiritual coping more frequently in response to racial discrimination is associated with a more complex RSA trajectory during recovery from a stressor. Perhaps spiritual coping is specifically associated with a more complex trend component (i.e. a quadratic, cubic, or higher-order slope), however this cannot be explicitly verified in the present study as de-trending was necessary. While non-constant variance across time may also necessitate a higher order of differencing, overall variance in RSA was not significantly associated with spiritual coping. However, this may mean that time specific variability is associated with spiritual coping. Without more specific quantification of these complex processes, this result is open to various interpretations and future research. The present literature offers no guidance to explain this result.

More frequent use of active coping was significantly predictive of increased variability in RSA during recovery. Within limits, greater variability in RSA is ideal during recovery as this demonstrates increased engagement to environmental demands. If the environment is perceived as safe, restorative and digestive states compatible with optimal PNS functioning during recovery from stress will be initiated. Therefore, active coping may be effective due to its

association with more flexible and adaptive responses to environmental stress. However, this relationship did not extend directly or indirectly to symptoms of depression or anxiety in the present sample. Therefore this assertion requires further empirical examination. More frequent use of social support was significantly associated with decreased RSA variability during recovery. Perhaps social support is a coping strategy that has differential associations with the flexibility of responses to stress. Because social support is a means of mobilizing resources to seek solutions or emotional support from others, it may not be as associated with RSA during recovery from a stressor in which the option for seeking social support is not available. In the absence of the option to seek social support in this situation, perhaps those individuals who most frequently use social support in response to racial discrimination do not evidence as optimal a recovery from stress in the short-term. As this study did not examine short-term versus long-term responses to the stressor, this assertion requires further empirical examination. Furthermore, it should be noted that these are preliminary insights into very complex processes. These suggestions assume variance is constant across time and future work should explore complexities in the shape of these trajectories of RSA as they relate to such variability.

Contrary to my hypothesis, the number of autoregressive terms estimated for each RSA time series was significantly associated with symptoms of anxiety such that those individuals who required more autoregressive terms to explain autocorrelation in their time series reported fewer symptoms of anxiety. The number of autoregressive terms may be seen as the degree of inertia in a given time series, the degree to which values at one second in time significantly predict values later in time. The duration of significant unique autocorrelation across seconds of the time series is exemplified by the number of autoregressive terms required to sufficiently capture this inertia. While it is difficult to visually separate the components of a time series,

Figure 12 presents characteristic series for participants whose optimal models contained no autoregressive terms (no significant autocorrelation was detected once fitting a regression model), one autoregressive term (the lower limit) and four autoregressive terms (the upper limit). Series which required four autoregressive terms exemplified significant second to second correlations across greater lags of time, such that their RSA values were more inert across recovery. Perhaps it is the seemingly unrelated fluctuations in RSA from second to second that represent a degree of higher sensitivity to internal and/or external environmental demand. As symptoms of anxiety are associated with hypervigilance, extreme fluctuations that are less correlated over time may be in turn associated with greater symptoms of anxiety. However, as with discussion of other results, such explanations are preliminary generalizations of more complex time-specific processes and should not be without further empirical examination and support. Indeed, while I controlled for the degree of differencing required, the substantive interpretation of first-differenced variables is different than the interpretation of non-differenced variables.

Contributions

This investigation makes several contributions to the literature on coping strategies and mental health symptoms by bridging the clinical and physiological literatures. John Henryism was the only coping strategy examined that was associated with symptoms of depression after accounting for other coping variables as well as RSA estimates during recovery. This finding provides an exciting new direction for future research and adds to a body of literature aimed at clarifying the relationship between coping strategies and mental health symptoms. John Henryism has been studied extensively with Black samples in relation to physical health outcomes, most notably cardiovascular risk (e.g. James, 1994; James et al., 1987; LeBron,

Schulz, Mentz, & Perkins, 2015). However, in the present sample more frequent use of John Henryism was found to be associated with Black college students' *mental health*, an extension of the literature on John Henryism that has not been previously reported. In the current full sample, the observed mean score on John Henryism was 48.23 points (for n=133 who had complete data on John Henryism), while the true scale median score was 24 points. While the true scale ranged from 12-60 points, the observed scores ranged from 26-60 points. The majority of the current sample of Black undergraduate students at a predominantly White institution self-reported moderate to very frequent use of John Henryism, and this coping strategy was used more often than active coping, spiritual coping, or social support according to self-reports. John Henryism may be an especially relevant coping strategy for Black college students who are at risk for experiencing racial discrimination in addition to working to adjust to the educational transition that all college students experience. Indeed, perfectionism in a college context, a construct that may share similarities to John Henryism, has been found to be associated with increased mental health symptoms and reduced mental health support seeking for Black college students at predominantly White institutions (e.g., DiBartolo & Rendon, 2012).

It is important to note that use of John Heryism has been found to have long-term implications for cardiovascular health risk in samples of middle-aged and older Black adults (e.g. Merritt et al., 2004). The allostatic load model (McEwen & Stellar, 1993) may be used to explain these findings. According to this model, pervasive experiences of chronic stressors may cause a 'wear and tear' on bodily systems responsible for initiation and regulation of responses, resulting in an increased susceptibility for the development of poor health outcomes. It is reasonable to assume that in contexts of prolonged stress the use of John Henryism as a high-effort coping strategy may put additional strain on physiological systems by demanding increased activation of

bodily systems responsible for responding to threat. It should be noted that this environment of prolonged stress may stem from the predominantly White institution in which Black college students may face heightened risk of experiencing racial discrimination and stress due to the college transition, or may be a function of experiences of racial discrimination and/or other forms of disenfranchisement or familial lack of resources during childhood and adolescence. Indeed, work with Black adolescents supports the idea that experiencing racial discrimination increases the probability of disease risk in the long-term by creating wear and tear on biological systems and stress responses (Brody et al., 2014).

However, negative physical health impacts of the use of John Henryism during young adulthood have not been supported. For example, in one nationally representative study of Black young adults ages 18-30, use of John Henryism was not significantly associated with higher blood pressure (McKetney & Ragland, 1996). The authors argue that limited evidence of hypertension in young adult populations may obscure the gradual impact of the potential wear and tear of use of John Henryism over the long term. Additionally, the health consequences of John Henryism may emerge at older ages due to increased developmental demands that create greater environmental demand upon physiological systems already compromised by experiences of discrimination, including direct responsibilities in the family and professional sphere. While we did not measure these outcomes in the present investigation, we may not yet see sufficient evidence of cardiovascular health difficulties. Furthermore, from a developmental perspective, it may be that high-effort coping may reduce symptoms of depression and anxiety by inspiring persistence and determination to successfully meet the challenging environmental demands of racial discrimination. However, in the long term such excessive effort when too frequently applied may initiate prolonged bodily activation and condition a dysregulated, hypervigilant and

hyperactivated stress response that may result in poorer mental and physical health. Thus, further research should examine the long-term impact of frequent utilization of John Henryism beginning in college in order to ensure its protective utility in the short- and long-term for both mental and physical health before recommendations on the benefits of John Henryism may be made.

Another significant contribution of this study to the literature is its use of individually derived estimates of RSA using time series analysis. While a majority of studies assume equivalence between intraindividual and interindividual variability in their statistical approaches, scholars have found this assumption to be erroneous (e.g. Molenaar, 2004). In the present study, time series analyses were conducted to arrive at best-fitting individual-level models. While the study results could be different if the same models were fit at the intergroup level, an argument for the present analytic strategy is its sensitivity to the necessity of person-specific models while other strategies such as multilevel models may assume the same model is applicable for each individual. Generally, examination of individuals' physiological recovery from stress at an intraindividual level did support that estimates of trajectories of RSA recovery were associated with increased risk for anxiety symptoms and that coping strategies did impact numerical estimates of RSA trajectories during recovery. While partial mediation was not supported, direct relationships between coping strategies and RSA variables and between RSA variables and mental health outcomes were supported. Preliminary insights as to the meaning of these results are suggested above, yet now that initial support for relations between RSA estimates during recovery, coping strategies, and mental health, these complex processes are ripe for further investigation.

Limitations

There are several limitations to the work presented above. Most notably, while the use of an *in vivo* task that mimicked elements of a racial discrimination experience above self-report measures was a strength of the present study, this task was not experimentally manipulated. Though the task was previewed and approved by a small focus group of Black students on campus, we cannot conclude with certainty that responses to and recovery from this task were equivalent to responses to and recovery from an experience of racial discrimination above and beyond another type of potentially stress-inducing task. Similarly, an assumption of this study is that the task was sufficiently stressful to activate a physiological response. By controlling for RSA at stress, any lack of change from stress to recovery was partialed out. Furthermore, as can be seen in Figure 5, it appears that most participants did respond to the task. However, we cannot say this response is specific to racial discrimination. Future research should experimentally manipulate the task and incorporate a measure of the perceived stressfulness of the task into analyses (though this measure may be highly susceptible to social desirability). Additionally the present study measured recovery from an acute experience, however prior and/or chronic experiences of racial discrimination have been found to alter the way in which the body responds to experiences of racial discrimination in the present (e.g. Brody et al., 2014). Therefore, the present study would benefit from the additional incorporation of self-report measures of prior/chronic experiences or longitudinal measures of acute experiences as a control variable.

Other limitations also pertain to study measures. As an initial investigation, the broadness of the items which assessed active coping, social support, and spiritual coping were ideal. However, further research should build upon present results by seeking to understand what elements of active coping and social support were driving the relationship between these coping

strategies and RSA variability during recovery from stress. Elements of these strategies could then be incorporated into interventions and clinical practice. Similarly, spiritual coping approximated a bimodal distribution in the present sample. As spiritual coping was measured broadly using two items which did not pinpoint specific behaviors, thoughts, or emotions, a better measure of spiritual coping that is specifically related to Black college students may be more optimal in future investigations. Additional limitations concerning the in vivo stress task include limited external validity, as only one situation was presented in structured format which may not be as directly applicable to Black college students' experiences on campus. Furthermore, to what internal thoughts and emotions and external stimuli participants were responding during recovery was not assessed. While all protocol was standardized and distractors in the physical environment were removed, I am making the assumption that time series of RSA during recovery is an approximate representation of recovery from stress. However this recovery period could also include responsivity to other unobserved conditions. Finally, coping measures are used in the present analysis as trait-level variables and were not measured with regard to the specific stressor with which participants were presented in the laboratory. Therefore, while associations between coping strategies, RSA, and mental health symptoms were observed, RSA during recovery was not directly influenced by each coping strategy. Participants could have used a variety of coping strategies to recover from the stress task in the laboratory, some of which were not included in the present analyses. Similarly, the notion that participants may use multiple strategies in their natural environments should be explored in future research.

Finally, as mentioned above, the use of person-specific intraindividual estimates was both a strength and a limitation. Most notably, in the current study the complexity of each time series

could not be adequately captured due to a lack of comparable estimates across participants. This may be a reflection of the limitations of the laboratory context in which participants were asked to recover rather than a limitation of this method. For example, decreasing the length of time participants were allotted for recovery and increasing the uniformity of the context in which participants were asked to recover (i.e. instead of being left alone in a bare room for five minutes, participants could be instructed to engage in a soothing activity or listen to relaxing music) may have provided less variability in participants' time series. As is, this variability cannot be completely understood as true variability in recovery from the stress task rather than variability due to responses to other internal or external environmental stimuli. More generally, case-by-case sensitivity to the data may result in overfitting the data to an idiosyncratic case. As model estimation was iterative, while governed by established procedures for time series analysis including several model tests (i.e. Ljung-Box, Augmented Dickey-Fuller), a degree of measurement error was necessarily introduced via this selection method.

General Conclusion and Future Directions

The findings of the present study are globally consistent with the previous literature. Expanding upon these findings, this investigation was the first step in examining the complex processes by which coping strategies may or may not impact mental health symptoms. Notably, this study demonstrated that John Henryism was uniquely associated with reduced symptoms of depression and anxiety for Black college students. Active coping and social support were both related to elements of physiological recovery from a stressor that mimics elements of racial discrimination, and elements of this physiological recovery were in turn related to symptoms of anxiety.

The present study provides several unique contributions to the literature. While John Henryism has been examined with respect to the physical health outcomes of middle-aged and older Black populations, the present study is the first to examine its association with mental health outcomes for Black college students attending a predominantly White institution. Results suggest that the short- and long-term impacts of frequent use of John Henryism merits further longitudinal investigation, as allostatic load theory would suggest that a long-term cost might be associated with frequent employment of such high-effort coping. Furthermore, to the author's knowledge investigations specifically focused on RSA during recovery from a stressor have not been examined. However, now that the importance of examining estimates of RSA during recovery has received initial support, joint impacts of RSA reactivity to a stressor and subsequent recovery from that stressor may be examined to glean important information about complex person- and time-specific relations between coping strategies and stress processes.

Future research should seek to refine the present model to include the perceived stressfulness of the task, additional targeted measures of spiritual coping, and temporal measures of depression and anxiety symptoms. Careful attention to the length and conditions of the recovery period for those investigators interested in examination of intraindividual differences in second-by-second RSA is suggested. Furthermore, experimental manipulation of the *in vivo* task is the necessary next step in assessing the uniqueness of the present results for experiences of racial discrimination as compared to other stressful experiences.

Taken together, this work informs our understanding of the coping strategies that Black college students employ at a predominantly White institution. While physiology was not found to facilitate coping in the present investigation, students' use of John Henryism to cope with experiences of racial discrimination may be a key to understanding health risk for this population

in both the short- and long-term. Moving forward, as our understanding of intraindividual elements of trajectories of RSA during recovery grows, we may be able to pinpoint specific physiological factors that impact mental health symptoms for Black college students attending a predominantly White institution which could subsequently inform culturally-appropriate interventions on biofeedback or mindfulness training.

Table 1.
Descriptive Statistics (n=116)

Variable	**N**	**M (*SD*)**	**Observed Sample Range**	**Scale True Range**
RSA at time of stress (control)	112	4.31 (*1.16*)	.20 - 6.82	N/A
Active Coping	115	2.33 (*1.83*)	0-6	0-6
Social Support	115	5.05 (*3.81*)	0-12	0-12
Spiritual Coping	115	2.64 (*2.44*)	0-6	0-6
John Henryism	85	48.04 (*6.11*)	26-59	12-60
Number of AR terms	114	2.46 (*.88*)	0-4	N/A
Number of MA terms	114	1.15 (*1.03*)	0-4	N/A
Order of Differencing	114	1.04 (.88)	0-2	N/A
Variance of RSA series – square root	114	Original: .28 (*.23*) .49 (*.20*)	Original: .01-1.29 .12-1.14	N/A
Mean of RSA series	114	5.50 (*1.07*)	3.07-8.57	N/A
Depression Symptoms	116	9.44 (5.50)	1-25	0-36
Anxiety Symptoms – square root	116	Original: 8.16 (*5.22*) 2.55 (*1.28*)	Original: 0-40 0-6.32	Original: 0-63 0 -7.94

Table 2.

Intercorrelations amongst Study Measures (n=116)

		1	2	3	4	5	6	7	8	9	10	11	12
1.	Active Coping												
2.	Social Support	.51***											
3.	Spiritual Coping	.24**	.43***										
4.	John Henryism	.16	-.03	.22*									
5.	RSA at Stress	-.17	.04	.11	.07								
6.	RSA Recovery Mean	-.16	.04	-.02	.03	.56***							
7.	RSA Recovery Variance (sqrt)	.22*	-.03	.10	.04	-.13	-.16						
8.	Number of AR terms	.06	-.004	-.17	-.02	-.08	.08	-.23*					
9.	Number of MA terms	.03	.09	.13	-.01	-.03	-.01	.04	-.36***				
10.	Order of differencing	-.16	-.13	.16	.11	.10	-.04	.17	-.50**	-.27*			
11.	Depression	-.10	-.004	-.05	-.25*	.01	.09	.001	-.05	-.02	-.004		
12.	Anxiety (sqrt)	-.01	.15	.16	-.16	-.05	-.02	.05	-.19*	-.005	.05	.52***	

*p<.05, **p<.01, ***p<.001

Table 3.
Descriptive Statistics (n=205)

Variable	**N**	**M (*SD*)**	**Observed Sample Range**	**Scale True Range**
RSA at time of stress (control)	112	4.31 (*1.16*)	.20 - 6.82	N/A
Active Coping	185	2.54 (*1.80*)	0-6	0-6
Social Support	185	5.03 (*3.81*)	0-12	0-12
Spiritual Coping	185	2.54 (*2.31*)	0-6	0-6
John Henryism	133	48.23 (*6.30*)	26-60	12-60
Number of AR terms	114	2.46 (*.88*)	0-4	N/A
Number of MA terms	114	1.15 (*1.03*)	0-4	N/A
Order of Differencing	114	1.04 (.88)	0-2	N/A
Variance of RSA series – square root	114	Original: .28 (*.23*) .49 (*.20*)	Original: .01-1.29 .12-1.14	N/A
Mean of RSA series	114	5.50 (*1.07*)	3.07-8.57	N/A
Depression Symptoms	205	9.76 (5.77)	0-25	0-36
Anxiety Symptoms – square root	205	Original: 8.16 (*5.28*) 2.53 (*1.29*)	Original: 0-40 0-6.32	Original: 0-63 0 -7.94

Table 4.
Intercorrelations amongst Study Measures (n=205)

		1	2	3	4	5	6	7	8	9	10	11	12
1.	Active Coping												
2.	Social Support	.50***											
3.	Spiritual Coping	.27**	.36***										
4.	John Henryism	.06	-.18*	.12									
5.	RSA at Stress	-.17	.04	.11	.07								
6.	RSA Recovery Mean	-.16	.04	-.02	.03	.56***							
7.	RSA Recovery Variance (sqrt)	.22*	-.03	.10	.04	-.13	-.16						
8.	Number of AR terms	.06	-.004	-.17	-.02	-.08	.08	-.23*					
9.	Number of MA terms	.03	.09	.13	-.01	-.03	-.01	.04	-.36***				
10.	Order of differencing	-.15	-.05	.24*	.11	.16	.01	.17	-.50**	-.19*			
11.	Depression	-.10	.09	-.01	-.28**	.01	.09	.001	-.05	-.004	-.05		
12.	Anxiety (sqrt)	-.02	.13	.14	-.22*	-.05	-.02	.05	-.19*	-.01	.10	.51***	

*p<.05, **p<.01, ***p<.001

Table 5.
Results of Model Addressing Specific Aim 1: Full Sample (n=205)

Nature of Relation	Path	Standardized Estimate	S.E.	p-value
Control	Sex to Active Coping	.09	.16	.577
Control	Sex to Social Support	-.23	.16	.138
Control	Sex to Spiritual Coping	-.39	.15	.012*
Control	Sex to John Henryism	-.03	.18	.887
Control	Age to Active Coping	-.02	.02	.479
Control	Age to Social Support	-.009	.02	.714
Control	Age to Spiritual Coping	-.02	.02	.453
Control	Age to John Henryism	.00	.03	.998
Control	Parental Education to Active Coping	.03	.07	.648
Control	Parental Education to Social Support	.03	.07	.623
Control	Parental Education to Spiritual Coping	.05	.06	.428
Control	Parental Education to John Henryism	-.007	.08	.924
Focal Relation	Active Coping to Depression	-.15	.08	.081
Focal Relation	Social Support to Depression	.12	.09	.199
Focal Relation	Spiritual Coping to Depression	.006	.08	.939
Focal Relation	John Henryism to Depression	-.23	.09	.010**
Focal Relation	Active Coping to Anxiety	-.08	.08	.306
Focal Relation	Social Support to Anxiety	.07	.09	.408
Focal Relation	Spiritual Coping to Anxiety	.16	.08	.042*
Focal Relation	John Henryism to Anxiety	-.23	.09	.010**
Covariance	Depression with Anxiety	.48	.06	.000***
Covariance	Active Coping with Social Support	.45	.06	.000***
Covariance	Active Coping with Spiritual Coping	.28	.07	.000***
Covariance	Active Coping with John Henryism	.05	.09	.558
Covariance	Social Support with Spiritual Coping	.34	.07	.000***
Covariance	Social Support with John Henryism	-.18	.09	.044*
Covariance	Spiritual Coping with John Henryism	.12	.09	.165

*p<.05, **p<.01, ***p<.001

Table 6.
Results of Model Addressing Specific Aim 1: Sub-Sample (n=115)

Nature of Relation	Path	Standardized Estimate	S.E.	p-value
Control	Sex to Active Coping	-.05	.10	.603
Control	Sex to Social Support	-.14	.09	.128
Control	Sex to Spiritual Coping	-.24	.09	.008**
Control	Sex to John Henryism	-.90	1.50	.548
Control	Age to Active Coping	-.05	.10	.667
Control	Age to Social Support	-.03	.10	.773
Control	Age to Spiritual Coping	-.10	.09	.284
Control	Age to John Henryism	-.06	.11	.606
Control	Parental Education to Active Coping	.05	.10	.643
Control	Parental Education to Social Support	-.004	.10	.965
Control	Parental Education to Spiritual Coping	.03	.09	.744
Control	Parental Education to John Henryism	-.03	.11	.801
Focal Relation	Active Coping to Depression	-.09	.11	.398
Focal Relation	Social Support to Depression	.03	.12	.810
Focal Relation	Spiritual Coping to Depression	.01	.12	.948
Focal Relation	John Henryism to Depression	-.21	.11	.052 †
Focal Relation	Active Coping to Anxiety	-.90	.11	.410
Focal Relation	Social Support to Anxiety	.12	.12	.316
Focal Relation	Spiritual Coping to Anxiety	.16	.10	.119
Focal Relation	John Henryism to Anxiety	-.16	.11	.172
Covariance	Depression with Anxiety	.50	.07	.000***
Covariance	Active Coping with Social Support	.51	.07	.000***
Covariance	Active Coping with Spiritual Coping	.24	.09	.008**
Covariance	Active Coping with John Henryism	.14	.11	.188
Covariance	Social Support with Spiritual Coping	.40	.08	.000***
Covariance	Social Support with John Henryism	-.05	.12	.673
Covariance	Spiritual Coping with John Henryism	.21	.11	.056 †

*p<.05, **p<.01, ***p<.001, †p<.07

Table 7.
Model Comparison – Fit Criteria

Fit Index	**Model Full Sample (n=205)**	**Model Sub-Sample (n=115)**
Chi-Square Test of Model Fit	$\chi^2(6)= 8.92$, $p= .1780$	$\chi^2(6)= 9.99$, $p=.1248$
RMSEA	.05, 90% CI [.00, .11], p=.444	.08, 90% CI [.00, .16], p=.255
CFI	.98	.96
TLI	.89	.78
SRMR	.030	.030

Table 8.
Model Information (n=115)

Participant ID	RSA Recovery Mean	RSA Value at Stress	Model R^2 **(bold=selected)**	Stationarity-Final ADF	ARIMA Model	Final Ljung-Box
302	5.35	4.93	Linear= .09 **Quadratic= .28** Cubic= .28	*ADF*= -3.83, *p*= 0.019	(3, 0, 2) AIC= -1218.33	χ^2= 2.34, *p*= 0.126
401	6.00	Missing	Linear= .04 **Quadratic= .41** Cubic= .42	*ADF*= -3.78, *p*= 0.022	(4, 1, 0) AIC= -1152.45	χ^2= 1.16, *p*= 0.281
402	4.90	3.02	Linear= .14 Quadratic= .66 **Cubic= .89**	*ADF*= -4.10, *p*<0.001	(0, 0, 0)	N/A
403	6.19	5.16	Linear= .02 Quadratic= .04 **Cubic= .16**	*ADF*= -6.48, *p*<0.001	(2, 2, 0) AIC= -1446.04	χ^2= 1.33, p= 0.250
404	6.03	5.06	Linear= .008 **Quadratic= .12** Cubic= .13	*ADF*= -4.41, *p*<0.001	(3, 0, 2) AIC= -1212.09	χ^2= 3.63, p= 0.057
405	4.05	3.79	Linear= .17 Quadratic= .19 **Cubic= .47**	*ADF*= -5.26, *p*<0.001	(2, 2, 1) AIC= -1068.73	χ^2=0.87, *p*= 0.351
406	5.68	4.52	Linear= .54 **Quadratic= .74** Cubic= .76	*ADF*= -5.40, *p*<0.001	(2, 2, 0) AIC= -1296.01	χ^2= 3.12, *p*= 0.077
407	8.41	6.47	Linear= .10 Quadratic= .11 **Cubic= .17**	*ADF*= -3.80, *p*= 0.021	(2, 0, 3) AIC= -941.20	χ^2= 3.64, *p*= 0.056
408	5.56	4.64	Linear= .16 **Quadratic= .40** Cubic= .44	*ADF*= -3.76, *p*= 0.023	(2, 0, 4) AIC= -1327.06	χ^2= 1.82, *p*= 0.178
409	4.76	4.98	Linear= .04 Quadratic= .10 **Cubic= .48**	*ADF*= -4.18, *p*<0.001	(1, 2, 2) AIC= -1193.60	χ^2= 1.46, *p*= 0.227
410	5.52	4.42	**Linear= .28** Quadratic= .29 Cubic= .29	*ADF*= -3.65, *p*= 0.031	(3, 1, 1) AIC= -1001.51	χ^2= 0.30, *p*= 0.586
411	3.56	2.21	Linear= .19 Quadratic= .64 **Cubic= .73**	*ADF*= -3.61, *p*= 0.035	(2, 0, 2) AIC= -1056.49	χ^2= 1.21, *p*= 0.272
412	5.75	4.75	Linear= .12 Quadratic= .29 **Cubic=.42**	*ADF*= -3.22, *p*= 0.048	(4, 1, 0) AIC= -1234.31	χ^2= 0.08, *p*= 0.778
413	5.00	3.03	Linear=.07 **Quadratic=.44**	*ADF*= -4.72, *p*<0.001	(1, 2, 2) AIC=	χ^2= 3.39, *p*= 0.066

			Cubic=.47		-942.40	
414	5.20	2.85	Linear=.25 Quadratic=.26 **Cubic=.30**	*ADF*= -4.13, *p*<0.001	(4, 0, 0) AIC= -1311.56	χ^2= 0.05, *p* = 0.822
415	6.83	4.69	Linear= .03 Quadratic= .22 **Cubic= .64**	*ADF*= -5.95, *p*<0.001	(2, 2, 1) AIC= -1249.88	χ^2= 1.13, *p*= 0.288
416	6.63	5.94	Linear=.25 Quadratic=.28 **Cubic=.37**	*ADF*= -3.92, *p*= 0.015	(2, 1, 2) AIC= -1122.69	χ^2= 0.313, *p*= 0.576
417	5.37	4.92	Linear=.34 Quadratic=.36 **Cubic=.42**	*ADF*= -5.52, *p*<0.001	(2, 0, 4) AIC= -1371.02	χ^2= 0.27, *p*= 0.601
419	5.62	5.37	Linear= .01 **Quadratic=.34** Cubic=.38	*ADF*= -4.10, *p*<0.001	(3, 0, 1) AIC= -1344.93	χ^2= 0.97, *p*= 0.325
420	3.15	2.04	Linear= .14 Quadratic= .24 **Cubic=.55**	*ADF*= -3.93, *p*= 0.015	(4, 0, 1) AIC= -1363.65	χ^2= 0.51, *p*= 0.476
423	7.09	Missing	Linear=.01 Quadratic=.09 **Cubic=.66**	*ADF*= -6.50, *p*<0.001	(2, 2, 2) AIC= -956.24	χ^2 = 2.41, *p*= 0.121
424	5.62	5.49	Linear= .01 Quadratic=.28 **Cubic=.32**	*ADF*= -5.11, *p*<0.001	(2, 2, 1) AIC= -1087.60	χ^2= 3.94, *p*= 0.047
425	6.07	3.74	Linear=.44 Quadratic=.49 **Cubic=.54**	*ADF*= -5.51, *p*<0.001	(2, 1, 1) AIC= -1396.66	χ^2= 0.007, *p*= 0.933
426	5.45	4.98	Linear= .73 Quadratic= .75 **Cubic= .78**	*ADF*= -3.75, *p*= 0.023	(2, 1, 1) AIC= -1143.53	χ^2= 2.06, *p*= 0.151
427	4.98	4.00	Linear= .004 **Quadratic= .68** Cubic= .68	*ADF*= -5.88, *p*<0.001	(2, 2, 2) AIC= -1072.82	χ^2= 0.90, *p*= 0.342
428	5.25	4.88	Linear= .08 Quadratic= .20 **Cubic= .41**	*ADF*= -3.50, *p*=0.044	(3, 1, 1) AIC= -1348.35	χ^2= 0.36, *p*= 0.550
429	4.52	3.14	Linear=.42 Quadratic=.48 **Cubic= .53**	*ADF*= -6.12, *p*<0.001	(3, 2, 0) AIC= -999.04	χ^2= 0.90, *p*= 0.342
430	4.17	3.99	Linear= .03 **Quadratic=.20** Cubic= .20	*ADF*= -4.09, *p*<0.001	(3, 1, 0) AIC= -1091.5	χ^2= 3.28, *p*= 0.070
433	7.63	6.82	Linear= .09 Quadratic=.09 **Cubic=.38**	*ADF*= -7.54, *p*<0.001	(2, 2, 1) AIC= -1328.44	χ^2= 0.02, *p*= 0.877

435	5.00	2.43	Linear= .17 **Quadratic= .33** Cubic=.33	*ADF*= -3.87, *p*=0.018	(4, 0, 0) AIC= -1246.90	χ^2= 3.61, *p*= 0.057
436	6.39	6.40	Linear= .02 Quadratic=.03 **Cubic=.11**	*ADF*= -4.43, *p*<0.001	(3, 0, 0) AIC= -1072.59	χ^2= 0.30, p= 0.584
438	5.22	2.56	Linear=.004 **Quadratic=.15** Cubic=.16	*ADF*= -5.66, *p*<0.001	(1 , 2, 2) AIC= -1137.80	χ^2= 3.80, *p*= 0.051
439	5.28	2.46	Linear= .05 Quadratic= .06 **Cubic=.70**	*ADF*= -3.56, *p*= 0.040	(3, 1, 0) AIC= -1353.19	χ^2= 1.85, *p*= 0.174
441	5.53	3.71	Linear= .10 Quadratic= .16 Cubic= .52	*ADF*= -3.31, *p*= 0.042	(3, 1, 0) AIC= -1190.06	χ^2= 1.05, *p*= 0.305
442	4.08	3.28	Linear= .41 **Quadratic= .57** Cubic= .59	*ADF*= -6.81, *p*<0.001	(3, 2, 0) AIC= -1251.93	χ^2= 0.61, *p*= 0.435
443	4.23	3.64	Linear= .14 Quadratic= .35 **Cubic= .38**	*ADF*= -4.14, *p*<0.001	(3, 0, 2) AIC= -1132.93	χ^2= 3.63, *p*= 0.057
444	3.11	3.47	Linear= .01 Quadratic=.19 **Cubic=.25**	*ADF*= -4.06, *p*<0.001	(3, 0, 1) AIC= -1026.69	χ^2= 0.43, *p*= 0.512
445	6.73	6.14	Linear= .74 Quadratic= .77 **Cubic= .93**	*ADF*= -4.71, *p*<0.001	(3, 0, 1) AIC= -1324.87	χ^2= 1.35, *p*= 0.246
446	4.57	5.32	Linear= .44 Quadratic= .45 **Cubic= .83**	*ADF*= -3.76, *p*= 0.023	(3, 0, 1) AIC= -1040.35	χ^2= 2.06, *p*= 0.151
447	3.77	2.81	Linear= .06 Quadratic= .06 **Cubic= .27**	*ADF*= -4.401, *p*<0.001	(2, 2, 1) AIC= -1232.77	χ^2= 0.06, *p*= 0.803
448	5.48	1.73	Linear= .07 Quadratic= .06 **Cubic= .28**	*ADF*= -3.46, *p*= 0.049	(3, 0, 2) AIC= -1106.12	χ^2= 1.28, *p*= 0.257
450	6.78	5.48	Linear= .04 Quadratic= .12 **Cubic= .17**	*ADF*= -3.62, *p*= 0.034	(3, 0, 2) AIC= -1120.64	χ^2= 2.33, *p*= 0.127
451	5.80	4.60	Linear= .03 Quadratic= .06 **Cubic= .10**	*ADF*= -4.32, *p*<.001	(3, 0, 3) AIC= -1163.10	χ^2=0.74, *p*= 0.389
452	7.15	5.80	Linear= .02 Quadratic= .21 **Cubic= .86**	*ADF*= -4.36, *p*< 0.001	(4, 0, 1) AIC= -1255.06	χ^2= 0.21, p= 0.650
453	5.55	4.65	Linear= .06	*ADF*= -3.70,	(3, 0, 3)	χ^2= 0.55,

			Quadratic= .08 **Cubic= .12**	*p*= 0.026	AIC= -1087.61	*p*= 0.457
454	5.02	3.90	Linear= .27 Quadratic= .37 **Cubic= .45**	*ADF*= -6.61, *p*<0.001	*(3, 2, 1)* *AIC=* *-1118.33*	χ^2= 1.27, *p*= 0.260
456	5.92	4.91	Linear= .60 **Quadratic= .70** Cubic= .71	*ADF*= -3.71, *p*=0.026	(3, 1, 1) AIC= -1288.71	χ^2< 0.001, *p*= 0.991
457	5.00	3.51	Linear= .003 Quadratic= .02 **Cubic=.21**	*ADF*= -5.62, *p*<0.001	(1, 2, 3) AIC= -1230.20	χ^2= 1.26, *p*= 0.261
458	4.10	3.74	Linear= .14 **Quadratic= .41** Cubic= .44	*ADF*= -5.85, *p*= 0.01	(1, 2, 2) AIC= -1171.83	χ^2= 1.91, *p*= 0.167
459	6.58	4.86	Linear= .03 Quadratic= .08 **Cubic= .54**	*ADF*= -7.08, *p*<0.001	(2, 2, 0) AIC= -1222.42	χ^2= 0.37, *p*= 0.543
460	4.28	3.30	Linear= .38 Quadratic= .47 **Cubic=.64**	*ADF*= -6.92, *p*<0.001	(2, 2, 0) AIC= -1152.71	χ^2= 1.77, *p*= 0.183
500	6.01	5.26	Linear= .15 **Quadratic= .46** Cubic= .45	*ADF*= -3.59, *p*=0.037	(4, 0, 0) AIC= -1365.77	χ^2= 0.44, *p*= 0.509
501	6.99	5.90	Linear= .10 **Quadratic= .16** Cubic= .16	*ADF*= -4.55, *p*<0.001	(3, 2, 0) AIC= -1375.95	χ^2= 0.04, *p*= 0.839
504	3.87	3.26	Linear= .33 **Quadratic= .51** Cubic= .52	*ADF*= -3.87, *p*= 0.018	(2, 1, 1) AIC= -949.23	χ^2= 2.10, *p*= 0.147
505	4.80	4.29	Linear= .43 **Quadratic= .69** Cubic= .70	*ADF*= -6.67, *p*<0.001	(2, 2, 0) AIC= -1256.24	χ^2= 0.74, *p*= 0.389
506	7.50	5.65	Linear= .16 Quadratic= .16 **Cubic= .20**	*ADF*= -7.51, *p*<0.001	(3, 2, 0) AIC= -1067.46	χ^2= 0.41, *p*= 0.524
508	6.47	0.20	Linear= .04 Quadratic= .15 **Cubic= .26**	*ADF*= -3.76, *p*= 0.023	(3, 0, 3) AIC= -1294.87	χ^2= 2.26, *p*= 0.133
510	6.37	3.96	Linear= .01 Quadratic= .14 **Cubic= .21**	*ADF*= -3.64, *p*= 0.032	(3, 1, 0) AIC= -1180.40	χ^2 = 1.10, *p*= 0.295
514	4.46	3.90	Linear= .30 Quadratic= .30 **Cubic= .36**	*ADF*= -3.99, *p*= 0.012	(3, 0, 3) AIC= -1261.00	χ^2= 0.29, *p*= 0.588
516	6.68	6.22	Linear= .54 **Quadratic= .75**	*ADF*= -4.97, *p*<.001	(2, 2, 1) AIC=	χ^2= 1.52, *p*= 0.217

			Cubic= .75		-1076.84	
517	5.58	3.48	Linear= .59 Quadratic= .76 **Cubic= .81**	*ADF*= -4.17, *p*<0.001	(3, 0, 1) AIC= -1315.95	χ^2= 2.77, *p*= 0.096
25897	6.08	3.57	Linear= .04 Quadratic= .10 **Cubic= .19**	*ADF*= -5.49, *p*<0.001	(2, 2, 1) AIC= -1177.84	χ^2=0.01, *p*= 0.906
26035	5.06	4.74	Linear <.001 Quadratic=.51 **Cubic= .59**	*ADF*= -4.14, *p*<0.001	(3, 1, 1) AIC= -1340.95	χ^2= 0.26, *p*= 0.612
26104	4.80	3.43	Linear= .14 Quadratic= .20 **Cubic= .24**	*ADF*= -3.56, *p*= 0.040	(4, 1, 0) AIC= -1257.02	χ^2= 0.07, *p*= 0.798
26116	5.53	4.40	**Linear= .23** Quadratic= .25 Cubic= .25	*ADF*= -3.66, *p*= 0.03042	(3, 1, 2) AIC= -1169.86	χ^2= 3.48, *p*= 0.062
26146	4.57	4.38	Linear= .02 Quadratic= .36 **Cubic= .41**	*ADF*= -4.69, *p*< 0.001	(1, 2, 2) AIC= -1024.30	χ^2= 3.44, *p*= 0.064
26152	6.13	3.48	Linear= .002 Quadratic= .59 **Cubic= .76**	*ADF*= -4.88, *p*< 0.001	(2,1,1) AIC= -1271.57	χ^2= 2.04, *p*= 0.153
26194	6.61	5.84	Linear= .03 Quadratic= .04 **Cubic= .15**	*ADF*= -4.30, *p*< 0.001	(3, 1, 1) AIC= -1423.77	χ^2= 0.75, *p*= 0.388
26242	5.56	4.63	Linear= .24 Quadratic= .33 **Cubic= .59**	*ADF*= -3.76, *p*= 0.023	(3, 1, 1) AIC= -1158.93	χ^2= 1.03, *p*= 0.311
26269	4.02	3.70	Linear= .33 Quadratic= .48 **Cubic= .59**	*ADF*= -6.49, *p*< 0.001	(2, 2, 0) AIC= -924.39	χ^2= 1.67, *p*= 0.196
26275	4.32	5.63	Linear= .06 Quadratic= .16 **Cubic= .70**	*ADF*= -3.5, *p*= 0.045	(2, 1, 1) AIC= -1078.7	χ^2= 3.99, *p*= 0.046
26371	6.72	5.76	Linear= .05 Quadratic= .08 **Cubic= .69**	*ADF*= -6.81, *p*< 0.001	(1, 2, 3) AIC= -1270.6	χ^2=0.26, *p*= 0.608
26452	7.44	6.13	Linear= .06 Quadratic= .17 **Cubic= .21**	*ADF*= -3.50, *p*= 0.045	(1, 2, 2) AIC= -1164.16	χ^2 = 1.41, *p*= 0.236
26458	5.02	4.95	Linear= .02 Quadratic= .02 **Cubic= .16**	*ADF*= -6.40, *p*< 0.001	(3, 2, 0) AIC= -1269.4	χ^2=0.07, *p*= 0.785
26461	4.35	3.26	Linear= .18 Quadratic= .18	*ADF*= -3.46, *p*= 0.048	(3, 0, 1) AIC=	χ^2=0.85, *p*= 0.358

			Cubic= .25		-921.54	
26527	6.60	5.63	Linear= .24 Quadratic= .33 **Cubic= .58**	*ADF*= -5.85, *p*< 0.001	(2, 1, 1) AIC= -1323.63	χ^2=0.02, *p*= 0.889
26701	5.49	4.33	Linear= .001 Quadratic= .18 **Cubic= .53**	*ADF*= -4.26, *p*< 0.001	(2, 0, 3) AIC= -1191.61	χ^2= 2.02, *p*= 0.155
26713	4.99	5.17	Linear= .64 Quadratic= .69 **Cubic= .78**	*ADF*= -6.75, *p*< 0.001	(2, 2, 1) AIC= -1229.33	χ^2= 0.49, *p*= 0.482
26734	6.06	6.28	Linear= .48 Quadratic= .55 **Cubic= .58**	*ADF*= -4.14, *p*< 0.001	(2, 1, 2) AIC= -1464.58	χ^2= 1.89, *p*= 0.169
26746	5.34	4.81	Linear= .08 Quadratic= .25 **Cubic= .33**	*ADF*= -3.44, *p*= 0.060	(3, 1, 0) AIC= -1221.43	χ^2<.001 *p*= 0.997
26821	5.33	3.63	Linear= .35 Quadratic= .46 **Cubic= .58**	*ADF*= -3.58, *p*= 0.03791	(2, 0, 3) AIC= -1182.43	χ^2= 1.21, *p*= 0.271
26872	8.57	5.09	Linear= .07 Quadratic= .16 **Cubic= .27**	*ADF*= -4.06, *p*< 0.001	(4, 0, 0) AIC= -1473.5	χ^2= 2.48, *p*= 0.116
26956	5.83	3.68	Linear= .41 Quadratic= .64 **Cubic= .68**	*ADF*= -4.18, *p*< 0.001	(3, 1, 0) AIC= -1378.47	χ^2= 1.05, *p*= 0.306
27070	4.78	4.7096	Linear= .05 Quadratic= .20 **Cubic= .68**	*ADF*= -7.03, *p*< 0.001	(2, 2, 1) AIC= -1076.06	χ^2=0.34, *p*= 0.560
27076	5.71	3.7947	Linear= .003 Quadratic= .004 **Cubic= .01**	*ADF*= -3.54, *p*= 0.041	(3, 0, 2) AIC= -1020.99	χ^2=0.87, *p*= 0.352
27082	3.79	2.8616	Linear= .07 Quadratic= .08 **Cubic= .37**	*ADF*= -3.87, *p*= 0.018	(3, 1, 1) AIC= -1160.81	χ^2=0.26, *p*= 0.610
27205	6.34	5.6796	Linear= .43 **Quadratic= .53** Cubic= .53	*ADF*= -6.38, *p*< 0.001	(1, 2, 2) AIC= -1027.65	χ^2= 2.97, *p*= 0.085
27265	3.07	2.5629	Linear= .38 Quadratic= .44 **Cubic= .75**	*ADF*= -4.26, *p*< 0.001	(3, 0, 2) AIC= -1133.84	χ^2= 2.36, *p*= 0.124
27289	5.23	4.9515	Linear= .58 Quadratic= .61 **Cubic= .64**	*ADF*= -5.30, *p*< 0.001	(2, 2, 0) AIC= -1399.83	χ^2=0.55, *p*= 0.459
27673	4.83	4.4304	Linear= .32	*ADF*= -5.95,	(3, 2, 0)	χ^2=0.09,

			Quadratic= .20 **Cubic= .73**	*p*< 0.001	AIC= -1187.46	*p*= 0.759
27685	5.42	4.6663	Linear= .27 Quadratic= .47 **Cubic= .62**	*ADF*= -6.66, *p*< 0.001	(1, 2, 2) AIC= -1059.67	χ^2= 3.01, *p*= 0.083
27784	5.00	2.6494	Linear= .14 Quadratic= .41 **Cubic= .46**	*ADF*= -3.52, *p*= 0.043	(2, 1, 2) AIC= -1191.19	χ^2= 1.40, *p*= 0.237
28348	5.15	3.9454	Linear= .12 **Quadratic= .26** Cubic= .29	*ADF*= -6.79, *p*< 0.001	(1, 2, 2) AIC= -1186.19	χ^2= 3.66, *p*= 0.056
28651	7.04	5.0534	Linear= .04 Quadratic= .09 **Cubic= .17**	*ADF*= -3.47, *p*= 0.048	(3, 1, 1) AIC= -1115.72	χ^2= 0.40, *p*= 0.525
28792	5.59	3.5734	Linear= .08 Quadratic= .19 **Cubic= .79**	*ADF*= -3.74, *p*= 0.024	(3, 0, 1) AIC= -1040.21	χ^2= 3.75, *p*= 0.053
29014	4.86	3.7105	Linear= .002 **Quadratic= .51** Cubic= .52	*ADF*= -5.16, *p*< 0.001	(2, 2, 1) AIC= -1241.9	χ^2= 1.28, *p*= 0.258
29212	6.21	4.0638	Linear= .43 **Quadratic= .56** Cubic= .56	*ADF*= -3.91, *p*= 0.015	(4, 0, 0) AIC= -1221.48	χ^2=0.01, *p*= 0.933
29275	6.61	2.8706	Linear= .01 Quadratic= .01 **Cubic= .33**	*ADF*= -3.68, *p*= 0.029	(3, 1, 0) AIC= -1215.58	χ^2=0.06, *p*= 0.810
29293	6.01	4.7214	Linear<.001 Quadratic=.001 **Cubic= .40**	*ADF*= -5.46, *p*< 0.001	(3, 2, 0) AIC= -1242.18	χ^2=0.01, *p*= 0.904
29341	6.10	4.5358	Linear= .10 Quadratic= .10 **Cubic= .33**	*ADF*= -4.35, *p*< 0.001	(1, 2, 2) AIC= -1237.14	χ^2= 2.92, *p*= 0.087
29419	6.26	4.1157	Linear= .76 **Quadratic= .81** Cubic= .81	*ADF*= -6.86, *p*< 0.001	(3, 2, 0) AIC= -1149.17	χ^2=0.05, *p*= 0.821
29617	5.54	4.9604	Linear= .52 Quadratic= .58 **Cubic= .66**	*ADF*= -6.71, *p*< 0.001	(2, 2, 1) AIC= -1335.51	χ^2=0.22, *p*= 0.636
29686	6.28	4.3192	Linear= .02 Quadratic= .05 **Cubic= .46**	*ADF*= -3.48, *p*= 0.047	(3, 0, 2) AIC= -1106.54	χ^2= 3.72, *p*= 0.054
29698	4.98	Missing	Linear= .04 Quadratic= .05 **Cubic= .12**	*ADF*= -6.05, *p*< 0.001	(2, 2, 0) AIC= -1072.49	χ^2= 2.83, *p*= 0.093

29761	5.75	5.2076	Linear= .21 Quadratic= .28 **Cubic= .42**	*ADF*= -3.62, *p*=0.034	(3, 0, 1) AIC= -1146.7	χ^2= 2.94, *p*= 0.087
29791	5.86	3.8181	Linear<.001 **Quadratic= .28** Cubic= .30	*ADF*= -6.62, *p*< 0.001	(2, 2, 0) AIC= -1155.54	χ^2=0.69, *p*= 0.405
30550	5.89	3.5470	Linear= .02 Quadratic= .03 **Cubic= .37**	*ADF*= -4.13, *p*< 0.001	(2, 1, 1) AIC= -511.53	χ^2= 1.62, *p*= 0.203
30559	6.06	6.5737	Linear= .02 Quadratic= .16 **Cubic= .29**	*ADF*= -5.44, *p*< 0.001	(2, 2, 1) AIC= -1295.18	χ^2= 0.18, *p*= 0.673
31039	5.59	2.6637	Linear= .002 Quadratic= .002 **Cubic= .10**	*ADF*= -3.80, *p*= 0.021	(2, 1, 2) AIC= -1057.47	χ^2= 0.53, *p*= 0.466
31471	3.27	3.5099	Linear= .11 Quadratic= .12 **Cubic= .32**	*ADF*= -5.8394, *p*< 0.001	(1, 2, 3) AIC= -1055.16	χ^2= 1.10, *p*= 0.29
31588	6.35	4.7352	Linear= .23 Quadratic= .26 **Cubic= .41**	*ADF*= -3.64, *p*= 0.032	(4, 0, 0) AIC= -1380.22	χ^2= 1.73, *p*= 0.19
31750	5.25	3.6975	Linear= .003 **Quadratic= .70** Cubic= .70	*ADF*= -4.01, *p*= 0.011	(3, 0, 2) AIC= -1241.41	χ^2= 2.43, *p*= 0.119

Table 9.

Results of Model Addressing Specific Aim 3: All Coping Strategies

Nature of Relation	Path	Standardized Estimate	S.E.	p-value
Control	Sex to Active Coping	-.05	.10	.629
Control	Sex to Social Support	-.14	.09	.128
Control	Sex to Spiritual Coping	-.18	.07	.013*
Control	Sex to John Henryism	-.06	.11	.586
Control	Age to Active Coping	-.04	.10	.654
Control	Age to Social Support	-.03	.10	.773
Control	Age to Spiritual Coping	-.10	.09	.286
Control	Age to John Henryism	-.06	.12	.580
Control	Parental Education to Active Coping	.04	.10	.674
Control	Parental Education to Social Support	-.004	.10	.966
Control	Parental Education to Spiritual Coping	.03	.09	.733
Control	Parental Education to John Henryism	-.03	.11	.780
Control	Sex to RSA at Stress	.17	.10	.076
Control	Age to RSA at Stress	-.05	.10	.605
Control	Parental Education to RSA at Stress	-.02	.10	.843
Control	RSA at Stress to Depression	-.05	.11	.635
Control	RSA at Stress to Anxiety	-.12	.11	.265
Control	RSA at Stress to Mean RSA	.55	.07	.000***
Control	RSA at Stress to RSA Variance	-.07	.10	.489
Control	RSA at Stress to # of AR terms	-.04	.10	.669
Control	RSA at Stress to # of MA terms	-.05	.10	.617
Control	RSA at Stress to Order of Differencing	.11	.09	.233
Focal Relation	Mean RSA to Depression	.13	.11	.257
Focal Relation	RSA Variance to Depression	.03	.10	.771
Focal Relation	# of AR terms to Depression	-.17	.13	.193
Focal Relation	# of MA terms to Depression	-.09	.11	.434
Focal Relation	Order of Differencing to Depression	-.13	.12	.281
Focal Relation	Mean RSA to Anxiety	.06	.11	.592

Focal Relation	RSA Variance to Anxiety	.02	.10	.821
Focal Relation	# of AR terms to Anxiety	-.26	.12	.029*
Focal Relation	# of MA terms to Anxiety	-.14	.10	.188
Focal Relation	Order of Differencing to Anxiety	-.07	.12	.543
Focal Relation	Active Coping to Mean RSA	-.12	.10	.224
Focal Relation	Social Support to Mean RSA	.13	.10	.181
Focal Relation	Spiritual Coping to Mean RSA	-.12	.09	.205
Focal Relation	John Henryism to Mean RSA	.04	.10	.678
Focal Relation	Active Coping to RSA Variance	.29	.11	.005**
Focal Relation	Social Support to RSA Variance	-.25	.11	.032*
Focal Relation	Spiritual Coping to RSA Variance	.14	.11	.176
Focal Relation	John Henryism to RSA Variance	-.03	.12	.808
Focal Relation	Active Coping to # of AR terms	.10	.11	.406
Focal Relation	Social Support to # of AR terms	.02	.12	.866
Focal Relation	Spiritual Coping to # of AR terms	-.20	.11	.067
Focal Relation	John Henryism to # of AR terms	-.03	.12	.792
Focal Relation	Active Coping to # of MA terms	-.04	.11	.712
Focal Relation	Social Support to # of MA terms	.07	.12	.564
Focal Relation	Spiritual Coping to # of MA terms	.12	.11	.289
Focal Relation	John Henryism to # of MA terms	.007	.12	.955
Focal Relation	Active Coping to Order of Difference	-.16	.11	.133
Focal Relation	Social Support to Order of Difference	-.09	.11	.425
Focal Relation	Spiritual Coping to Order of Difference	.28	.10	.003**
Focal Realtion	John Henryism to Order of Difference	.06	.11	.563
Focal Relation	Active Coping to Depression	-.09	.12	.448
Focal Relation	Social Support to Depression	.01	.12	.906
Focal Relation	Spiritual Coping to Depression	.03	.11	.764
Focal Relation	John Henryism to Depression	-.23	.09	.009*
Focal Relation	Active Coping to Anxiety	-.11	.11	.355

Focal Relation	Social Support to Anxiety	.13	.12	.251
Focal Relation	Spiritual Coping to Anxiety	.16	.11	.127
Focal Relation	John Henryism to Anxiety	-.24	.09	.011*
Covariance	Depression with Anxiety	.50	.07	.000***
Covariance	# AR terms with # MA terms	-.35	.08	.000***
Covariance	# AR terms with order of difference	-.47	.07	.000***
Covariance	# MA terms with order of difference	-.23	.09	.009**
Covariance	Active Coping with Social Support	.51	.07	.000***
Covariance	Active Coping with Spiritual Coping	.23	.09	.009**
Covariance	Active Coping with John Henryism	.14	.11	.195
Covariance	Social Support with Spiritual Coping	.40	.08	.000***
Covariance	Social Support with John Henryism	-.04	.12	.744
Covariance	Spiritual Coping with John Henryism	.21	.11	.049*

*p<.05, **p<.01, ***p<.001, †p<.07

Table 10.

Results of Model Addressing Specific Aim 3: All Coping Strategies with Robust Standard Errors

Nature of Relation	Path	Standardized Estimate	S.E.	p-value
Control	Sex to Active Coping	-.05	.09	.591
Control	Sex to Social Support	-.14	.09	.099
Control	Sex to Spiritual Coping	-.18	.07	.011*
Control	Sex to John Henryism	-.06	.13	.636
Control	Age to Active Coping	-.04	.12	.712
Control	Age to Social Support	-.03	.10	.780
Control	Age to Spiritual Coping	-.10	.12	.396
Control	Age to John Henryism	-.06	.11	.563
Control	Parental Education to Active Coping	.04	.09	.649
Control	Parental Education to Social Support	-.004	.09	.964
Control	Parental Education to Spiritual Coping	.03	.08	.707
Control	Parental Education to John Henryism	-.03	.10	.764
Control	Sex to RSA at Stress	.17	.10	.088
Control	Age to RSA at Stress	-.05	.10	.643
Control	Parental Education to RSA at Stress	-.02	.10	.850
Control	RSA at Stress to Depression	-.05	.11	.624
Control	RSA at Stress to Anxiety	-.12	.10	.222
Control	RSA at Stress to Mean RSA	.55	.10	.000***
Control	RSA at Stress to RSA Variance	-.07	.09	.461
Control	RSA at Stress to # of AR terms	-.04	.09	.661
Control	RSA at Stress to # of MA terms	-.05	.10	.638
Control	RSA at Stress to Order of Differencing	.11	.10	.270
Focal Relation	Mean RSA to Depression	.13	.11	.251
Focal Relation	RSA Variance to Depression	.03	.10	.788
Focal Relation	# of AR terms to Depression	-.17	.12	.178
Focal Relation	# of MA terms to Depression	-.09	.11	.422

Focal Relation	Order of Differencing to Depression	-.13	.12	.255
Focal Relation	Mean RSA to Anxiety	.06	.09	.539
Focal Relation	RSA Variance to Anxiety	.02	.09	.814
Focal Relation	# of AR terms to Anxiety	-.26	.09	.004**
Focal Relation	# of MA terms to Anxiety	-.14	.11	.181
Focal Relation	Order of Differencing to Anxiety	-.07	.10	.463
Focal Relation	Active Coping to Mean RSA	-.12	.10	.228
Focal Relation	Social Support to Mean RSA	.13	.09	.153
Focal Relation	Spiritual Coping to Mean RSA	-.12	.10	.234
Focal Relation	John Henryism to Mean RSA	.04	.10	.653
Focal Relation	Active Coping to RSA Variance	.29	.09	.001**
Focal Relation	Social Support to RSA Variance	-.25	.10	.018*
Focal Relation	Spiritual Coping to RSA Variance	.14	.12	.210
Focal Relation	John Henryism to RSA Variance	-.03	.12	.803
Focal Relation	Active Coping to # of AR terms	.10	.11	.366
Focal Relation	Social Support to # of AR terms	.02	.12	.862
Focal Relation	Spiritual Coping to # of AR terms	-.20	.11	.079
Focal Relation	John Henryism to # of AR terms	-.03	.10	.741
Focal Relation	Active Coping to # of MA terms	-.04	.11	.698
Focal Relation	Social Support to # of MA terms	.07	.12	.562
Focal Relation	Spiritual Coping to # of MA terms	.12	.11	.296
Focal Relation	John Henryism to # of MA terms	.007	.10	.947
Focal Relation	Active Coping to Order of Difference	-.16	.10	.115
Focal Relation	Social Support to Order of Difference	-.09	.11	.405

Focal Relation	Spiritual Coping to Order of Difference	.28	.09	.002**
Focal Realtion	John Henryism to Order of Difference	.06	.10	.530
Focal Relation	Active Coping to Depression	-.09	.11	.396
Focal Relation	Social Support to Depression	.01	.13	.911
Focal Relation	Spiritual Coping to Depression	.03	.11	.766
Focal Relation	John Henryism to Depression	-.23	.09	.008**
Focal Relation	Active Coping to Anxiety	-.11	.13	.406
Focal Relation	Social Support to Anxiety	.13	.12	.281
Focal Relation	Spiritual Coping to Anxiety	.16	.10	.103
Focal Relation	John Henryism to Anxiety	-.24	.11	.028*
Covariance	Depression with Anxiety	.50	.09	.000***
Covariance	# AR terms with # MA terms	-.35	.09	.000***
Covariance	# AR terms with Order of Difference	-.47	.09	.000***
Covariance	# MA terms with Order of Difference	-.23	.09	.013*
Covariance	Active Coping with Social Support	.51	.07	.000***
Covariance	Active Coping with Spiritual Coping	.23	.10	.015*
Covariance	Active Coping with John Henryism	.14	.11	.196
Covariance	Social Support with Spiritual Coping	.40	.08	.000***
Covariance	Social Support with John Henryism	-.04	.11	.723
Covariance	Spiritual Coping with John Henryism	.21	.11	.061

*p<.05, **p<.01, ***p<.001, † p<.07

Table 11.
Results of Model Addressing Specific Aim 3: Just John Henryism

Nature of Relation	Path	Standardized Estimate	S.E.	p-value
Control	Sex to John Henryism	-.04	.11	.699
Control	Age to John Henryism	-.09	.11	.459
Control	Parental Education to John Henryism	-.03	.11	.765
Control	Sex to RSA at Stress	.17	.10	.078
Control	Age to RSA at Stress	-.05	.10	.604
Control	Parental Education to RSA at Stress	-.02	.10	.834
Control	RSA at Stress to Depression	-.05	.11	.689
Control	RSA at Stress to Anxiety	-.10	.11	.392
Control	RSA at Stress to Mean RSA	.56	.07	.000***
Control	RSA at Stress to RSA Variance	-.12	.09	.220
Control	RSA at Stress to # of AR terms	-.08	.09	.412
Control	RSA at Stress to # of MA terms	-.03	.10	.780
Control	RSA at Stress to Order of Difference	.16	.09	.074
Focal Relation	Mean RSA to Depression	.13	.11	.226
Focal Relation	RSA Variance to Depression	.02	.10	.835
Focal Relation	# of AR terms to Depression	-.17	.13	.180
Focal Relation	# of MA terms to Depression	-.08	.11	.452
Focal Relation	Order of Difference to Depression	-.11	.12	.342
Focal Relation	Mean RSA to Anxiety	.06	.11	.590
Focal Relation	RSA Variance to Anxiety	.005	.09	.959
Focal Relation	# of AR terms to Anxiety	-.25	.12	.037*
Focal Relation	# of MA terms to Anxiety	-.10	.11	.361
Focal Relation	Order of Difference to Anxiety	-.02	.12	.877
Focal Relation	John Henryism to Mean RSA	-.008	.10	.933
Focal Relation	John Henryism to RSA Variance	.08	.11	.472
Focal Relation	John Henryism to # of AR terms	-.08	.12	.506
Focal Relation	John Henryism to # of MA terms	.02	.11	.834
Focal Relation	John Henryism to Order of Difference	.12	.11	.274
Focal Relation	John Henryism to Depression	-.27	.08	.001**
Focal Relation	John Henryism to Anxiety	-.11	.11	.008**
Covariance	Depression with Anxiety	.49	.07	.000***
Covariance	# AR terms with # MA terms	-.36	.08	.000***
Covariance	# AR terms with Order of Difference	-.50	.07	.000***
Covariance	# MA terms with Order of Difference	-.20	.10	.031*

*p<.05, **p<.01, ***p<.001, †p<.07

Table 12.

Results of Model Addressing Specific Aim 3: Just John Henryism with Robust Standard Errors

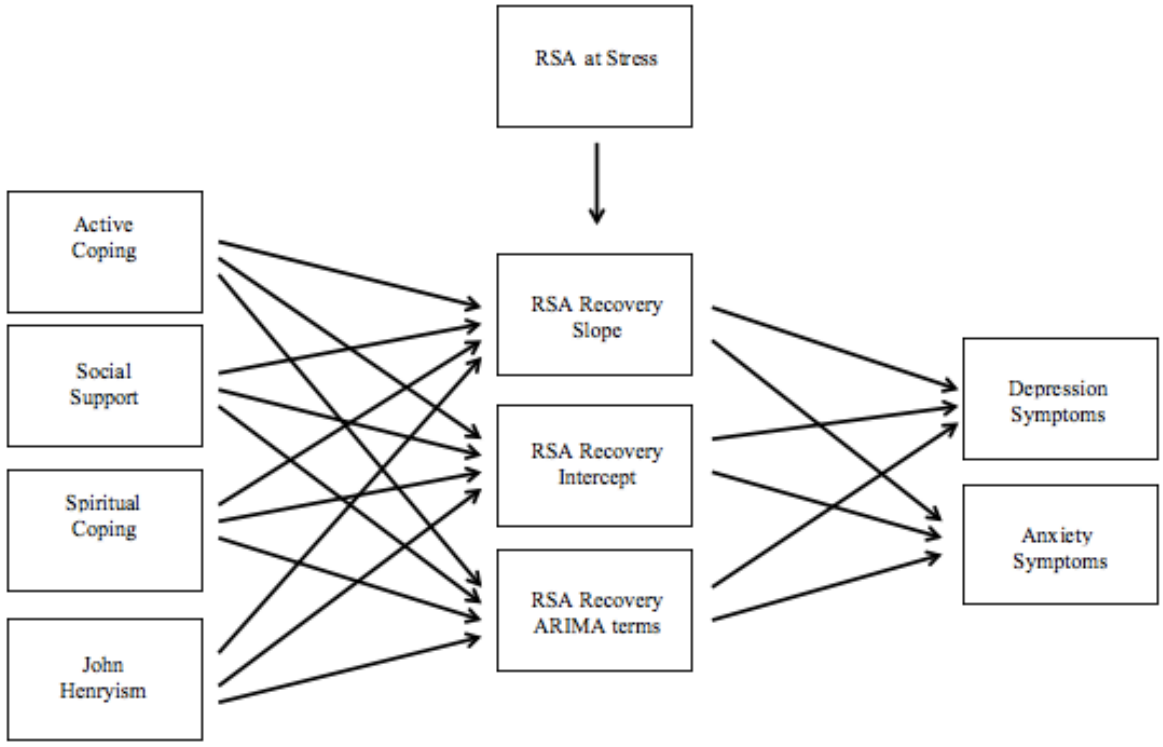
Nature of Relation	Path	Standardized Estimate	S.E.	p-value
Control	Sex to John Henryism	-.04	.13	.732
Control	Age to John Henryism	-.09	.11	.430
Control	Parental Education to John Henryism	-.03	.10	.748
Control	Sex to RSA at Stress	.17	.10	.089
Control	Age to RSA at Stress	-.05	.11	.641
Control	Parental Education to RSA at Stress	-.02	.10	.841
Control	RSA at Stress to Depression	-.05	.10	.666
Control	RSA at Stress to Anxiety	-.10	.09	.300
Control	RSA at Stress to Mean RSA	.56	.09	.000***
Control	RSA at Stress to RSA Variance	-.12	.09	.213
Control	RSA at Stress to # of AR terms	-.08	.09	.398
Control	RSA at Stress to # of MA terms	-.03	.10	.784
Control	RSA at Stress to Order of Difference	.16	.09	.074
Focal Relation	Mean RSA to Depression	.13	.11	.213
Focal Relation	RSA Variance to Depression	.02	.11	.852
Focal Relation	# of AR terms to Depression	-.17	.12	.154
Focal Relation	# of MA terms to Depression	-.08	.11	.437
Focal Relation	Order of Difference to Depression	-.11	.11	.301
Focal Relation	Mean RSA to Anxiety	.06	.10	.547
Focal Relation	RSA Variance to Anxiety	.005	.10	.960
Focal Relation	# of AR terms to Anxiety	-.25	.09	.007**
Focal Relation	# of MA terms to Anxiety	-.10	.11	.376
Focal Relation	Order of Difference to Anxiety	-.02	.10	.853
Focal Relation	John Henryism to Mean RSA	-.008	.09	.930
Focal Relation	John Henryism to RSA Variance	.08	.10	.472
Focal Relation	John Henryism to # of AR terms	-.08	.09	.407
Focal Relation	John Henryism to # of MA terms	.02	.09	.805
Focal Relation	John Henryism to Order of Difference	.12	.10	.233
Focal Relation	John Henryism to Depression	-.27	.08	.001**
Focal Relation	John Henryism to Anxiety	-.24	.10	.018*
Covariance	Depression with Anxiety	.49	.09	.000***
Covariance	# AR terms with # MA terms	-.36	.09	.000***
Covariance	# AR terms with Order of Difference	-.50	.09	.000***
Covariance	# MA terms with Order of Difference	-.20	.10	.045*

*p<.05, **p<.01, ***p<.001, † p<.07

Table 13.
Model Comparison – Fit Criteria

Fit Index	**Model All Coping Strategies**	**Model John Henryism**
Chi-Square Test of Model Fit	$\chi^2(32)= 46.24$, $p= .050$	$\chi^2(29)= 35.63$, $p=.18$
RMSEA	.05, 90% CI [.00, .08], $p=.547$	.03, 90% CI [.00, .07], $p=.77$
CFI	.95	.96
TLI	.84	.92
SRMR	.05	.06

Figure 1. *Initial empirical model.*



Note: For simplification, potential quadratic slopes and controls (age, sex, and parental education) are not included and ARIMA terms have been collapsed. Covariances will be estimated amongst all coping strategies, amongst all RSA recovery terms, and between depression and anxiety symptoms.

Figure 2. *Specific Aim 1.*

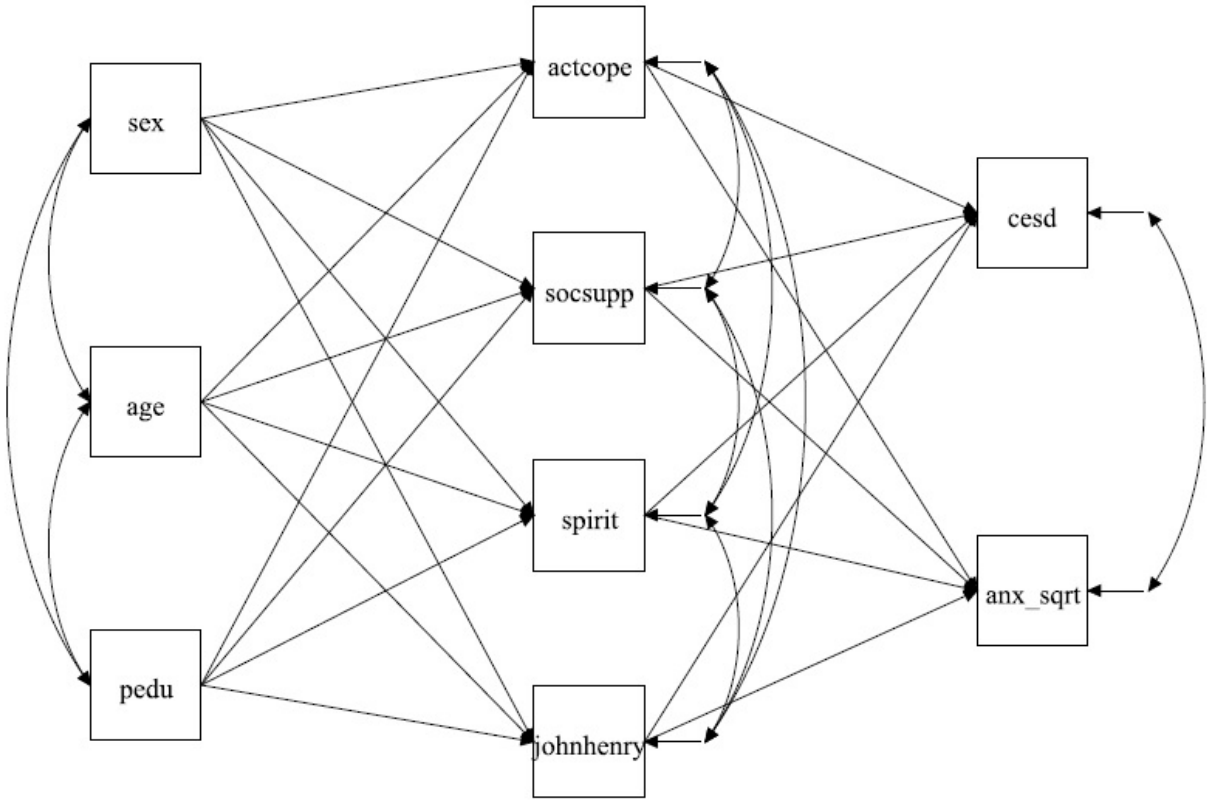
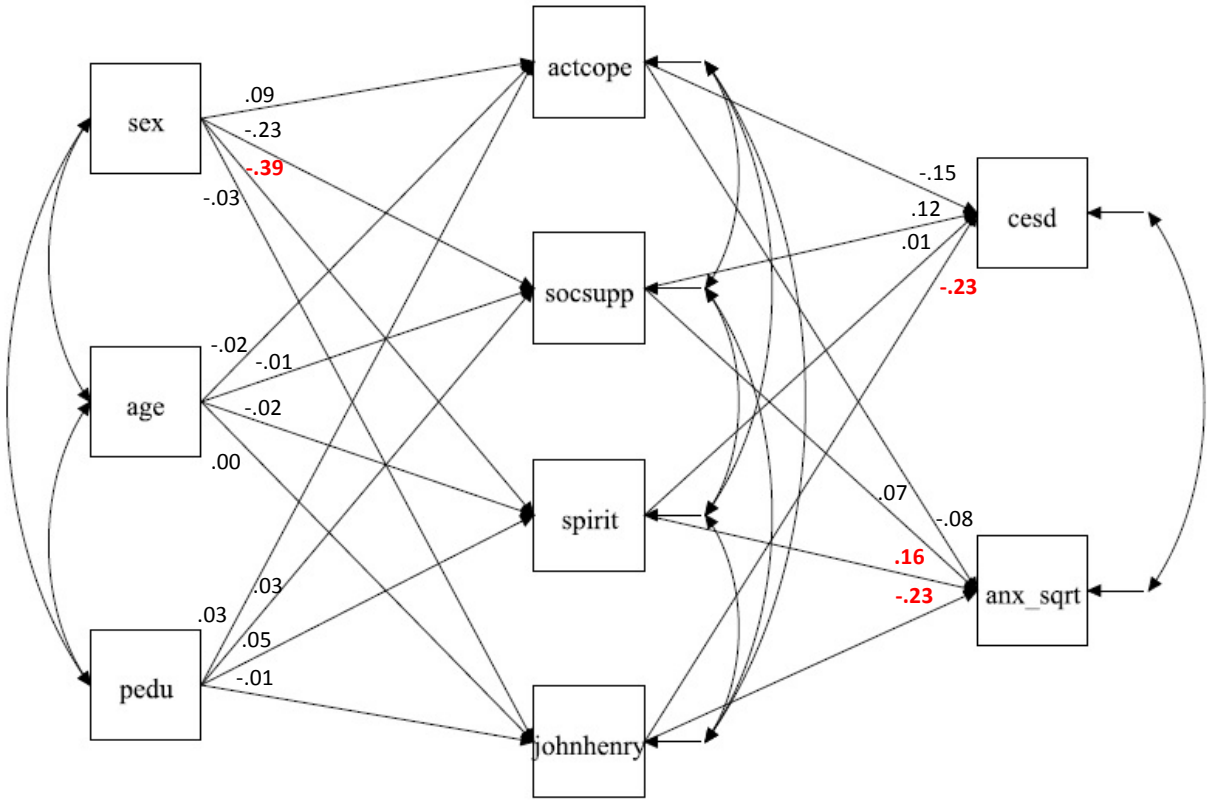
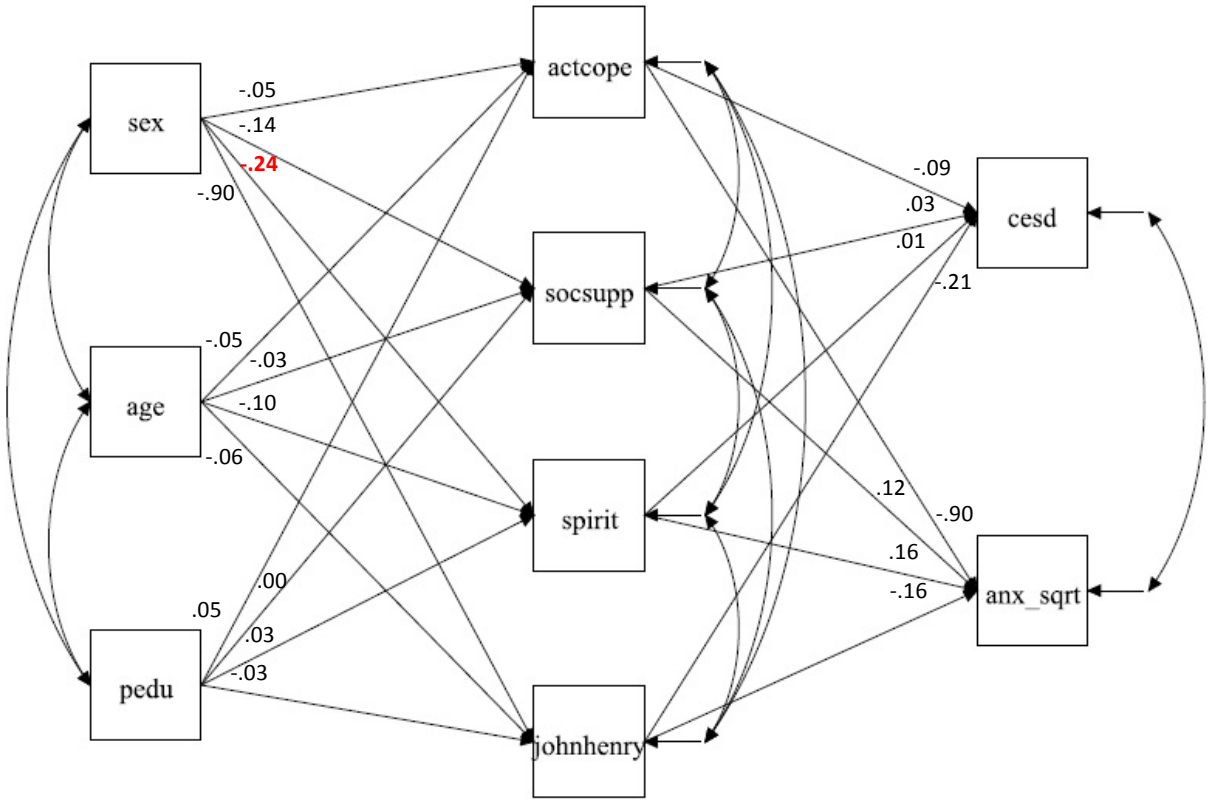


Figure 3. *Specific Aim 1 – Significant Paths (n=205).*



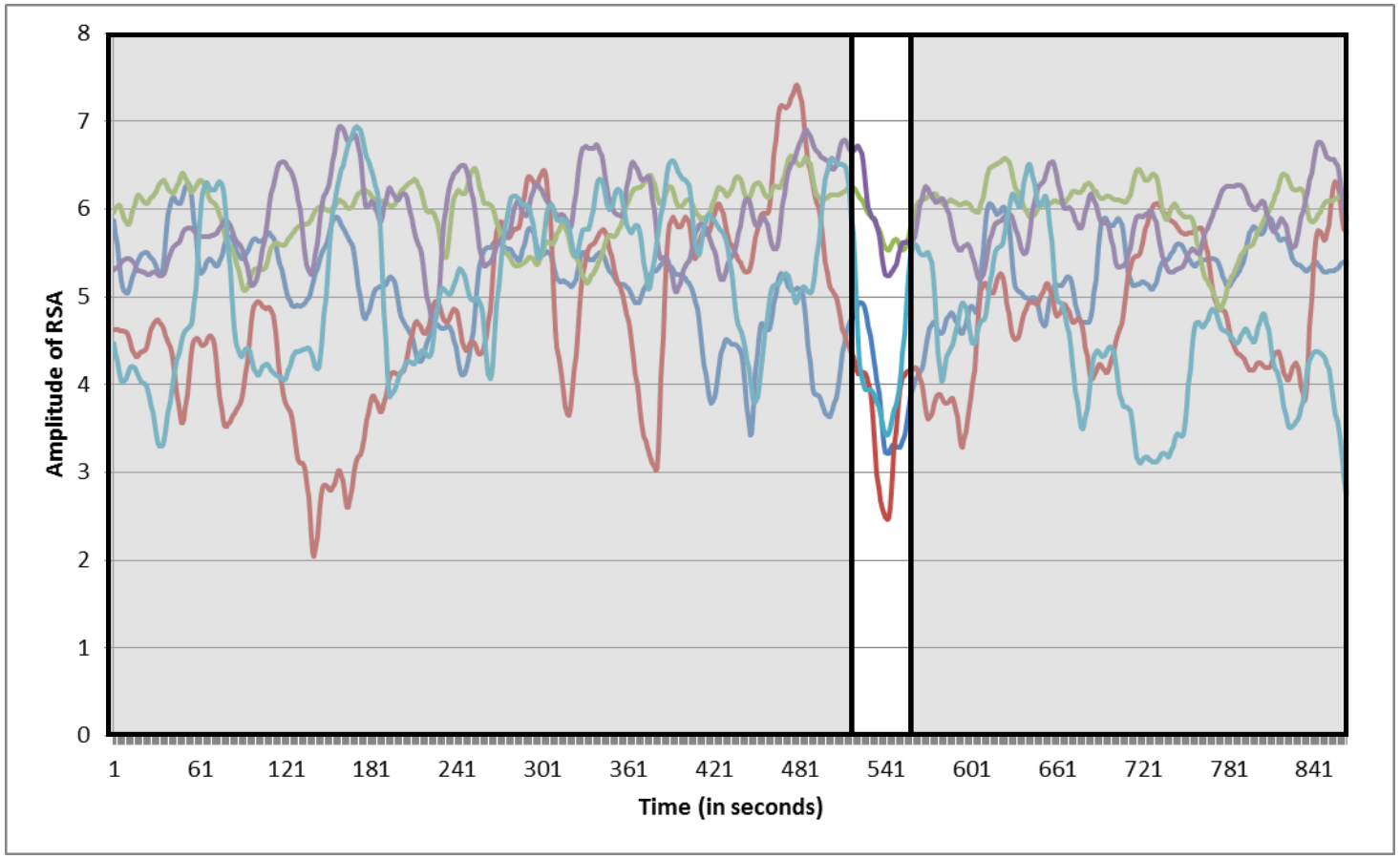
Note: Statistically significant standardized estimates are indicated in red.

Figure 4. *Specific Aim 1 – Significant Paths (n=115).*



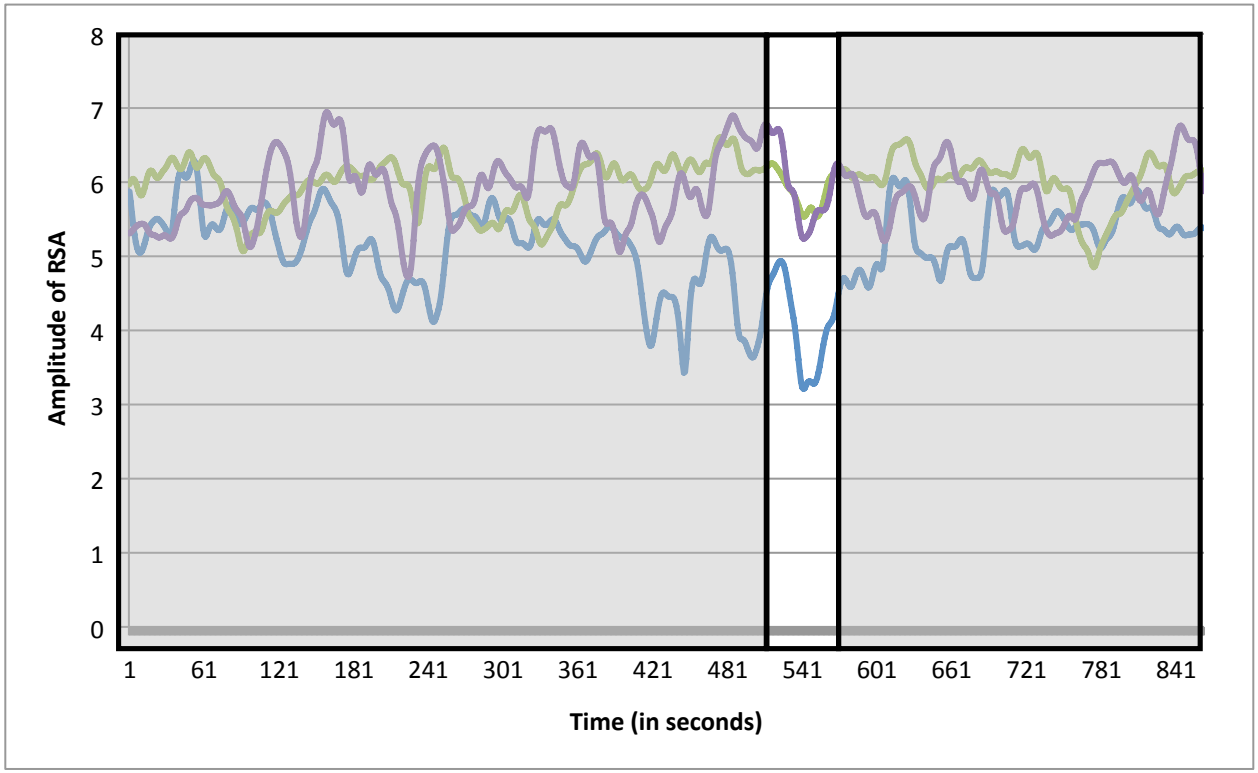
Note: Statistically significant standardized estimates are indicated in red.

Figure 5. *Spaghetti Plots of RSA across Laboratory Paradigm*



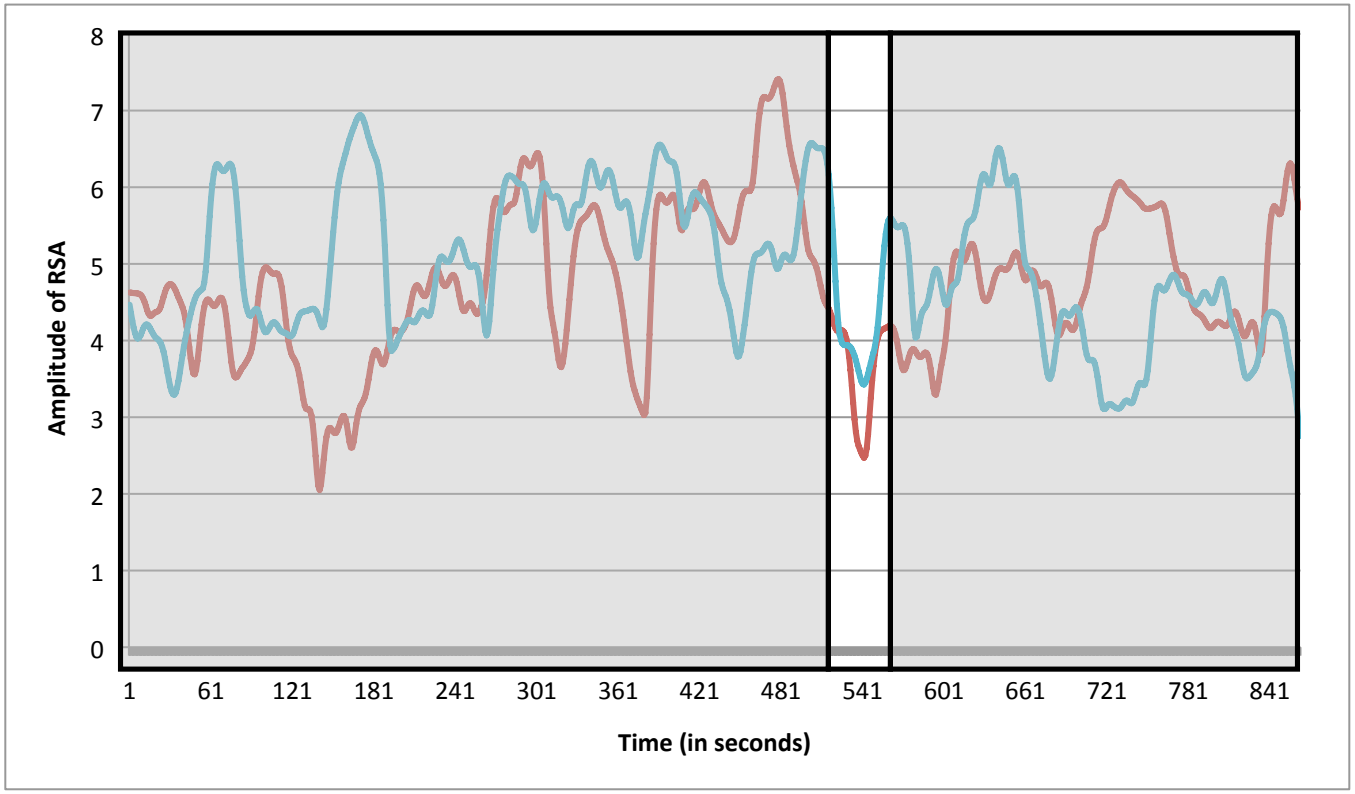
Note: RSA values at stress are indicated in the clear window. Time in seconds is graphed on the horizontal axis, however the duration of the stress task varied from participant to participant, therefore each RSA series has been graphed with respect to RSA value at stress.

Detail – Subset of participants (from above) with small to moderate degree of variability across the laboratory paradigm



Note: RSA values at stress are indicated in the clear window. Time in seconds is graphed on the horizontal axis, however the duration of the stress task varied from participant to participant, therefore each RSA series has been graphed with respect to RSA value at stress.

Detail – Subset of participants (from above) with large degree of variability across the laboratory paradigm



Note: RSA values at stress are indicated in the clear window. Time in seconds is graphed on the horizontal axis, however the duration of the stress task varied from participant to participant, therefore each RSA series has been graphed with respect to RSA value at stress.

Figure 6. *Time Series Decomposition for Participant 506.*

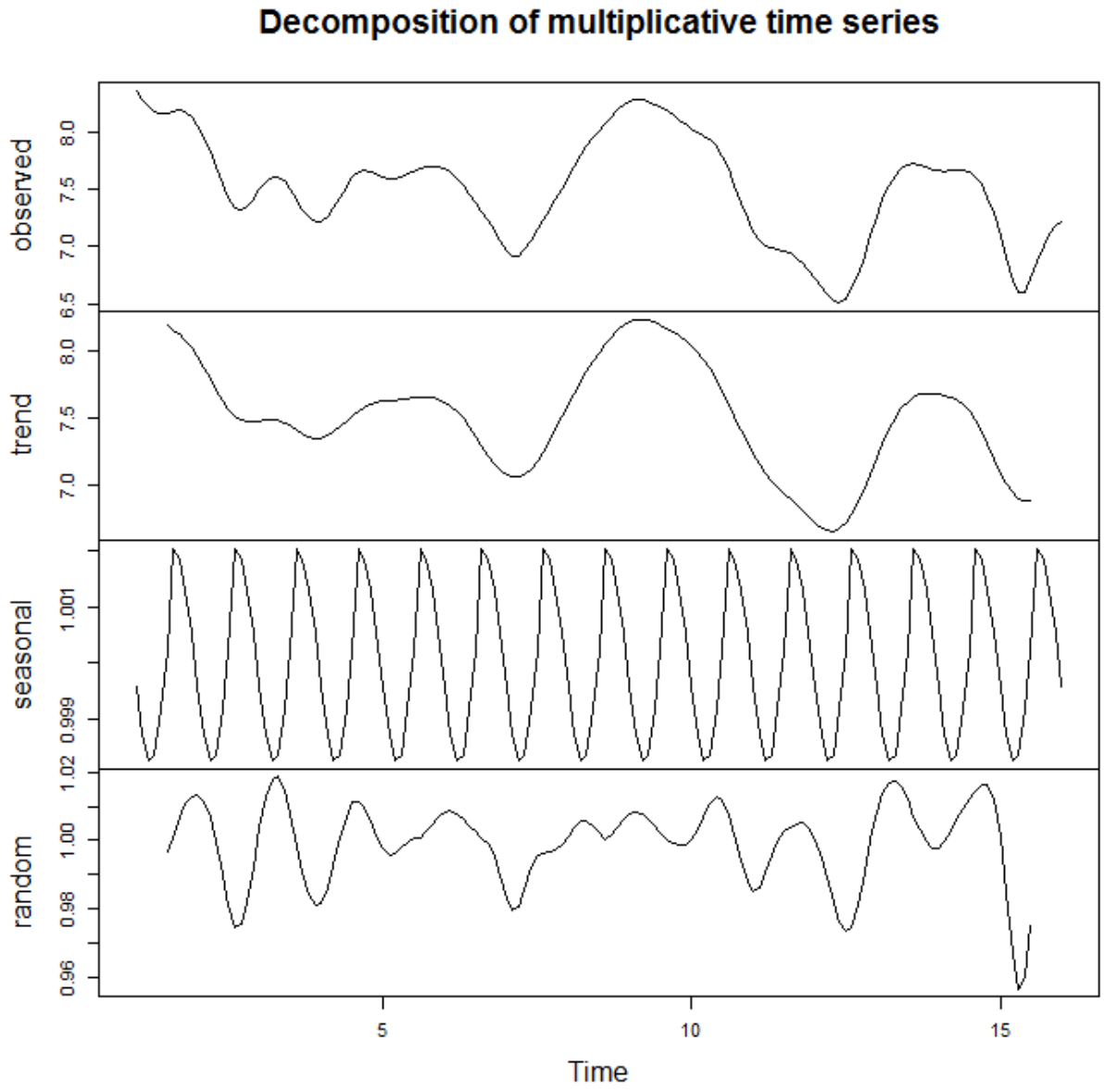


Figure 7. *Partial Autocorrelation Function Plot of Residuals of Cubic Regression Model for Participant 506.*

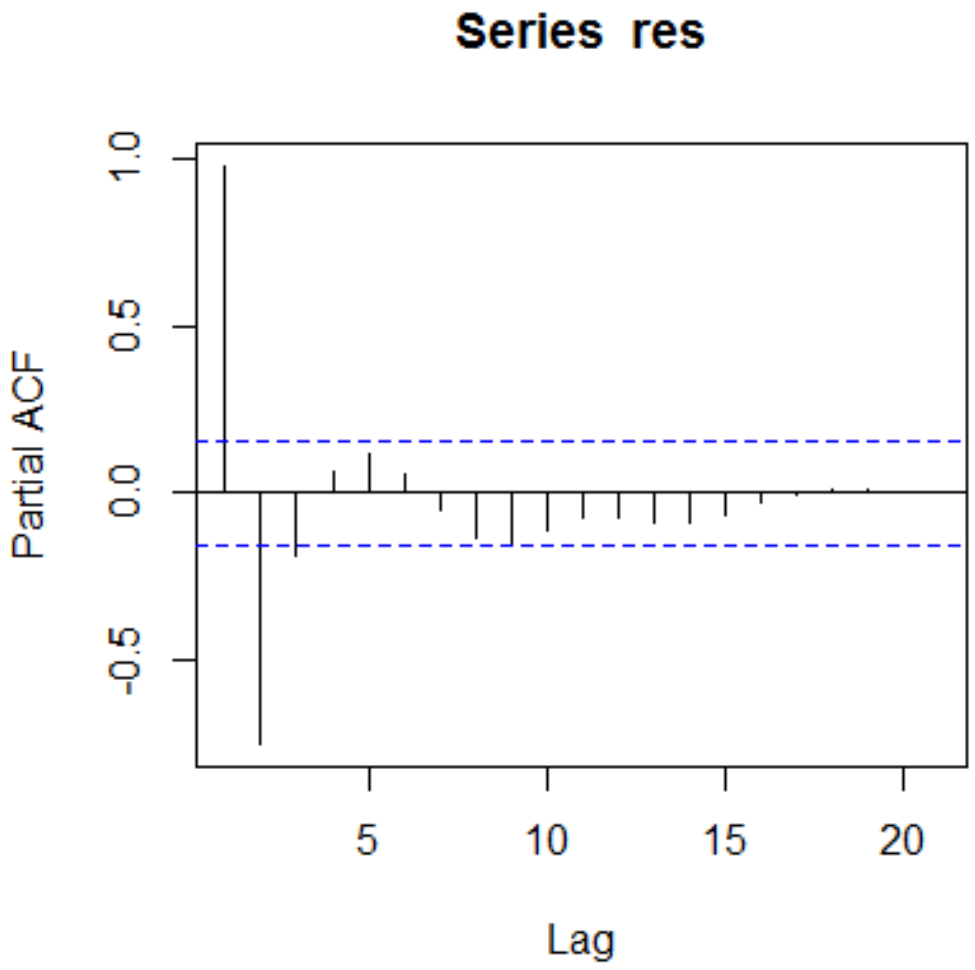
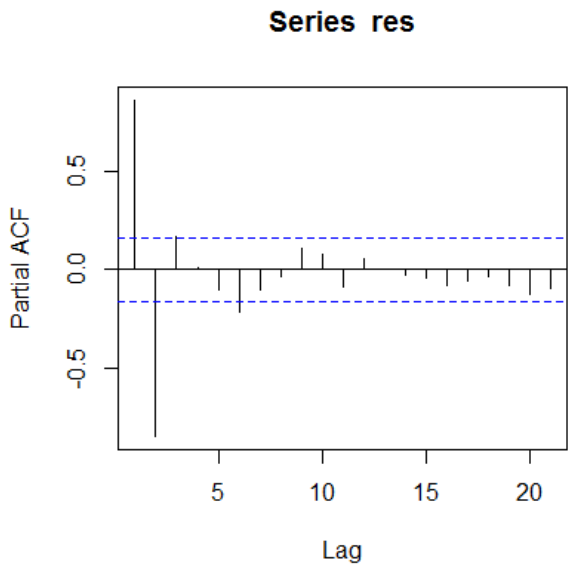


Figure 8. *Partial Autocorrelation Function and Autocorrelation Function Plots of Residuals of Baseline ARIMA model for Participant 506.*



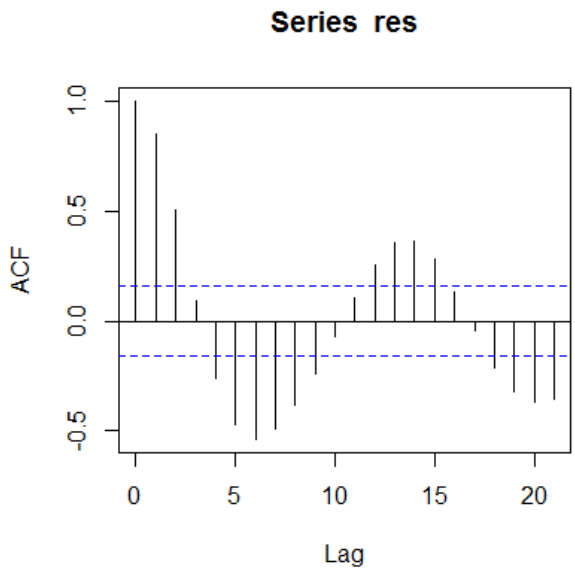


Figure 9. *Specific Aim 3 Model Including All Coping Strategies*

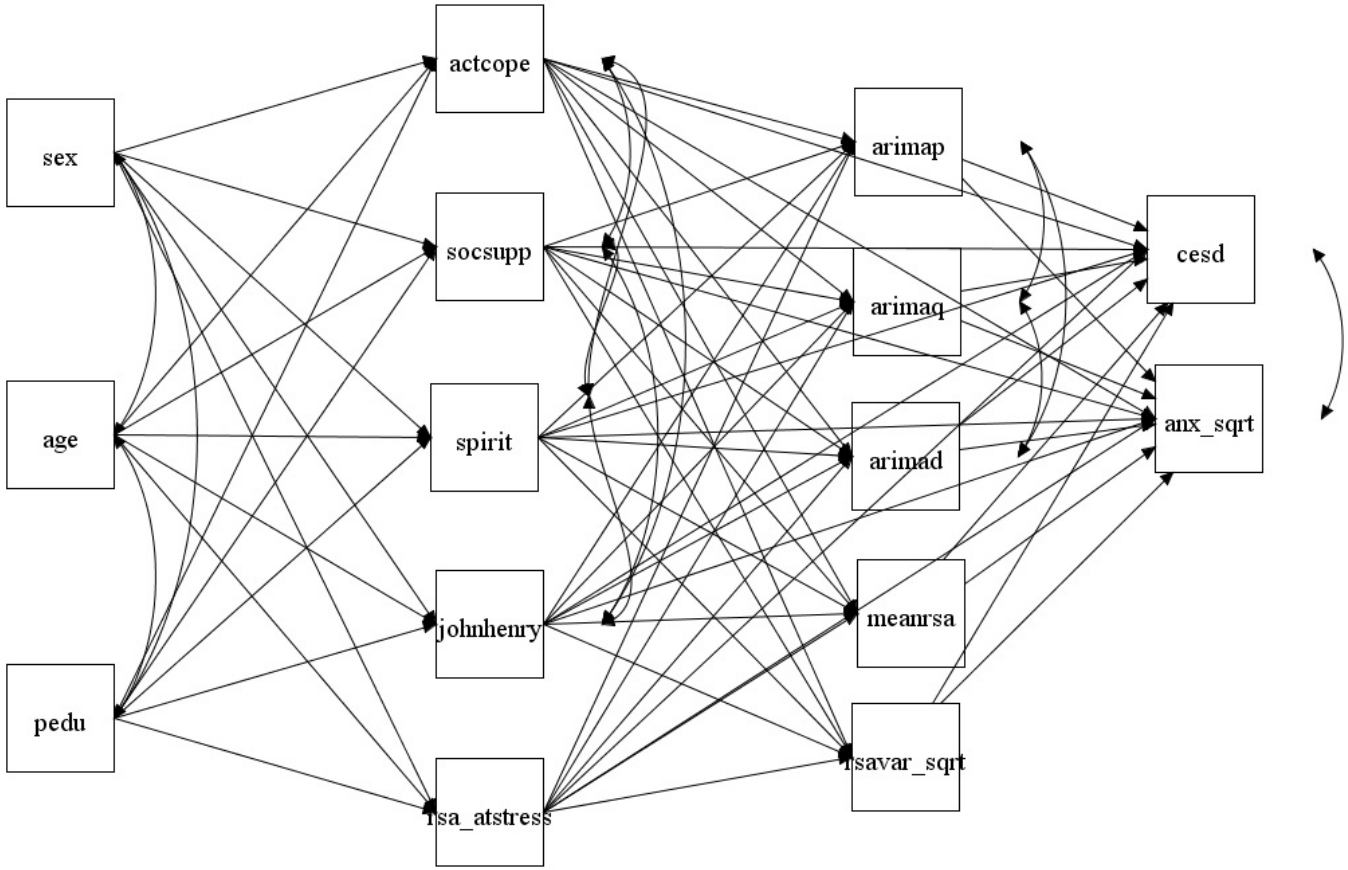
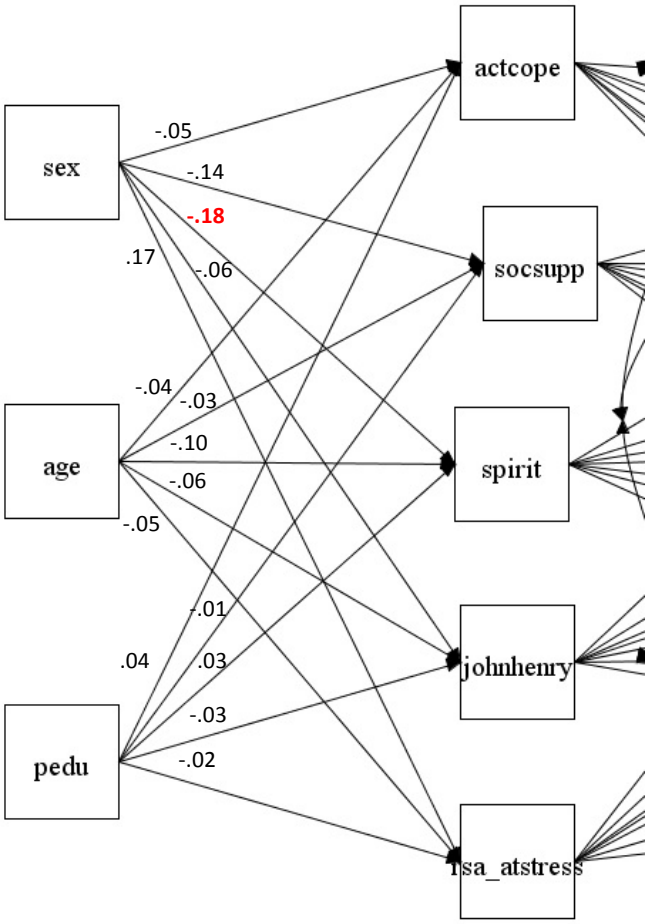
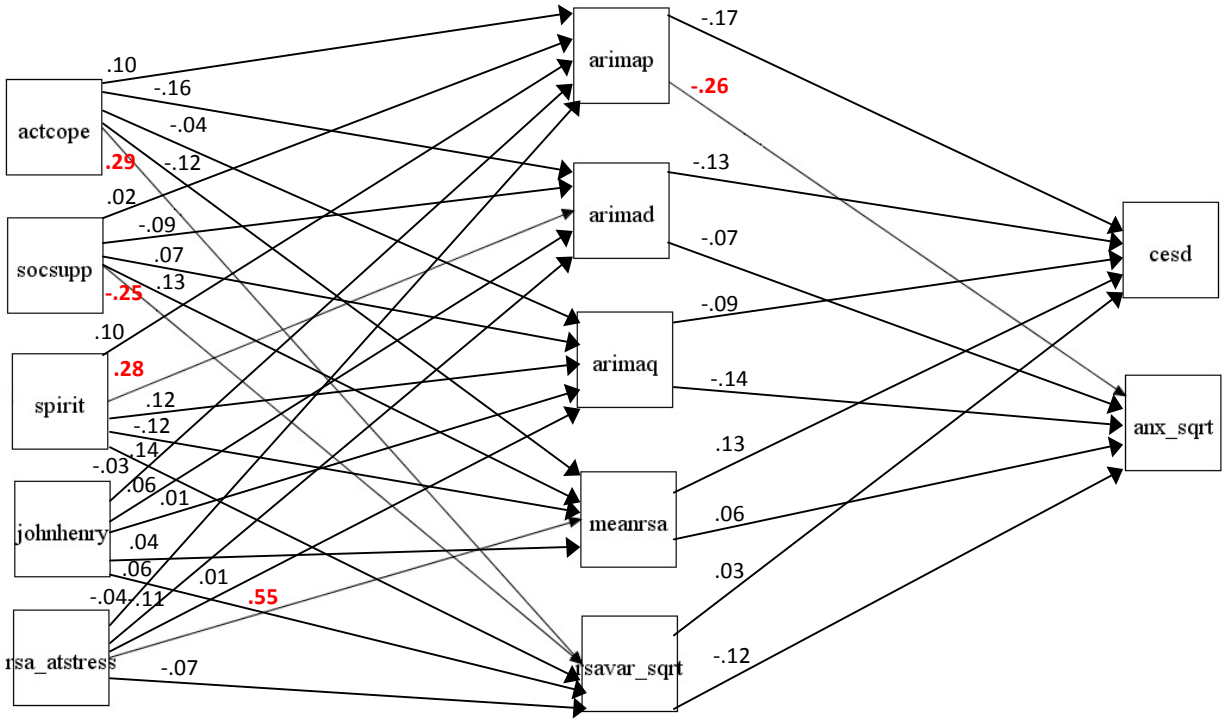


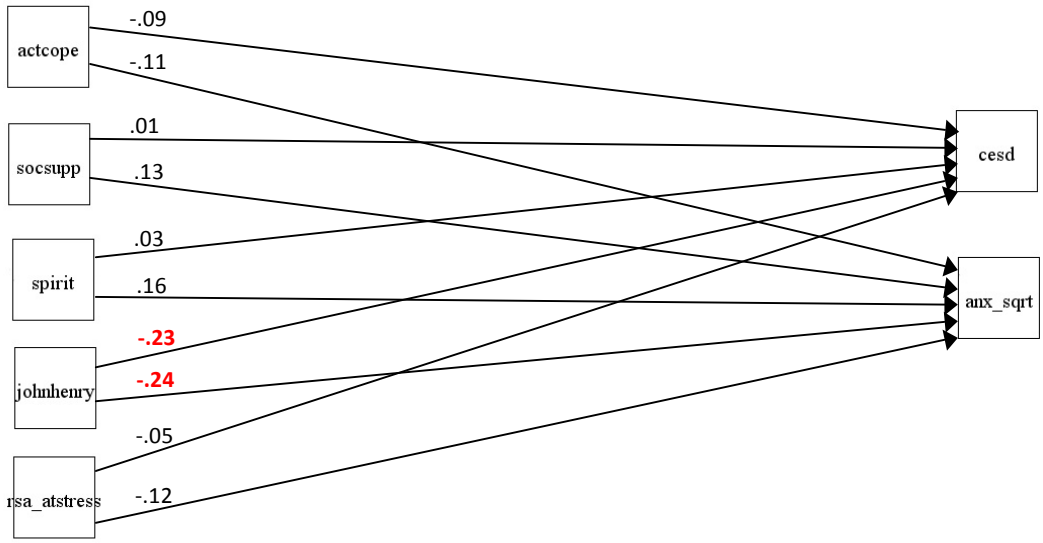
Figure 10. *Specific Aim 3 Model Including All Coping Strategies Including Estimates*



Note: Detail of model – demographic control variables and their standardized estimates of coping strategies and RSA at stress. Statistically significant standardized estimates are indicated in red.

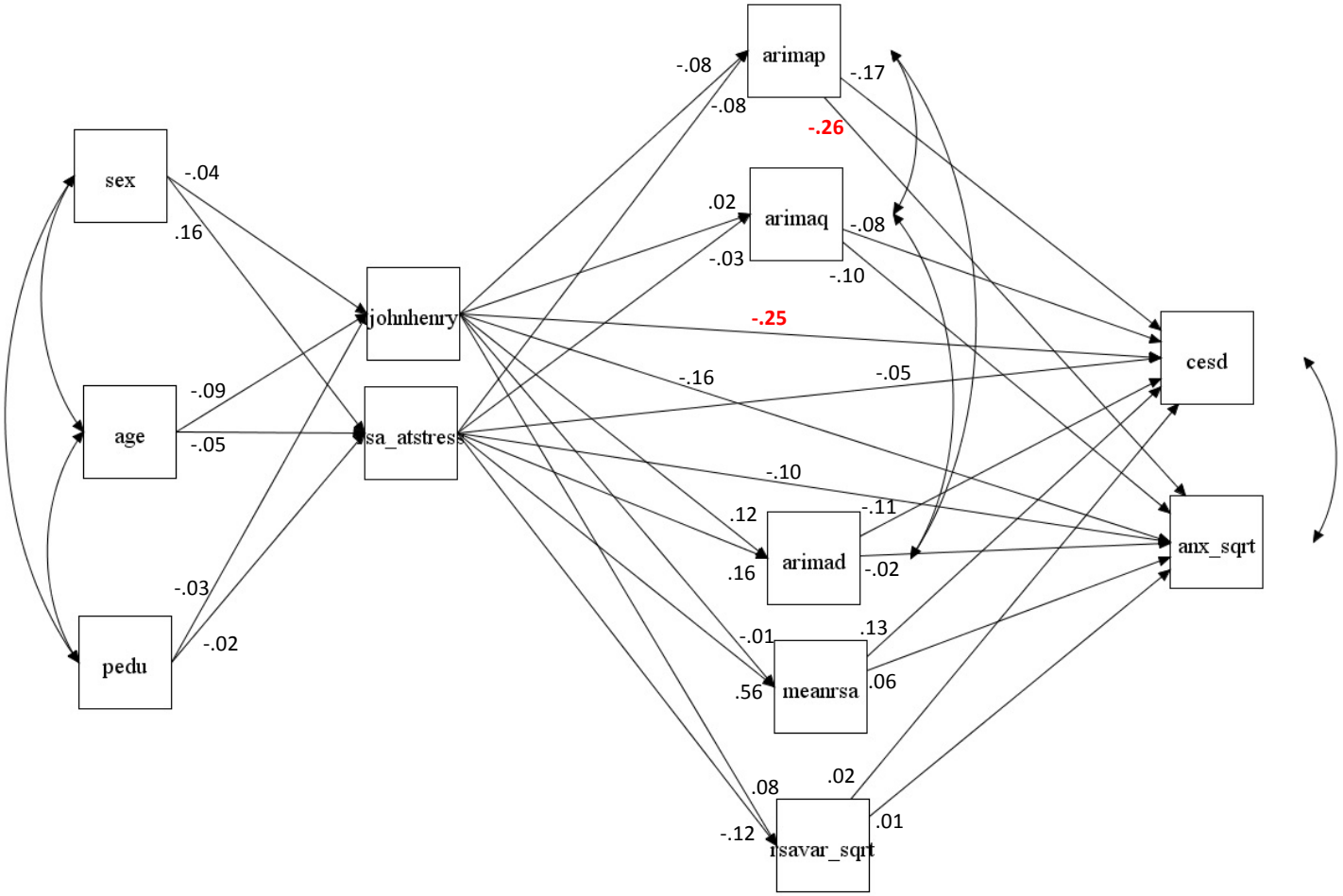


Note: Detail of model – standardized estimates of relationships between coping strategies and RSA at stress variables and RSA mediators, and RSA mediators and mental health symptoms. Statistically significant standardized estimates are indicated in red.



Note: Detail of model – direct relationships between coping strategy and RSA at stress variables and mental health symptoms. Statistically significant standardized estimates are indicated in red.

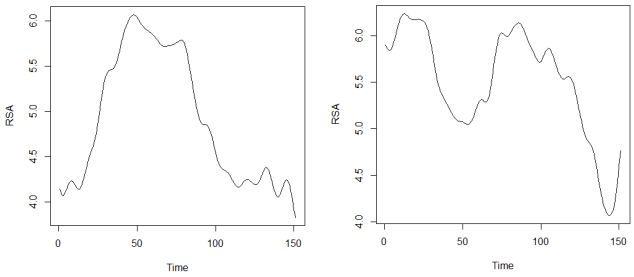
Figure 11. *Specific Aim 3 Model Including Only John Henryism*



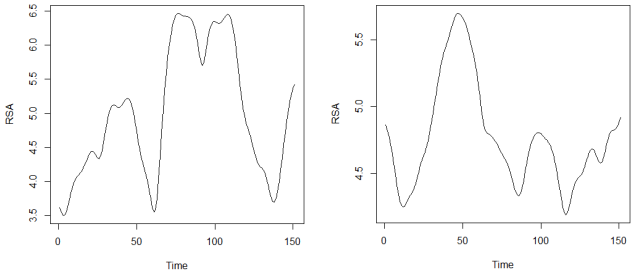
Note: Statistically significant standardized estimates are indicated in red.

Figure 12. *Comparison of Time Series Models with 0, 1, and 4 AR Components*

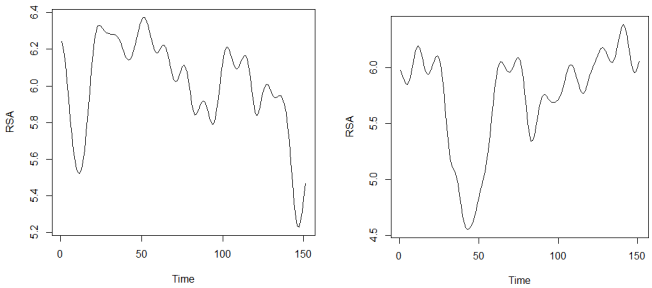
0 AR Components



1 AR Component



4 AR Components



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