PERSONAL CONTROL LEVEL AND CHANGE AS PREDICTORS OF INFLAMMATORY DYSREGULATION

Moira Pauline Johnson

A thesis submitted to the faculty at the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Master of Arts in the Sociology department in the Graduate School.

Chapel Hill
2015

Approved by:

Michael Shanahan

Kathleen Mullan Harris

Guang Guo

Lilly Shanahan

ABSTRACT

Moira Pauline Johnson: Personal Control Level and Change as Predictors of Inflammatory Dysregulation
(Under the direction of Michael Shanahan)

Biological mechanisms linking individual sense of control to physical health outcomes remain understudied. Research offers reason to expect that chronic low-grade inflammation may account for some of the association between sense of control and morbidity. To better understand why and how personal control affects patterns of health disparities, this thesis evaluates whether level and change in the sense of control predict three biomarkers of inflammation using data from the Midlife in US Study. Findings show that average sense of control predicts levels of interleukin-6, and that sense of control mediates the association between income and inflammation. The positive association involving sense of control is stronger at lower income levels, particularly for men. Positive change in sense of control over time provides an additional protective effect against elevated IL6, but this association is not mediated or moderated by SES.

ACKNOWLEDGEMENTS

First, I would like to express my sincere gratitude to my advisor Prof. Mike Shanahan for the continuous support of my Master's and PhD study and related research, for his patience, motivation, and immense knowledge. His guidance helped me in all the time of research and writing of this thesis.

Besides my advisor, I would like to thank the rest of my thesis committee: Prof. Kathie Harris, Prof. Guang Guo, and Prof. Lilly Shanahan for their insightful comments and encouragement, but also for their questioning of my research proposal which helped me to produce a stronger final product.

This material is based upon work supported by the National Science Foundation Graduate Research Fellowship under Grant No. DGE-1144081. This research is also supported by the Carolina Population Center NICHD-NRSA training grant 5-T32-HD07168, University of North Carolina at Chapel Hill.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

BMI	Body mass index
CES-D	20-item Center for Epidemiological Studies Depression Inventory
CRP	C-reactive protein
HPA	Hypothalamic-pituitary-adrenal axis
IL-6	Interleukin-6
MIDUS	National Survey of Midlife Development in the United States
SES	Socioeconomic status
SNS	Sympathetic nervous system

SECTION 1: INTRODUCTION

Life expectancy and other key health outcomes vary greatly by socioeconomic status. Greater exposure to stress-inducing circumstances and environments heighten the risk of chronic stress-related illness and increase the likelihood of premature death among those with fewer socioeconomic resources (Link and Phelan 1995). However, some people who experience the chronic stress-inducing challenges associated with cycles of poverty and other low-status positions are able to avoid or delay the onset of these negative health outcomes (Lachman and Weaver 1998; Pudrovska et al 2005).

Many factors have been proposed as sources of such resilience. Prominent among these explanations is internal sense of control (also known as personal control), the belief in one's ability to exert an influence over important aspects of life (Thoits 2010). Personal control provides an important pathway in the link between social status and indicators of health and well-being. While people with fewer socioeconomic resources have lower average levels of personal control, lower-status individuals who nevertheless maintain a strong sense of control achieve health outcomes equal to those of their high-status peers (Mirowsky and Ross 2007; Pearlin et al. 2007).

Personal control likely impacts health by altering the likelihood that people will avoid and/or effectively cope with chronic and acute stressors. If so, personal control should predict low-grade inflammation, a biological symptom of immune dysregulation resulting from repeated or enduring stress activation. This hypothesis has not yet been evaluated in a representative sample of US adults. To address this research gap, I will test whether (1) level

and change in personal control predict inflammation. Given the important role of stress process as a determinant of major health inequalities, my second aim will be to assess whether or not (2) change and level of personal control mediate and moderate the association between current socioeconomic status and inflammation. That is, while people with lower socioeconomic status in adulthood may generally be likely to have low levels of personal control and to experience declines in personal control over time (mediation), having a strong sense of control may have a greater protective effect on inflammatory outcomes for people with lower SES compared to their more well-off counterparts (moderation).

Data come from waves one and two of the National Survey of Midlife Development in the United States (MIDUS) to conduct the analyses for this project. MIDUS provides a national sample of adults ages 35-84 in 2005. The MIDUS data offers three major strengths for this investigation. **1)** The survey's explicit focus on midlife allows for an assessment of how personal control relates to physiological outcomes at a critical developmental period during which health disparities by social status are at their peak. **2)** MIDUS provides 10-year follow-up data on all psychosocial and demographic measures, allowing for an assessment of how long-term changes in sense of control independently affect physiological outcomes. **3)** The MIDUS Biomarker Project (Project 4) added biological assessments for a subsample of MIDUS respondents in order to identify biopsychosocial pathways that contribute to diverse health outcomes. The biomarkers reflect functioning of the hypothalamic-pituitary-adrenal axis, the autonomic nervous system, the immune system, cardiovascular system, musculoskeletal system, antioxidants, and metabolic processes. The Biomarker Project specimens allow for assessment of multiple indicators within these systems, making MIDUS

a strategic data resource to assess the shared and unique associations between personal control and multiple markers of inflammation.

In the sections that follow I will first provide background information on the definition and operationalization of personal control in addition to two other, related psychosocial resources: self-efficacy and locus of control. After summarizing existing findings for the association between personal control and health, I will introduce theories and findings in psychobiology that provide the biological basis to understand how personal control may affect physical health. I then discuss the role of socioeconomic status in these relationships. After specifying the hypotheses and the proposed models, I will address the data source, variables to be used in the analysis, and offer preliminary cross-sectional and descriptive results. Findings will provide a better understanding of the specific inflammatory pathways affected by level and change in sense of control, and whether these relationships vary by adult SES.

SECTION 2: BACKGROUND

Sociologically, personal control reflects the lived experiences and opportunities afforded to people on the basis of social status. Having a low sense of control is a central form of alienation strongly linked to the structural constraints people face in their daily lives (Mirowsky and Ross 2003; Mirowsky and Ross 2007; Pearlin et al. 1981). Sociologists have added greatly to the study of stress and resilience by focusing on how the structured arrangements of people's lives alter the likelihood of stress exposure, as well as the development, maintenance, and significance of coping resources, including personal control (Pearlin 1989; Thoits 2010). In addition to documenting the socially structured patterns of stress exposure and its negative health effects, stress research in sociology has contributed to a growing body of work focused on factors that may weaken these disparities. In particular, findings indicate that personal control acts as a particularly effective stress buffer (Pearlin 1981; Pearlin 1989; Thoits 2010).

Personal control can be defined as the "the perceived ability to significantly alter [important] events [in one's life]" (Skinner 1996). Personal control is just one of several closely related psychological attributes linked to a sense of overall 'positive self-concept,' each with their own conceptual strengths and weaknesses. For the purposes of this analysis I will focus on sense of control, largely because of its widespread acceptance as a key psychological resource for health in both psychology and sociology.

Locus of control and self-efficacy have overlapping and closely connected meanings to the sense of control, and have sometimes been grouped together as measures of "positive

self-concept" (Johnson and Barer 1993; Judge and Bono 2001; Lachman et al. 2011). Rotter first conceived of locus of control in the 1960s to measure the extent to which someone attributes desired outcomes to "internal or external circumstances" (Van Liew 2013). Originally, psychologists viewed locus of control as a "learned, generalized expectation" that did not change over time within individuals, in much the same way that researchers understood personality characteristics and IQ to be fixed stable traits (DeLamater and Ward 2006). However, more recent work in psychology and sociology has acknowledged that personal control and related concepts vary within individuals across time based on personal experience and objective constraints such as those resulting from financial hardship, illness, and/or the aging process (Mirowsky and Ross, 2007; Pearlin et al., 2007).

While locus of control scales include questions about the ability of people to alter outcomes in a general sense (e.g. not specific to that individual's belief about their own abilities), personal control focuses solely on questions pertaining to self-perceptions (Bandura 1986; Ross and Mirowsky 2006). In addition, measures of locus of control sometimes focus on domain-specific questions. That is, measures for locus of control may include questions about the belief in one's ability to exert control in separate domains of life such as at work, at home, or in his or her personal life. In contrast, personal control and Bandura's concept of self-efficacy represent global measures concerned with one's ability to exert control in a general sense without differentiating by context. While self-efficacy reflects someone's perceived ability to perform the specific steps needed to successfully complete a given challenging goal or task, personal control is not task-specific (DeLamater and Ward 2006). The most commonly used personal control index contains items on both mastery (the sense of control) and constraints (lack of control).

Table 1. Measures of Positive Self-Concept

Construct	Conceptual Definition	Operationalization	Citation
Personal Control	Perceived ability to significantly alter [important] events [in one's life]	"What happens in my life is often beyond my control." "Whether or not I am able to get what I want is in my own hands."	Pearlin & Schooler (1978).
Locus of Control	Extent to which someone attributes desired outcomes to internal or external circumstances	"People's misfortunes result from the mistakes they make" "There will always be wars, no matter how hard people try to prevent them. "	Rotter (1966).
Self-Efficacy	Perceived ability to perform the specific steps needed to successfully complete a given challenging goal or task	"It is easy for me to stick to my aims and accomplish my goals." "I can remain calm when facing difficulties because I can rely on my coping abilities."	Schwarzer & Jerusalem (1995).

SECTION 3: LITERATURE AND HYPOTHESES

Section 3.1 Personal Control, Social Status, and Health and Well-Being

Personal control predicts educational attainment, job satisfaction, and overall well-being (Judge and Bono 2001; Mirowsky and Ross 2007; Peterson 1999). Personal control also predicts mental health outcomes, including depressive symptoms, clinical depression, anxiety, psychological distress, and overall satisfaction and well-being (Caputo 2003; Keith 2004; Mirowsky and Ross 2003; Pearlin 1981; Schieman 2002; Turiano et al. 2014; Turner and Lloyd 1999). Additionally, personal control is associated with self-rated health and a wide range of physical health outcomes including physical functioning, diagnosed health conditions, acute and chronic symptoms, and mortality (Caputo 20003; Krause 2006; Lachman and Weaver 1998; Pudrovska et al. 2005; Pearlin et al. 2007;Schieman 2002; Turiano et al. 2014). Under most circumstances, a stronger sense of control predicts better health and well-being outcomes, relative to those with a lower sense of control (See Baltes 1995; Bisconti et al. 2006; Pagel, Becker and Coppel 1985; Skaff 2007; Wrosch et al., 2006 for descriptions of the key exceptions).

Personal control also varies by socioeconomic status. Opportunities fostering personal control become more or less likely based on indicator's of socioeconomic status including education, family income, individual earnings, occupational prestige, and economic hardship (Ross and Mirowsky 2013; Wallerstein 2002). In previous studies, current and lifetime financial strain decreased personal control, and every four years of additional education increased control by .6 standard deviations for young adults

(Mirowsky and Ross 2007; Pearlin 2007; Pudrovska et al., 2005; Wolinsky and Stump 1996). Additionally, level of personal control varies across income brackets (Lachman and Weaver 1998; Mirowsky and Ross 1998). With few, if any, exceptions, research shows that higher status predicts higher average levels of personal control compared to lower status positions, regardless of how SES is operationalized. Thus, although extant evidence does not warrant strong causal conclusions (owing to non-experimental research designs), it is nevertheless consistent with the proposition that current SES is positively associated with personal control, which is in turn positively associated with health.

In fact, personal control mediates the association between socioeconomic status and health outcomes. Differences in average levels of personal control explain at least some of the disparities in levels of chronic depression, self-rated health, and self-reported functional status by SES (Lachman and Weaver 1998; Pearlin et al., 2007; Taylor and Seeman, 1999). In addition to mediating the association between SES and health, the strength of the association between personal control and health also varies by adult social status. While findings show that low socioeconomic status is associated with lower average levels of personal control, low-SES people who develop and maintain a strong sense of personal control appear to experience a greater health-protective effect compared to high-SES people with an equally strong sense of personal control (Lachman and Weaver 1998). The effect of personal control on depression/anxiety and self-reported physical symptoms (e.g. headaches and shortness of breath) is stronger for people with current or previous economic hardship than for people without any lifetime experience of economic hardship (Pudrovska et al. 2005). Personal control also predicts mortality risk, but only at low levels of educational attainment (Turiano et al. 2014). These intriguing

findings thus suggest that personal control mediates the connection between SES and health, and this meditational link may be moderated, such that it accentuates at lower levels of SES.

Additional findings show that change in personal control over time predicts health outcomes independent of baseline sense of control. Trajectories of sense of control predict depression, mortality, anxiety and functional status (Infurna et al. 2012; Price et al. 2002). While fewer studies have evaluated the significance of individual trajectories in personal control for health, evidence points to the fact that change, in addition to level of control, varies by SES.

The few studies that have explicitly examined the role of SES in the association between change in personal control and health show that the rate and direction of change in sense of control varies by social status. One study demonstrated that among adults 18-95 years of age, adults with the lowest levels of education experienced the sharpest declines in sense of control over a six-year period. Meanwhile, adults with at least a high school degree showed a continued increase in sense of control until late middle age, but the gaps between college educated and high school graduates rose over time, suggesting that the most well-off people experience the greatest positive change in sense of control even after finishing school, and maintain a strong sense of control into older ages (Mirowksy and Ross 2007).

Additionally, results from a one-year follow-up survey of a sample of adults 18-55 in Toronto found that adults with incomes in the highest third of the population ($95,000 and above) experienced increases in mastery from time 1 to time 2, while those in the lower two-thirds of income ($54,000 and less) experienced average declines in

mastery over the year, with the greatest declines for those in the lowest third of income levels (Schieman 2003). These findings suggest that adults with the most income and education have opportunities to continue to develop and increase their sense of control, even after their formal education has ended (potentially through better-quality jobs that afford more autonomy). Conversely, adults with lower socioeconomic status tend to experience stagnation and/or decline over time, possibly as a result of accumulated and persistent stressors that erode existing personal control (Mirowsky and Ross, 2007; Schieman et al. 2003).

While current research clearly demonstrates significant connections between SES, personal control, and physical and mental health, the question remains: *Why* would personal control matter for health at a biological level?

Section 3.2 Biological Pathways from Personal Control to Health

The stress process model provides the primary framework for understanding how the social stratification of life stressors leads to inequalities in mental and physical health. The model suggests that external stressors (anything that threatens an individual or obstructs their ability to achieve a desired outcome) can trigger both stress (physiological and/or emotional arousal, or activation) and distress (failure to re-achieve homeostasis, whether physical or emotional), the effects of which accumulate over time to alter physical and mental health (Turner and Roszell 1994). When an individual perceives an environmental or social circumstance as stressful, this perception triggers the activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS), both of which have regulatory effects on other systems throughout the body. Upon activation, the HPA axis and SNS signal the release of higher

or lower levels of 'stress hormones' including cortisol and norepinephrine. These neurotransmitter hormones then alter the functioning of the immune, cardiovascular, and metabolic systems to prepare the body for 'fight or flight' response (McEwen 2012).

Indeed, low SES people often suffer from chronically activated fight or flight response, which is in turn associated with immune dysregulation (Segerstrom and Miller 2004). The physiological, biological, and emotional changes triggered by stress activation increase the chances of survival when temporary threats require an immediate short-term reaction, such as in the event of an attempted violent assault or robbery. However, these same biological processes have numerous negative health implications when triggered repeatedly or without resolution, as may occur in the case of chronic stress exposure. Long-term or repeated triggering of the biological stress response systems due to chronic rather than acute stress exposure can result in a failure of the HPA axis or SNS regulators to de-activate and return to 'normal' pre-stress baseline functioning. This dysregulation can result in continued inefficient or over-active responses from the immune, cardiovascular, and metabolic systems indicative of future chronic disease and mortality. The inability to self-regulate one's biological systems because of repeated, ongoing, or inefficient stress activation can be referred to as 'physiological dysregulation' (Juster et al. 2010; Seeman et al. 2010; McEwen and Seeman, 1999; Karatsoreos and McEwen 2011).

One particularly important aspect of physiological dysregulation is immune system function and inflammatory response (Seeman et al. 2010). While acute inflammation signals a necessary immune response to illness or infection, chronic low-grade inflammation results from the "inappropriate deployment of host defenses" due to

chronic stress overload (Libby 2007).

The immune system encompasses two separate types of response: natural immunity and specific immunity. The natural immunity cells provide a generalized defense against a number of different pathogens quickly with little use of excess energy to fight off potential invaders in the body. The response process of the natural immunity produces inflammation and fever to ward off infection. Specific immunity on the other hand In contrast, specific immunity provides greater specificity, but less speed than the natural immune response process.

The signaling of stress hormones by the HPA axis such as cortisol alters the immune system by up-regulating natural immunity and down-regulating specific immunity. This response can be adaptive as part of an acute fight or flight response. The natural immunity cells are best at quickly and effectively fighting off infections related to scrapes, cuts, and burns, and other acute threats with little use of excess energy. However, the more chronic the stressor, the less effective this strategy becomes. Prolonged stress response in the immune system function (as might happen in the result of recurring or ongoing daily stressors) can increase vulnerability to auto-immune and allergic diseases, in addition to the other pernicious effects of chronic low-grade inflammation (Segerstrom and Miller 2004).

Chronic low-grade inflammation is a significant determinant of morbidity and mortality. Measures of chronic inflammation are associated with diabetes mellitus, coronary heart disease, stroke, mortality, cancer, hypertension, depression, rheumatoid arthritis, and autoimmune diseases, periodontal disease, cognitive ability, periodontal disease, and chronic fatigue syndrome (Goldman et al., 2006; Hasson et al. 2009; Juster

et al. 2010; Kaptoge et al. 2010; Karlamangla et al. 2006; Seplaki et al. 2004; Yang et al. 2013).

The crucial role of inflammation as a mediator in the association between SES and morbidity and mortality is now well established (Harris et al.1999), but the psychosocial conditions associated with inflammatory markers are not yet well understood. The stress-process model asserts that psychosocial resources such as personal control may alter the biological processes associated with chronic and acute stress response described above by influencing the way people perceive events as more or less stressful. This then leads to altered behavioral, physiological, and neuro-endocrine responses to stress (Aneshensel and Mitchell 2014; Taylor and Seeman 1999). Researchers have hypothesized that measures of positive self-concept such as personal control may lead to decreased physiological stress reactivity, resulting in lower inflammatory dysregulation over time. This may occur because people with a greater sense of control more successfully avoid stressors and/or reinterpret stressful situations as manageable by employing active coping strategies to overcome challenges (Geronimus et al. 2006; Karatsoreos and McEwen 2011).

Most studies evaluating the links between personal control and biological functioning have used summary measures of global dysregulation, so they do not offer significant insight into the links between sense of control and individual system-level dysregulation. A few preliminary analyses provide support for the hypothesis that higher levels of sense of control predict lower levels of inflammatory dysregulation. Three cross-sectional studies have found that sense of control is inversely associated with biomarkers of inflammation (Garvin et al. 2009; Roepke 2011; Srogren et al. 2006). In

addition, similar psychosocial resources including locus of control and summary measures of psychosocial functioning have also been linked to inflammation (Gale et al. 2008; Taylor et al. 2006).

However, there are major limitations to the existing findings that necessitate further research. One of the most complete studies in the literature to date only included a measure of mastery as part of a larger summary measure of psychosocial functioning which also incorporated measures of depression and positive and negative social contacts, making it impossible to parse out the unique effects of sense of control (Taylor et al. 2006). Additionally, most studies examining the biological basis behind psychosocial resources have examined only a single inflammatory measure at a time. Multiple measures are necessary to best understand how sense of control 'gets under the skin'.

Since each inflammatory marker reflects different stages of the inflammatory process, there may be differences in the strength of the association between various markers and psychosocial resources including personal control. For instance, some evidence suggests that IL-6 may be more strongly linked to stress-related health outcomes than either CRP or fibrinogen, which may suggest that the links between each marker and health protective psychosocial resources such as sense of control may be different (Friedman and Herd, 2010; Kritchevsky et al. 2005).

If the theories and preliminary findings outlined above hold true, adults with a strong sense of personal control may exhibit a more adaptive stress response, thereby delaying or decreasing the inflammatory stress response that puts individuals at higher risk of experiencing death and disease. ***Hypothesis 1a. (Main Effect Hypothesis):*** *Average level of personal control is negatively associated with inflammatory dysfunction*.

This hypothesis can be studied in a growth curve framework. No studies appear to have examined the association between intra-individual change in mastery over time and inflammation. However, given that change in mastery predicts important physical and mental health outcomes independent of baseline mastery, the effects of change in mastery on health should also operate through the biological mechanisms associated with the stress response process. Moreover, pattern of change is considered a more accurate measurement strategy when contrasted with single-point assessments.

Change in sense of control may exert an independent effect on mortality on other health outcomes by altering how people avoid, react to, and cope with daily stressors over time. If one perceives a decreased ability to exert control, this may result in behavioral changes such as lowered levels of planfulness that may decrease the likelihood of stress avoidance. Declining sense of control may also heighten the level of anxiety associated with stressors and decrease the perceived ability to cope with stressors, even when the level of objective stress exposure remains the same (Infurna et al. 2012; Skinner 1995). These considerations suggest ***Hypothesis 1b:*** *Positive intra-individual change in mastery is associated with increased inflammatory dysfunction.*

Variations in the Inflammatory Markers

The complex mechanisms involved in the inflammatory process are not yet completely understood (Kritchevsky et al. 2005). In particular, the variations and interconnections between separate markers of inflammation remain understudied (Friedman et al. 2010). The elevation of IL6, an inflammatory cytokine triggers changes in protein synthesis directly responsible for increases in C-reactive protein and fibrinogen. As a more direct measure of the initial inflammatory response process, IL-6

may be more strongly associated with health outcomes than CRP and fibrinogen. Previous studies suggest that IL-6 is a better predictor of future coronary heart disease and/or cardiovascular mortality than either CRP or fibrinogen (Cesari et al. 2003; Harris et al.1999; Herd and Friedman et al. 2010; Kritchevsky et al. 2005).

Section 3.3 The Role of Socioeconomic Status

Physiological dysregulation is more common among lower socioeconomic status groups (Seeman et al. 2010). From childhood to old age, there are substantial differences in biomarkers of physical functioning by SES across the cardiovascular, immune, and metabolic systems. People with lower incomes, fewer years of education, or who have socioeconomic hardship in childhood or adulthood, have greater levels of dysregulation across multiple biological systems, with effects worsening for people who have experienced the most prolonged socioeconomic disadvantage over the life course (Evans and English 2002; Gruenewald et al. 2012; Gustafsson et al. 2011; Hu et al. 2007; Merken et al. 2014; Seeman et al. 2008; Seeman et al. 2004).

Differences in average levels of sense of control by social status in adulthood may account for some of the disparities in inflammatory dysfunction by socioeconomic status. The structural amplification model suggests that the factors that make a situation less damaging are also less common among those in the most need. At an early age, people from a lower status background are less likely to develop and maintain a strong sense of control. By not developing a sense of control or other effective psychosocial traits, poorly educated and low resourced adults experience stressful situations such as single parenthood, economic hardship, or neighborhood disorder, which further erodes their sense of control, thus widening health disparities between adults with high and low

socioeconomic status (Mirowsky and Ross 2005). Based on the structural amplification hypothesis, I predict ***Hypothesis 2a (Mediation pathway):*** *Average level of personal control mediates the association between current SES and inflammation.*

In support of this model, Taylor et al. (2006) found that a summary measure of psychosocial functioning (which included personal control) partially mediated the association between childhood SES and C-reactive protein in adulthood. However, this study did not allow for an analysis of the unique role of personal control independent of other psychosocial factors. Other previous studies that have evaluated the association between sense of control and inflammation did not control for socioeconomic status or evaluate the potential mediating role of sense of control (Garvin et al. 2009; Sjogren et al. 2006). Studies evaluating the links between sense of control and general level of physiological dysregulation (rather than inflammation specifically) are also lacking in this regard. A study of stressors and dysregulation among older Taiwanese adults controlled for years of education and occupational prestige and found that internal locus of control was negatively associated with a global measure of physiological dysregulation net of SES indicators (Glei et al. 2007). However, as with most other existing studies, the mediation and moderation pathways from SES to dysregulation via sense of control or other psychosocial resources were not examined.

Evidence for the role of intra-personal change in personal control as a mediator of the SES-inflammation association is quite sparse. Extant research suggests that declines in personal control are more rapid and more likely among people from a lower socioeconomic status (Schieman et al. 2003). High levels of baseline educational attainment and income predict positive change in mastery over time while lower SES

predicts earlier and faster decline in personal control with age. This suggests a potential structural amplification mechanism. Persistent limitations in choices and opportunities over multiple aspects of life may erode existing levels of personal control over time in low-SES people, while people with higher education or income-levels may actually increase their sense of control over time as their financial and human capital resources allow them to accumulate further achievements through occupational mobility and job autonomy (Schieman and Meersman 2004; Mirowsky 1995; Ross et al. 2001; Schieman 200l). Based on the structural amplification hypothesis, I predict ***Hypothesis 2b***: Intra-individual c*hange in personal control mediates the association between current SES and inflammation, independent of baseline personal control.*

Given the relative lack of literature on how the relationship between personal control and inflammation may vary by socioeconomic status, I draw on the resource substitution hypothesis (Shanahan et al. 2014) as the primary basis for expectations about variations by SES in the effect of sense of control on inflammatory outcomes. The resource substitution model hypothesizes that the effect of a given psychosocial resource on health outcomes varies based on an individual's current socioeconomic status. Individuals with many resources available to them will experience a smaller loss in the quality of their health due to a deficit in some psychosocial factor (such as sense of control) than those with fewer resources to make up for the same deficit. In other words, the presence of multiple resources (income, education, social supports, etc.) improves the ability to compensate for a lack of other resources that may matter for health. Conversely, although the likelihood of possessing a given psychosocial resource is lower in lower-status adults (see the structural amplification hypothesis above), the presence of

protective psychosocial resources may be more strongly predictive of health outcomes among the less privileged. Given that they have fewer resources to 'substitute' for one another, the presence of any one positive (or negative) psychosocial trait may be more significant for this group (Mirowsky and Ross 2005; Ross and Mirowsky 2011). This brings me to ***Hypothesis 3a (Moderation):*** *Baseline personal control is more strongly associated with inflammatory outcomes at lower levels of SES.* If the resource substitution hypothesis holds, then low-SES people who nonetheless maintain a strong sense of control should experience a stronger protective health effect compared to their high-SES counterparts with an equally strong sense of control.

While the hypothesized buffering effect of personal control may decline over time, especially for lower status adults, I also expect that changes in personal control (whether positive or negative) will be more important for the inflammatory outcomes of low-SES people, based on the resource substitution hypothesis described above. This brings me to ***Hypothesis 3b****: Positive change in personal control is more strongly linked to inflammation outcomes at lower levels of SES.* That is, while baseline sense of control may be lower on average, and negative changes in personal control more likely for people with lower SES, decreasing or strengthening sense of control over time will be a more important predictor for inflammation outcomes with lower social status given that sense of control may help 'substitute' for a lack of financial resources by altering health behaviors, stress exposure, and coping strategies.

Section 3.4 The Role of Gender and Age

Women's average sense of control is generally lower than men's. Unequal access to economic, education, and health opportunities throughout the life course may

decrease the health benefits of having a strong sense of control among women, particularly among older cohorts (Ross and Mirowsky 2002). Persistent structural limitations, especially among older cohorts of women may decrease the health benefits of having a strong internal sense of control. Additionally, women in midlife naturally experience higher levels of inflammation compared to men due to hormonal fluctuations (Yang et al. 2013). Given these two factors, the protective effect of sense of control against elevated inflammation at lower-income levels may be lesser for women relative to men, a three-way interaction **(Hypothesis 4).**

The aging process itself alters the development of mastery over time. Average levels of mastery decline in old age. Typically mastery begins to decline after age 50, typically a reflection of declining physical well-being (Reynolds et al. 2007). However, studies have also shown that by shifting goals towards the achievement of small autonomous tasks (such as taking care of a garden or preparing meals for oneself) rather than dwelling on losses in previously important domains (such as formal employment) elderly people may still maintain high mastery at least until extreme old age (Pudrovska et al. 2005). Given Mirowsky and Ross' resource substitution hypothesis, maintaining a strong sense of control may offer a greater protective effect for the health of adults over age 60, despite their greater risk of experiencing declines in control and health, a two-way interaction (**Hypothesis 5**).

In summary, the analyses that follow will assess whether average sense of control and/or change in the sense of control predict inflammatory dysregulation (hypotheses 1a and 1b). Subsequent analyses will test whether average personal control and/or amount of change in personal control mediate the association between SES and inflammation

(hypotheses 2a and 2b) Tests will also determine whether average and/or change in personal control moderate the association between SES and inflammation (hypotheses 3a and 3b). Finally, the role of gender and age will also be explored (hypotheses 4 and 5).

Table 2. Summary of Hypotheses	
Number	**Statement**
Hypothesis 1a (Main Effect)	Average personal control is negatively associated with inflammatory dysfunction.
Hypothesis 1b	Positive change in mastery is associated with increased inflammatory function.
Hypothesis 2a (Mediation)	Average level of personal control mediates the association between SES and inflammation.
Hypothesis 2b	Positive change in personal control mediates the association between SES and inflammation.
Hypothesis 3a (Moderation)	Average personal control is more strongly linked to inflammatory outcomes at lower SES.
Hypothesis 3b	Positive change in personal control is more strongly linked to inflammatory outcomes at lower SES.
Hypothesis 4	The protective effect of personal control by SES will be lower for women (a three-way interaction).
Hypothesis 5	The protective effect of personal control will be stronger for adults over age 60 (a two-way interaction).

SECTION 4: DATA AND METHODS

Section 4.1 The Data

This project uses data from MIDUS I and II. The purpose of the MIDUS study was to investigate the role of behavioral, psychological, and social factors in understanding age-related differences in physical and mental health. Began in 1994, a longitudinal follow-up of the original MIDUS respondents was conducted in 2004-2006. The MIDUS II Biomarker Project (Project 4) added comprehensive biological assessments for a subsample of MIDUS respondents, facilitating analyses that integrate behavioral and psychosocial factors with biology.

The Biomarker Project (Project 4) of MIDUS II contains data from 1,255 respondents. These respondents include two distinct subsamples, all of whom completed the Project 1 MIDUS II Survey: (1) longitudinal survey sample (n = 1,054) and (2) Milwaukee sample (n = 201). In the analyses that follow, I will use data only from the 1,054 participants who had data on sense of control collected in both waves of MIDUS.

Biomarker data collection was carried out at three General Clinical Research Centers (at UCLA, University of Wisconsin, and Georgetown University). The biomarkers reflect functioning of the hypothalamic-pituitary-adrenal axis, the autonomic nervous system, the immune system, cardiovascular system, musculoskeletal system, antioxidants, and metabolic processes. The specimens (fasting blood draw, 12-hour urine, saliva) allow for assessment of multiple indicators within these major systems. The protocol also included assessments by clinicians or trained staff, including vital signs, morphology, functional

capacities, bone densitometry, medication usage, and a physical exam. Project staff obtained indicators of heart-rate variability, beat-to-beat blood pressure, respiration, and salivary cortisol assessments during an experimental protocol that included both a cognitive and orthostatic challenge.

Of the 7,108 participants in MIDUS I, 4,963 were successfully contacted to participate in another phone interview of about 30 minutes in length (70% response rate). All respondents from the MIDUS II and Milwaukee samples were invited to participate in the biomarker study.

Data collection for the MIDUS, Milwaukee, and biomarker studies was approved by Institutional Review Boards at the University of Wisconsin, Madison, as well as the University of California, Los Angeles, and Georgetown University, which served as additional sites of data collection for the biomarker sub-study. All participants provided informed consent. The response rate for the biomarker study was 39.3 percent for each of the 2 sub-samples.

For both the MIDUS II and Milwaukee samples, individuals who participated in the biological assessment were significantly more likely to have a college degree relative to non-participants; no significant differences were observed for age, gender, marital status, or employment status (see Love et al., 2010 and 2011 for more details on the study sample).

Section 4.2 Evaluation of the Data for the Current Project

The MIDUS study provides several advantages for conducting this analysis. First, MIDUS offers data on sense of control at two time-points 10-years apart. While other samples provide shorter one and two-year time intervals, a longer time frame may be needed to capture major changes in psychosocial resources resulting from chronic stress and aging

processes. The MIDUS study also provides multiple biomarkers for each biological system. The availability of multiple inflammatory markers allows for an assessment of whether and how the effect of sense of control varies within the same biological system. Findings may allow for a more nuanced assessment of the specific immunological pathways affected by sense of control. Additionally, the middle age sample allows for an examination of these pathways at a time when health disparities by SES are at their peak (DiPrete and Eirich 2006; Wilson et al. 2007).

There are several limitations to the data. MIDUS only offers one time point of data collection for each inflammatory measure, making it impossible to assess how level and change in sense of control may alter trajectories of inflammation over time. Additionally, the respondents with baseline and follow-up data on sense of control measures are almost all white, preventing an analysis of variations by race and ethnicity. Despite these drawbacks, an analysis of the MIDUS data will still allow for an assessment of the key hypotheses concerning the role of sense of control as a potential buffer against stress-induced inflammatory dysregulation. Finally, the substantial ten-year time gap between wave 1 and wave 2 prevents an exploration of medium-term fluctuations in both health and sense of control, which may be critical for understanding these life course dynamics as fully as possible.

Section 4.3 Measures

Focal Independent Variables

a) ***Average Sense of Control***- The sense of control will be operationalized with two dimensions: personal mastery and perceived constraints. Two sub-scales were collected to measure the sense of control in MIDUS I and MIDUS II, one for mastery and one for

constraints (Lachman and Weaver 1998; Pearlin and Schooler 1978). Personal mastery refers to one's sense of efficacy or effectiveness in carrying out goals. Perceived constraint indicates to what extent one believes there are obstacles or factors beyond one's control that interfere with reaching goals. The mastery scale consists of a total of 4 items, and the constraints scale includes 8 items, assessed at time 1 and time 2. Prior confirmatory factor analysis reveals that the two-dimensional approach fits the data well (RMSEA of .047 and CFI of .979). Generally an RMSEA of < .1 and a CFI score of >.9 indicate good model fit (Marsh et al. 2004).

The measure of individuals' level of sense of control was computed by averaging scores on each sub scale at both wave 1 and wave 2. The score for level of personal mastery was calculated by adding each person's mastery score at each wave, divided by two. The same procedure was used to calculate each individual's level of constraints. The mean scores across each wave were used to indicate level of sense of control instead of just the baseline (time 1) score by itself to adjust for the fact that including a covariate of just baseline sense of control could produce spurious results in the change score models in the presence of measurement error on the time 1 measure (Glymour et al., 2005; Lee 2015; Turiano et al., 2011). The use of the mean score across both waves avoids this issue, but also leads to an underestimate of the true effect of change (Cain, Kronmal, & Kosinski 1992; Turiano et al. 2011).

b) ***Change in Sense of Control***- I will use change scores to evaluate the independent effects of change in constraints and mastery over time. To operationalize change in sense of control, I will assign each respondent change scores on both mastery and constraints by subtracting each person's wave 1 scores from their wave 2 scores on each dimension. A

positive change score indicates an increase in sense of control from wave 1 to wave 2, a negative change score indicates a decrease in sense of control from wave 1 to wave 2 and a score of 0 indicates stability across the two waves. The change score reflects intra-individual deviation over time, and will reflect trait-level fluctuations over time net of individuals' 'average' sense of control. Correlations between wave 1 and wave 2 sense of control are moderate (.51).

c) ***Construct Validity-*** To ensure the validity of the overall sense of control measure, the two sub-scales balance statements of control and constraint, and balance negative and positive outcomes. This ensures that the index captures perceived control over both positive and negative life outcomes, to avoid capturing only self-blame or defensive mechanisms (DeLamater and Ward, 2006; Mirowsky and Ross 1991). Perceived mastery and perceived constraints have previously been analyzed as two separate psychological indicators, and together as a single summary measure, with similar results (Mirowsky and Ross 1991; Turiano et al., 2014; Pearlin et al., 2005; Lachman and Weaver 1998). Summary measures of sense of control correlate with the other measures of 'positive self-concept' (see section 2 above). However, correlations are modest and usually run from .3 to .6, suggesting that while these measures of positive self-concept are conceptually linked, personal control scales capture a unique psychological disposition (Judge et al. 2003; Mirowsky and Ross 2006).

d) ***Reliability of Measurements-***The mastery and constraints items used in MIDUS provide stable and reliable responses, as shown in the use of the same items in previous research and survey data collection. Cronbach's alpha determines the internal consistency or average correlation of items in a survey instrument to gauge its reliability. Alpha coefficients range in value from 0 to 1 and can be used to describe the reliability of items from

dichotomous and/or multi-point questionnaires. Higher scores represent higher levels of reliability. Commonly, alpha reliability scores of >.7 are considered acceptable, >.8 is good, and > .9 is excellent (Santos 1999).

In previous studies, scales for constraints and mastery using similar items to those in MIDUS have attained alpha reliability scores ranging from .7-.9 (Lachman and Weaver 1998; MIDUS documentation). A recent test of these items from the Health and Retirement Study (which uses the same items as those included in MIDUS) yielded an alpha reliability score of .88 for the constraints scale and .89 for the mastery scale (Brim et al., 2010; Smith et al., 2013).

e) ***SES***- Socioeconomic status is a multifaceted (and contested) concept, which makes operationalization of SES quite difficult without making use of a range of different measures of multiple indicators simultaneously (Allin et al. 2009; Robert and House 1996). To fully and accurately capture socioeconomic status differentials in the US adult population, a range of measures for socioeconomic status will be used including highest level of education, household income, and financial hardship/well-being. For the purposes of this analysis, I will only examine current socioeconomic status at wave 2.

1. Education- As part of the telephone interview, participants were asked their highest level of educational attainment. Responses were grouped into 12 categories ranging from "no school/some grade school" (category 1) to "PhD, MD, JD, or other professional degree" (category 12). This 12- category variable will be used to examine linear associations between education and inflammatory proteins. A set of dummy-coded variables will be used to examine non-linear associations. The following four response categories were dummy-

coded with college graduate status acting as the reference category: less than a high school education, high school graduate, some college, and 4-year college degree and beyond.

2. Income- Information on pretax household income from wages, pensions, social security, and government assistance was obtained from mail surveys. Income will be adjusted for household size by dividing by the square root of the number of individuals in the household. Income will be treated as a continuous variable in analyses. To allow for non-linear associations, the analysis will also examine associations using income quintiles. The income ranges that corresponded to each quintile were: Q1: ≤$23,500; Q2: 23,501-46,250; Q3: $46,251–$70,000 Q4: $70,001–$105,000 Q5: $105,001-300,000. Household income was top-coded at $300,000 at the time of data collection. Each quartile will be dummy-coded and will be included in statistical analyses with the top quartile serving as the reference category. Information on total household income was available for all but 22 respondents out of the 1,052 eligible respondents who completed the biomarkers survey and participated in both waves 1 and 2 (2.09% missing responses).

3. Financial Hardship Financial hardship/well-being will be measured using a summary measure that combines the scores on three separate items. The first item is a dichotomous variable asking whether the family has enough money to get by. The second item assesses the level of difficulty paying bills, and the third item asks respondents to rate their financial situation. The summary measure is coded from 2- 16 so that a 16 indicates the greatest level of financial well-being. Data are available on each of these measures for 99% of respondents in the biomarkers sub-sample, with only 3-9 individual refusals per question.

Dependent Variables

I will analyze the associations between sense of control/SES and three markers of inflammation known to be associated with CVD and other chronic health outcomes: C-reactive protein (CRP), interleukin-6 (IL-6), and fibrinogen. Fasting serum samples were assayed for CRP, IL-6, and fibrinogen. Each inflammatory marker will be coded as a continuous variable. In regression models, CRP and IL-6 will be log-transformed to account for non-normality in the distributions. Additionally, a summary score of overall inflammation burden will be examined. The summary score will range for 0-3, with one point for each inflammatory marker above the sample median (Slopen et al. 2010). Similar indices have been used in previous studies of inflammation and physiological dysregulation (Yang and Kozloski 2011; 2012; Yang et al. 2013).

a) ***Il-6*** is an interleukin (secreted proteins and signaling molecules expressed by white blood cells) that acts as both a pro-inflammatory cytokine and an anti-inflammatory myokine (Pradhan et al. 2001). T cells and macrophages secrete IL-6 to stimulate immune response, during infection and after trauma. IL-6 also plays a role in fighting infection (Ferguson-Smith et al. 1988; van der Poll et al. 1997).

b) ***C-Reactive protein*** is a plasma protein synthesized by the liver, and is a sensitive marker of inflammation. It activates primarily as a result of IL-6 response (Pradhan et al., 2001). A high-sensitivity CRP (hs-CRP) test measures low levels of CRP with a sensitivity to detect C-reactive protein concentrations as low as 0.04 mg/L. (Olshaker 1999; Thompson et al. 1999).

c) ***Fibrinogen*** (factor I) is a glycoprotein that helps in the formation of blood clots. It is a general measure of inflammation that may be elevated regardless of the site/type of inflammation in the body (Lang et al. 2009).

Covariates

a) ***Gender***- Previous findings suggest that women may naturally experience higher inflammation levels compared to men at mid-life (Yang et al. 2013). As such, gender will be an important demographic control in the study. Previous findings do not point to any reason to expect a significant moderation effect of gender on the strength of the association between SES and inflammation or between personal control and inflammation outcomes.

b) ***Race/Ethnicity***- Unfortunately, the respondents from the Milwaukee subsample of the Biomarker Project (described above) do not have valid data from wave 1 of MIDUS. As such, a large proportion of the African American sample is not available for the section of the analysis examining change in sense of control over time. The remaining sample (n= 1,052) is 93% white, so significant insights into variations in the relationships between SES, personal control, and inflammation measures by race will likely not be possible. However, race/ethnicity will still be included as a control measure to account for potential confounding with demographic and health variables likely to affect the results, including SES. The original MIDUS questionnaire includes five racial origins categories: White, Black, Native American, Asian, and Other. For the analyses below I will include two dummy variables, one for blacks, and one for the 'other' category (a combination of Native American, Asian, and Other due to small cell sizes), with a dummy measure for white as the reference category.

c) ***Other Demographic Covariates***- A dichotomous measure for age will be used, reflecting whether individuals are over the age of 60. Previous findings show that certain

medications affect circulating markers of inflammation (Doggrell et al. 2005; Serebruany et al. 2003; Slopen et al. 2010).

d) ***Medications*** - Respondents' use of four classes of medications will also be statistically controlled for: use of anti-hypertensive, cholesterol lowering, corticosteroid, and anti-depressant medications will be indicated with dummy variables. All respondents participating in the biomarkers project were instructed to bring all their medications, in the original bottles, to the General Clinical Research Center when they came for their visit. Recorders at the clinic took down information on all FDA approved prescription medications, over the counter medications, and alternatives including herbs, herbal blends, and homeopathic remedies. For each item, medication name, dosage, route of administration, frequency and duration of use, and self-reported reason for taking the medication were recorded (Ryff et al. 2010). While this method of data collection may still lead to over or under-estimates of actual medication adherence, objective information from the medication bottles provides an extra check on respondents' information and increases the accuracy of the recorded data, relative to self-reported data alone (Chen et al. 2001).

e) ***Health Status***- Respondents reported whether they had received a physician's diagnosis for 20 chronic conditions. An index of chronic disease burden will be constructed from these responses containing multiple diseases associated inflammation (autoimmune disorders, cardiovascular and cerebrovascular disease, hypertension, arthritis, asthma, diabetes, gastrointestinal diseases, liver disease, and cancer). Depressive symptoms in the past week will also be statistically controlled for using a continuous measure for the number of symptoms in the past week based on the 20-item Center for Epidemiological Studies Depression Inventory (CES-D) (Slopen et al. 2010; Turiano et al. 2014). Height and weight

were measured by laboratory staff and were used to calculate body mass index (BMI) (weight in kilograms divided by the square of height in meters). A log-transformed continuous measure of BMI will be included in the analyses.

f**) *Health Behaviors*-** Health behavior indicators will be based on self-reported information from questionnaires completed by project 4 participants at each laboratory. Smoking status will be captured using a dummy variable having ever smoked regularly. Variables for the number of alcoholic drinks consumed during a typical week, as well as a dichotomous variable for regular physical activity, defined as moderate, vigorous, or light activity at least 3 times per week, will also be included.

g) ***Epinephrine/Norepinephrine-*** Norepinephrine and epinephrine levels were adjusted in the models. Both are indicators of sympathetic nervous system (SNS) activity. SNS activation has been found in previous studies to affect inflammatory outcomes due to its significance in the body's stress response (Yang et al. 2014).

SECTION 5: ANALYTIC STRATEGY

Analyses for this project will be performed using Stata 13.Table 2 reports descriptive statistics. Descriptive statistics have been generated for all variables (means and standard deviations for continuous variables and proportions for categorical variables), included in Table 2 below. Linear regression models will be used to estimate the effect of level and change in sense of control on inflammation, using separate models for each individual inflammation measure, as well as for the composite score of overall inflammatory dysregulation. All models will be adjusted for socioeconomic status, demographic characteristics, and health status.

Section 5.1 Regression Equations

The generalized ordinary least squared model is:

$$Y_i = \beta_0 + \beta_1 X_1 + \epsilon_i$$

The following equations represent the procedures and hypotheses outlined above:

1a) $Y = \beta_0 + \beta_1 C + \beta_2 S + \beta_3 x' + \epsilon_1$

1b) $Y = \beta_0 + \beta_1 C + \beta_2 S + \beta_3 x' + \beta_4 \Delta C + \epsilon_1$

2a)

1. $Y = \beta_0 + \beta_a S + \beta_3 x' + \epsilon_1$
2. $Y = \beta_0 + \beta_b S + \beta_2 C + \beta_3 x' + \epsilon_1$
3. $C = \beta_0 + \beta_c S + \beta_3 x' + \epsilon_1$

2b)

1. $Y = \beta_0 + \beta_a S + \beta_3 x' + \epsilon_1$
2. $Y = \beta_0 + \beta_b S + \beta_2 C + \beta_3 \Delta C + \epsilon_1$
3. $\Delta C = \beta_0 + \beta_c S + \beta_3 x' + \epsilon_1$

3a) $Y = \beta_0 + \beta_1 C + \beta_2 S + \beta_3 x' + \beta_4 CS + \epsilon_1$

3b) $Y = \beta_0 + \beta_1 C + \beta_2 S + \beta_3 x' + \beta_4 \Delta\text{CS} + \epsilon_1$

4) $Y = \beta_0 + \beta_1 C + \beta_2 S + \beta_3 G + \beta_4 x' + \beta_5 \text{CSG} + \epsilon_1$

5) $Y = \beta_0 + \beta_1 C + \beta_2 A + \beta_3 x' + \beta_4 \text{CA} + \epsilon_1$

Y represents the dependent variables (inflammatory markers). C represents averaged personal control (operationalized by the index of constraints or mastery). S is a summary measure for current socioeconomic status, and *x* is a vector of all other covariates. ϵ_1 represents the unobserved random error.

Hypothesis 1a (that baseline personal control predicts inflammation) is represented by equation 1a. Hypothesis 1b (that change in personal control predicts inflammation) is represented by equation 1b.

The first mediation hypothesis (hypothesis 2a) is represented by the series of equations in line 2a. If sense of control partially mediates the association between socioeconomic status and inflammation, then 1) SES should significantly affect inflammation outcomes (equation 2a #1), 2) the effect of SES on inflammation outcomes should decline when sense of control if controlled for (equation 2a #2), and 3) SES should predict the level of sense of control (equation 2a #3). The overall effect of SES on inflammation is represented by β_a in equation 2a #1. The effect of SES on inflammation controlling for sense of control is represented by β_b , and the effect of SES on sense of control is represented by β_c in equation 2a #3. Hypothesis 2b (that change in sense of control also mediates the association between SES and inflammation) is represented by the series of equations in 2b.

Hypothesis 3a (average sense of control*SES moderation) is represented by equation 3a and hypothesis 3b (change score*SES moderation) is represented by equation 3b. While

averaged sense of control reflects responses at both time 1 and time 2, all other covariates are from wave 2 data only.

Finally hypotheses 4 and 5 are represented by equations 4 and 5. β_5 in equation 4 represents the three-way interaction between control, SES and gender. β_4 in equation 5 represents the two-way interaction between control and age.

Supplementary analyses will be run using error in variables regression, which adjusts the above models based on the known level of measurement error in sense of control as given by Cronbach's alpha measure of reliability (Hardin 2003; Stefanski 2000). Additional model specifications will also be examined to test for the models' robustness.

Section 5.2 Conditional Change Score with Averaged Sense of Control

All models analyzing the effect of change (1b-3b) will be adjusted for underlying level of sense of control. In the models, the mean of time 1 and time 2 sense of control scores represent trait level and the difference scores represent trait change. The mean of the sense of control will be used to represent level of a given trait because adjustment for baseline scores (measures at time 1 only) can lead to spurious results in models analyzing change if there is measurement error in observed sense of control (Cain et al. 1992; Glymour et al. 2005). Inclusion of the averaged score for constraints and mastery from both time points avoids this issue but also leads to an underestimate of the true effect of change (Cain et al. 1992; Turiano 2014).

Section 5.3 Missing and Clustered Data

Multiple imputation will be used to estimate missing values for respondents with incomplete data. Employing multiple imputation reduces bias by making more complete use of the existing data, and does not assume the data is missing completely as random (Allison

2002). In addition, clustering may be an issue given that roughly 37% of the subjects for the biomarker project were chosen from the MIDUS I twin and sibling samples. Supplementary analyses will employ generalized estimating equations (GEE) to account for potential clustering of the results due to relatedness (Slopen et al. 2010).

SECTION 6: DESCRIPTIVE STATISTICS

Tables 3 and 4 below show the descriptive characteristics of the MIDUS respondents with valid responses for both the MIDUS I and MIDUS II general questionnaires and the project 4 biomarkers data (n = 1,052). Overall this is a relatively well-off and healthy sample population. The average age of the sample respondents is 55. Just over half of the sample (55%) is female. About 80% of the sample has at least some college education. Mean total household income is $76,506. On the scale for financial well-being situation, where 16 is the best, the mean score was 10.7. 79% of respondents report that they exercise at least 20 minutes for 3 times a week, and the majority of participants never or rarely eat fast food during the average week. Average BMI is 29.25 (overweight), and average number of chronic conditions is 2.3 out of a possible total of 30.

Average baseline levels of overall sense of control are relatively high (11.39 out of 14 possible points). Over 80% of the sample experienced change in mastery and/or constraints over time. The direction of change is relatively evenly split, with a slightly greater percentage reporting declining sense of control over time.

Figures A1-A3 (see appendix, p. 42) show the frequency distributions for the three dependent variables. The sample distributions for IL-6 and CRP are both positively skewed, and will need to be log-transformed in the analyses. The summary score for inflammation burden shows that the sample is relatively evenly split, with about 25% of the sample fitting into each category for overall inflammation burden (0-3). The distribution for fibrinogen is relatively normal however. Additionally, figures A4-A6 show that the distributions for wave

1 and wave 2 sense of control are both negatively skewed, due to high average internal control ratings. However, the variable for change in control is normally distributed.

The relatively high levels of education and income and the relative lack of racial diversity may make it harder to detect meaningful differences in health outcomes based on socioeconomic status in the biomarkers subsample population. However, simple cross-tabulation results indicate that average sense of mastery is lower for non-college graduates compared to college graduates (5.77 versus 5.86), and average constraints are also higher for non-college graduates compared to college graduates (2.24 for graduates versus 2.58 for non-college graduate). Although the absolute differences in these values are slight, even small differences in average level of sense of control may be enough to have significant effects on health outcomes. Indeed, summary analyses indicate that even relatively small differences in the level of mastery are associated with altered the inflammation burden scores. The average inflammation burden (across each of the three measures) for individuals with at least a college education is 1.31 compared to 1.62 for those with less than a college education. Additionally, the inflammation burden for those with a mastery score below the average of 5.81 is 1.5 compared to 1.46 for people with a mastery score just above the sample average. These preliminary results suggest that variations in inflammatory outcomes by both education-level and sense of control persist in the sample, despite a relative lack of racial and ethnic or socioeconomic diversity in the longitudinal MIDUS biomarkers subsample.

SECTION 7: MULTIVARIATE RESULTS

Results in table 5 (see p. 35 below) demonstrate the findings for hypothesis 1A. Average sense of control is significantly associated with decreased levels of interleukin-6 in the full cross-sectional biomarkers sample. Additionally, being older than the sample average, or nonwhite was significantly associated with increased levels of IL6 net of other controls, as was taking blood pressure medications, having more existing symptoms and conditions, drinking more often, or having a higher BMI or norepinephrine. Meanwhile, regular exercise was significantly associated with decreased levels of iL6. The effects of gender, smoking, and SES were not significant in the final model. Average sense of control was not significantly associated with fibrinogen or CRP outcomes in the full sample. Results for the longitudinal white-only sample were not significant.

Table 6 shows the results for hypothesis 2A. Findings show that the protective effect of income on inflammation levels declines to non-significance when sense of control is included in the model for interleukin-6, suggesting that personal control does indeed mediate the association between socioeconomic status and inflammation in the full sample. The effects of having a high school degree or lower or of having difficulty paying household bills were not significant in the final model regardless or whether average sense of control was included or not. Additionally, when sense of control is included in the model, the significance and magnitude of the effect of total symptoms and conditions on inflammation decreases slightly. This finding echoes existing theories that a strong sense of control may

protect against physiological dysregulation even in the face of existing conditions due to its role in the body's stress response process.

Table 7 shows results for hypothesis 3A. The interaction between sense of control and income is significant for the top two income quintiles. While each additional increase in sense of control is associated with a -.484 decrease in IL6 levels for individuals in the lowest income quintile, this protective effect diminishes and even reverses for respondents with the highest income levels. As graph 1 indicates, the returns to a medium (versus low) sense of control are much greater for individuals whose income is in the bottom 30 percent for the sample, while this effect diminishes and even appears to reverse for individuals whose income levels fall in the top third for the sample. While individuals from the lowest income bracket with the strongest sense of control still have slightly higher levels of IL6 relative to their high-income counterparts, the gap in inflammation levels by income diminishes greatly in individuals with a medium to high sense of control.

Findings in table 11 and graphs 2-3 below show support for the hypothesis that the income by sense of control interaction varies significantly by gender (hypothesis 4). Low-income men appear to experience the greatest benefit from a strong sense of control. That is, men with low income but a strong sense of control actually have lower levels of IL6 compared to medium or high-income men with an equally strong sense of control. Low-income women appear to experience a curvilinear effect whereby those with a low or high sense of control (relative to a medium or average sense of control) are both at risk of elevated IL6 levels. Meanwhile women in the highest income bracket experience greater protection at low-control levels compared to medium control levels.

Results in table 12 and graph 4 demonstrate support for hypothesis 5 that the effect of sense of control on inflammation varies by age. Individuals over age 60 experience a significantly greater protective effect as they move from lower to medium levels of control while the effect of greater sense of control appears to be less dramatic for younger respondents.

Table 8 shows support for the hypothesis that a positive change in sense of control over time offers a significant protective effect against elevated inflammation levels net of average sense of control (hypothesis 1B). Positive change in sense of control over time is significantly associated with lower IL6 and fibrinogen levels. The effect of gender is not significant in the model for IL6. However, being female is significantly associated with increased fibrinogen levels. Additionally, being older than the sample average and having only a high school degree or less are both significantly positively associated with fibrinogen levels, but is not significantly associated with interleukin outcomes. Finally, cholesterol medication-use is significantly positively associated with fibrinogen outcomes (but not IL6), while depression and blood pressure medications are significantly positively associated with IL6.

The effect of SES on inflammation does not seem to be mediated by change in mastery (hypothesis 2B). Income and difficulty paying bills are not significantly associated with IL6 or FGN outcomes either before or after change in sense of control is included in the models. Additionally, the effect of having only a high school diploma or less on fibrinogen outcomes does not decline once positive change in mastery is included in the model. (See table 9).

As shown in table 10 below, there is also no support for the hypothesis that the

effect of change in mastery on inflammation levels varies by SES. None of the interaction effects for control by income quintile are significant in either model. The null findings for hypotheses 2B and 3B suggest that the effect of positive change in mastery does not vary by SES, and that change in mastery does not act as a significant mechanism in the SES-inflammation pathway.

The results for IL6 and fibrinogen above were robust to different model specifications. Findings remained significant after adjusting for potential clustering by laboratory test site, or respondent relatedness. The findings remained significant with or without inclusion of the twins-subsample, and in fact were slightly stronger, suggesting a slight suppression effect. Results using errors in variables regression (to adjust for random measurement error in values for sense of control) did not vary significantly from the results using traditional ordinary least squares regression. The effect of change in sense of control remained significant after controlling for wave 1 prior symptoms and conditions. Results for CRP remained insignificant whether individuals with CRP-levels greater than 10 were included or excluded in the sample. As with the findings for CRP and sense of control using the MIDUS data set, results examining the relationship between personal control and CRP using HRS data found no significant findings. Results using a continuous inflammatory factor score measure were not significant.

SECTION 8: DISCUSSION

This project aimed to further understandings of how internal sense of control affects morbidity and mortality outcomes by examining the relationships between personal control and inflammation across multiple points in time. The cross-sectional analyses confirmed that sense of control is significantly associated with decreased inflammation (hypothesis 1A). This effect also mediates the association between income and inflammation (but not other socioeconomic markers such as education), demonstrating partial support for hypothesis 2A. The results also show that the strength and significance of these effects are heightened at lower income brackets, particularly for lower-income men as opposed to women, (hypotheses 3A and 4). As expected based on previous findings in the literature, the protective effect of sense of control was stronger for respondents over age 60 (hypothesis 5). However, these results only hold for interleukin-6, while the cross-sectional effects of sense of control on fibrinogen and C-reactive protein were not significant.

The complex inflammatory process remains incompletely understood. However, as a pro-inflammatory cytokine, IL-6 may have a more direct relationship to the biological processes associated with chronic-stress related disease. Previous research findings show that IL-6 has a more robust association with incident coronary heart disease and cardiovascular mortality than CRP and fibrinogen (Cesari et al. 2003; Harris et al. 1999). Additionally, past research suggests that IL-6 may have a unique role in the SES- inflammation relationship. Findings of a significant association between SES and CRP/fibrinogen may be attributed to the fact that IL-6 is both linked to SES, and triggers CRP/fibrinogen, rather than due to

independent associations between SES and these more distal inflammatory factors (Friedman and Herd 2010).

The change-score analyses also showed some support for the original hypotheses. A positive change in mastery over time was significantly associated with decreased levels of fibrinogen and IL6 net of individual average control (hypothesis 1B). The protective effects of change in mastery however did not appear to be a significant mediator or moderator in the inflammation-SES relationship, necessitating a rejection of hypotheses 2B and 3B.

Several possible explanations may account for the null findings for hypotheses 2B and 3B (that change in sense of control mediates and moderates the association between SES and inflammatory dysfunction). The greater homogeneity among respondents for whom data on sense of control were available at more than one time point may account for the inability to find significant evidence of sample mediation among this sub-sample. Alternatively, other unmeasured life experiences and individual dispositions may alter the likelihood of increased sense of control over time in a way that is not as closely linked to income or education levels. Career development and satisfaction strengthens the sense of control, and this effect may continue to strengthen over time as one accumulates responsibilities and seniority while the effects of education and/or income on the sense of control may remain relatively stagnant over time (Finch et al. 1991). Additionally, new life roles associated with middle and older age may enhance the sense of control over time regardless of socioeconomic position. Successfully navigating the role of empty-nester, becoming a part-time caretaker for grandchildren, or planning for a secure retirement may grant a sense of fulfillment and accomplishment that increase the sense of control regardless of one's socioeconomic standing.

The use of multiple inflammatory markers yielded important insights for future research. First, prior research examining the biological underpinnings of the health effects of psychosocial resources most frequently examined the effects of CRP. In these analyses, the internal sense of control and change in control over time had the weakest association with C-reactive protein and the strongest link to interleukin-6. This finding upholds previous work suggesting that IL-6 may have stronger links to both SES and cardiovascular health outcomes (Friedman et al., 2010; Kritchevsky et al. 2005). The differences in the associations between personal control and inflammatory outcomes demonstrate the need to further examine the specific biological mechanisms linking internal resources to physical health. The three markers of inflammation used in the analysis are related, yet they each play distinct roles in the immunological stress response process. Given the findings, more work should be done to further explore the distinctions and connections between these three inflammatory measures.

Second, the examination of the associations between change in sense of control over time and inflammatory outcomes added considerable insight into how fluctuations in internal resources patterned over the life course may alter health outcomes over and above the effects of psychological functioning at one point in time. The fact that change in mastery was significantly associated with fibrinogen levels, while underlying average sense of control was not clearly demonstrates this fact.

There are several limitations to this research that should be addressed in future work. First, The findings from the multivariate analyses are associational in nature and do not establish a causal framework. Second, the relative racial and socioeconomic homogeneity of the longitudinal sample in particular limits our ability to determine to what extent these findings (and the non-significance of certain findings) can be applied to the broader US

population as a whole. Second, because the biological markers were only observed at one time point, it was impossible to analyze the effects of change in internal control on change in physiological functioning over time. This also limited the ability to determine to what extent these biomarkers are good indicators of long-term physiological dysregulation. In addition, we were unable to fully rule-out potential confounders in the control-inflammation relationship such as previous inflammatory-related conditions. While current conditions and medication-use were controlled for, there may be other factors that influence inflammation that date further back in individuals' life histories.

Overall this study provided support for the inflammation pathway as a link to understanding how internal resources such as sense of control 'get under the skin' to influence physical health. Further work should be done to re-assess these findings using a more diverse sample and with more waves of data to better elucidate the complex pathways from sense of control at baseline to individual indicators of inflammation. Additionally, future analyses using measures of inflammation at multiple time-points would demonstrate more clearly whether and how shifts in one's internal outlook across the life course contribute to prospective changes in physiological functioning.

APPENDIX 1: TABLES AND FIGURES

Table 3. Descriptive Statistics for Focal Variables

Variable	N	%	Mean	Std. Dev.	Min	Max
Mastery, Control, Constraints						
Wave 1						
Control	1,020		11.39	1.79	4.12	14
Mastery	1,020		5.85	0.97	1.75	7
Constraints	1,020		5.54	1.12	1.5	7
Wave 2						
Control	1,049		11.35	1.85	2.25	14
Mastery	1,049		5.77	1.01	1.25	7
Constraints	1,049		5.58	1.11	1	7
Change Scores						
Control Change	1,017		-0.021	1.71	-7	8.37
Mastery Change	1,017		0.054	1.07	-3.87	5.25
Positive		38.50%				
Negative		42.30%				
No Change		19.20%				
Constraint Change	1,017		0.05	1.07	-3.87	5.25
Positive		47.80%				
Negative		43.40%				
No Change		8.90%				
Inflammation Measures						
IL6 (pg./mL)	1,052		2.85	2.86	0.16	23
Fibrinogen (mg/dL.)	1,038		341.02	83.76	45	759
CRP (ug/mL)	1,052		2.8	4.34	0.14	61.7
Inflammation Burden	1,038					
0		26.10%				
1		24.90%				
2		23.80%				
3		25.10%				

Table 4. Descriptive Statistics for Covariates						
Variable	N	%	Mean	Std. Dev.	Min	Max
Basic Demographic Covariates						
Age	1,052		55.28	11.78	34	84
Gender	1,052					
Male		45.25				
Female		54.75				
Race	1,049					
White		93.04				
Black		2.57				
Other		4.39				
Socioeconomic Status Covariates						
Education	1,049					
Less than HS		3.53				
High School		20.59				
Some College		29.57				
College+		46.71				
Financial Well-Being	1,042		10.7	3.28	2	16
Income	1,030		76,506	60,252	0	300,000
Health Status and Health Behaviors						
Physical SRH	1,052					
Excellent		19.77				
Very Good		41.83				
Good		28.9				
Fair/Poor		9.51				
Mental SRH	1,052					
Excellent		29.47				
Very Good		38.5				
Good		25.67				
Fair/Poor		6.37				
CES-D Score	1,048		8.02	7.7	0	49
Chronic Conditions	1,052		2.3	2.34	0	29
BMI	1,052		29.25	6.4	14.99	100
Regular Exercise	1,052					
Yes		78.9				
No		21.1				
Ever Smoked Regularly	1,052					
Yes		47.3				
Drinks Per Week	1,052					
Never Drinks		0.4				
<1 drink		26.9				
1-4 drinks		25.1				
5 or more		12.3				
Medications						
Anti-Hypertensive	1,052	35.27				
Anti-Depressant	1,052	4.37				
Cholesterol Lowering	1,052	15.59				

Table 5. Hypothesis 1A Control Predicts Inflammation	
Variable	Full Sample IL6
Avg. Control	-0.233**
Female	0.104
Older	0.372+
Nonwhite	0.810***
Income Quintile	-0.11
High School/Less	-0.038
Difficulty Pay Bills	-0.304
Blood Pressure Med	0.426*
Cholesterol Med	-0.066
Depression Med	0.23
Steroids	0.068
Total Sympt/Conditions	0.106+
Ever Smoke	0.176
BMI	0.068***
Avg. Alcohol	0.068+
Norepinephrine	0.026***
Regular Exercise	-0.593**
_cons	1.43
N	1233
r2	0.13

Table 6. Hypotheses 1A and 2A Main Effects and Mediation

Variable	Full Sample	
	Model 1	Model 2
Avg. Control		-0.233**
Female	0.115	0.104
Older	0.309	0.372+
Nonwhite	0.783***	0.810***
Income Quintile	-0.140*	-0.11
High School/Less	0.026	-0.038
Difficulty Pay Bills	-0.155	-0.304
Blood Pressure Med	0.416*	0.426*
Cholesterol Med	-0.008	-0.066
Depression Med	0.24	0.23
Steroids	0.28	0.068
Total Sympt/Conditions	0.124*	0.106+
Ever Smoke	0.167	0.176
BMI	0.069***	0.068***
Avg. Alcohol	0.059	0.068+
Norepinephrine	0.026***	0.026***
Regular Exercise	-0.607**	-0.593**
_cons	0.22	1.43
N	1233	1233
r2	0.128	0.13

Table 7. Hypothesis 3A Control by SES		
Variable		Full Sample IL6
Avg. Control		-0.484***
Income#Control		
	1st Quintile	(base)
	2nd Quintile	0.121
	3rd Quintile	0.238
	4th Quintile	0.464*
	5th Quintile	0.548*
Female		0.105
Older		0.385+
Nonwhite		0.780***
Income Quintile		-0.919**
High School/Less		-0.062
Difficulty Pay Bills		-0.32
Blood Pressure Med		0.431*
Cholesterol Med		-0.05
Depression Med		0.277
Steroids		0.1
Total Sympt/Conditions		0.099
Ever Smoke		0.178
BMI		0.067***
Avg. Alcohol		0.064
Norepinephrine		0.025***
Regular Exercise		-0.584**
_cons		3.775**
N		1233
r2		0.138

Table 8. Hypothesis 1B Control-Change Predicts Inflammation		
Variable	IL6	FGN
Positive Change Mastery	-0.318*	-8.314+
Average Control	-0.026	2.205
Female	0.163	18.725***
Older	0.31	13.959*
Nonwhite	-0.309	17.421+
Income Quintile	-0.002	0.621
High School/Less	0.115	18.547**
Difficulty Pay Bills	-0.04	-2.537
Blood Pressure Med	0.549*	-7.252
Cholesterol Med	0.185	11.771
Depression Med	0.464+	10.632
Steroids	0.453	-9.559
Total Sympt/Conditions	0.05	2.181
Ever Smoke	0.294+	0.489
BMI	0.077***	3.014***
Avg. Alcohol	-0.06	-2.196
Norepinephrine	0.024***	0.878***
Regular Exercise	-0.372	-6.02
_cons		201.355***
N	1033	1033
r2	0.125	0.138

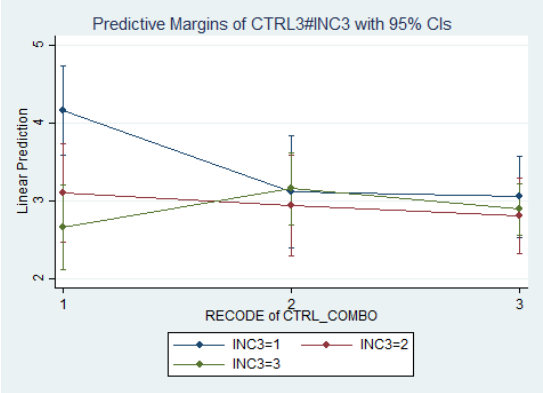
Table 9. Hypothesis 2B Control-Change Mediates SES- Inflammation				
Variable	IL6		FGN	
	Model 1	Model 2	Model 1	Model 2
Positive Change Mastery		-0.318*		-8.314+
Average Control	0	-0.026	3.489	2.205
Female	0.157	0.163	18.663***	18.725***
Older	0.301	0.31	13.991*	13.959*
Nonwhite	-0.291	-0.309	17.895+	17.421+
Income Quintile	0.001	-0.002	0.747	0.621
High School/Less	0.118	0.115	18.491**	18.547**
Difficulty Pay Bills	-0.055	-0.04	-2.778	-2.537
Blood Pressure Med	0.545*	0.549*	-7.423	-7.252
Cholesterol Med	0.179	0.185	11.671	11.771
Depression Med	0.452	0.464+	10.513	10.632
Steroids	0.422	0.453	-10.44	-9.559
Total Sympt/Conditions	0.057	0.05	2.309	2.181
Ever Smoke	0.279	0.294+	0.047	0.489
BMI	0.076***	0.077***	2.979***	3.014***
Avg. Alcohol	-0.06	-0.06	-2.192	-2.196
Norepinephrine	0.024***	0.024***	0.851***	0.878***
Regular Exercise	-0.405	-0.372	-6.705	-6.02
_cons	-0.381		193.637***	201.355***
N	1033	1033	1033	1033
r2	0.121	0.125	0.136	0.138

Table 10. Hypothesis 3B PC Change-Inflammation Moderated by SES			
Variable		IL6	FGN
Positive Change Mastery		-0.522+	-1.716
Average Control		-0.063	2.25
Income#Control-Change			
	2nd Quintile	0.309	-3.431
	3rd Quintile	-0.159	-4.878
	4th Quintile	0.56	-10.257
	5th Quintile	0.173	-20.212
Female		0.158	18.369**
Older		0.324	13.403*
Nonwhite		-0.334	17.932+
Income Quintile		-0.022	1.975
High School/Less		0.1	19.073**
Difficulty Pay Bills		-0.043	-2.637
Blood Pressure Med		.552*	-6.96
Cholesterol Med		0.185	12.065+
Depression Med		0.451+	10.24
Steroids		0.463	-9.811
Total Sympt/Conditions		0.047	2.211
Ever Smoke		0.302+	0.55
BMI		0.077	3.006***
Avg. Alcohol		-0.065	-2.128
Norepinephrine		0.025***	0.866***
Regular Exercise		-0.367+	-6.286
_cons		0.085	198.051***
N		1033	1033
r2		0.128	0.14

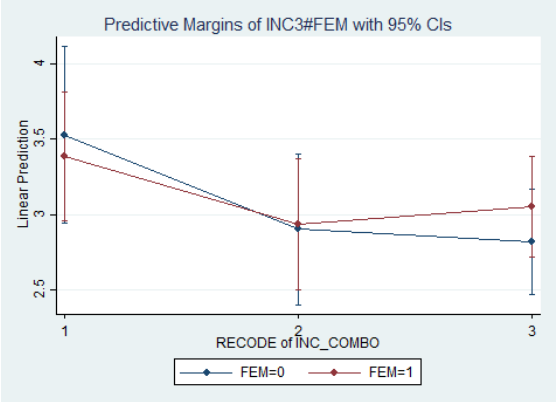
Table 11. Hypothesis 4 Effect of Control by Income and Gender		
Variable		Full Sample
		IL6
Avg. Control		-0.658***
Female#Control#Income		
	Male	0.147*
	Female	0.154**
Female		-0.028
Older		0.39
Nonwhite		0.789***
Income Quintile		-0.955**
High School/Less		-0.037
Difficulty Pay Bills		-0.325
Blood Pressure Med		0.425*
Cholesterol Med		-0.075
Depression Med		0.264
Steroids		0.096
Total Sympt/Conditions		0.104
Ever Smoke		0.171
BMI		0.067***
Avg. Alcohol		0.065
Norepinephrine		0.025***
Regular Exercise		-0.579**
_cons		3.882**
N		1191
r2		0.135

Table 12. Hypothesis 5 Control by Age	
Variable	Full Sample IL6
Avg. Control	-0.137
Older#Control	-0.368+
Female	0.092
Older	2.456*
Nonwhite	.806***
Income Quintile	-0.113+
High School/Less	-0.049
Difficulty Pay Bills	-0.291
Blood Pressure Med	0.416*
Cholesterol Med	-0.086
Depression Med	0.245
Steroids	0.074
Total Sympt/Conditions	0.109+
Ever Smoke	0.175
BMI	.069***
Avg. Alcohol	0.065
Norepinephrine	0.026***
Regular Exercise	-0.593**
_cons	0.878
N	1233
r2	0.133

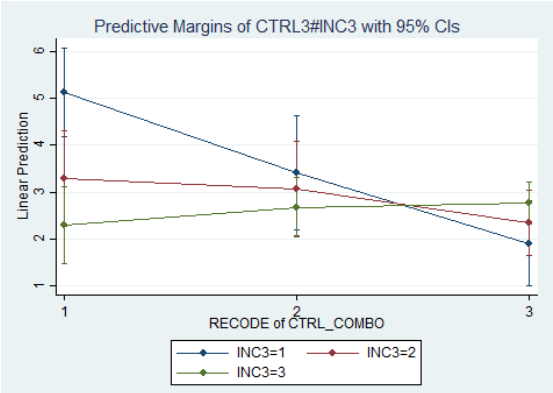
Graph 1: Effect of Sense of Control on IL6 by Income



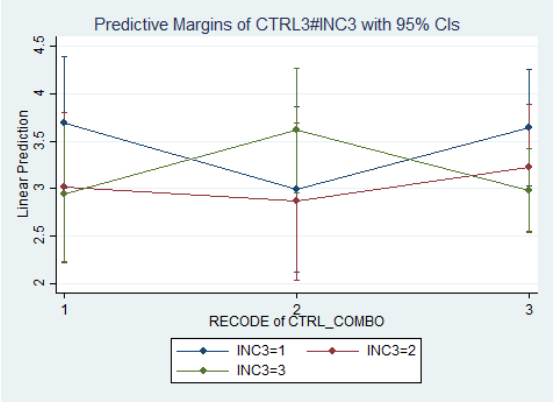
Graph 2: Income by Gender



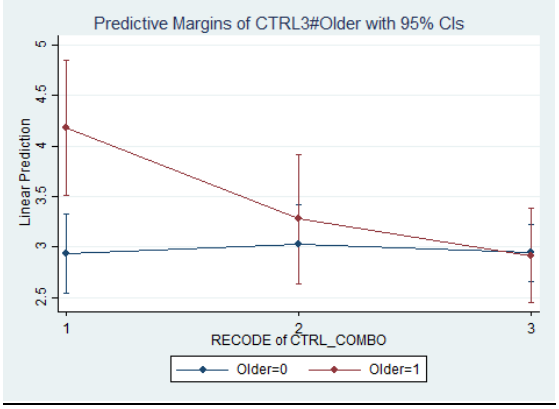
Graph 3: Control by Income: Men



Graph 4: Control by Income: Women



Graph 5. Control by Age



APPENDIX 2: ADDITIONAL TABLES AND FIGURES

Figure 1 Sense of Control Documentation of Sub-Scales

SENSE OF CONTROL

Scales/Items:

Personal Mastery [B1SMASTE] (M1 scale name: A1SMASTE):

Items: 4 items - Self-Administered Questionnaire, Section E, Question 4 (c, f, h, l)

c. "I can do just about anything I really set my mind to."

f. "When I really want to do something, I usually find a way to succeed at it."

h. "Whether or not I am able to get what I want is in my own hands."

l. "What happens to me in the future mostly depends on me."

Perceived Constraints [B1SCONST] (M1 scale name: A1SCONST):

Items: 8 items - Self-Administered Questionnaire, Section E, Question 4 (a, b, d, e, g, i, j, k)

a. "There is little I can do to change the important things in my life."

b. "I often feel helpless in dealing with the problems of life."

d. "Other people determine most of what I can and cannot do."

e. "What happens in my life is often beyond my control."

g. "There are many things that interfere with what I want to do."

i. "I have little control over the things that happen to me."

j. "There is really no way I can solve the problems I have."

k. "I sometimes feel I am being pushed around in my life."

Coding: 1 Strongly agree; 2 Somewhat agree; 3 A little agree; 4 Neither agree or disagree; 5 A little disagree; 6 Somewhat disagree; 7 Strongly disagree.

Scaling: Scales are constructed by calculating the mean across each set of items. Items were recoded so that high scores reflect higher standing in each dimension.

Perceived Control [B1SCTRL] (M1 scale name: A1SACTRL):

Items: 12-item scale combining the 4 "personal mastery" items and the 8 "perceived constraints" items.

Scaling: [B1SCTRL] is constructed by calculating the mean of the 12 items. Items from "personal mastery" were reverse-coded so that higher scores represent higher levels of the overall perceived control.

Missing Values: The scales are computed for cases that have valid values for at least half of the items on the particular scale. Scale scores are not calculated for cases with fewer than half of the items on the scales, and coded as "8" for "NOT CALCULATED (Due to missing data)."

26

Figure 2 CFA Structure

Model C- Two Dimensional Correlated Structure

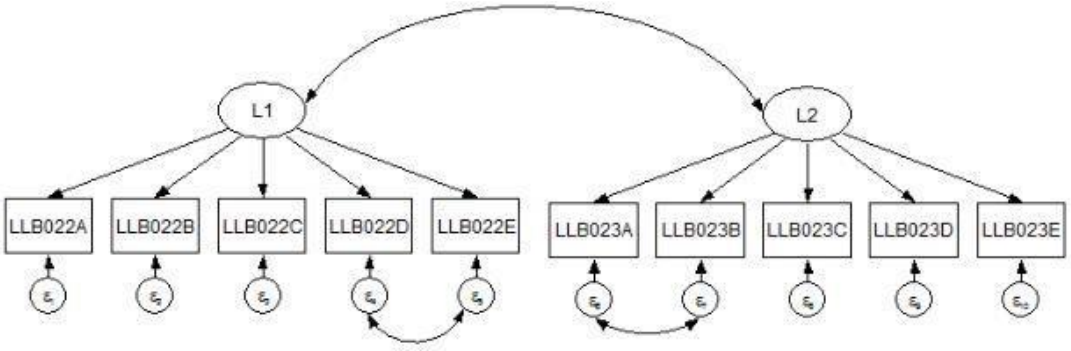


Table 13. CFA Model Fit Statistics				
Model	Estimator	RMSEA	CFI	TLI
C	MLMV	0.047	0.979	0.971

REFERENCES

Allin, S., C. Masseria and E. Mossialos. 2009. "Measuring Socioeconomic Differences in use of Health Care Services by Wealth Versus by Income." *American Journal of Public Health* 99(10): 1849-1855. doi: 10.2105/AJPH.2008.141499 [doi].

Allison, Paul D. "Missing data: Quantitative applications in the social sciences." *British Journal of Mathematical and Statistical Psychology* 55, no. 1 (2002): 193-196.

Aneshensel, Carol S. and Uchechi A. Mitchell. 2014. "Stress Process, the." *The Wiley Blackwell Encyclopedia of Health, Illness, Behavior, and Society*.

Baltes, Margret M. 1995. "Dependency in Old Age: Gains and Losses." *Current Directions in Psychological Science*:14-19.

Bailis, Daniel S., Alexander Segall, Michael J. Mahon, Judith G. Chipperfield, and Elaine M. Dunn. 2001. "Perceived Control in Relation to Socioeconomic and Behavioral Resources for Health." *Social Science & Medicine* 52(11): 1661-1676.

Bandura, Albert. 1986. *Social foundations of thought and action*. Englewood Cliffs, New Jersey: Prentice-Hall.

Bolger, Niall and Adam Zuckerman. 1995. "A Framework for Studying Personality in the Stress Process." *Journal of Personality and Social Psychology* 69(5):890.

Bovier, Patrick A., Eric Chamot and Thomas V. Perneger. 2004. "Perceived Stress, Internal Resources, and Social Support as Determinants of Mental Health among Young Adults." *Quality of Life Research* 13(1):161-170.

Brim, Orville G, et al. 2009. "Documentation of Scales and Constructed Variables in MIDUS 2." *Institute of Aging,* University of Wisconsin.

Cain, Kevin C., Richard A. Kronmal and Andrzej S. Kosinski. 1992. "Analysing the Relationship between Change in a Risk Factor and Risk of Disease." *Statistics in Medicine* 11(6):783-797.

Caplan, Leslie J. and Carmi Schooler. 2003. "The Roles of Fatalism, Self-Confidence, and Intellectual Resources in the Disablement Process in Older Adults." *Psychology and Aging* 18(3):551.

Caputo, Richard K. 2003. "The Effects of Socioeconomic Status, Perceived Discrimination and Mastery on Health Status in a Youth Cohort." *Social Work in Health Care*37(2):17-42.

Case, Anne, Angela Fertig and Christina Paxson. 2005. "The Lasting Impact of Childhood Health and Circumstance." *Journal of Health Economics* 24(2):365-389. Retrieved 8/18/2014 3:50:17 PM.

Cesari M., Penninx B.W., Newman A.B., et al. (2003) Inflammatory markers and onset of cardiovascular events: results from the Health ABC study. Circulation108:2317–2322.

Chen, Y-F, ME Dewey and AJ Avery. 2001. "Self-reported Medication use for Older People in England and Wales." *Journal of Clinical Pharmacy and Therapeutics* 26(2):129-140.

Crimmins, E. M., J. K. Kim and T. E. Seeman. 2009. "Poverty and Biological Risk: The Earlier "Aging" of the Poor." *The Journals of Gerontology.Series A, Biological Sciences and Medical Sciences* 64(2):286-292. doi: 10.1093/gerona/gln010 [doi].

DeLamater, John D. and Amanda Ward. 2006. *Handbook of Social Psychology*.Springer.

DiStefano, Christine, Min Zhu and Diana Mindrila. 2009. "Understanding and using Factor Scores: Considerations for the Applied Researcher." *Practical Assessment, Research & Evaluation* 14(20):1-11.

Duncan, Greg J., Kathleen M. Ziola Guest and Ariel Kalil. 2010. "Early Childhood Poverty and Adult Attainment, Behavior, and Health." *Child Development* 81(1):306-325. Retrieved 8/18/2014 4:21:08 PM.

Evans, Gary W. and Kimberly English. 2002. "The Environment of Poverty: Multiple Stressor Exposure, Psychophysiological Stress, and Socioemotional Adjustment." *Child Development* 73(4):1238-1248.

Feinglass, J., S. Lin, J. Thompson, J. Sudano, D. Dunlop, J. Song and D. W. Baker. 2007. "Baseline Health, Socioeconomic Status, and 10-Year Mortality among Older Middle-Aged Americans: Findings from the Health and Retirement Study, 1992 2002." *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 62(4):S209-17. doi: 62/4/S209 [pii].

Finch, Michael D., Michael J. Shanahan, Jeylan T. Mortimer, and Seongryeol Ryu.1991. "Work Experience and Control Orientation in Adolescence." *American Sociological Review*: 597-611.

Folkman, Susan, Richard S. Lazarus, Christine Dunkel-Schetter, Anita DeLongis and Rand J. Gruen. 1986. "Dynamics of a Stressful Encounter: Cognitive Appraisal, Coping, and Encounter Outcomes." Journal of Personality and Social Psychology 50(5):992. Retrieved 8/18/2014 5:21:53 PM.

Geronimus, Arline T., Margaret Hicken, Danya Keene, and John Bound. 2006. "“Weathering” and age patterns of allostatic load scores among blacks and whites in the United States." *Journal Information* 96, no. 5

Glei, D. A., N. Goldman, Y. L. Chuang and M. Weinstein. 2007. "Do Chronic Stressors Lead to Physiological Dysregulation? Testing the Theory of Allostatic Load."*Psychosomatic Medicine* 69(8):769-776. doi: PSY.0b013e318157cba6 [pii].

Glymour, M. M., J. Weuve, L. F. Berkman, I. Kawachi and J. M. Robins. 2005. "When is Baseline Adjustment Useful in Analyses of Change? an Example with Education and Cognitive Change." *American Journal of Epidemiology* 162(3):267-278. doi: kwi187 [pii].

Goldman, N., C. M. Turra, D. A. Glei, C. L. Seplaki, Y. H. Lin and M. Weinstein. 2006. "Predicting Mortality from Clinical and Nonclinical Biomarkers." *The Journals of Gerontology Series A, Biological Sciences and Medical Sciences* 61(10):1070-1074. doi: 61/10/1070 [pii]

Gruenewald, Tara L., Arun S. Karlamangla, Perry Hu, Sharon Stein-Merkin, Carolyn Crandall, Brandon Koretz and Teresa E. Seeman. 2012. "History of Socioeconomic Disadvantage and Allostatic Load in Later Life." *Social Science & Medicine* 74(1):75-83.

Guilley, Edith, Matthias Bopp, David Faeh and Fred Paccaud. 2010. "Socioeconomic Gradients in Mortality in the Oldest Old: A Review." *Archives of Gerontology and Geriatrics* 51(3):e37-e40.

Gustafsson, P. E., U. Janlert, T. Theorell, H. Westerlund and A. Hammarstrom. 2011. "Socioeconomic Status Over the Life Course and Allostatic Load in Adulthood: Results from the Northern Swedish Cohort." *Journal of Epidemiology and Community Health* 65(11):986-992. doi: 10.1136/jech.2010.108332 [doi].

Hardin, James W., Henrik Schmiediche and Raymond J. Carroll. 2003. "The Regression-Calibration Method for Fitting Generalized Linear Models with Additive Measurement Error." *Stata Journal* 3(4):361-372.

Harris T.B., FERRUCCI L., TRACY R.P., et al. (1999) Associations of elevated interleukin-6 and C-reactive protein levels with mortality in the elderly. Am. J. Med. 106:506–512

Hart, Patricia L. and Cecelia G. Grindel. 2010. "Illness Representations, Emotional Distress, Coping Strategies, and Coping Efficacy as Predictors of Patient Outcomes in Type 2 Diabetes." Journal of Nursing and Healthcare of Chronic Illness 2(3):225-240. Retrieved 8/13/2014 1:58:14 PM.

Hasson, D., U. Von Thiele Schwarz and P. Lindfors. 2009. "Self-Rated Health and Allostatic Load in Women Working in Two Occupational Sectors." *Journal of Health Psychology* 14(4):568-577. doi: 10.1177/1359105309103576 [doi].

Hatcher, L. (1994). A step-by-step approach to using the SAS(R) system for factor analysis and structural equation modeling. Cary, NC: SAS Institute.

Heckhausen, Jutta. 1997. "Developmental Regulation Across Adulthood: Primary and Secondary Control of Age-Related Challenges." *Developmental Psychology* 33(1):176.

Health and Retirement Study, public use dataset. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740). Ann Arbor, MI, (2010).

Friedman, Elliot M., and Pamela Herd. "Income, education, and inflammation: differential associations in a national probability sample (the MIDUS study)." *Psychosomatic medicine* 72, no. 3 (2010): 290.

Hokayem, Charles and Misty L. Heggeness. 2014. "Living in Near Poverty in the United States: 1966-2012." Census Current Population Reports.

Hoshino, Takahiro and Peter M. Bentler. 2011. "Bias in Factor Score Regression and a Simple Solution." *Department of Statistics, UC*

Hu, Peifeng, Naveed Wagle, Noreen Goldman and TERESA E. SEEMAN. 2007. "The Associations between Socioeconomic Status, Allostatic Load and Measures of Health in Older Taiwanese Persons: Taiwan Social Environment and Biomarkers of Aging Study." *Journal of Biosocial Science* 39(04):545-556.

Infurna, Frank J., Nilam Ram and Denis Gerstorf. 2012. "Level and Change in Perceived Control Predict 19-Year Mortality: Findings from the Americans' Changing Lives Study."

Infurna, Frank J., Denis Gerstorf and Steven H. Zarit. 2011. "Examining Dynamic Links between Perceived Control and Health: Longitudinal Evidence for Differential Effects in Midlife and Old Age." *Developmental Psychology*47(1):9.

Johnson, Colleen L. and Barbara M. Barer. 1993. "Coping and a Sense of Control among the Oldest Old: An Exploratory Analysis." *Journal of Aging Studies* 7(1):67-80.

Judge, Timothy A. and Joyce E. Bono. 2001. "Relationship of Core Self-Evaluations traits—self-Esteem, Generalized Self-Efficacy, Locus of Control, and Emotional stability—with Job Satisfaction and Job Performance: A Meta-Analysis." *Journal of Applied Psychology* 86(1):80. Retrieved 9/8/2014 11:09:30 AM.

Judge, Timothy A., Amir Erez, Joyce E. Bono and Carl J. Thoresen. 2003. "The Core self evaluations Scale: Development of a Measure." *Personnel Psychology* 56(2):303-331.

Juster, Robert-Paul, Bruce S. McEwen and Sonia J. Lupien. 2010. "Allostatic Load Biomarkers of Chronic Stress and Impact on Health and Cognition." *Neuroscience & Biobehavioral Reviews* 35(1):2-16.

Juster, Robert-Paul, Shireen Sindi, Marie-France Marin, Andrea Perna, Alireza Hashemi, Jens C. Pruessner and Sonia J. Lupien. 2011. "A Clinical Allostatic Load Index is Associated with Burnout Symptoms and Hypocortisolemic Profiles in Healthy Workers." *Psychoneuroendocrinology* 36(6):797-805. Retrieved 8/18/2014 4:41:32 PM.

Kaptoge, S., E. Di Angelantonio, G. Lowe, M. B. Pepys, S. G. Thompson, R. Collins and J. Danesh. 2010. "C-Reactive Protein Concentration and Risk of Coronary Heart Disease, Stroke, and Mortality: An Individual Participant Meta-Analysis." *Lancet* 375(9709):132-140. doi: 10.1016/S0140-6736(09)61717-7 [doi]. Emerging Risk Factors Collaboration.

Karlamangla, A. S., B. H. Singer and T. E. Seeman. 2006. "Reduction in Allostatic Load in Older Adults is Associated with Lower all-Cause Mortality Risk: MacArthur Studies of Successful Aging." *Psychosomatic Medicine*68(3):500-507. doi: 68/3/500 [pii].

Karatsoreos, Ilia N., and Bruce S. McEwen. 2011. "Psychobiological allostasis: resistance, resilience and vulnerability." *Trends in cognitive sciences* 15, no. 12:576-584.

Keith, Pat. 2004. "Resources, Family Ties, and Well-being of Never-Married Men and Women." *Journal of Gerontological Social Work* 42(2):51-75.

Krause, N. 2007. "Age and Decline in Role-Specific Feelings of Control." *The Journals of Gerontology.Series B, Psychological Sciences and Social Sciences* 62(1):S28-35. doi: 62/1/S28 [pii].

Kristenson, Margareta, Hege R. Eriksen, Judith K. Sluiter, Dagmar Starke, and Holger Ursin. 2004. "Psychobiological Mechanisms of Socioeconomic Differences in Health." *Social Science & Medicine* 58(8): 1511-1522.

Kritchevsky SB, Cesari M, Pahor M. Inflammatory markers and cardiovascular health in older adults. *Cardiovasc Res*. 2005;66:265–275.

Lachman, Margie E., and Suzanne L. Weaver. 1998. "The Sense of Control as a Moderator of Social Class Differences in Health and Well-being." *Journal of Personality and Social Psychology* 74(3): 763.

Lachman, Margie E. and Stefan Agrigoroaei. 2010. "Promoting Functional Health in Midlife and Old Age: Long-Term Protective Effects of Control Beliefs, Social Support, and Physical Exercise." PloS One 5(10):e13297.

Lachman, Margie E., Shevaun D. Neupert and Stefan Agrigoroaei. 2011. "The Relevance of Control Beliefs for Health and Aging." *Handbook of the Psychology of Aging* 7:175-190.

Leopold, Liliya and Henriette Engelhartdt. 2013. "Education and Physical Health Trajectories in Old Age. Evidence from the Survey of Health, Ageing and Retirement in Europe (SHARE)." *International Journal of Public Health* 58(1):23-31.

Lee, Pai-Lin. 2015. "Control Beliefs Level and Change as Predictors of Subjective Memory Complaints." *Aging & Mental Health* (ahead-of-print):1-7.

Libby, Peter. 2007. "Inflammatory Mechanisms: The Molecular Basis of Inflammation and Disease." *Nutrition Reviews* 65(suppl 3):S140-S146.

Link, Bruce G. and Jo Phelan. 1995. "Social Conditions as Fundamental Causes of Disease." *Journal of Health and Social Behavior*:80-942

Marsh, Herbert W., Kit-Tai Hau and Zhonglin Wen. 2004. "In Search of Golden Rules: Comment on Hypothesis-Testing Approaches to Setting Cutoff Values for Fit Indexes and Dangers in Overgeneralizing Hu and Bentler's (1999) Findings." *Structural Equation Modeling* 11(3):320-341.

McEwen, Bruce S. and Peter J. Gianaros. 2010. "Central Role of the Brain in Stress and Adaptation: Links to Socioeconomic Status, Health, and Disease." *Annals of the New York Academy of Sciences* 1186(1):190-222.

McEwen, B. S. 2012. "Brain on Stress: How the Social Environment Gets Under the Skin." *Proceedings of the National Academy of Sciences of the United States of America* 109 Suppl 2:17180-17185. Retrieved 8/18/2014 4:14:36 PM. doi: 10.1073/pnas.1121254109 [doi].

McEwen, Bruce S. and Teresa Seeman. 1999. "Protective and Damaging Effects of Mediators of Stress: Elaborating and Testing the Concepts of Allostasis and Allostatic Load." *Annals of the New York Academy of Sciences* 896(1):30-47.

McLeod, Jane D. 2012. "The Meanings of Stress Expanding the Stress Process Model." *Society and Mental Health* 2(3):172-186. Retrieved 8/18/2014 4:05:40 PM.

Merkin, Sharon S., Arun Karlamangla, Ana V. Diez Roux, Sandi Shrager and Teresa E. Seeman. 2014. "Life Course Socioeconomic Status and Longitudinal Accumulation of Allostatic Load in Adulthood: Multi-Ethnic Study of Atherosclerosis." *American Journal of Public Health* 104(4):e48-e55. Retrieved 8/18/2014 4:40:56 PM.

Miller, G. E., M. E. Lachman, E. Chen, T. L. Gruenewald, A. S. Karlamangla and T. E. Seeman. 2011. "Pathways to Resilience: Maternal Nurturance as a Buffer Against the Effects of Childhood Poverty on Metabolic Syndrome at Midlife." *Psychological Science* 22(12):1591-1599. Retrieved 8/18/2014 4:28:36 PM. doi: 10.1177/0956797611419170 [doi].

Mirowsky, John and Catherine E. Ross. 1991. "Eliminating Defense and Agreement Bias from Measures of the Sense of Control: A 2 x 2 Index." *Social Psychology Quarterly*:127-145.

Mirowsky, John, and Catherine E. Ross. 2003. Social causes of psychological distress. *Transaction Publishers.*

Mirowsky, John, and Catherine E. Ross. 2005. "Education, Learned Effectiveness and Health." *London Review of Education* 3(3):205-220

Mirowsky, John, and Catherine E. Ross. 2007. "Life Course Trajectories of Perceived Control and Their Relationship to Education." *American Journal of Sociology* 112(5): 1339-1382.

Moon, J. R., M. M. Glymour, SV Subramanian, Mauricio Avendaño and Ichiro Kawachi. 2012. "Transition to Retirement and Risk of Cardiovascular Disease: Prospective Analysis of the US Health and Retirement Study."*Social Science & Medicine* 75(3):526-530.

Pagel, Mark D., Joseph Becker and David B. Coppel. 1985. "Loss of Control, Self-Blame, and Depression: An Investigation of Spouse Caregivers of Alzheimer's Disease Patients." *Journal of Abnormal Psychology* 94(2):169.

Pearlin, Leonard I., Elizabeth G. Menaghan, Morton A. Lieberman and Joseph T. Mullan. 1981. "The Stress Process." *Journal of Health and Social Behavior*:337-356. Retrieved 8/12/2014 11:21:16 AM.

Peterson, Christopher. 1999. "15 Personal Control and Well-being." *Well-being: Foundations of Hedonic Psychology*:288.

Phelan, J. C., B. G. Link and P. Tehranifar. 2010. "Social Conditions as Fundamental Causes of Health Inequalities: Theory, Evidence, and Policy Implications." *Journal of Health and Social Behavior* 51 Suppl:S28-40. Retrieved 8/18/2014 4:38:28 PM. doi: 10.1177/0022146510383498 [doi].

Pearlin, Leonard I., Kim B. Nguyen, Scott Schieman, and Melissa A. Milkie. 2007. "The Life-course Origins of Mastery among Older People." *Journal of Health and Social Behavior* 48(2): 164-179.

Pudrovska, Tetyana, Scott Schieman, Leonard I. Pearlin, and Kim Nguyen. 2005. "The Sense of Mastery as a Mediator and Moderator in the Association between Economic Hardship and Health in Late Life." *Journal of Aging and Health* 17(5): 634-660.

Reynolds, John R., Stephanie W. Burge, Cheryl L. Robbins, Emily M. Boyd and Brandy Harris. 2007. "Mastery and the Fulfillment of Occupational Expectations by Midlife." *Social Psychology Quarterly* 70(4):366-383. Retrieved 8/13/2014 1:55:40 PM.

Robert, S. and J. S. House. 1996. "SES Differentials in Health by Age and Alternative Indicators of SES." *Journal of Aging and Health* 8(3):359-388.

Rodin, J. 1986. "Aging and Health: Effects of the Sense of Control." *Science (New York, N.Y.)* 233(4770):1271-1276.

Ross, Catherine E., and John Mirowsky. "Age and the Gender Gap in the Sense of Personal Control." *Social Psychology Quarterly* (2002): 125-145.

Ross, Catherine E., and John Mirowsky. 2013. "The sense of personal control: Social structural causes and emotional consequences." In *Handbook of the sociology of mental health*, pp. 379-402. Springer Netherlands.

Ross, Catherine E., and John Mirowsky. 2011. "The Interaction of Personal and Parental Education on Health." *Social Science & Medicine* 72(4):591-599.

Ross, Catherine E. and John Mirowsky. 2006. "Social Structure and Psychological Functioning" in *Handbook of Social Psychology*, DeLamater, John D. and Amanda Ward eds. Springer.

Ryff, Carol D, Seeman, Teresa, Weinstein, Maxine. 2010. "Medication Data." *National Survey of Midlife Development in the United States (MIDUS II): Biomarker Project, 2004-2009.*

Santos, J. R. A. 1999. "Cronbach's Alpha: A Tool for Assessing the Reliability of Scales." *Journal of Extension* 37(2):1-5.

Schieman, Scott, Kim Nguyen and Diana Elliott. 2003. "Religiosity, Socioeconomic Status, and the Sense of Mastery." *Social Psychology Quarterly*:202-221.

Schneiderman, N., G. Ironson and S. D. Siegel. 2005. "Stress and Health: Psychological, Behavioral, and Biological Determinants." *Annual Review of Clinical Psychology* 1:607-628. doi: 10.1146/annurev.clinpsy.1.102803.144141 [doi].

Seeman, T. E., B. S. McEwen, J. W. Rowe and B. H. Singer. 2001. "Allostatic Load as a Marker of Cumulative Biological Risk: MacArthur Studies of Successful Aging." *Proceedings of the National Academy of Sciences of the United States of America* 98(8):4770-4775. doi: 10.1073/pnas.081072698 [doi].

Seeman, Teresa E., Eileen Crimmins, Mei-Hua Huang, Burton Singer, Alexander Bucur, Tara Gruenewald, Lisa F. Berkman and David B. Reuben. 2004. "Cumulative Biological Risk and Socio-Economic Differences in Mortality: MacArthur Studies of Successful Aging." *Social Science & Medicine* 58(10):1985-1997.

Seeman, Teresa, Sharon S. Merkin, Eileen Crimmins, Brandon Koretz, Susan Charette and Arun Karlamangla. 2008. "Education, Income and Ethnic Differences in Cumulative Biological Risk Profiles in a National Sample of US Adults: NHANES III (1988–1994)." *Social Science & Medicine* 66(1):72-87.

Seeman, Teresa, Elissa Epel, Tara Gruenewald, Arun Karlamangla and Bruce S. McEwen. 2010. "Socio-economic Differentials in Peripheral Biology: Cumulative Allostatic Load." *Annals of the New York Academy of Sciences*1186(1):223-239.

Segerstrom, Suzanne C. and Gregory E. Miller. 2004. "Psychological Stress and the Human Immune System: A Meta-Analytic Study of 30 Years of Inquiry." *Psychological Bulletin* 130(4):601.

Seplaki, C. L., N. Goldman, M. Weinstein and Y. H. Lin. 2004. "How are Biomarkers Related to Physical and Mental Well-being?" *The Journals of Gerontology.Series A, Biological Sciences and Medical Sciences* 59(3):201-217.

Shanahan, Michael J., Patrick L. Hill, Brent W. Roberts, Jacquelynne Eccles, and Howard S. Friedman. 2012. "Conscientiousness, Health, and Aging: The Life Course of Personality Model." *Developmental Psychology*.

Shanahan, M. J., Bauldry, S., Roberts, B. W., Macmillan, R., & Russo, R. (2014). Personality and the reproduction of social class. *Social Forces*, 93(1), 209-240.

Skaff, Marilyn M. 2007. "Sense of Control and Health." *Handbook of Health Psychology and Aging*:186-209.

Skinner, Ellen A. "A guide to constructs of control." *Journal of personality and social psychology* 71, no. 3 (1996): 549.

Smith, Jacqui, et al. 2013. "HRS Psychosocial and Lifestyle Questionnaire 2006-2010 Documentation Report." Institute for Social Research, University of Michigan. http://hrsonline.isr.umich.edu/sitedocs/userg/HRS2006-2010SAQdoc.pdf

Stefanski, LA. 2000. "Measurement Error Models." *Journal of the American Statistical Association* 95(452):1353-1358.

Tavakol, Mohsen and Reg Dennick. 2011. "Making Sense of Cronbach's Alpha." *International Journal of Medical Education* 2:53.

Taylor, Shelley E., and Teresa E. Seeman. 1999. "Psychosocial Resources and the SES-Health Relationship." *Annals of the New York Academy of Sciences* 896(1): 210-225.

Taylor, Shelley E., Jennifer S. Lerner, David K. Sherman, Rebecca M. Sage and Nina K. McDowell. 2003. "Are Self-Enhancing Cognitions Associated with Healthy Or Unhealthy Biological Profiles?" *Journal of Personality and Social Psychology* 85(4):605.

Taylor, Shelley E. 2011. "How Psychosocial Resources Enhance Health and Well-being." Applied Positive Psychology: Improving Everyday Life, Health, Schools, Work, and Society:65-78. Retrieved 8/18/2014 4:09:08 PM.

Taylor, Shelley E. and Teresa E. Seeman. 1999. "Psychosocial Resources and the SES-health Relationship." *Annals of the New York Academy of Sciences* 896(1):210-225. Retrieved 8/12/2014 1:55:53 PM.

Taylor, Shelley E. and Annette L. Stanton. 2007. "Coping Resources, Coping Processes, and Mental Health." *Annu.Rev.Clin.Psychol*. 3:377-401.

Taylor, Shelley E., Lisa J. Burklund, Naomi I. Eisenberger, Barbara J. Lehman, Clayton J. Hilmert and Matthew D. Lieberman. 2008. "Neural Bases of Moderation of Cortisol Stress Responses by Psychosocial Resources." *Journal of Personality and Social Psychology* 95(1):197.

Thoits, Peggy A. 1995. "Stress, Coping, and Social Support Processes: Where are we? what Next?" *Journal of Health and Social Behavior*:53-79. Retrieved 8/12/2014 11:50:17 AM.

Tucker-Seeley, Reginald D., Yi Li, SV Subramanian and Glorian Sorensen. 2009. "Financial Hardship and Mortality among Older Adults using the 1996–2004 Health and Retirement Study." *Annals of Epidemiology*19(12):850-857.

Turiano, Nicholas A., Benjamin P. Chapman, Stefan Agrigoroaei, Frank J. Infurna, and Margie Lachman. (2014). "Perceived Control Reduces Mortality Risk at Low, Not High, Education Levels."

Turner, R. J. 2010. "Understanding health disparities: The promise of the stress process model." Pp. 3-21 in Advances in the Conceptualization of the Stress Process."Understanding health disparities: The promise of the stress process model."Springer. Retrieved 8/18/2014 4:16:50 PM.

Turner, R. J. and Donald A. Lloyd. 1999. "The Stress Process and the Social Distribution of Depression." *Journal of Health and Social Behavior*:374-404.

Turner, R. J. and Patricia Roszell. 1994. "Psychosocial resources and the stress process." Pp. 179-210 in *Stress and mental health*." Psychosocial resources and the stress process."Springer.

Turiano, N. A., L. Pitzer, C. Armour, A. Karlamangla, C. D. Ryff and D. K. Mroczek. 2012. "Personality Trait Level and Change as Predictors of Health Outcomes: Findings from a National Study of Americans (MIDUS)." *The Journals of Gerontology.Series B, Psychological Sciences and Social Sciences* 67(1):4-12. doi: 10.1093/geronb/gbr072 [doi].

van de Mheen, H., K. Stronks, C. W. Looman and J. P. Mackenbach. 1998. "Does Childhood Socioeconomic Status Influence Adult Health through Behavioural Factors?" *International Journal of Epidemiology* 27(3):431-437. Retrieved 8/18/2014 3:51:33 PM.

Van Liew, Julia R. 2013. "Locus of Control." Pp. 1171-1171 in *Encyclopedia of Behavioral Medicine*. Springer.

Wheaton, Blair. 2010. "The stress process as a successful paradigm." Pp. 231-252 in *Advances in the Conceptualization of the Stress Process.* Springer.

Wallerstein, Nina. 2002. "Empowerment to Reduce Health Disparities." *Scandinavian Journal of Public Health* 30(59): 72-77.

Wolinsky, Fredric D. and Timothy E. Stump. 1996. "Age and the Sense of Control among Older Adults." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 51(4):S217-S220.

Wolinsky, F. D., K. W. Wyrwich, A. N. Babu, K. Kroenke and W. M. Tierney. 2003. "Age, Aging, and the Sense of Control among Older Adults: A Longitudinal Reconsideration." *The Journals of Gerontology.Series B, Psychological Sciences and Social Sciences* 58(4):S212-20.

Wrosch, Carsten, Jutta Heckhausen and Margie E. Lachman. 2006. "Goal Management Across Adulthood and Old Age: The Adaptive Value of Primary and Secondary Control.".

Wrosch, Carsten, Richard Schulz, Gregory E. Miller, Sonia Lupien and Erin Dunne. 2007. "Physical Health Problems, Depressive Mood, and Cortisol Secretion in Old Age: Buffer Effects of Health Engagement Control Strategies." *Health Psychology* 26(3):341.

Yang, Y. C., M. K. McClintock, M. Kozloski and T. Li. 2013. "Social Isolation and Adult Mortality: The Role of Chronic Inflammation and Sex Differences." *Journal of Health and Social Behavior* 54(2):183-203. doi: 10.1177/0022146513485244 [doi].

Yang, Yang C., Kristen Schorpp and Kathleen M. Harris. 2014. "Social Support, Social Strain and Inflammation: Evidence from a National Longitudinal Study of US Adults." *Social Science & Medicine* 107:124-135.