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THE IMPACT OF ATTRIBUTING PAIN TO AGING ON CHRONIC PAIN-RELATED OUTCOMES, PHYSICAL ACTIVITY, AND SEDENTARY TIME

by

Matthew Schumann, M.A.

A dissertation

Submitted in partial fulfillment

of the requirements for the degree of

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To the Graduate Faculty:

The members of the committee appointed to examine the dissertation of Matthew Schumann, M.A. find it satisfactory and recommend that it be accepted.

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February 18, 2016

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RE: regarding study number IRB-FY2016-261 : Health, Pain, and Aging 2016

Dear Mr. Schumann:

I have reviewed your request for expedited approval of the new study listed above. This is to confirm that I have approved your application.

Notify the HSC of any adverse events. Serious, unexpected adverse events must be reported in writing within 10 business days.

You may conduct your study as described in your application effective immediately. The study is subject to renewal on or before Feb 17, 2017, unless closed before that date.

Please note that any changes to the study as approved must be promptly reported and approved. Some changes may be approved by expedited review; others require full board review. Contact Tom Bailey (208-282-2179; email humsubj@isu.edu) if you have any questions or require further information.

Sincerely,

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ABSTRACT

The belief that "aging causes pain" is a common attribution endorsed by individuals across the lifespan. In line with Attributional Theory (Weiner, 1985), causal attributions regarding pain and aging could potentially immobilize motivation to improve one's health (Stewart, Chipperfield, Perry, & Weiner, 2012). Endorsing age-related stigmas and stereotypes such as "aging causes illness" has been shown to negatively affect potentially modifiable lifestyle behaviors that can improve functioning (Stewart et al., 2012). Specifically, extant research has demonstrated that this self-directed age stereotype is associated with less health behaviors (e.g., physical activity) and shortened longevity (Stewart et al., 2012; Gagliese, 2009; Miaskowski, 2000). However, current research has not investigated the impact of endorsing the age-related stereotype "aging causes pain." The current study aimed to examine ways in which this belief impacts pain interference, overall health status, and reports of physical activity and sedentary time using a novel seven-item measure, *Aging Causes Illness – Pain* (ACI-P). The sample consisted of 370 participants over the age of 45 who responded to either a mail-out or online questionnaire. Results indicated the ACI-P was an internally consistent measure that yielded a single "aging causes pain" factor. Moreover, individuals with chronic pain reported significantly higher ACI-P scores (i.e., holding the belief that aging causes pain to a greater extent) than individuals who did not endorse chronic pain. Higher ACI-P scores and chronic pain also significantly predicted pain interference for individuals with and without chronic pain, and pain catastrophizing significantly mediated this relationship. Age and sex were not significant moderators. While individuals with

chronic pain reported more health conditions, individuals with high ACI-P scores did not endorse significantly more health conditions compared to individuals with low ACI-P scores. Further, individuals with high and low ACI-P scores did not significantly differ with regard to physical activity and sedentary time. These findings suggest that endorsing the belief that aging causes pain is associated with the cognitive aspects of the "vicious cycle of pain" and not the biological or physical aspects of the pain experience. Implications of the potential clinical and empirical utility of the findings are discussed.

CHAPTER I

INTRODUCTION AND REVIEW OF THE LITERATURE

Introduction

Chronic pain is a biopsychosocial experience that is reported by approximately 116 million American adults per year (Jensen & Turk, 2014). Chronic pain has been consistently shown to impact physical and psychological well-being (e.g., quality of life, sleep, physical activity, depression, anxiety, etc.; Molton & Terrill, 2014). From a larger socioeconomic perspective, chronic pain has an annual economic cost in the United States between $560 and $635 billion dollars (Gatchel, McGeary, McGeary, & Lippe, 2014). Despite the prevalence of chronic pain, interventions remain inadequate or only provide modest improvements in quality of life (Turk, Wilson, & Cahana, 2011). Given the impact of chronic pain on an idiographic and nomothetic level, primary and secondary prevention is necessary to decrease the interference that chronic pain causes.

The purpose of this paper is to investigate how self-directed stereotypes toward aging ("aging causes pain") may exacerbate the pain experience and foil chronic pain prevention efforts. In order to do so, this paper will determine how self-directed stereotypes impact pain interference, physical activity, and sedentary behavior. Given the complexity of chronic pain in research, this paper will begin with a brief overview of pain, pain coping strategies, and methods to assess pain. The paper will continue with the rationale behind focusing on cognitive aspects regarding chronic pain framed in a historical perspective of pain research and various theorized pain models. The paper

closes with the rationale, hypotheses, methods, results, and a discussion of the implications of findings of this study.

Brief Overview of Pain

Pain is a complex, unpleasant sensory experience that is associated with biological, psychological, and social factors that may mitigate or exacerbate the experience (Gatchel, McGeary, McGeary, & Lippe, 2014). The term pain is broadly used to discuss a variety of different types of unpleasant experiences that are biological, psychological, and social in nature. For example, biological or somatosensory experiences of pain occur through nociception within the nervous system encoding and processing of stimuli causing harm to the body (Dubin & Patapoutian, 2010). Psychological pain is commonly thought to be the combination of nociception, perception of pain, and suffering (Caudill, 2009). With psychological pain, cognitive components such as pain beliefs, coping strategies, and catastrophizing appraisals of stress and pain impact functioning as an individual attempts to make sense of the pain experience (Turk & Okifuji, 2002). Social pain is caused by events such as the experience of empathy, isolation, rejection, bullying, romantic break-ups, or grief over the loss of a loved one. While social pain has been shown to activate similar areas within the central nervous system such as the posterior insular cortex and the secondary somatosensory cortex (Novembre, Zanon, & Silani, 2014), social pain is not the result of tissue damage or injury whereas physiological and psychological pain typically are.

Not only are there biopsychosocial distinctions in the pain experience, pain can also be classified on a temporal basis. This classification is primarily categorized between

short-term (acute) and long-term (chronic) experiences of pain. The International Association for the Study of Pain (IASP, 2014) differentiates acute and chronic pain using guidelines based on onset as well as temporality. Acute pain is typically elicited by an injury and activation of pain receptions with a short course and remission after the tissue heals. One can think of acute pain as the pain experience associated with burning a hand on a stove, falling and scraping a knee, breaking a limb, or any other tissue damage or injury. In contrast, chronic pain is persistent or recurrent pain with a duration of the pain experience lasting longer than the expected healing period. Chronic pain is typically worsened by factors distal to the cause of pain (e.g., cognitive interpretations of pain and lack of social support), and not explained by the underlying pathology (Jensen & Turk, 2014). Due to causes of chronic pain that are unknown or an individual is unaware of, research criteria for defining chronic pain has been inconsistent or subjective (IASP, 2003). As a means to improve the study of chronic pain, the IASP (2014) provides a widely used definition of chronic pain. This definition classifies chronic pain as pain without apparent biological value (i.e., not a nociceptive signal of structural damage) that has persisted beyond the normal healing – approximately three months longer than the normal expected healing time.

Chronic pain is often categorized further into cancer-related and non-cancer pain. Cancer-related pain consists of persistent pain associated with tissue damage (e.g., tumor invasion of tissue, compression of nerves, organ obstruction) or painful diagnostic procedures or treatments (e.g., chemotherapy or radiation therapy) as a result of cancer (Lucas, 2006). Within research, this type of pain is typically differentiated due to the

known cause of pain and course of available treatment. Chronic non-cancer pain (CNCP) typically describes individuals with pain not closely associated with the extent or presence of injury and pathology and is persistent for months to years (Portenoy, Payne, & Passik, 2005).

CNCP consists of a variety of pain sites and pain conditions such as somatic pain, neuropathic pain, and visceral pain. Somatic pain is nociception associated with a location on the body surface or musculoskeletal tissues. The most common somatic chronic pain conditions include lower back pain, myofascial pain, osteoarthritis, and central pain following spinal cord injury or stroke (Lucas, 2006). Visceral pain is associated with pain in the chest, abdomen or pelvic areas. Common chronic visceral pain conditions include non-cardiac chest pain, diabetes pain, abdominal wall pain, ulcer or irritable bowel syndrome, and bladder pain (Labus et al., 2015). With neuropathic chronic pain, the nerve fibers themselves might be damaged, dysfunctional, or injured. This type of pain may be localized or widespread. Common chronic neuropathic pain conditions include fibromyalgia, post-operative pain, multiple sclerosis, phantom limb pain, and complex regional pain syndrome (Humble, Dalton, & Li, 2015). Given the heterogeneity of types of CNCP and the variety of factors that influence the chronic pain experience, this paper will utilize the IASP (2014) definitions of chronic pain without a specific focus on one particular type of CNCP.

Coping with Chronic Pain

Symptoms of pain and the chronicity of the pain experience can be exacerbated or mitigated by the ways in which an individual copes with or confronts pain (Ehde,

Dillworth, & Turner, 2014). This is a critical reason why the assessment of pain acknowledges the inextricable connection between coping strategies and the pain experience. The repertoire of cognitions and behaviors an individual employs to cope with or confront pain are called coping strategies. The most frequently used classification systems of coping strategies is the distinction between *active* and *passive* strategies for coping with chronic pain (Brown & Nicassio, 1987; Riley & Robinson, 1997; Comeche Moreno, Díaz García, Vallejo Pareja, 1999; Kraaimaat & Evers, 2003; Jensen et al., 2011).

Active strategies are conceptualized as strategies that individuals with chronic pain engage in that assume responsibility of the pain experience and ensure that an individual takes instrumental actions to manage their pain. Examples of active coping strategies include positive self-statements (e.g., "I'm going to fine," "This too shall pass"), distracting oneself from the pain, seeking medical attention, and pacing of physical activity. These strategies are considered adaptive and have been shown to be positively associated with measures of quality of life and decreased pain interference (Jensen et al., 2011). In contrast, passive strategies are conceptualized as strategies that an individual engages in to avoid rather than address the pain experience, displace of responsibility for the actions to be taken to address the pain, situate the control of pain to external sources and often are associated with feelings of hopelessness. Examples of passive coping strategies include negative self-statements regarding the pain experience (i.e., catastrophizing or overestimation of disability), pain behaviors (i.e., guarding or obtaining a posture that avoids pain and distorted or unbalanced movement), sedentary

behavior, withdrawal, and isolation. Passive coping strategies have consistently been found to be associated with negative pain outcomes (Jensen et al., 2011).

It is important to note that there is variability in the combination of active and passive coping strategies an individual can utilize. For this reason, self-report measures have been constructed to determine the extent to which individuals utilize different cognitive and behavioral coping strategies. One of the most widely used measures of coping strategies, the Coping Strategies Questionnaire – Revised (CSQ-R; Riley & Robinson, 1997), has an established six-factor model to determine the extent which an individual utilizes adaptive and maladaptive pain-coping strategies. These factors include Distraction, Coping Self-Statements, Ignoring Pain, Distancing from the Pain, Praying, and Catastrophizing. Subsequent research has determined that individuals who score high on the catastrophizing scale and low on coping self-statements typically have poor adjustment to chronic pain (Thorn, 2004). Conversely, individuals who endorse coping self-statements and ignoring pain tend to have better adjustment (Riley, Robinson, & Geisser, 1999; Jensen et al., 2011). These results have been found in samples of heterogeneous chronic pain, fibromyalgia, and chronic lower back pain (Stewart, Harvey, & Evans, 2001; Goldenberg, Burckhardt, & Crofford, 2004; Riley, Robinson, & Geisser, 1999).

In summary, when assessing chronic pain, it is important for health professionals to consider the way in which an individual reacts to their pain experience. Research has consistently shown that the way in which a person copes with chronic pain impacts the level of adjustment and is predictive of positive or negative outcomes. Specifically,

individuals who take responsibility and the necessary actions to alter their pain experience tend to do so in ways that improve chronic pain symptomatology. Those who take a more passive stance and engage in less positive self-statements tend to adjust poorly to their chronic pain.

Assessing Pain

Difficulties with assessing chronic pain lie in the subjective nature of the experience (Jensen & Karoly, 2001; Tait & Chibnall, 2014; IASP, 2014). From an empirical standpoint, the assessment of pain is complicated by the lack of objective tools. While neuroimaging technology has been successful in associating neural activity to the pain experience (Davis, Racine, & Collett, 2012), it is not feasible to apply these tools to a clinical setting (Tait & Chibnall, 2014). Further, assessing the causes of pain (e.g., childbirth, kidney stones, post-surgical pain, or injury) yield inconsistent or non-significant associations to pain intensity (Beattie & Meyers, 1998), and individuals often report high levels of pain in the presence or absence of diagnostic medical findings such as x-ray images of spinal injuries in individuals with chronic back pain (Rhodes et al., 1999). Aside from the lack of objective evidence, research has indicated that the interpretation of pain is the combination of one's expectations (i.e., thoughts about pain and past experiences of pain) and the actual nociceptive sensory experience (i.e., an objective measurement of neural activity; Brown et al., 2008). Thus, compared to other factors relevant to the assessment of chronic pain, cognitive factors are among the most critical as pain is a perceptual phenomenon (Comeche Moreno, Díaz García, & Vallejo Pareja, 1999).

The most common assessment of pain is self-reported categorizations or ratings of pain using visual analog scales (VAS, Jensen & Karoly, 2001; Tait & Chibnall, 2014). These scales typically provide numerical rating between 0 ("no pain") and 10 ("worst possible pain") and to address issues with language proficiency or cognitive impairments, these scales commonly include pictures associated with each numerical rating (e.g., smiling face to frowning and crying face, see Figure 1). However, this means that assessing pain is limited by its unidimensionality and uncertainty regarding magnification of symptoms (Tait & Chibnall, 2014). Specifically, VAS for pain are simply a rating of an individual's pain experience compared to other pain experiences they have had, and common experiences among individuals must be inferred.

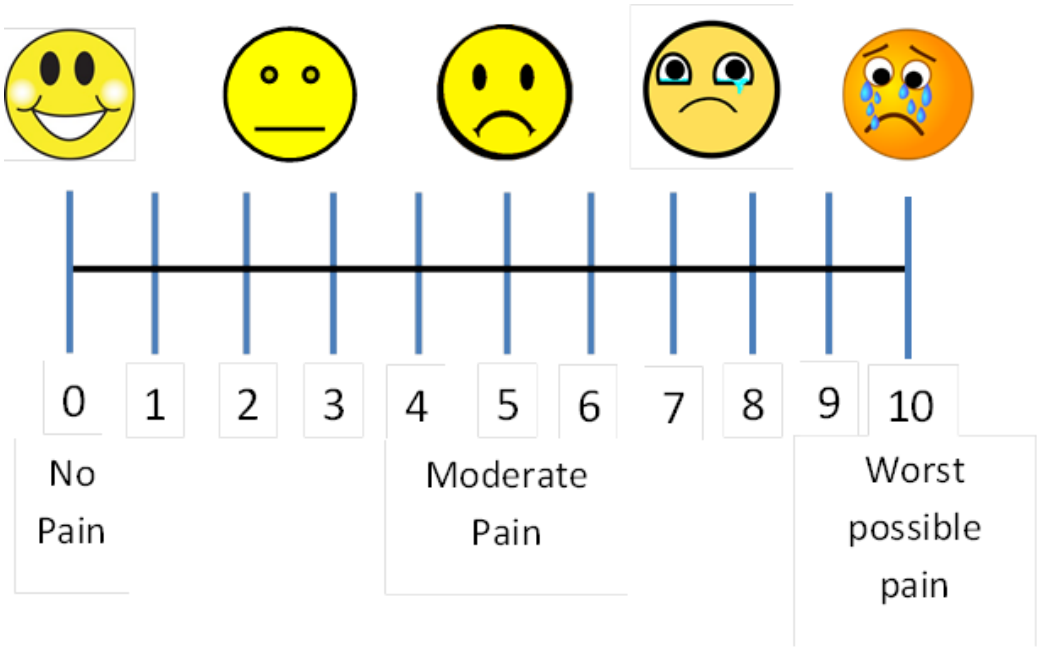

Figure 1. Example of a common self-report visual analog scale (VAS) for pain.

As discussed above, the experience of pain is multidimensional. To address the limitations of a unidimensional measure such as the VAS, research has indicated that a

multidimensional approach to assessment is critical, especially in chronic pain rehabilitation (Hooten et al., 2013). A multidimensional approach consists of questions regarding the chronicity and severity of pain, location/distribution of pain, etiology, and the mechanism of injury (as to provide insight into possible experiences of psychological trauma). It is also important to assess factors that contribute to pain or other associated factors such as access to treatment, medical and mental health conditions, and social support. Finally, and perhaps most importantly, one should determine the quality of life (i.e., general well-being and functioning) of an individual experiencing persistent pain to determine the level of interference that chronic pain is causing. Commonly used measures of pain that utilize a multidimensional approach that focuses on pain interference in daily activities include the West Haven-Yale Multidimensional Pain Inventory **(**WHYMPI; Kerns, Turk, & Rudy, 1985) and the Brief Pain Inventory (BPI, Cleeland & Ryan, 1994). Other measures focusing on the cognitive aspects, beliefs, and strategies to cope with pain include the Survey of Pain Attitudes-Brief Version (SOPA-B, Tait & Chibnall, 1997**),** The Chronic Pain Acceptance Questionnaire (CPAQ; McCracken, Vowles, Eccleston, 2004), and Coping Strategies Questionnaire -- Revised (CSQ-R; Riley & Robinson, 1997).

Taken together, the measurement of pain is an imperfect process. Rather than focusing on the intensity, onset, location, and neural activity associated with chronic pain alone, it is important that researchers utilize multiple forms of assessment that focus on cognitive appraisals or beliefs about pain, how an individual copes with pain, pain interference, and quality of life. As is the case with the conceptualization of the pain

experience, the measurement of pain must consider the interactions between biological, psychological, and social factors that exacerbate the experience. In order to understand the misconceptions and difficulties in measuring chronic pain, it is important to first understand theories regarding chronic pain from a historical perspective.

Theoretical Models of Chronic Pain: A Historical Perspective

As a means to provide a concise description of the chronic pain experience, many physiological and psychological chronic pain models have been described over the past three hundred years (for a review see Jensen & Turk, 2014; Thorn, 2004). Since the time of Rene Descartes and his establishment of a pain pathway to the brain, models of pain and pain treatment were primarily focused on biomedical interventions to decrease the intensity of acute pain (Melzack, 1993). The "biomedical model" posits that severity of injury and intensity of nociceptive signal from the location of injury is associated with greater pain severity (Engel, 1977). According to this model, chronic pain is the product of nociception that never ceased or continues to send pain signals, and there should be some objective indictor of nociceptive malfunction. Hypothetically, successful intervention should arise if the pain signal is dulled (e.g., opioid medication or muscle relaxants) or the nerves delivering the pain signals are eliminated or destroyed to cease the nociceptive signal altogether (e.g., nerve lesion or spinal fusion). From this standpoint, in order for pain to be "real" or "justified" it must be quantifiable and viewable outwardly or by x-ray (Thorn, 2004). Due to the lack of nociception associated with chronic pain, individuals experiencing this type of pain are often considered hypochondriacs and "faking" their pain (Jensen & Turk, 2014).

However, medical intervention is not always needed following acute injury with the interpretation of pain serving as a better predictor of pain intensity. The case study of the city of Anzio, Italy by Henry Beecher (1946, 1959) was among the first documented cases of the importance of interpretation of pain toward the pain experience. Beecher (1946, 1959) researched the qualitative experiences between U.S. soldiers and the citizens of Anzio, Italy following a battle that resulted in many military and civilian casualties. He discovered through interviewing both soldiers and civilians that many of the injured citizens of Anzio with minor injuries experienced intense pain that required analgesic medicine. Interestingly, the U.S. soldiers that were severely injured (e.g., limb amputation) often cited their injuries as a means to leave war, and reported less pain and less pain medication use (Beecher, 1946, 1959).

By 1960, researchers began to recognize that the biomedical model was "purely mechanistic and reductionist" (Thorn, 2004, pg. 5). Scientific evidence began to mount demonstrating that the link between the experience of pain and the amount of tissue damage was tenuous at best. For instance, many biomedical treatments for pain were found to lack long-term benefits or introduced iatrogenic risks such as rebound pain (increased pain intensity following the use of opioids) or substance dependence (Chou, 2013; Deyo et al., 2011). Therefore, the biomedical model is inadequate as simply addressing the pain signals is ineffective (Chan & Peng, 2011; Deyo & Mirza, 2009), and lacking acknowledgement to a critical component to the pain experience.

Variability in pain responses to stimuli of varying intensity (e.g., gentle touch to traumatic amputation) prompted Melzack and Wall (1965) to challenge the biomedical

model by investigating the impact of psychological factors such as the perception of pain. The researchers proposed a signal-perception model called the *Gate Control Theory* (Melzack & Wall, 1965). The Gate Control Theory posits that perceptual processes in the brain dynamically interact with the nociceptive input from the body. Specifically, this theory hypothesizes that the amount of nociceptive input (e.g., information indicating damage or threat of further damage from the periphery) that reaches the brain is modulated at the dorsal horn in the spinal cord by afferent and efferent neuron activity. While injury or threat of further damage opens the pain gate, stimulating the nerve fibers associated with the pain signal (e.g., rubbing the location of the injury) sends signals to close the pain gate. Interestingly, top-down activity can also open the gate. For example, past aversive experiences and the amount of attention to pain can influence how open the pain gate would be (Melzack, 1993). Thus, the interaction between the processes that influence how open or closed the pain gate is impacts the intensity of the pain experience. This expanded the biomedical model by demonstrating the dynamic interplay of perception of neuronal activity, and legitimized the role of psychological factors associated with the pain experience.

Perhaps the most influential advance this model provided was the acknowledgement of psychological factors as key role in prolonging the pain experience after tissue damage or injury occurred (Jensen & Turk, 2014). This offered a new way to treat individuals with chronic pain in the form of psychosocial interventions. Psychosocial interventions for chronic pain such as contingency management, cognitive-behavior therapy (CBT), cognitive restructuring, coping skills training, mindfulness-

based stress reduction, and acceptance and commitment therapy (ACT) have flourished over the past 55 years due to the concept of pain being adopted to the biopsychosocial model of illness (Fordyce, Fowler, & DeLateur, 1968; Segal & Lachman, 1972; Beck, 1979; Ellis & Grieger, 1977; Turk, Meichenbaum, & Genest, 1983; Hayes, Follette, & Linehan, 2004; Thorn, 2004; Schmidt et al., 2011; McCracken & Vowles, 2014). The biopsychosocial model, first introduced by Engel (1977), offers an accommodating heuristic that describes pain and disability as a "complex and dynamic interaction among physiological, psychological factors that perpetuate, and even worsen, one another, resulting in chronic and complex pain syndromes" (Gatchel, McGeary, McGeary, & Lippe, 2014, pg. 120). From this perspective, the pain experience is the result of biological (e.g., genetic predisposition, nervous system experience, etc.), psychological (e.g., cognitions, emotional responses, pain behaviors, etc.) and social (e.g., culture, socioeconomic status, interpersonal relationships, social expectancies, etc.) factors dynamically interacting (Gatchel et al., 2007). Research has continued to support the biopsychosocial conceptualization of pain with Burton and colleagues (1995) finding that actual physical pathology accounted for 10% of disability after one year in workers with low back injuries. Further, 59% of the disability was explained by psychosocial variables (e.g., lack of coping strategies, negative affectivity, and fear-avoidance to activity).

A common example of how biopsychosocial factors interact to produce a chronic pain condition could be in the case of an individual with a lower back injury. Consider a person that is working a low-wage job that was injured lifting a heavy object at her/his place of work. This individual has been experiencing anxiety due to fears that she/he will

reinjure her/himself with thoughts that any lifting motion could be overexerting. Further movement is dissuaded and persistent distress regarding fears of re-injury and the presence of pain will inevitably lead to physical deconditioning (i.e., weakening of muscle groups that support the musculoskeletal system). Other social stressors include fear of losing her/his job due to avoidance of activities that require physical mobility and lifting. This psychosocial stress may lead to prescription opioid abuse or abuse of other substances (e.g., alcohol) to decrease the physical pain. It is common for individuals like to this to adopt a "sick role" due to their persistent pain and disability (Wright & Gatchel, 2002). Thus, this individual has relinquished her/his social and occupational responsibilities onto others (e.g., family members, co-workers, etc.). The reaction to this individual's pain by his social support system may enable further avoidance or put strains on interpersonal relationships due to the individual frequently soliciting remediation of her/his pain condition.

In the example above, the acute experience of a lower back injury was influenced by biological factors such as tissue damage, nociception, and physical deconditioning. Psychological factors also contributed to distress and disability through emotions and cognitions regarding the pain experience and future activity. Emotions such as anxiety are the moment-to-moment reactions to nociception, while the cognitions ascribe meaning to the emotional experience (Gatchel et al., 2014). Finally, the individual's social environment affects her/his level of distress and disability by the responses her/his social support system gives to the individual. Other higher-order social factors such as socioeconomic status and financial strain contribute to the stress an individual

experiences and influences the individual's thoughts regarding pain, as well as relying on substances to cope with the pain. Pain-related cognitions can trigger a cascade of other emotional and behavioral responses that amplify the experience of pain. Thus, a "vicious cycle" of pain that includes nociception, pain, distress, and disability continues (Gatchel et al., 2014). Taken together, acute experiences of pain may transition into chronic pain due to biological, behavioral, emotional, and cognitive changes. These changes are a result of vicious pain cycles that reinforce disability and prevent pain resolution (Hart, Martelli, & Zasler, 2000).

Evidence for a Biopsychosocial Model of Pain: Why Psychological Experiences Matter

It is one thing to provide an anecdote regarding the biopsychosocial interaction; it is another to provide evidence for such a model. Indeed, there exists a detailed literature describing the reciprocal interactions between biological, psychological, and social factors. Cognitive and emotional activity such as hypervigilance to pain, catastrophizing (i.e., thinking the worst about the pain experience), causal beliefs and control of pain has been shown to amplify pain signals and alter nervous system activity associated with pain perception (Thorn, 2004). This in turn increases the experience of pain as indicated by ratings of pain intensity and pain interference (Thorn, 2004).

Over the past half a century the Gate Control Theory has been refined to identify an etiological mechanism of chronic pain. The reformulation of the Gate Control Theory, called the "Neuromatrix" model, hypothesizes that the modulation of pain perception is associated with a widespread network of neural loops throughout the central nervous

system (Melzack, 1999; Jensen, 2010). Further, this model suggests that an individual possesses a predisposed sensitivity to pain that can be modified by experience (i.e., cognitive, behavioral and social factors; Melzack, Coderre, Katz, & Vaccarino, 2001). Current neurophysiological models of pain support this model with evidence of widespread, integrated cortical activity during the experience of pain (Apkarian, Baliki, & Geha, 2009). Specifically, the modulation of pain has been associated with the prefrontal cortex (PFC), the anterior cingulate cortex (ACC), insula, somatosensory cortex, the periaqueductal gray, and nucleus accumbens (Baliki et al., 2006; Bonifazi et al., 2006; Apkarian, Hashmi, & Baliki, 2011). Where the somatosensory cortex, periaqueductal gray, and nucleus accumbens are thought to be associated with acute responses to pain (e.g., avoidance of aversive stimuli, motivating movement, etc.), the other regions identified are believed to the associated with chronic experiences of pain due to nervous system changes (Flor, 2014).

Research has demonstrated that cognitive or emotional aspects of pain may be a better predictor of sensitivity to pain. For example, Ploghaus and colleagues (2001) used functional Magnetic Resonance Imaging (fMRI) research to determine that sensory processing of pain is relatively stable over time, while activity in more cognitive or emotional regions (i.e., the PFC, ACC, and insula) increased over the course of the pain experience. Specifically, these regions are associated with the ability to ascribe meaning to external and internal stimuli, attending to stimuli, and emotional responses to the intensity of pain (Price, 2000). Greater sensitivity to pain, whether that is allodynia (nonpainful stimulation such as mild touching resulting in pain) or hyperalgesia (mildly

painful stimuli producing intense pain), has been demonstrated in acute pain experiences and individuals with chronic pain. For example, Coghill, McHaffie, and Yen (2003) found that individuals who reported greater sensitivity to heat pain exposure displayed stronger brain activity in the PFC and ACC compared to those who reported less sensitivity. This pattern holds for individuals with chronic pain as Vachon-Presseau and colleagues (2013) showed greater pain sensitivity and activation in the PFC and ACC during pain exposure compared to individuals without chronic pain.

Central sensitization of the neuromatrix associated with cognitive and emotional factors is hypothesized to be the etiological source of chronic pain (Curatolo, Arendt-Nielson, & Petersen-Felix, 2006). Central sensitization is the modification of the nervous system due to a persistent state of reactivity. Individuals with chronic pain have been shown to have a shift or expansion in the representation of painful and non-painful stimuli in these areas as well as the sensorimotor cortex indicating a general hyperactivity to pain related stimuli **(**Buchgreitz et al., 2008; Richter et al., 2010; Moseley & Flor, 2012). Chronic pain is associated with altered grey matter density and changes in resting state activity in the PFC, ACC, and insula, suggesting long-lasting brain changes in the presence of chronic pain (Flor, 2014). Reduced connectivity, as indicated by decreased activity in tracts associated with descending pain modulation in chronic pain patients compared to non-diagnosed individuals suggests deficient cognitive pain control (Davis & Moayedi, 2013).

Consistent with research that has shown that prolonged exposure to psychosocial stressors increases hypervigilance to adverse stimuli over the lifespan (Danese &

McEwen, 2012), hypothalamus-pituitary-adrenal (HPA) axis activity is also altered in such a way that individuals with chronic pain have increased HPA activity and an increased sensitivity to pain (Wright & Gatchel, 2002; Flor, 2014). Stressful environment as indicated by socioeconomic status results in complex interactions between the nociceptive input and HPA axis as demonstrated by a line of research that has shown that presence of pain interferes with quality of life to a greater extent for individuals whose income is $\leq$ $25,000 and have a high school education or lower (Portenoy, Ugarte, Fuller, & Haas, 2004). Taken together, central sensitization serves as a hypothesized explanation for structural and functional changes within the nervous system that accounts for learning history, cognition, and emotional factors.

While this paper has described ways in which prolonged experiences of stress and pain can result in allodynia and hyperalgesia, exposure to psychological treatments for chronic pain have been shown to decrease hypervigilance toward pain, allodynia, and hyperalgesia (Flor, 2014). For example, Lackner and colleagues (2006) found that CBT techniques such as pain education, introducing cognitive coping strategies (e.g., distraction and thought redirection), and problem solving reduced neural activity in the ACC during pain induction (for the mentioned study visceral distention was used) from baseline to four month follow up as measured with positron emission topography (PET). Additionally, reduced neural activity in the ACC was associated with decreases in pain-related anxiety and worry. Another study by Rainville and colleagues (1997) attempted to selectively alter the emotional experience of chronic pain by using hypnosis. Rainville and colleagues (1997) found that altering the emotional component of pain through

guided meditation and hypnotic suggestions decreased activity in the ACC during pain induction (e.g., hand in "very hot" water) with somatosensory cortex activation remaining unaltered.

CBT has also been shown to increase PFC gray matter in patients with chronic pain. Seminowicz and colleagues (2013) used magnetic resonance imaging (MRI) scans to compare changes before and after an 11-week CBT treatment between 13 mixed chronic pain type individuals and 13 healthy controls. Their results demonstrated that CBT led to significant improvement in pain disability across both groups. Further, the individuals with chronic pain had increased gray matter in the PFC which was associated with increased pain-coping cognitions (e.g., decreasing catastrophizing).

In summary, research has consistently shown the psychological treatments described above have been more effective than no treatment or treatment as usual, and efficacious under varying treatment contexts (e.g., time sensitive, therapist involvement, and treatment outcome expectancies; Jensen & Turk, 2014). Cognitive attributions and interpretations of pain appear to be an important point of intervention with regard to the pain experience. Jensen (2011) identified that many of the current psychological treatments address three core factors: what people think, how people think, and what pain patients do. These factors have shown to be reciprocally related (Turner &Clancy, 1988; Wetherell et al., 2011; Jensen, 2011) as a change in thought content affects thought processes and thus behavior, and the same could be said about behavior affecting cognitive processes and content. The role of each of these components remains unclear,

and comparing each of these factors is important in determining what mechanism of change or improvement is operating (Jensen & Turk, 2014).

Taken together, the studies mentioned above illustrate that there is empirical evidence that cognitions and emotions are capable of producing physiological differences within the nervous system. In the case of chronic pain, the initial physiological response is layered with thoughts about the pain experience and social factors that may contribute to a vicious cycle that perpetuates the pain experience. The encouraging aspect of these findings is that these cycles are modifiable through CBT approaches. Thus, further examination of the cognitive aspects associated with pain (i.e., content and processes) serves as an apt point of intervention and will be the primary focus of this paper as a means to elucidate which aspects of cognitive-behavioral techniques decrease pain disability and improve quality of life. Specifically, the focus will be on how cognitive factors such as attributions toward pain affect the chronic pain experience.

Aging

Aging is a dynamic process defined by organismic changes due to passage of time. These changes are gradual and associated with functional and structural decline over the life span (Yin & Chen, 2005). There are two predominant perspectives that differ in their emphasis regarding the aging process: chronological age (CA) and biological age (BA; Levine, 2013). The CA perspective views aging along a fixed trajectory of inevitable declines that occurs due to the passage of time associated with Earth's orbital path around the Sun (i.e., years) since birth. One can conceptualize CA as the amount of time passed since birth; the number that answers the question, "How old are you?" In

contrast, BA refers to the description of an individual's development based on biomarkers (e.g., a recordable physiological event) and psychological functioning. The primary distinction between CA and BA is the emphasis on physical disposition rather than the passage of time.

The distinction between CA and BA has several important implications. CA does not recognize the influence of biological, psychological, and social factors that influence functional and structural decline. CA attributes the principal cause of chronic conditions (e.g., hypertension, heart disease, and stroke) to the passage of time. While it has been shown that health declines with chronological age, these declines are often associated with other biological processes such as atherosclerosis (Stout, 1990; Strehler, 1977), and have been shown to be attenuated and influenced by volitional behavior. The BA perspective views physiological decline as an age-related process influenced by biopsychosocial factors (Fries, 1980) rather than predominately age-determined. Lifestyle factors such as poor diet, limited physical activity, and tobacco use have consistently been associated with exacerbating serious health conditions and mortality as well as the broader socio-cultural conditions that strongly affect lifestyle such as material and social deprivation (Johnson et al., 2014; Holt-Lunstad, Smith, & Layton, 2010).

Health and Aging Stereotypes. In much of the developed world, CA is emphasized to a greater extent than BA, thus perpetuating the notion that the aging process is largely out of human control (Gorman, 1999). Misconceptions such as these often lead to stereotypic views which in turn can lead to prejudicial attitudes. Such stereotypic views and prejudicial attitudes exist in Western cultures (Rowe & Kahn,

1998). Ageism is a term that was coined to describe these prejudicial attitudes and behaviors toward older people and the aging process (Butler, 1969). Several widespread ageist misconceptions include the belief that older adults are lonely, old age is a dreary time, older adults are incompetent, and aging causes illness (Ory et al., 2003; Rowe & Kahn, 1998; Stewart et al., 2012).

Aging-related stereotypes have broad implications for both treatment seeking as well as services provided, especially for individuals with chronic pain. From a healthcare provider's perspective, uncertainties and ambiguity regarding the pain assessment process (see Assessing Pain section above) foster clinical judgments that are influenced by stereotyping (Tait & Chibnall, 2014). Unlike other types of explicit biases that are commonly disavowed (e.g., racism and sexism), ageist beliefs are commonly endorsed in both tests of explicit and implicit bias. One study investigated health provider bias by having providers view a series of pained expressions, estimate the level of pain the patient experienced (a question without a "correct" answer), and then rate the influence of elements of the picture on their judgments. This study found that age was one of those elements that influenced greater patient pain estimations (Hirsh, Jensen, & Robinson, 2010). These findings suggest that biases related to patient age may be prominent in practitioner decision making about pain assessment and treatment.

Self-perceptions of aging (i.e., beliefs about their age and the aging process) have been shown to be an important correlate of well-being and health across the lifespan (Rothermund & Brandtstädter, 2005). Specifically, individuals that hold more positive self-perceptions of aging of various ages (e.g., younger, middle, and older adulthood) are

likely to rate themselves as having a higher quality of life, better overall health, and expected longevity (e.g., Levy, Slade, & Kasl, 2002; Levy et al., 2006; Uotinen, Rantanen, & Suutama, 2005). Further, research has shown that individuals who hold negative stereotypes regarding aging to a greater extent report more health conditions and possess a lower desired age (Kotter-Grühn & Hess, 2012). Thus, activation of both positive (e.g., "to be old is to be wise") and negative (e.g., "to be old is to be senile") stereotypes influences behavior and health outcomes.

Self-perceptions of aging have also been shown to be manipulated by priming. Interestingly, there are contrasting patterns for activating positive and negative stereotypes separately. Hess and Hinson (2006) conducted a priming study that consisted of activating positive stereotypes to both middle-age and older adults. Their results demonstrated that both age groups reported feeling younger than control groups (e.g., no stereotype activation). Additionally, the researchers showed that being primed with negative age stereotypes led to participants reporting lower memory controllability and greater age-related concerns such as disability (Hess & Hinson, 2006). Mock and Eibhach (2010) conducted a 10-year longitudinal study that supported the established cross-sectional research. These researchers found that older subjective age (i.e., how old an individual feels) was moderated by aging attitudes such that individuals felt pessimism regarding the aging process predicted older subjective age, but favorable aging attitudes did not. Furthermore, negative aging attitudes were associated with greater reported negative aging stereotypes, lower life satisfaction, and higher negative affect (Mock & Eibach, 2011). These findings held when priming middle-aged and older adults compared

to adults in control conditions (Eibach, Mock, & Courtney, 2010). Taken together, holding and acquiring stereotypical views, both positive and negative, is modifiable and may serve as a point of intervention to improve health outcomes and life satisfaction.

While aging-related stereotypes are typically investigated in concert with measures of aging expectations (e.g., Expectations Regarding Aging-12, Sarkisian, Hayes, & Mangione, 2002) or aging satisfaction (e.g., Philadelphia Geriatric center Morale Scale, Lawton, 1975; Liang & Bollen, 1983) there is a paucity of research focusing on specific aging stereotypes. It remains unclear whether self-directed stereotypes such as "pain is to be expected as an individual ages," serves as a socio-cognitive factor that is associated with distress/disability or whether this attribution serves as a buffer of psychological distress by decreasing emotional reactivity (Molton & Terrill, 2014). However, there remains no quantitative evidence to substantiate either notion.

In summary, negative age-related stereotypes (i.e. "to be old is to be in pain") exert influences on physiological and psychosocial mechanisms. The physiological mechanism affects the autonomic nervous system and responses to stress associated with engaging in activities that may potentially lead to pain. The psychosocial mechanisms may operate through self-fulfilling prophecies (i.e., expecting pain leads to pain) and health practices (Levy et al., 2009). When individuals view chronic pain as part of aging, these individuals may consider healthy behaviors futile and no longer see the purpose of health behaviors in addressing their pain conditions. Therefore, these individuals might eat healthy diets less, sleep less, or engage in physical activity less (Levy et al., 2009).

Next, the paper will continue with a discussion of a theoretical model that accounts for the interaction between self-directed aging stereotypes, pain beliefs, and pain interference – Attribution Theory.

Causal Attributions about Pain

Patients hold personal representations about the cause, meaning, and appropriate treatment of their pain condition (Cedraschi et al., 2013). These personal representations, called causal attributions, are beliefs about the pain experience that stem from cognitions regarding pain (e.g., cause of pain, catastrophizing, pain coping, and locus of control), but also can be informed by cultural expectations (Thorn, 2004). There is an established literature that focuses on explanatory thinking and causal attributions (Abramson, Garber, & Seligman, 1980; Weiner, 1985, 1995; Weiner, Perry & Magnusson, 1988), and studies have shown that upwards to 95% of chronic pain patients reported causal attributions (Hiller et al., 2010). Further, older adults who frequently endorse causal attributions about pain tend to report greater pain interference and pain-related difficulties than younger adults (Gagliese & Melzack, 2003).

As a means to assess pain-related self-directed stereotypes such as "aging causes pain," one must evaluate the extent to which an individual attributes the onset of pain to the advanced age. The belief in the self-directed stereotype that aging causes pain can be conceptualized as a causal attribution. Attributions Theory has conceptualized that causal attributions affect cognitive and behavioral responses along three dimensions (Weiner, 1985): Locus of causality, stability, and controllability. When applied to the specific area of chronic pain, locus of causality refers to the cause of pain residing within the person

(e.g., self-blame or other cognitive factors) or outside a person (e.g., traumatic injury). Stability refers to the perceived degree of permanence (i.e., stable) or changeability (i.e., unstable) and modulating expectancies over time of the pain experience. Controllability refers to the cause of pain being volitionally controlled by self or others (i.e., controllable) or the pain cannot be controlled (i.e., uncontrollable). The combination of internal, unstable, and controllable attributions have been shown to facilitate positive coping strategies to improve psychological functioning, illness-associated disability, and help-seeking behavior for individuals with chronic illness and chronic pain (Hiller et al., 2010; Higgins et al., 2015). In contrast, individuals with predominately external, stable, and uncontrollable attributions regarding chronic illness have worse psychological adjustment and more avoidant coping behaviors (Roesch & Weiner, 2001).

The causal attribution of "aging causes pain" can be applied to this model as advancing age may be considered internal, stable, and uncontrollable by self and others (i.e., thinking of this cause as part of the individual that cannot be changed or controlled). This sense of uncontrollability toward an individual's pain may contribute to a sense of learned helplessness, and lead to using less adaptive coping strategies to mitigate or improve the pain experience (Seligman, 1975; Weiner & Litman-Adizes, 1980). In fact, such causal attributions have been shown to serve as a barrier to motivating factors and goal-directed behavior such as diet, exercise and medical treatment (Bennett & Elliot, 2005; Gjorup, Henrickson, Lund, & Stromgard, 1987; Rakowski & Hickey, 1992; Stewart et al., 2012). There remains a paucity of research that has assessed for the role of aging as a causal attribution, and this study intends to address this gap in the research

given the prevalence of the belief that "to be old is to be in pain." This will be discussed further in the Purpose of the Current Study section.

Physical Activity, Sedentary Time, and Pain

As individuals age, a combination of physiological, psychological and social factors determine how individuals react to pain signals. One common result of prolonged pain is avoidance of physical activity and increased sedentary time especially in advanced age (Stubbs et al., 2013). Decreased physical activity is a major public health concern and has been documented as the fourth leading cause of global mortality (World Health Organization [WHO], 2010). The Center for Disease Control and Prevention (CDC, 2011) currently recommends 2.5 hours of moderate to vigorous physical activity (MVPA) a week for adults ages 18 and above. MVPA is defined as engaging in physical activity that requires a moderate amount of effort that noticeably accelerates heart rate (WHO, 2010). Examples of MVPA include walking at a brisk pace (i.e., approximately 4 miles per hour), dancing, gardening, housework and domestic chores, light effort bicycling, and carrying/moving objects between 10 and 40 pounds. Previous research has demonstrated an increased risk of death for individuals who are reporting less than the weekly recommended MVPA (Leitzmann et al., 2007; Lollgen Bockenhoff, & Knapp, 2009). However, for middle-age to older adults any dose of MVPA has been shown to be beneficial in reducing mortality rates. Specifically, mortality was reduced by 22% for individuals engaging in minimal weekly MVPA, 28% for individuals followed the weekly recommendations and 35% for those who exceeded the weekly recommendations (Hupin, et al., 2015).

Physical activity and sedentary time, while often related (Kujala, Kaprio, Sarna, & Koskenvuo, 1998), can influence health independently. For example, Koster and colleagues (2012) showed that sedentary time was a significant predictor of mortality while controlling for moderate to vigorous physical activity (MVPA). Sedentary behavior refers to "any waking behavior characterized by an energy expenditure ≤1.5 METs [Metabolic Equivalent of Task] while in a sitting or reclining posture" (Sedentary Behaviour Research Network, 2012, p. 540). Common sedentary behaviors include engaging in screen time while seated/reclining (e.g., television, computer, tablet, phone), reading, and driving. Seated activities that utilize more than 1.5 METs such as bicycling or rowing are not considered sedentary behavior. Sedentary living is associated with higher risk for various chronic health conditions (Tremblay et al., 2011; Frei et al., 2011; Sawatzky, Liu-Ambrose, Miller, & Marra, 2007) such as cancer, diabetes, and cardiovascular disease. Taken together, promoting the increase of physical activity and decreases in sedentary time is crucial for primary prevention.

An important component of the vicious cycle of chronic pain includes physical disability associated with deconditioning (i.e., loss of muscle strength or endurance due to inactivity) which is a common side effect of having too little physical activity and spending too much time being sedentary. It is not uncommon for individuals with chronic pain to initially decrease physical activity and notice improvement in pain level. Following the improvement in pain level, individuals will attempt to increase physical activity, which leads to experiencing an increase in pain levels. Therefore, physical activity becomes associated with pain and further declines in endurance and strength lead

to fear-avoidance of any physically demanding task (see Figure 2). This behavioral cycle may repeat several times leading to decreases in self-efficacy and pain control; perpetuating the negative cognitive and emotional effects of the pain experience.

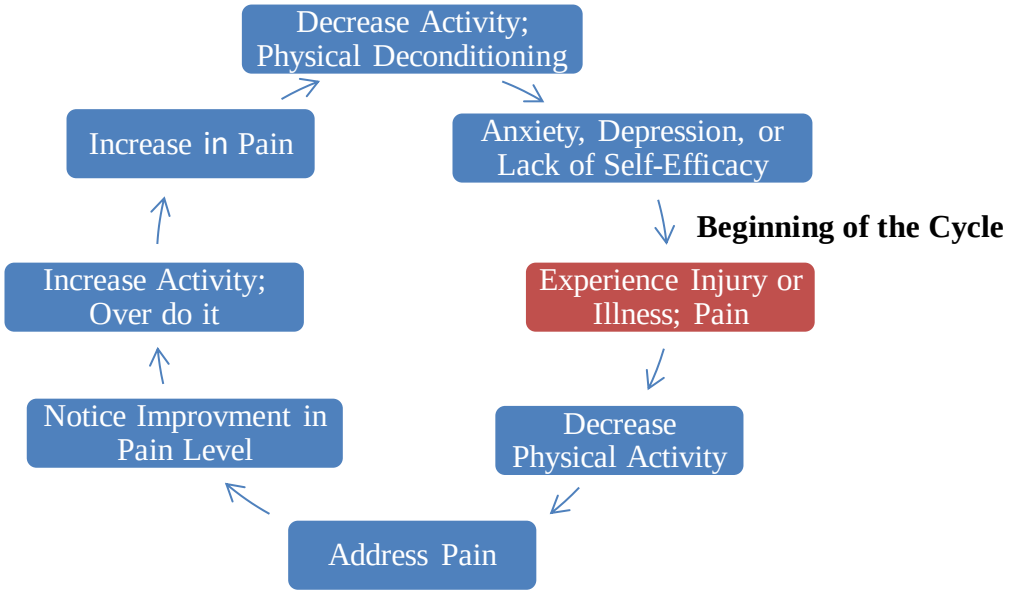

Figure 2. Example behavioral cycle of physical deconditioning and exacerbation of the pain experience.

This cycle often coincides with the Fear-Avoidance Model of chronic pain (Vlaeyen et al., 1995; Vlaeyen & Linton, 2000). This model provides a useful framework to understand how physical activity avoidance is often due to fear of increased pain or bodily harm that is exacerbated by psychosocial factors. Fear-avoidance has been shown to be an critical treatment target for improvement of physical, psychological, and social aspects related to pain (Gatchel et al., 2007; Leeuw et al., 2007). Fear-avoidance models (see Figure 3) suggest that psychological factors such as appraisals and attributions regarding the pain experience impact any movement or activity that may provoke pain.

Individuals that have adaptive attributions or appraisals (e.g., pain is temporary, changeable, and controllable) tend to end the vicious cycle of chronic pain discussed above. However, individuals that have maladaptive attributions or appraisals such as a tendency to catastrophize (i.e., think the worst possible outcome about the pain experience) or overestimate the level of disability become less likely to engage in physical activity.

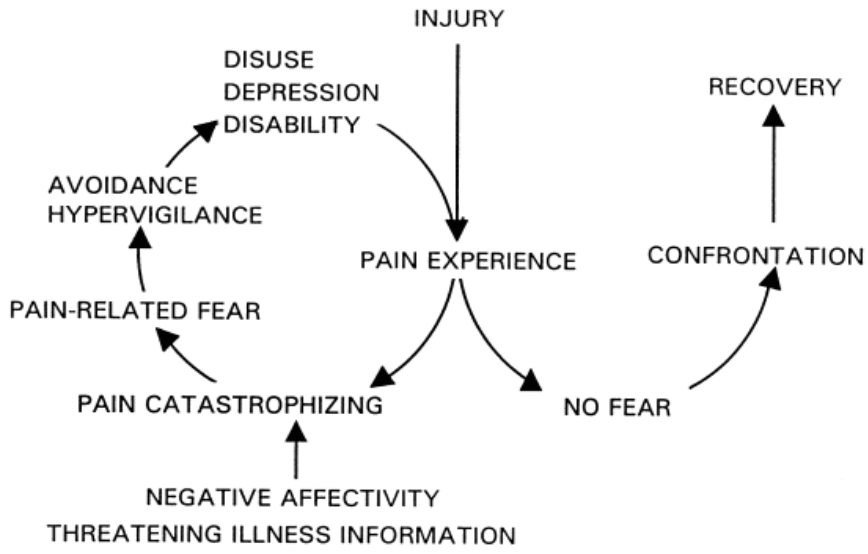

Figure 3. The fear-avoidance model from Vlaeyen & Linton (2000, pg. 329).

As mentioned above, avoidance of physical activity leads to deconditioning and negative emotional outcomes (e.g., anxiety and depression; Ramírez-Maestre, Esteve, López-Martínez, 2014). The set of negative emotional outcomes about the pain perpetuates this cycle by expecting negative events to occur the next time pain is experienced. Perhaps the most emphasized point for individuals with chronic pain attempting to engage in physical activity is to pace their activity (i.e., attempt to be increasingly active over time) and to avoid vigorous activity until an individual's strength

and endurance can compensate for such activity. Pacing and engaging in activities that are not too strenuous have been shown to maintain self-efficacy and avoid negative emotional outcomes (Leveille, Cohen-Mansfield, & Guralnik, 2003). In summary, the combination of psychological factors such as the way an individual ascribes meaning to the pain experience can lead to deconditioning and further avoidance of movement that is perceived to contribute to more pain.

Physical activity has consistently been shown to be positively related to a variety of quality of life measures and is found to reduce symptom impairment associated with chronic pain (Stewart et al., 1994; VanBuskirk et al., 2014). Exercise-based treatments that promote activity over inactivity have been successful in reducing pain intensity, pain interference, and psychological distress (Mannion et al., 2001). Research has also shown that exercise may compliment psychological treatments for chronic pain such as CBT and ACT (VanBuskirk et al., 2014). Interventions that focus on increasing physical activity have also been shown to improve quality of life, physical and mental wellness in non-clinical samples (Warburton, Nicol, & Bredin, 2006). While deconditioning as a mechanism of chronic pain disability has had moderate support in research, some researchers argue that it is the combination of conditioning and flexibility that improves the chronic pain experience (Bousema et al., 2007). For example, Geisser and colleagues (2004) found that pain-related fear was significantly associated with musculoskeletal abnormalities associated with deconditioning and limited flexibility for individuals with chronic musculoskeletal low back pain.

Aside from the effects of deconditioning and limited flexibility on chronic pain-related disability, the intensity and duration of physical activity can contribute to pain interference. Specifically, Heneweer, Vanhees, and Picavet (2009) conducted a study that investigated the risk factors associated with excessive activity and inactivity for individuals with chronic low back pain. The results of their cross-sectional study found that the greatest risk for chronic pain was for those who engaged in sedentary lifestyles and those involved in strenuous physical activities. The researchers demonstrated that the risk for chronic pain was had a U-shaped relation. Interestingly, the researchers also found that women on both ends of the physical activity spectrum have a greater risk for chronic pain (Heneweer, Vanhees, & Picavet, 2009). Therefore, a "virtuous cycle" of physical activity (see Figure 4) can occur with proper attention to psychosocial factors. For example, injury can occur and an individual can take the necessary steps to heal the injury (i.e., treatment seeking, non-opioid analgesics, rest, etc.). By engaging in pacing and moderate levels of exercise or physical activity, an individual will increase aerobic fitness and in turn self-efficacy related to physical activity. Self-efficacy is associated with optimism, and the virtuous cycle of physical activity continues with physical activity being integrated into an individual's daily routine.

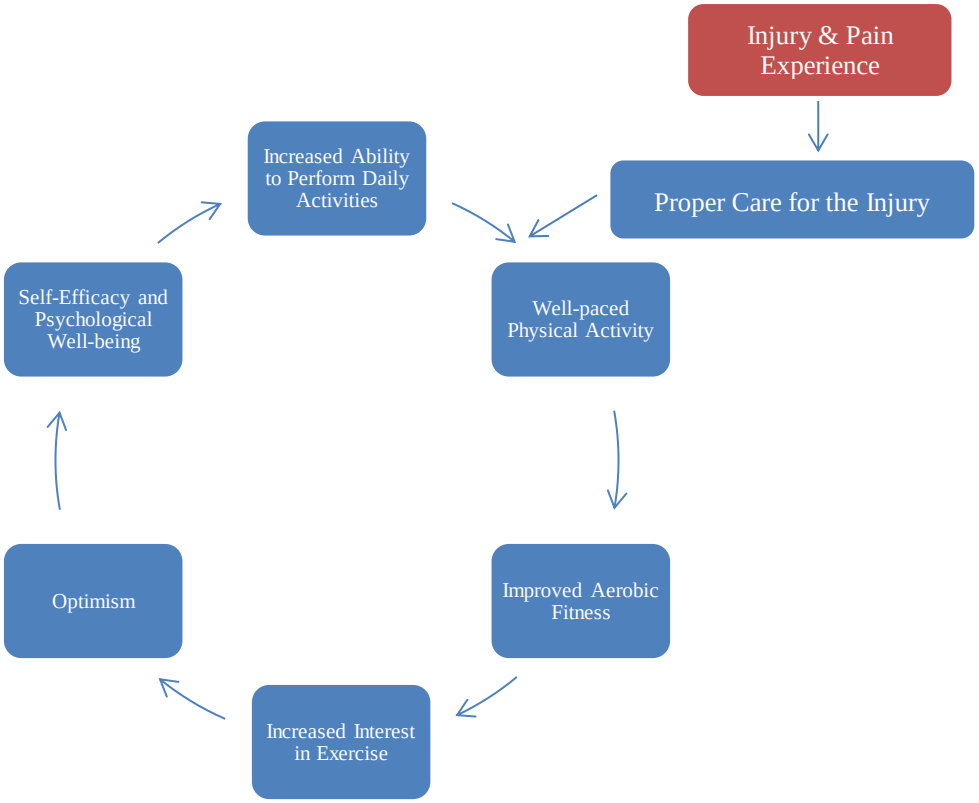

Figure 4. The "virtuous cycle" of physical activity following a pain experience.

The Interaction between Aging, Physical Activity, and Chronic Pain.

As individuals age, physiological factors such as osteoporosis, health complications, and lack of social support contribute to inactivity. With increasing age, walking, yard work, and household chores are among the most common forms of physical activities (Moore et al., 2014). However, by age 75, about one in three men and one and two women engage in no physical activity (WHO, 2010). Research has shown that pain has been one of the main contributors to decreased physical activity and increased functional disability (Bryant et al., 2007). Specifically, Bryant and colleagues (2007) found that pain interference was a significant predictor for physical impairment for older adults over a 22-month span. Fear-avoidance may also contribute to a lack of

physical activity for older adults as fear of injuries due to falls have been shown to contribute to lack of physical activity (Tennstedt et al., 1998).

Another contributor to increased pain interference with age is the loss of muscle mass and bone density. Decreased muscle mass and bone density have been associated with a susceptibility to muscle strains and tears, as well as bone fractures (Layne & Nelson, 1999). Women tend to experience greater losses in muscle mass and bone density compared to men, with significant loss above the age of 55 (National Institute of Health, 2011). Considering that individuals with chronic pain have a tendency to avoid postures that are perceived to lead to pain as well as moving in ways that do not ergonomically distribute their weight, the loss of muscle mass and bone density may interact to perpetuate the pain cycle for older adults.

Research has demonstrated that physical activity not only improves muscle mass, but bone density as well. Jessup and colleagues (2003) revealed that strength and aerobic exercises were effective in significantly improving bone density, muscle strength and self-efficacy among older women between the ages of 65 and 75. Regular MVPA has been shown to improve muscle mass, bone density, and pain symptoms across heterogeneous presentations of chronic pain (Hagen et al., 2002; Nelson et al., 2007). Thus, previous research has established that preventative or rehabilitative actions can be taken to address losses in muscle mass and bone density.

Remaining active may have a dose-dependent effect on successful aging (i.e., physical, psychological, and social satisfaction). Dogra and Stathokostas (2012) investigated the role that physical activity and sedentary behavior serves in successful

aging. The researchers discovered that physical activity and sedentary time independently predict successful aging in middle-aged and older adults such that decreasing sedentary time was significantly associated with successful aging beyond the effects of physical activity. Specifically, middle-aged and older adults that were least sedentary were each 43% more likely to age successfully (Dogra & Stathokostas, 2012). There remains a lack of research investigating how chronic pain may influence this relationship.

Social factors also influence the level of impairment with regard to physical activity due to chronic pain as individuals age. Perceived social support has been shown to be associated with less pain and better physical and psychological well-being (Jensen et al., 2011; Molton & Terrill, 2014), and this is important to consider as an individual ages. Changes in social support networks have been consistently documented in empirical literature with older adults tending to have a few close friends or family members rather than broader networks of support (Huxhold, Fiori, & Windsor, 2013) with these smaller networks being more sensitive to losses or strain (Molton & Terrill, 2014). Research has shown that the extent to which an older adult has an active or inactive support network that is physically active contributes to the amount of physical activity an individual engages in (Resnick et al., 2002). With increasing age, individuals within a given social network have a greater chance of experiencing health problems and disability. Thus, an older adult with chronic pain may view inactivity as the "social norm," and be less motivated to adhere to an exercise regimen.

In summary, biological and psychosocial factors affect the amount of physical activity an individual with chronic pain engages in which in turn contribute to pain

interference. Past research has demonstrated that sedentary behavior and physical activity independently predict physical, psychological, and social well-being. However, there remains a lack of empirical evidence investigating the intersecting relationships between aging (especially comparing middle-aged and older adults), physical activity, sedentary behavior, and chronic pain.

The Current Study: "Aging Causes Pain"

The belief that aging causes pain" is widespread. Research by Sarkisian, Hays, and Mangione (2002) revealed that as many as 87% of a community dwelling sample of older adults endorsed that aches and pains increase with age. While the impact of this belief is complex, further investigation into how the belief is associated with pain-related interference is warranted. In line with the Attributional Theory of Motivation and Emotion (Weiner, 1985) causal attributions regarding pain and aging could potentially produce a state of learned helplessness or immobilize motivation to improve health (Stewart, Chipperfield, Perry, & Weiner, 2012). Holding age-related stigmas and stereotypes such as "aging causes illness" has been shown to negatively affect potentially modifiable lifestyle behaviors that can improve functioning (Stewart et al., 2012). Specifically, extant research has demonstrated that this self-directed age stereotype regarding illness and pain is associated with decreased exercise, less restful sleep, and less probable pain reporting or treatment seeking (Stewart et al., 2012; Gagliese, 2009; Miaskowski, 2000). This may be the cause with regard to the belief that aging causes pain and pain interference.

Another aspect to consider is that the belief that aging causes pain is often endorsed by health professionals as a way to promote patient acceptance of pain and ensure that patients continue to engage in daily tasks (Weiner, 2006; Gagliese, et al., 2009). This strategy on the part of health professionals is corroborated by the finding that normalizing pain may decrease depressed affect (Williamson, 2000), and lead to individuals eliciting less emotional reactivity during sensations of pain (Williamson & Schulz, 1992; 1995). Thus, the belief that aging causes pain may be both adaptive and maladaptive for chronic pain sufferers (Molton & Terrill, 2014). Figure 5 illustrates how negative self-stereotypes regarding aging impact the "vicious cycle" of chronic pain from a fear-avoidance perspective. Conversely, Figure 5 also illustrates how believing that aging causes pain to a lesser extent can contribute to a "virtuous cycle" of pain. However, research has yet to 1) Examine the differences between chronic pain sufferers who do, compared to those who do not, believe that their pain is due to aging, and 2) Simultaneously investigate how holding the belief that aging causes pain can affect adaptive behaviors such as physical activity. The purpose of this study is to address these two issues.

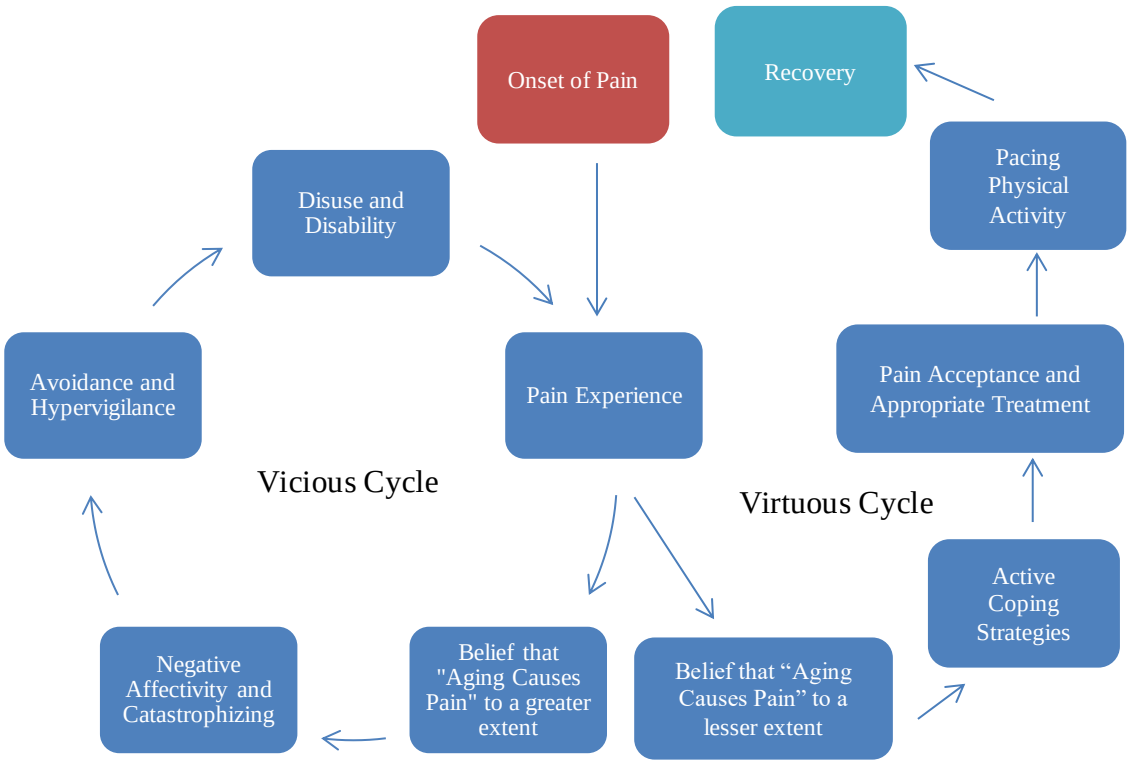

Figure 5. Hypothesized impact of self-directed aging stereotypes as they contribute to "vicious and virtuous cycles" of chronic pain. *

***Figure Note:** While the specific pathways of Figure 5 will not be tested in this study, the aim of this study is to evaluate to what extent aging-related beliefs about pain contribute to vicious and virtuous cycles regarding pain. Based on the review of the literature, the hypotheses reflect significantly worse pain and physical activity outcomes for individuals who endorse the belief that aging causes pain to a greater extent. Conversely, individuals who not attribute their pain to aging to a lesser extent will reported significantly better pain and physical activity outcomes in general.

Given that self-perceptions of aging or aging-related stereotypes have been shown to be associated with negative health outcomes, this study will be the first to this author's knowledge to investigate differences in the prevalence of the self-directed stereotype that

"to be old is to be in pain" between an endorsing chronic pain sample and non-endorsing sample. This study will also attempt to determine differential outcomes (e.g., general health status, pain interference, and pain coping strategies) associated with holding such a belief. Specifically, health status pertains to the number of past or current medical conditions endorsed such as hypertension, heart disease, and diabetes (see Appendix H). Pain interference is a term used to refer to functional disability and barriers to a higher quality of life associated with pain (Wilson, 2014). This common outcome measurement in clinical research focuses on an individual's general activity level, engagement in social and recreational activities, enjoyment of life, and the ability to complete work and daily living tasks (Thomas et al., 2004). Finally, pain coping strategies refers to common ways in which individuals adapt to their pain experience. These strategies can be either adaptive (e.g., acceptance, praying, distraction, and positive self-statements) or maladaptive (e.g. catastrophizing and negative self-statements; Robinson et al., 1997).

Individual differences such as age and sex may be associated with self-directed stereotypes regarding pain. For example, research findings suggest that older adults are less likely to report pain symptoms (Helme & Gibson, 2001). Sex differences have also been found with regard to pain reporting, with females having a greater tendency to report pain than men (Bartley & Fillingim, 2013). Women are also more likely to use passive or maladaptive coping strategies, resulting in poorer daily functioning than men (El-Shormilisy, Strong, & Meridith, 2015). Therefore, it is relevant to explore possible differences in holding a self-directed stereotype regarding pain across different age groups and sex.

Additionally, research is limited in determining how the belief that pain is a natural part of aging and physical activity beliefs differ among individuals with and without chronic pain. Thus, it remains unclear whether the belief that aging causes pain is adaptive or maladaptive. One way to determine the role this self-directed belief holds in chronic pain outcomes is to focus on its relation to an adaptive behavior: physical activity. This justifies further investigation of how the self-directed stereotype of "aging causes pain" impacts the lives of those who hold this belief compared to those that do not.

Specific Aims and Hypotheses for the Present Study are:

Specific Aim 1a) To examine whether the aging-related pain stereotype, aging causes pain is more prevalent in individuals that endorse a chronic pain condition compared to those that do not endorse a pain condition.

Hypothesis 1a) Individuals who endorse chronic pain will endorse that aging causes pain to a significantly greater extent than individuals who do not endorse chronic pain.

Specific Aim 1b) Assess whether individual differences (i.e., age and sex) contribute to attributing pain to aging.

Hypothesis 1b) Aging-related beliefs about pain will be significantly associated with pain interference, and age and sex will moderate this relationship.

Specific Aim 2) To determine how believing aging causes pain can differentially impact health status, level of pain interference, and pain coping strategies for individuals with chronic pain.

Hypothesis 2a) Individuals with high aging-related pain beliefs (ACI-P scores) for who endorsed or did not endorse chronic pain will endorse significantly more health conditions (i.e., poorer health status) compared to individuals with low aging-related pain beliefs who did or did not endorse chronic pain.

Hypothesis 2b) Individuals with high aging-related pain beliefs (ACI-P scores) who endorsed chronic pain will endorse greater pain interference compared to individuals with lower levels of aging-related pain beliefs who did not endorse chronic pain.

Hypothesis 2c) Pain coping strategies will significantly mediate the relationship between aging-related pain beliefs and pain interference (see Figure 6 below).

Specific Aim 3a) Assess whether individuals that attribute pain to aging engage in differential amounts of physical activity.

Hypothesis 3a) Individuals with high aging-related beliefs about pain (ACI-P scores) who endorsed or did not endorse chronic pain will endorse significantly less physical activity compared to individuals with low ACI-P scores who did or did not endorse chronic pain.

Specific Aim 3b) Assess the extent to which individuals that believe that aging causes pain are more sedentary than those who do not hold this aging-related stereotype.

Hypothesis 3b) Individuals with high aging-related beliefs about pain (ACI-P scores) who did or did not endorse chronic pain will endorse significantly more daily sedentary time compared to individuals with low ACI-P scores who did or did not endorse chronic pain.

CHAPTER II

Methods

This chapter provides a review of the study design, sampling strategy, and participants.

Design

This study employed a cross-sectional design as a means to provide an initial understanding of the relationship between the attribution "aging causes illness," pain-related variables, and self-reported physical activity and sedentary time. The advantages and disadvantages of this design will be addressed in the Discussion section.

Sampling. To explore the associations among identified variables in Chapter I, participants were recruited though the national online research recruitment program, Amazon Mechanical Turk (MTurk),as well as from an existing pool of participants enrolled in an ongoing longitudinal study on aging at Idaho State University, *Aging In Idaho*.

Amazon Mechanical Turk (MTurk). MTurk is a crowdsourcing web service that a variety of fields of research (e.g., economics, sociology, and psychology) have used to collect self-report data from "workers" who are compensated for participating in such research (Mason & Suri, 2012). Researchers are called "requesters" and anonymously post surveys that workers can complete for compensation. Requesters provide a title and description of the survey and set the compensation rate. The survey and the workers are assigned identification numbers so anonymity can be kept throughout the research process. There are several advantages to using MTurk. First, online recruitment offers

ease of subject pool access and study feasibility given a constricted timeline. Second, the diversity of the subject pool and low cost of access offers an acceptable supplement to the *Aging in Idaho* sample that can bolster the generalization of results. While MTurk workers tend to be between the ages of 18 and 30 years (Ross, Zaldivar, Irani, & Tomlinson 2010), MTurk demographic information indicates that participants well above the age of 60 are present. Consistent with the expected advantages of utilizing MTurk, data for this study was collected in full in May 2016 with sufficient participants over the age of 45 (see Participants section below). Previous studies on the use of MTurk for cross-sectional self-report research recommend the use of manipulation checks to ensure that the participants understand and are attending to the posted questionnaires (Casler, Bickel, & Hackett, 2013). Consistent with this recommendation, the current study utilized eight forced choice manipulation checks (these are described in the Procedures section and results of these checks including data exclusion are reported in the Participants section below).

Aging in Idaho. The *Aging in Idaho* sample consists of individuals from Southeastern Idaho that have completed previous research focusing on overall health and aging. At the time of initial inclusion in Fall 2013, the sample's age ranged from 45 to 65 years (Aging in Idaho, $N = 427$; 59.1% women; age $M = 55.15$, $SD = 5.79$). Each participant from the Aging in Idaho sample provided consent to be contacted for future research studies.

***Inclusion/Exclusion Criteria*.** Inclusion criteria for this study included individuals who are English-speaking, residing within the United States, and above the

age 45. Participants with and without chronic pain status were eligible for the study. However, for those who did endorse chronic pain, inclusion criteria (consistent with IASP guidelines for defining chronic pain in research; IASP, 2014) for the chronic pain group meant the individuals had reported persistent pain for at least three months (excluding malignancies such as cancer pain), and reported either a past or current chronic pain condition diagnosis. Exclusion criteria consisted of physical disabilities as indicated by WHYMPI response, reporting being unable to engage in physical activity, and failing 25% (or more) of the manipulation checks (for the MTurk sample).

Power Analysis

In order to estimate the necessary sample size, an a priori power analysis was conducted. Given the novel research question, a review of past research focusing on treatment effectiveness, pain appraisal, and aging stereotypes was deemed appropriate to determine established effect sizes and recommended sample sizes to determine ample power for primary analyses.

A meta-analysis of 22 studies focusing on the relative effect for evidenced-based psychological treatments for individuals with chronic pain compared to wait-list and healthy controls on pain interference variables (e.g., quality of life, pain intensity, and depression) found the effect to be approximately $d = .41$ with a mean sample size of 79.42 (SD = 59.50; Hoffman, Papas, Chatkoff, & Kerns, 2007). With regard to threat pain appraisals (i.e., fear of potential future damage), challenge pain appraisal (i.e., opportunity for growth due to pain) and pain outcomes, Jackson, Wang, and Fan (2014) conducted a meta-analysis focusing on the chronic pain literature. Across 59 studies with

a mean sample size of 138.41 (*SD* = 112.01), the researchers found threat pain appraisal-outcome associations and challenge appraisal-outcome associations to be medium in effect size (r = .29, p<.001 and r = -.40, p<.001, respectively). Other studies have looked at attributional style and negative outcomes. In particular, Hu, Zhang, & Yang (2015) found there to be a medium effect size (r = .25) for stable, internal causal beliefs and psychopathology across 86 studies. With respect to aging attributions, it has been established that attributing illness to "old age" predicts a decrease in health maintenance behavior (β = -.24) and increased risk of mortality (β = .24) at two year follow-up utilizing multiple regression analysis (Stewart et al., 2012). The effect was found with a study recruitment of 71 participants.

Taken together, the current study took a conservative approach to determining sample size due to the paucity of research on this specific topic. Past research has utilized an average sample size between 80 and 150 to obtain a medium effect size in the variables the current study is interested in investigating. G*Power analyses corroborated these estimates with a sample size between 55 and 240 necessary to arrive at a medium effect size across the statistical analyses conducted (e.g., multiple regression, ANOVA, and ANCOVA). In an attempt to be fully powered ($1 - \beta$ = .8), an aim was established to recruit 400 total participants across both *Aging in Idaho* and MTurk samples (characteristics of both samples are discussed below). This oversampling approach increases the probability of detecting an effect, and will establish a benchmark for power in future research.

Participants

MTurk Participants. Participants included 373 individuals that responded to the MTurk Human Intelligence Task (HIT) titled "Health, Pain and Aging 2016." Of the 373 that responded, 15 participants withdrew from full completion of the study. Of the remaining 356, 22 participants were excluded due to failing the reliability checks (e.g., admitting they were not taking the survey seriously, incorrectly responding to 25% (or more) of the reliability checks, and/or reporting to be under the age of 45). An additional 42 participants were excluded for identifying as physically disabled/unable to engage in physical activity. The final sample included 294 participants after all exclusions (see Table 1). The average age was 52.94 years (*SD* = 7.05, age ranging from 45 to 79 years). The majority of the sample endorsed chronic pain (58.5%) compared to those who did not endorse experiencing chronic pain (41.5%). The average rating of current pain for all participants (from 0 = no pain to 6 = very intense pain) was 1.60 (*SD* = 1.38). The sample was mostly women (54.4%), married (51%), employed (73.5%) and white (81%). Other ethnicities represented by participants were African American (8.2%), Asian American (4.4%), Latino (3.7%), American Indian (3.1%), and Asian or Pacific Islander (1%). With regard to relationship status, 51% of participants reported being married, 21.8% single, 18.3% divorced or separated, 5.4% in domestic partnership, and 3.4% widowed. Participants reported the highest level of education they completed was a four year college (36.7%) followed by completing graduate school (17.3%), completing some college (16.0%), completing two years of college (15.6%), graduating from high school or completing a GED (9.9%), and some graduate school (4.4%). Other participant

demographics include 9.5% currently or having previously served in the United States military.

***Aging In Idaho* Participants.** Participants were mailed packets including study materials. Of the 200 mailed packets, 78 participants returned completed surveys (39% response rate). This rate was comparable to previous mail-out questionnaire *Aging in Idaho* studies. Two participants were excluded for identifying as physically disabled/unable to engage in physical activity. A total of 76 participants were included in the analyses. The average age was 59.46 years (*SD* = 7.27, age ranging from 47 to 68 years).The majority of the sample endorsed chronic pain (60.5%) compared to those who did not endorse experiencing chronic pain (39.5%). The average rating of current pain for all participants (from 0 = no pain to 6 = very intense pain) was 1.68 (*SD* = 1.43). The sample was mostly women (57.3%), employed (61.3%) and white (97.3%); 2.7% identified as Hispanic/Latino. With regard to relationship status, 80.0% of participants reported being married, 6.6% divorced or separated, 5.3% dating or in domestic partnership, 4.0% widowed, and 3.9% single. Participants reported the highest level of education they completed was a four year college degree (26.7%) followed by completing some college (19.7%), completing high school or GED (16.0%), completing a two years of college degree (13.3%), completing some graduate school and some graduate school (13.3%), completing a graduate degree (10.7%), and completing less than a high school degree (1.3%). Other participant demographics include 10.4% currently or having previously served in the United States military.

Measures

Aging Causes Illness - Pain (adapted from Stewart, Chipperfield, Perry, & Weiner, 2012; Stewart & Levy, 2014). The Aging Causes Illness – Pain (ACI-P) was an adapted scale used for the purposes of this study (see Appendix B). In the original measure (ACI), participants were asked to answer seven items, rating the extent that the illness conditions they have experienced are the result of old age (e.g., "Most people are ill because of old age"). ACI-P was adapted by inserting the word "pain" or "in pain" in place of "ill" or "illness" (e.g., "Most people are in pain because of old age"). Thus, the ACI-P was used to assess the belief that aging causes pain. This measure consists of seven items rated on a five point Likert scale from 1 ("strongly disagree") to 5 ("strongly agree"). Example items include "It is impossible to escape being in pain when you are old" and "Most people are in pain because of old age." The original measure has been shown to have high internal consistency (α = .88) and has been shown to be related to other relevant constructs in the field of health and aging: the Expectations Regarding Aging scale (Sarkisian et al., 2002; Sarkisian, Steers, Hays, & Mangione, 2005) and the External Health Locus of Control scale (Wallston, Wallston, & Devellis, 1978). The ACI has also been shown to predict increased reported health conditions and decreased health lifestyle behaviors among middle-aged adults. The ACI was shown to have a single factor structure. The factor structure and internal consistency of the ACI-P is shown in the results section below.

Past research has implicated the belief that pain is age-normative in pain outcomes (Molton & Terril, 2014), however a specific measure that permits investigation

of the impact that this belief has on pain outcomes does not exist. Most studies that attempt to assess this belief rely on general attitudes about pain control, acceptance, and functional impairment (i.e., Survey of Pain Attitudes, Tait & Chibnall, 1997; Pain Beliefs Questionnaire; Edwards, Pearce, Turner-Stokes, & Jones, 1992), use qualitative interviews to determine if the belief is present (Gagliese, 2009; Miaskowski, 2000), or do not explicitly assess participants' age normative beliefs (Sarkisian et al., 2002). The ACI-P explicitly tests how this belief affects individuals with chronic pain as they age, and allows for a better understanding of how "age normative" beliefs affect chronic pain outcomes. The ACI-P also measures duration of pain symptoms and provides a visual representation adapted from the Brief Pain Inventory (Cleeland & Ryan, 1994) to provide a fuller conceptualization of pain beliefs and chronic pain.

Coping Strategies Questionnaire -- Revised (CSQ-R; Riley & Robinson, 1997). The CSQ-R is a widely used self-report measure of adaptive and maladaptive pain coping strategies. This measure consists of 27-items, and participants are asked to rate the extent to which various cognitive and behavioral coping strategies are typically used on a 7-point Likert scale from 0 ("never do") to 6 ("always do"). The CSQ-R items are grouped into six specific strategies: Distraction, Catastrophizing, Ignoring Pain, Distancing from the Pain, Coping Self-Statements, and Praying. Greater use of the coping strategy assessed is reflected in higher scores. For the purposes of this study, the CSQ-R helps determine whether the belief that aging causes pain is associated with specific adaptive and maladaptive cognitions and behaviors (see Appendix C). The CSQ-R has been shown to have robust internal consistency, predictive validity for several measures

of adjustment including pain severity, depression, state anxiety, and physical functioning, and has sound factor structure with subscale reliability ranging from α = .72 to .86 (Robinson et al., 1997; Riley & Robinson, 1997; Riley, Robinson, & Geisser, 1999). Hastie, Riley, and Fillingim (2004) retained the revised factor structure in a sample of heterogeneous chronic pain with good overall (α = .85) and subscale (α = .83 to .91) reliability.

For this study the internal consistency was good for the Distraction (α = .87), Catastrophizing (α = .89), Ignoring Pain (α = .88), Distancing from the Pain (α = .88) subscales. The Coping Self-Statements subscale (α = .71) yielded acceptable internal consistency, while the Praying subscale yielded excellent internal consistency (α = .91). The overall internal consistency was good (α = .88).

West Haven-Yale Multidimensional Pain Inventory (WHYMPI; Kerns, Turk, & Rudy, 1985). The WHYMPI is a 52-item comprehensive self-report pain assessment that consists of 12 subscales (see Appendix D). The inventory is divided into three parts: The Pain Experience, Perceptions of Social Support, and Engagement in Everyday Activities. For the purposes of this study Part I and Part III were used. Part I (The Pain Experience) consists of questions regarding pain interference in work, social, family/marital functioning, support or concern from others, pain severity, perceived life-control, and affective distress. Part III (Engagement in Everyday Activities) includes subscales related to engagement in household chores, outdoor work, activities away from home, social activities, and general activities. Each question is rated on a 7-point Likert scale from 0 (the absence of...) to 7 (the extreme presence of …). On the Pain

Interference, Pain Severity, and Affective Distress subscales, high scores indicate greater pain-related interference and distress. In contrast, higher scores on the Support, Life-control, and Part III subscales indicate a greater degree of pain-related adjustment in the form of greater social support, more life-control, and greater activity involvement.

The WHYMPI has been demonstrated to be applicable across a variety of pain conditions including low back pain, temporomandibular disorders, headaches and fibromyalgia (Turk & Rudy, 1988; 1990; Turk et al., 1996). Kerns, Turk and Rudy (1985) demonstrated that the internal reliability coefficients of all WHYMPI scales range from Cronbach's $\alpha = .70$ to .90. With regard to test-retest reliability, the WHYMPI scales range from .62 to .91 over a 2-week interval. Of particular relevance for this study, the Interference and General Activity subscales demonstrate good internal consistency (α's between .86 to .90 for Interference and .74 to .78 for General Activity) with two week stability ranging from .85 to .87 for Interference and .80 to .87 for General Activity (Ebert & Kerns, 2010). The WHYMPI Interference Subscale has also shown to be positively correlated with criterion measures of psychological well-being (i.e., anxiety and depression), social support satisfaction, pain severity, and health locus of control (Deisinger et al., 2001). The WHYMPI General Activity Subscale has been associated with functional restoration among chronic pain samples (Turner-Stokes et al., 2003). The WHYMPI Life Control and Affective Distress subscales have been associated with psychological adjustment and functioning post-chronic pain treatment (Nielson, & Jensen, 2004).

For this study the internal consistency was excellent for the WHYMPI Pain Interference (α = .94) and Pain Severity (α = .91) subscales. The WHYMPI General Activity (α = .84) and Perceptions of Social Support subscale yielded good internal consistency. The WHYMPI Life Control (α = .75) and WHYMPI Affective Distress (α = .77) subscale yielded acceptable internal consistency.

The Chronic Pain Acceptance Questionnaire (CPAQ; McCracken, Vowles, Eccleston, 2004). The CPAQ is a 20-item self-report measure of pain acceptance (see Appendix E). The CPAQ consists of two subscales associated with activity engagement (e.g., "It's a great relief to realize that I don't have to change my pain to get on with life") and pain willingness (e.g., "I have to struggle to do things when I have pain"). Individuals rate items on a seven-point Likert scale from 0 ("Never true") to 6 ("Always true."). Items on the activity engagement scale are summed, while the pain willingness subscale is reversed scored and then summed. Higher scores are associated with greater activity engagement and pain willingness. Total scores are derived by adding the sum of the activity engagement and pain willingness subscales with higher scores indicating greater pain acceptance. This measure has acceptable to excellent internal consistency (Cronbach's alpha = .72 to .91; McCracken, Vowles, Eccleston, 2004) and has been associated with measures of avoidance distress and daily functioning (Wicksell, Olsson, & Melin, 2009).

For this study, the internal consistency was good for the CPAQ total score (α = .81). The activity engagement (α = .87) and pain willingness (α = .84) subscales yielded good internal consistency.

Global Physical Activity Questionnaire (World Health Organization, 2005). The GPAQ was developed by the World Health Organization as a measure that recognizes physical activity as a chronic disease risk factor and attempts to assess various aspect of the physical activity construct. The GPAQ consists of 16-items that focus on different behavior domains associated with physical activity. Specifically, participants indicate whether they are active at work, in transport, and in recreational time. Within these domains, questions assess the frequency and duration of vigorous or moderate-to-vigorous physical activity. Each participant is presented with "Show Cards" with examples of vigorous or moderate-to-vigorous activity to provide a visual anchor for this type of behavior (see Appendix F). This measure also contains an item that assess the duration of daily sedentary time. This measure has shown moderate validity when compared to other objective (e.g., accelerometer or pedometer) and subjective measures of physical activity with correlations between r = .40 to .63 (Herrmann et al., 2013; Thuy, 2012). The short-term and long-term (i.e., three months following initial administration) test-retest reliability has been shown to be robust with r =.83 to .96 and r = .53 to .83, respectively (Armstrong & Bull, 2006; Herrmann et al., 2013).

Demographic and General Health Questionnaire. A brief questionnaire was developed for this study for the purposes of assessing participant demographics. Specifically, questions include participant age, sex, education level, income, relationship, and veteran's status (see Appendix G). Participants were also asked general health questions related to height, weight, tobacco and alcohol use, and other preexisting health conditions (e.g., heart disease, diabetes, and stroke; see Appendix H).

The Social Desirability Scale – 17 (SDS-17; Stöber, 2001). Past research has indicated that individuals with chronic pain and a social desirability response bias report less psychological distress, but higher levels of pain severity (Deshields, Tait, Gfeller, & Chibnall, 1995). Therefore, these individuals tend to have a "cry for help" pattern of responding that may over exaggerate physical disability associated with pain. Given the self-report nature of this study, the SDS-17 (Stöber, 2001) was used to control for social desirability response bias. The SDS-17 is an impression management and social desirability measure that consists of 17 statements such as "I always eat healthy" or "I'm always objective in arguments." Respondents must indicate whether each item is a true or false description of themselves. Total scores are summed across items with true scores on items 2, 3, 5, 7, 8, 9, 11, 12, 13, and 14, and false responses on items 1, 5, 6, 10, 14, and 16 worth a score of 1 each (See Appendix I). The SDS-17 has been shown to be psychometrically sound with Cronbach's alpha = .80 and convergent validity with other established impression management scales (e.g., Eysenck Personality Questionnaire and the Marlow-Crowne Scale; Stöber, 2001). The internal consistency of the SDS-17 for this study was poor ($\alpha = .59$). Of note, this measure was not significantly related to the other measures within this study, and thus, not controlled for. Due to the poor internal consistency found, the SDS-17 may have been an inadequate measure of social desirability, and future studies may opt to utilize different social desirability measures for this type of sample.

Procedures

The questionnaires were administered via two methods: A mail-out to the Aging in Idaho sample, and an electronic questionnaire administered through MTurk. For the Aging in Idaho sample, 200 questionnaire packets were prepared. The packets consisted of a personalized cover letter stating appreciation of the participant's prior participation and instructions to complete and return the packet, each of the measures, and a large return envelope with postage. The participants were randomly selected from the original 426 participants who completed the first Aging in Idaho study and consented to participate in future studies.

For the MTurk sample, a project titled "Health, Pain, and Aging 2016" was created on MTurk to recruit participants. On the webpage for this study, participants were provided with a description of the questionnaire content along with a link to the questionnaires. The questionnaires utilized for this study was created and hosted on SurveyMonkey.com. The project was coded to request participants above the age of 45. All participants had to first read and agree to the informed consent before being able to access the questionnaires on SurveyMonkey.com. To ensure that the MTurk workers understood the instructions of each questionnaire and attended to the question prompts, eight reliability checks were included. The first technique consisted of a force choice answer requesting a specific response to a given question (ex. "Please select the number 4). This technique was used within eight administered measures (the demographics questionnaire did not include a reliability check). A final reliability check was a question asking the participant if they took the survey seriously and provided quality answers. The

questionnaire titles were altered to limit the participant's ability to predict what construct the questionnaire was investigating. While the true titles of the questionnaires are found in the Measures section above, the altered titles appear in the Appendices (See Appendices B through I).

For both the Aging in Idaho and MTurk samples, the questionnaires were presented in a uniform order with the social desirability scale first to control for response bias, followed by the ACI-P, CSQ-R, WHYMPI, CPAQ, and GPAQ. The questionnaire concluded with the demographics and general health form. The questions for the MTurk sample were forced choice (i.e., participants were required to answer the question in order to progress in the questionnaire), although (as noted in the informed consent), participants were allowed to discontinue participation at any point without penalty (participants were still compensated even if they did not complete all questionnaires). Issues related to participants' nonresponse to questions and/or attrition was addressed in data cleaning (e.g., all included participants from the MTurk sample had fully completed the questionnaires, while for the *Aging in Idaho* sample multiple imputation was conducted due to 2.6% of possible responses missing and appeared to be missing completely at random). Each MTurk responder received $1.00 for completing the questionnaire (typical payment for MTurk responders is $0.75 per half hour; Mason & Suri, 2012). This was an acceptable amount given the average compensation on MTurk and the length of the questionnaires. The average time of HIT completion was 28 minutes and 43 seconds. The amount compensated was also similar to that of other studies (e.g., Casler, Bickel, & Hackett, 2013). For U.S. participants, research has shown that the level

of compensation does not greatly impact the quality of data. For instance, Litman, Robinson, and Rosenzweig (2015) used instructional manipulation checks to evaluate participants' inattentiveness (e.g., whether they responded differently to two similar questions that were worded slightly different). They found no significant difference between U.S. participants paid $1.00USD and those who were paid a lesser amount (e.g., $0.05USD). For the *Aging in Idaho* sample, participants were entered into a raffle in which 17 participants were randomly selected to receive one of 10 $10.00 gift cards to a large department store or one of seven $15.00 gift cards to a large department store. It is recognized that the form and content of the compensation is different across participants contacted through mail-out vs. MTurk. Nonetheless, it is suggested that the common practices of these two separate recruitment methods dictate different norms of compensation.

Analyses

Data analyses were performed using IBM SPSS Statistics 20 (IBM Corporation, 2011), with statistical significance set at alpha level of $p = 0.05$. First, a series of multiple regression analyses were conducted with demographic variables (age, sex, ethnicity, social desirability, and socioeconomic status) as predictors and study relevant variables (e.g., ACI-P score, WHYMPI-Pain Interference Subscale, CSQ-R subscale scores, GPAQ daily physical activity time, GPAQ daily sedentary time) as the criterion. These analyses were utilized to help determine whether the demographic variables should be added as covariate(s) in subsequent analyses.

To determine if interaction effects exist between the Aging in Idaho and MTurk samples, a series of one-way ANOVAs were conducted with sample group as the independent variable and study relevant variables (e.g., ACI-P score, WHYMPI-Pain Interference score, CSQ-R Catastrophizing subscale, CSQ-R Coping Self-Statements and GPAQ physical activity and sedentary time totals) as the dependent variable. Reliability, validity, and factor analyses were conducted to verify the suitability of the ACI-P for use with individuals with chronic pain. Specifically, the original ACI had a single factor structure; therefore, a confirmatory factor analysis was conducted to ensure that the ACI-P maintains the single factor structure. Correlation analyses were conducted to determine whether the measure is construct valid (i.e., comparing them to CPAQ total scores) as well as pain interference.

To evaluate Hypothesis 1a, an independent samples *t*-test was used to determine if the ACI-P mean score differences between individuals endorsing chronic pain and non-endorsing individuals are significant. Further, a regression model controlling for demographics (as needed) was used to determine to what extent chronic pain status (dummy code 0=non-endorsed chronic pain, 1= endorsed chronic) predicts ACI-P score.

To evaluate Hypothesis 1b, two moderation models were used to determine whether age and/or sex influence the relationship between ACI-P total score and WHYMPI Pain Interference (WHYMPI-PI) subscale score. For both moderator models, ACI-P served as the independent variable (IV) and WHYMPI-PI subscale served as the dependent variable in the direct path. Due to insufficient group sample size for a dichotomous categorization of age into middle and older adulthood, age was maintained

as a continuous variable. Sex was dichotomized between female and male. Moderation models were evaluated with hierarchical multiple regression with IV (ACI-P total score), the grand mean centered moderator interaction term (ACI-P*Age or ACI-P*Sex), and the DV (WHYMPI-PI total). In order to test for a moderated effect, the interaction term entered into the regression model should be statistically significant. A positive value for the age moderation model would imply the higher the age, the higher the ACI-P score. A positive value for the sex moderation model would imply that women endorse higher ACI-P scores than men." Change in variance explained (ΔR^2) was utilized to determine the linear contribution of variables added into the regression model when the covariates are held constant.

For the ANOVA/ANCOVA analyses below, individuals with high and low ACI-P scores were separated based on the observed median score for the total sample. This was done to compare group differences between individuals who hold "aging causes pain" beliefs to higher and lower extents. To test hypothesis 2a and 2b, a 2 (ACI-P total: low = 0, high =1) by 2 (chronic pain: non-endorsed = 0, endorsed =1chronic pain) factorial ANCOVA, controlling for covariates (as needed), were used to determine significant mean differences in number of health conditions (Hypothesis 2a) and pain interference associated with WHYMPI Total scores (Hypothesis 2b). Tukey's HSD were used for post-hoc tests to determine specific group differences.

For Hypothesis 2c, mediation analyses were conducted with ACI-P serving as the independent variable, WHYPI-Pain Interference subscale score serving as the dependent variable, and CSQ-R subscale score as the mediator. The sequence of the regression

models was conducted as follows for both meditation analysis to determine a mediated effect: (1) ACI-P score predicting WHYMPI score (c path), (2) ACI-P score predicting CSQ-R subscale score (alpha path), and (3) CSQ-R subscale score predicting WHYMPI score while controlling for ACI-P score (beta path). To test the significance of the mediated effect the Sobel test was utilized. The mediated effect is equal to the beta weight of the alpha path multiplied by the beta path. A z-test with a critical value of +/- 1.96 was used to determine if the mediated effect is significant at p = .05 level. This is calculated by the formula: $\frac{a*b}{\sqrt{(a^2 s_b^2 + b^2 s_a^2)}}$ (MacKinnon, Fairchild, & Fritz, 2007). The Sobel test is an overly conservative method that assumes the product of coefficients is normally distributed. A strategy that was used to account for these issues is the PRODCLIN (distribution of the product confidence limits for indirect effects) method. This method consists of using the values for the alpha and beta paths and their standard errors to compute a distribution of the product confidence limits (MacKinnon, Fritz, Williams, & Lockwood, 2007). This approach acknowledges the typically non-normal distribution of the mediated effect and can result in more accurate Type I error rates. The strength of mediation analysis lies in its ability to determine causal mechanisms within a three or more variable relationship. In this case, one can determine whether this belief is associated with pain coping skills which in turn lead to less pain interference (See Figure 6).

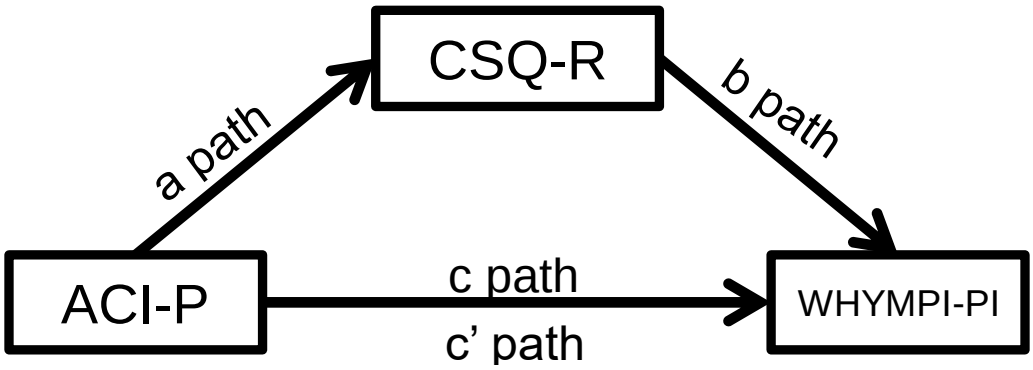

Figure 6. Proposed mediation model to test Hypothesis 2c.

To evaluate Hypothesis 3, five 2 (ACI-P: low = 0, high = 1 ACI-P) by 2 (chronic pain: non-endorsed = 0, endorsed =1) factorial ANCOVAs were conducted, controlling for covariates (as needed). The first four 2 x 2 factorial ANCOVAs were used to determine if there were significant group differences in mean GPAQ physical activity time. This tested for significant differences in mean time spent engaging in total daily physical activity, vigorous work, moderate-to-vigorous work, vigorous recreation, and moderate-to-vigorous recreation activity. Multiple regression models were used to determine if ACI-P scores predict the four domains of physical activity. The final 2 x 2 factorial ANCOVA was used to determine if there were significant group differences in total daily GPAQ sedentary time. This tested for significant differences in mean daily time spent engaging in sedentary behavior. Again, multiple regressions were used to determine if ACI-P predicts daily time spent engaging in sedentary behavior.

It is worth noting that there is an increased potential for alpha inflation given the number of group comparisons conducted in this study. One factor that this study offers to protect against alpha inflation is that each hypothesis was determined *a priori*. The *a priori* nature of the hypotheses provides a way to protect against test-wise Type I errors. Tukey's HSD were used to control for Type I error when making simple comparisons.

This method takes into account the number of means being compared, and adjusts for the total number of tests to make all simple comparisons conducted within this study (Kromrey & La Rocca, 1995). The strength utilizing this method comes from its robustness to violations of normality and homogeneity assumptions, and relative insensitivity to skewness.

CHAPTER III

Results

This chapter provides the results of the analyses conducted to test the hypotheses stated in Chapter I. All analyses were conducted in IBM SPSS Statistics 20. Prior to conducting analyses the data was screened for accuracy of input, missing data, univariate and multivariate outliers, as well as parametric assumptions. All included participants from the MTurk sample had fully completed the questionnaires, while for the *Aging in Idaho* sample multiple imputation was conducted due to 2.6% of possible responses missing and appeared to be missing completely at random. No univariate or multivariate outliers were found for the variables within this study. The total physical activity (Skewness = 3.65, Standard Error of Skewness = 0.12; Kurtosis = 18.01, Standard Error of Kurtosis = 0.25), vigorous physical activity (Skewness = 3.39, Standard Error of Skewness = 0.12; Kurtosis = 13.93, Standard Error of Kurtosis = 0.25), and moderate physical activity (Skewness = 4.44, Standard Error of Skewness = 0.12; Kurtosis = 12.59, Standard Error of Kurtosis = 0.25) scores were significantly positively skewed as a result of a substantial number of zero or low count responses. Therefore, a reciprocal transformation was conducted. Normality was restored for the physical activity measures using this method. Analyses were run on the raw scores and on the transformed data, and no differences were found. Thus, the untransformed scores were used for all analyses for ease of interpretation.

The zero-order correlations among independent variables are found in Table 2. As displayed in Table 2, the SDS-17 was not significantly correlated with any variables of

interest, and thus not added as a covariate for any of the subsequent analyses. The MTurk, Aging *in Idaho*, and total sample were compared on demographic variables (see Table 1), and the descriptive statistics (means and standard deviations) for each subsample and the total sample can be found in Table 3. Univariate tests were conducted to determine if the demographic variables would serve as covariates in the analyses below. All analyses were one-tailed with a *p*-value of .05 for the hypothesized effects, and two-tailed with a *p*-value of .05 for the exploratory analyses.

Summary of Analyses

Factor Structure of Aging Causes Illness-Pain Measure. A confirmatory factor analysis (CFA) was conducted on the seven items of the revised Aging Causes Illness-Pain (ACI-P) measure to determine if the original single factor structure was upheld. The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis, KMO = .86, and all KMO values for individual items were >.81, which is well above the general convention of .60 (Field, 2009). The overall KMO indicates acceptable sampling adequacy of the scale and each item. Bartlett's test of sphericity χ^2 (21) = 1292.51, $p<.001$, indicated that correlations between items were sufficiently large for CFA. An analysis was run to obtain eigenvalues for each factor in the data. Consistent with the original Aging Causes Illness Scale, the analysis yielded a total of seven factors extracted with one factor obtaining an eigenvalue exceeding the convention of 1.00. The extracted factor accounted for 59.44% of the total variance in the original items. The scree plot revealed a clear inflection between the first factor and the other six. Given only one component was extracted, the scale was not rotated. The one factor that had an

eigenvalue exceeding one was labeled the ACI-P factor, and used for all further ACI-P measure analyses. Table 4 shows the eigenvalue and factor loadings of each item on the ACI-P factor. The ACI-P had high reliability with Cronbach's $\alpha = .88$. Table 5 contains the inter-item correlations for the ACI-P measure.

Hypothesis 1a: Individuals who endorse chronic pain will believe that aging causes pain to a significantly greater extent than individuals who do not endorse chronic pain. A regression analysis was conducted to evaluate the extent to which chronic pain status (dummy code 0 = non-endorsed chronic pain, 1 = endorsed chronic pain) predicts ACI-P total score. For the total sample, a significant regression equation was found (F (1,368) = 34.42, p<.001) with an R^2 = .09. Using the unstandardized beta, the participants' predicted ACI-P is equal to 17.28 + 3.01(chronic pain status). Thus, ACI-P total score increased by 3.01 for individuals endorsing chronic pain and chronic pain status was a significant predictor of ACI-P. For the MTurk sample, a significant regression equation was found (F (1,292) = 30.34, p<.001) with an R^2 = .09. Using the unstandardized beta, the participant's' predicted ACI-P is equal to 17.22 + 3.19(chronic pain status). Thus, ACI-P total score increased by 3.19 for individuals endorsing chronic pain and chronic pain status was a significant predictor of ACI-P. For the *Aging in Idaho* sample, a significant regression equation was found (F (1, 74) = 4.74, p<.05) with an R^2 = .06. Using the unstandardized beta, the participant's' predicted ACI-P is equal to 17.40 + 2.40(chronic pain status). Thus ACI-P total score increased by 2.40 for individuals endorsing chronic pain and chronic pain status was a significant predictor of ACI-P. Results of these analyses are found in Table 6. Taken together, hypothesis 1a was

supported as individuals with chronic pain endorsed significantly higher levels of aging-related pain beliefs, and chronic pain status significantly predicted between 6 to 9% of the variance in ACI-P. The means and standard deviations for the non-endorsed and endorsed chronic pain participants for each sample are found in Table 3.

Hypothesis 1b: Aging-related beliefs will be significantly associated with pain interference, and age and sex will moderate this relationship. Four moderation models were used to examine whether age or sex influence the relation between ACI-P total score and WHYMPI Pain Interference (WHYMPI-PI) subscale score. The analyses were conducted on the total sample. For each moderator model, ACI-P total score served as the independent variable and WHYMPI-PI served as the dependent variable in the direct path. An initial regression model supported the possibility of conducting a moderation analysis, as the direct path model was significant (R^2 = .10, F (1, 368) = 39.86, p<.001) with ACI-P total score serving as a significant predictor of WHYMPI-PI (B = .78, SE B = .12, β = .31, p<.001). For the first moderator model, ACI-P total score and the continuous variable, age, and a grand mean centered interaction term (ACI-P*Age given the total sample was included in the analyses) were entered in regression analysis. Age was measured as a continuous variable and remained continuous in the analyses due to 18 total participants reporting to be above the age of 65. Therefore, meaningful differences among age categories could not be made. The model was significant (R^2 = .10, F (1, 366) = 13.86, p<.001). However, contrary to the hypothesis that age would moderate the relationship between ACI-P and WHYMPI-PI, the

interaction term was not a significant predictor of WHYMPI-PI (B = -.01, SE B = .02, β = -.20, p = .641).

For the second moderator model, ACI-P total score, sex (dummy coded 0 = man, 1 = woman), and a grand mean centered interaction term (ACI-P*Sex) were entered in regression analysis. While the model was significant (R^2 = .11, F (1, 366) = 14.77, p<.001), the interaction term was not a significant predictor of WHYMPI-PI (B = .13, SE B = .26, β = .12, p = .641). ACI-P total remained a significant predictor of WHYMPI-PI (B = .73, SE B = .21, β = .29, p < .001).

Further analysis investigating the possible moderating effect of age and sex was conducted on individuals that endorsed chronic pain (i.e., excluding individuals that did not endorse chronic pain). Results of an initial regression found ACI-P total score to be a significant predictor (B = .41, SE B = .16, β = .17, p < .05) of WHYMPI-PI when entered alone, R^2= .03, F (1, 216) = 6.30, p<.05). For the third moderator model, ACI-P total score and the continuous variable, age, and a grand mean centered interaction term (ACI-P*Age) were entered in regression analysis. The model was significant (R^2 = .04, F (3, 214) = 2.88, p<.05). Again, the interaction term was not a significant predictor of WHYMPI-PI (B = -.01, SE B = .02, β = -.15, p = .802). A fourth moderator model was conducted with ACI-P total score, sex (dummy coded 0 = man, 1 = woman), and a grand mean centered interaction term (ACI-P*Sex) were entered in regression analysis. While the model was significant (R^2 = .04, F (3, 214) = 2.97, p<.05), the interaction term was not a significant predictor of WHYMPI-PI (B = .14, SE B = .34, β = .14, p = .686).

In summary, hypothesis 1b was partially supported. While aging-related pain beliefs significantly predicted 10% of the variance in the total sample and 3% of the variance of those who endorsed chronic pain, age and sex did not significantly moderate the relationship between aging-related pain beliefs and pain interference.

Hypothesis 2a: Individuals with high aging-related pain beliefs (ACI-P scores) for who endorsed or did not endorse chronic pain will endorse significantly more health conditions (i.e., poorer health status) compared to individuals with low aging-related pain beliefs who did or did not endorse chronic pain. To test hypothesis 2a, a 2 (ACI-P total: low = 0, high =1) by 2 (chronic pain: non endorsed = 0, endorsed chronic pain =1) factorial ANOVA was conducted. The dependent variable was total number of health conditions endorsed. The analyses were conducted on the total sample. The group means and standard deviations are provided and depicted in Figure 7. The results indicated there was a significant main effect of chronic pain status on health conditions reported, $F(1, 366) = 19.00$, $p<.001$, $\eta^2 = .05$. Tukey HSD revealed individuals with chronic pain ($M = 1.87$, $SD = 1.59$) endorsed significantly higher average number of health conditions than those who did not endorse chronic pain ($M = 0.99$, $SD = 1.73$). There was a non-significant main effect of high/low ACI-P group on number of health conditions, $F(1, 366) = 3.14$, $p = .08$, $\eta^2 = .01$, such that those with low aging-related pain beliefs ($M = 1.28$, $SD = 1.81$) did not significantly differ from those with high beliefs ($M = 1.77$, $SD = 1.54$). There was not a significant interaction effect, $F(1,366) = .42$, $p = .516$, $\eta^2 = .001$.

In summary, while individuals with chronic pain endorsed significantly more health conditions, hypothesis 2a was not supported as individuals with high ACI-P did not significantly differ from those with low ACI-P, and there was no interaction between chronic pain status and ACI-P groups.

Hypothesis 2b: Individuals with high aging-related pain beliefs (ACI-P scores) who endorsed chronic pain will endorse greater pain interference compared to individuals with lower levels of aging-related pain beliefs who did not endorse chronic pain. To test hypothesis 2b, a 2 (ACI-P total: low = 0, high =1) by 2 (chronic pain: non endorsed = 0, endorsed chronic pain =1) factorial ANOVA was conducted. The dependent variable was total WHYMPI-PI subscale score. The analyses were conducted on the total sample. The group means and standard deviations are provided and depicted in Figure 8. There was a significant main effect of chronic pain status on WHYMPI-PI, $F(1, 366) = 122.68$, $p<.001$, $\eta^2 = .25$. Tukey HSD revealed individuals with chronic pain ($M = 20.29$, $SD = 12.00$) endorsed significantly higher pain interference than those who did not endorse chronic pain ($M = 6.47$, $SD = 8.46$). There was a significant main effect of high/low ACI-P on WHYMPI, $F(1, 366) = 15.86$, $p < .001$, $\eta^2 = .04$, such that those with high aging-related pain beliefs ($M = 18.79$, $SD = 12.66$) had significantly greater pain interference compared to low beliefs ($M = 10.94$, $SD = 12.00$). There was not a significant interaction effect, $F(1,366) = .71$, $p = .401$, $\eta^2 = .002$. The results are depicted in Figure 8. Taken together, Hypothesis 2b was supported as individuals with high ACI-P scored significantly higher than those with low ACI-P for both chronic pain and non-chronic pain endorsed groups.

Hypothesis 2c: Pain coping strategies will significantly mediate the relationship between aging-related pain beliefs and pain interference. To evaluate the possible mediating effects pain coping strategies have on the established relationship between aging-related pain beliefs and pain interference (see Hypothesis 1b), a mediation analysis was conducted. The analyses were conducted on the total sample. A series of regression analyses as the second step in mediation were conducted to determine the relationship between the mediation *alpha path* (α). As noted in Table 7, which shows regression analyses of aging-related beliefs predicting pain coping strategies, the only significant mediation A path was between ACI-P total score and CSQ-R subscale scores was that of the Catastrophizing subscale, $R^2 = .04$, $F(1, 368) = 13.41$, $p < .001$, $B = .27$, $SE\ B = .07$, $\beta = .19$, $p < .001$. Four other regression models found that ACI-P did not serve as a significant predictor for the other pain coping strategy subscales. Therefore, one mediation analysis was conducted with ACI-P serving as the independent variable, WHYMPI-Pain Interference subscale serving as the dependent variable, and CSQ-R Catastrophizing subscale score as the mediator. To complete the *beta path* (β) of the mediation analysis, a regression analysis was conducted with the CSQ-R Catastrophizing and ACI-P total scores serving as predictors and WHYMPI-PI score as the outcome. This allows for CSQ-R to serve as an independent variable while controlling for the contribution of ACI-P on WHYMPI-PI. The full mediation model can be found in Figure 9. The significance of the mediated effect was tested by two methods: the Sobel test (Sobel, 1982) and MacKinnon's asymmetric confidence interval (MacKinnon et al., 2007; MacKinnon, 2008). The Sobel *z* statistic is defined as:

$$\frac{\alpha\beta}{\sqrt{(a^2 s_b^2 + b^2 s_a^2)}}$$

The z statistic is compared to critical values of the normal distribution. With the Sobel Test, the mediated effect is significant at $p < .05$ with a critical value of +1.96. A significant mediated effect would suggest that pain catastrophizing significantly mediates the relationship between ACI-P and WHYMPI-PI. However, the Sobel Test assumes the mediate effect is normally distributed, which is often not the case (MacKinnon et al., 2002). Further, the Sobel Test is highly conservative and offers low statistical power. Therefore, the asymmetric confidence interval was used which takes the shape of the distribution of the mediated effect into account when calculating confidence limits. The asymmetric confidence interval is less conservative and offers more accurate Type 1 error compared to the Sobel Test (MacKinnon et al., 2002; MacKinnon, 2008). Thus, the mediated effect was also tested using MacKinnon's asymmetric confidence interval using the PRODCLIN executable (MacKinnon et al., 2007). This program calculates the mediated effect at the 95% confidence interval. If the interval does not include zero, the mediated effect is statistically significant. The results of the Sobel Test indicated pain catastrophizing partially mediates the relationship between aging-related pain beliefs and pain interference ($z = 2.19$, $SE = .09$, $p < .05$). Additionally, the asymmetric confidence interval using PRODCLIN indicated that the indirect effect was significant with a 95% confidence interval ranging from .027 to .400. The effect size found for this model, $\kappa^2 = .088$, is a medium mediation effect size following the convention established by Preacher and Kelley (2011).

Taken together, hypothesis 2c was partially supported as one coping strategy, pain catastrophizing, was a significant mediator of the relationship between aging-related pain beliefs and pain interference. The other pain coping strategies were not significant mediators.

Hypothesis 3a: Individuals with high aging-related beliefs about pain (ACI-P scores) who endorsed or did not endorse chronic pain will endorse significantly less physical activity compared to individuals with low ACI-P scores who did or did not endorse chronic pain. To test hypothesis 3a, a 2 (ACI-P total: low = 0, high =1) by 2 (chronic pain: non endorsed = 0, endorsed chronic pain =1) factorial ANCOVA was conducted controlling for age and sex (univariate tests indicated age and sex significantly differed or predicted total physical activity). Total physical activity was initially tested, and given that there was not a statistical difference in utilizing the reciprocally transformed physical activity measures, the raw physical activity score was used for the following analyses of moderate and vigorous physical activity. The analyses for total, moderate, and vigorous physical activity was conducted on the total sample. The group means and standard deviations are provided and depicted in Figure 10. Total physical activity was computed as average daily vigorous and moderate physical activity in minutes. There was not a significant main effect for ACI-P group on total daily physical activity, $F(1, 364) = .68$, $p = .411$, $\eta^2 = .002$. There was also not a significant main effect of chronic pain group on total physical activity, $F(1, 364) = 1.17$, $p = .280$, $\eta^2 = .003$. The interaction between ACI-P group and chronic pain status was also non-significant, $F(1, 364) = 1.17$, $p = .280$, $\eta^2 = .003$. Group means are depicted in Figure 10.

Looking closer at average daily vigorous physical activity controlling for age and sex (again, univariate tests indicated age and sex significantly differed in or predicted vigorous physical activity, group means and standard deviations are provided and depicted in Figure 11), there was not a significant main effect for ACI-P group on daily vigorous physical activity, $F(1, 364) = .03$, $p = .874$, $\eta^2 = .000$. There was also not a significant main effect of chronic pain group on vigorous physical activity, $F(1, 364) = .002$, $p = .968$, $\eta^2 = .000$). The interaction between ACI-P group and chronic pain status was also non-significant, $F(1, 364) = 2.11$, $p = .147$, $\eta^2 = .006$. The same pattern held for average daily moderate physical activity (age and sex did not significantly differ in or predict daily moderate activity) as a 2 x 2 ANOVA indicated as there was a non-significant main effect for ACI-P group ($F(1, 366) = .70$, $p = .403$, $\eta^2 = .002$), chronic pain status ($F(1, 366) = .76$, $p = .384$, $\eta^2 = .002$), and interaction ($F(1, 366) = .10$, $p = .753$, $\eta^2 = .000$). The group means and standard deviations are provided and depicted in Figure 12.

Given the relatively high number of participants reporting no vigorous physical activity, additional analyses were conducted comparing individuals with any reported vigorous physical activity (minutes ≥ 1, $n = 67$) to individuals who had not engaged in vigorous physical activity (minutes = 0, $n = 303$). To retest hypothesis 3a on only individuals that had endorsed vigorous physical activity, a 2 (ACI-P total: low = 0, high =1) by 2 (chronic pain: non endorsed = 0, endorsed chronic pain =1) factorial ANCOVA was conducted controlling for age and sex, and total physical activity was the dependent variable. There was not a significant main effect for ACI-P group on total daily physical

activity, $F(1, 63) = .37$, $p = .547$, $\eta^2 = .006$, such that individuals with low ACI-P scores ($n = 33$, $M = 26.75$, $SD = 22.53$) did not significantly differ from individuals with high ACI-P scores ($n = 34$, $M = 30.43$, $SD = 19.07$) in total physical activity time. There was also not a significant main effect of chronic pain group on total physical activity, $F(1, 63) = .56$, $p = .457$, $\eta^2 = .009$, such that individuals who did not endorse chronic pain ($n = 27$, $M = 30.66$, $SD = 25.49$) did not significantly differ from individuals who endorsed chronic pain ($n = 40$, $M = 27.24$, $SD = 17.08$) in total physical activity time. The interaction between ACI-P group and chronic pain status was also non-significant, $F(1, 63) = 2.26$, $p = .138$, $\eta^2 = .035$.

A one-way ANOVA was conducted with vigorous activity group as the independent variable and ACI-P total and WHYMPI-Interference subscale as the dependent variables. The analyses revealed a non-significant main effect of vigorous activity group such that individuals with no vigorous activity ($M = 21.99$, $SD = 5.15$) and individuals who endorsed engaging in any vigorous activity ($M = 22.40$, $SD = 4.75$) did not significantly differ in ACI-P total score, $F(1,368) = .362$, $p = .548$, $\eta^2 = .001$. Further, there was not a significant main effect of vigorous activity group on WHYMPI-Interference score such that individuals with no vigorous activity ($M = 14.30$, $SD = 12.94$) and individuals who endorsed engaging in any vigorous activity ($M = 16.02$, $SD = 11.28$) did not significantly differ in ACI-P total score, $F(1,368) = 1.015$, $p = .548$, $\eta^2 = .003$. The vigorous activity groups described above did not significantly differ in the number of participants who endorsed chronic pain, $\chi^2 (370) = .021$, $p = .889$, $V = .007$.

A substantial number of participants (32.4%) reported receiving at or above the American College of Sports Medicine's Physical Activity recommended amount of physical activity per day (30 minutes for individuals above the age of 45, Pescatello, Arena, Riebe, & Thompson, 2014). Additional analyses were conducted comparing those who had endorsed $\geq$ 30 minutes of total physical activity (n = 120) to individuals who had endorsed $\leq$ 30 minutes of total physical activity (n =250) on the dependent measures of ACI-P total score and WHYMPI-Interference score. A one-way ANOVA was conducted. The analyses revealed a non-significant main effect of physical activity group such that individuals with who endorsed less than 30 minutes of physical activity (M = 21.76, SD = 5.17) and individuals who endorsed more than 30 minutes of physical activity (M = 22.71, SD = 4.84) did not significantly differ in ACI-P total score, $F(1,368) = 2.87$, $p = .09$, $\eta^2 = .008$. Further, there was not a significant main effect of physical activity group on WHYMPI-Interference score such that individuals with who endorsed less than 30 minutes of physical activity (M = 14.11, SD = 12.96) and individuals who endorsed more than 30 minutes of physical activity (M = 15.65, SD = 11.99) did not significantly differ in ACI-P total score, $F(1,368) = 1.20$, $p = .275$, $\eta^2 = .003$. Taken together, hypothesis 3a was not supported as there was not a main effect of ACI-P for total, vigorous, and moderate physical activity.

Hypothesis 3b: Individuals with high aging-related beliefs about pain (ACI-P scores) who did or did not endorse chronic pain will endorse significantly more daily sedentary time compared to individuals with low ACI-P scores who did or did not endorse chronic pain. To test hypothesis 3b, a 2 (ACI-P total: low = 0, high =1) by 2

(chronic pain: non endorsed = 0, endorsed chronic pain =1) factorial ANOVA was conducted. Average daily sedentary time was computed and tested. The analyses were conducted on the total sample. The group means and standard deviations are provided and depicted in Figure 13. There was not a significant main effect for ACI-P group on average sedentary time, $F(1, 366) = .04$, $p = .839$, $\eta^2 = .000$. There was also not a significant main effect of chronic pain group on average sedentary time, $F(1, 366) = 3.35$, $p = .068$ $\eta^2 = .01$. The interaction between ACI-P group and chronic pain status was also non-significant, $F(1, 366) = .84$, $p = .360$, $\eta^2 = .002$. Though differences in chronic pain status in daily sedentary time approached significance, hypothesis 3b was not supported as there was not a significant main effect of ACI-P group on sedentary time.

Exploratory Analyses. Exploratory analyses were conducted to determine how other multidimensional pain facets (i.e., general activity, life control, and affective distress) and pain acceptance may be impacted by aging-related pain beliefs and chronic pain status. A series of 2 (ACI-P total: low = 0, high =1) by 2 (chronic pain: non endorsed = 0, endorsed chronic pain =1) factorial ANOVAs were conducted to test the aforementioned impact. The statistical tests conducted for the exploratory analyses were from the total sample.

WHYMPI General Activity. There was significant main effect for ACI-P group on WHYMPI General Activity, $F(1, 366) = 5.65$, $p < .05$, $\eta^2 = .015$ such that individual with high ACI-P scores ($M = 2.82$, SD = 0.91) endorsed significantly higher WHYMPI General Activity than individuals with low ACI-P scores ($M = 2.82$, SD = 0.91). There was not a significant main effect of chronic pain group on WHYMPI General Activity,

$F(1, 366) = .11$, $p = .738$ $\eta^2 = .000$. The interaction between ACI-P group and chronic pain status approached significance, $F(1, 366) = 3.14$, $p = .08$, $\eta^2 = .009$. The group means and standard deviations are provided and depicted in Figure 14.

WHYMPI Life Control. There was not a significant main effect for ACI-P group on WHYMPI Life Control, $F(1, 366) = .001$, $p = .980$, $\eta^2 = .000$. There was a significant main effect of chronic pain group on WHYMPI Life Control, $F(1, 366) = 10.44$, $p < .01$, $\eta^2 = .028$. Tukey HSD revealed individuals with chronic pain ($M = 4.11$, $SD = 1.25$) endorsed significantly lower perceived life control over pain than those who did not endorse chronic pain ($M = 4.56$, $SD = 1.37$). The interaction between ACI-P group and chronic pain status was not significant, $F(1, 366) = 1.52$, $p = .219$, $\eta^2 = .004$. The group means and standard deviations are provided and depicted in Figure 15.

WHYMPI Affective Distress. There was not a significant main effect for ACI-P group on WHYMPI Affective Distress, $F(1, 366) = 1.29$, $p = .257$, $\eta^2 = .004$. There was a significant main effect of chronic pain group on WHYMPI Affective Distress, $F(1, 366) = 37.99$, $p < .001$, $\eta^2 = .094$. Tukey HSD revealed individuals with chronic pain ($M = 2.26$, $SD = 1.28$) endorsed significantly higher affective distress due to pain than those who did not endorse chronic pain ($M = 1.43$, $SD = 1.05$). The interaction between ACI-P group and chronic pain status was not significant, $F(1, 366) = 1.63$, $p = .203$, $\eta^2 = .004$. The group means and standard deviations are provided and depicted in Figure 16.

CPAQ Pain Acceptance Total. The main effect for ACI-P group on Total CPAQ score approached significance, $F(1, 366) = 3.62$, $p = .058$, $\eta^2 = .010$. There was a significant main effect of chronic pain group on Total CPAQ score, $F(1, 366) = 8.70$, p

<.01, η^2 = .023. Tukey HSD revealed individuals with chronic pain (*M* = 66.33, *SD* = 13.60) endorsed significantly higher total pain acceptance (consisting of both Pain Willingness and Activity Engagement subscales) than those who did not endorse chronic pain (*M* = 59.54, *SD* = 15.52). The interaction between ACI-P group and chronic pain status was not significant, *F*(1, 366) = .402, *p* =.527, η^2 = .001. The group means and standard deviations are provided and depicted in Figure 17.

CPAQ Pain Willingness. The main effect for ACI-P group on CPAQ Pain Willingness score was significant, *F*(1, 366) = 17.38, *p* < .001, η^2 = .045 such that individual with high ACI-P scores (*M* = 24.18, SD = 10.02) endorsed significantly higher WHYMPI General Activity than individuals with low ACI-P scores (*M* = 18.53, SD = 9.85). There was a significant main effect of chronic pain group on CPAQ Pain Willingness score, *F*(1, 366) = 10.10, *p* <.01, η^2 = .027. Tukey HSD revealed individuals with chronic pain (*M* = 22.97, *SD* = 9.77) endorsed significantly higher scores of recognition that avoidance and control are often unworkable methods of adapting to pain than those who did not endorse chronic pain (*M* = 18.59, *SD* = 10.54). The interaction between ACI-P group and chronic pain status was not significant, *F*(1, 366) = 2.17, *p* =.142, η^2 = .006. The group means and standard deviations are provided and depicted in Figure 18.

CPAQ Activity Engagement. The main effect for ACI-P group on CPAQ Activity Engagement score was not significant, *F*(1, 366) = .98, *p* = .324, η^2 = .003. There was not a significant main effect of chronic pain group on CPAQ Activity Engagement score, *F*(1, 366) = 1.31, *p* = .254, η^2 = .004. The interaction between ACI-P group and chronic

pain status was significant, $F(1, 366) = 4.01$, $p < .05$, $\eta^2 = .011$ where individuals with chronic pain and low ACI-P beliefs engaged in life activities regardless of pain to the greatest extent ($M = 44.70$, $SD = 10.39$), and the chronic pain/high ACI-P beliefs ($M = 40.72$, $SD = 10.72$) scored similarly in Activity Engagement to individuals who did not endorse chronic pain with high ($M = 41.86$, $SD = 10.40$) and low ($M = 40.51$, $SD = 15.28$) ACI-P beliefs. The group means and standard deviations are provided and depicted in Figure 19.

CHAPTER IV

Discussion

The purpose of the current study was to examine the ways in which stereotypic aging-related beliefs about pain impact an individual's experience of pain, their overall health status, and reports of physical activity and sedentary time, by using a cross-sectional, self-report design. As such, the study had several aims: 1) create a measure that reliably assesses aging-related beliefs about pain, *Aging Causes Illness – Pain* (ACI-P), 2) determine the ACI-P's ability to differentiate individuals who reported chronic pain from those who did not report chronic pain, 3) evaluate the extent the ACI-P predicts negative pain outcomes, specifically pain interference, and differentiates individuals by the amount of health conditions they endorse, 4) examine moderators (i.e., age and sex) and mediators (i.e., pain coping strategies) of the relationship between aging-related beliefs and pain interference, and 5) evaluate the utility of the measure in differentiating groups with regard to average daily physical activity and sedentary time. The findings, implications of the findings, limitations of the study, and directions for future research will be discussed below.

Measuring the belief that "aging causes pain" and its impact on pain outcomes

The results from the confirmatory factor analysis suggest the ACI-P yielded a single "aging causes pain" factor that possessed good internal consistency ($\alpha = .88$). The factor structure was similar to and complements the original *Aging Causes Illness* scale, which predicts objective physical illness (e.g., amount of physical activity, being overweight) and subjective self-rated health (Stewart & Levy, 2014). The ACI-P offers a

quantitative way to briefly assess a well-accepted implicit stereotype (i.e., "to be old is to be in pain") that often goes unchallenged (Stewart, Chipperfield, Perry, & Hamm, 2016). Additionally, support was found for hypothesis 1a, which posited that individuals who reported chronic pain would endorse the belief that aging causes pain to a greater extent than individuals who did not endorse chronic pain. This finding indicates that the ACI-P successfully discriminated chronic pain and non-chronic pain groups. The finding that individuals with chronic pain endorse aging-related causal pain beliefs offers a foundation for future research to further examine the impact of an aging causal attribution that is internal, stable, and uncontrollable (Abramson, Garber, & Seligman, 1980; Weiner, 1985, 1995; Weiner, Perry & Magnusson, 1988; Hiller et al., 2010) and how aging attributions related to pain may impact the aging process for these individuals.

The finding that individuals with chronic pain endorse higher levels of aging-related pain beliefs may be related to a predominately biomedical explanation of pain (e.g., aging is seen as a biomedical process), as individuals with chronic pain often desire a clear biological explanation for pain (Baird & Sheffield, 2016). Specifically, this finding aligns with research by Baird and Haslam (2013) that chronic low back pain patients endorsed significantly higher organic beliefs about pain (e.g., "pain is the result of damage to the tissues," "physical exercise makes pain worse," and "pain is a sign of illness") compared to a nonclinical community sample. Aging-related pain beliefs (i.e., a biological explanation for pain) as well as the biomedical model for pain are often reinforced throughout the lifespan by society at large as well as an individual's increased involvement in the health care system (Baird & Haslam, 2013). For example, traditional

medical treatment for persistent pain aims to reduce or manage the biological pain sensations with the goal of complete elimination of pain (Gatchel, Peng, Peters, & Turk, 2007). Primary care physicians are often overworked and overbooked resulting in a willingness to bring their patient's immediate relief in the form of prescription analgesics (often opioids). While these medications work in the short term, prescription analgesics alone are often ineffective in treating the distress and suffering associated with chronic pain (Morley, Eccleston, & Williams, 1999). As pain persists, unclear or inconsistent diagnoses are given to individuals with chronic pain in primary care settings, and it is not uncommon for these individuals to feel as though they are blamed or labeled as symptom magnifiers to medical professionals (e.g., called "frequent fliers" in medical facilities) and their social support network (Gatchel et al., 2007; Dima et al., 2013). Living in a society that reinforces the belief that individuals should be pain free to be happy may result in individuals with chronic pain subscribing to biomedical stereotypes and beliefs about pain (e.g., aging causes pain) to make sense of their condition. This may also prevent optimal emotional functioning as an individual internalizes the notion that to be happy one must be pain free. Therefore, by this logic, if one has chronic pain she or he cannot be happy.

Importantly, there is indication that believing that "aging causes pain" may be maladaptive. The pain interference piece of hypothesis 1b was supported, specifically the belief that "aging causes pain" significantly predicted pain interference (i.e., impairment in psychosocial functioning due to pain) for both the overall sample and individuals who reported chronic pain. The finding that the extent that old age is perceived as an

uncontrollable cause of pain fits with the broader literature concerning the relationship between perceived control and health **(**Wallston, 2004; Lachman et al., 2011). This literature has repeatedly demonstrated that motivation to engage in health behaviors is compromised when individuals believe that they have little control over illness (Roesch and Weiner, 2001; Turnquist et al., 1988). Exploratory analyses in the current study supported the relationship between decreased motivation in the face of perceived uncontrollability, as those with chronic pain had significantly lower perceived control over their pain, and, although not statistically significant, individuals with higher aging-related pain beliefs endorsed even lower perceived control over their pain. Thus, aging-related beliefs about pain may be related to the broader construct of external locus of control over their health (i.e., belief that an individual's health condition is out of their control or others "know all" in addressing an individual's health condition; Wallston, 1991). The research on health locus of control suggests positive health behaviors are more likely to occur if an individual believes positive outcomes are due to their own behavior and they are capable of enacting these behaviors (Bonetti et al., 2001; Pucheu, et al., 2004; Keedy, Keffala, Altmaier, & Chen, 2014). The decreased sense of control over one's pain may suggest a decreased sense of self-efficacy in managing pain, and thus lead to increased pain interference. Therefore, the ACI-P may be conceptualized as a measure that taps into control-related beliefs that also predicts pain interference. While research has established the importance of self-efficacy and internal locus of control in successfully managing chronic pain (Smith et al., 2015; Thompson, Broadbent, Bertino,

& Staiger, 2015), further research is needed to establish the link between aging-related pain beliefs and pain self-efficacy.

Regarding, the ability of the ACI-P to predict pain interference for the total sample, and specifically the chronic pain sample, the predictive variance was relatively low (between 6 to 9%). This amount of variance explained is lower than with other pain belief measures predicting chronic pain disability. Specifically, the external causal attribution subscales of the Pain Belief Questionnaire (Edwards, Pearce, Turner-Stoke, & Jones, 1992), Pain Beliefs and Perceptions Inventory (Mikail, D'Eon, & De Gagné, 1996), and Multidimensional Health Locus of Control measures have demonstrated a pain interference or disability predictive variance ranging from 11% to 22% of variance explained in pain interference and disability (Mikail, D'Eon, & De Gagné, 1996; Baird & Sheffield, 2016). However, the predictive variance was consistent with the amount of variance the original Aging Causes Illness measure explained in an individual's perceived disability (Stewart, Chipperfield, Perry, & Weiner, 2012; Stewart & Levy, 2014). The original ACI measure yielded a single factor with 50.27% of variance explained and an internal consistency of α = .88, while the ACI-P yielded a single factor structure with 59.44% of variance explained and α = .88. One explanation for this is, like the chronic pain experience, beliefs about pain are multidimensional in nature (Gatchel et al., 2007). There may be other nuanced causal beliefs about pain broader than aging specific pain beliefs that contribute to the negative impact of pain on an individual's life that contributed to the decreased predictive validity of the ACI-P. Therefore, this study

demonstrated how one specific external belief about pain contributes to the broader experience of pain.

While other measures possess scales that assess stereotypic traits of older adults (Levy, Kasl, & Gill, 2004), general beliefs about aging (Palmore, 1998), and expectations about aging (Sarkisian et al., 2005), this study was the first to produce a scale that specifically assesses aging causes pain beyond using a single-item indicator (i.e., "Is age the main cause of your pain?"). The ACI-P allows clinicians and researchers to utilize a brief measure with known reliability and emerging validity that assesses external, stable, and uncontrollable factors that impact the pain experience.

Hypothesis 1b was not fully supported due to the finding that age and sex did not significantly moderate the relationship between ACI-P score and pain interference. The finding suggests that the relationship between aging-related beliefs about pain and pain interference holds across middle-to-older adulthood and the pattern of impact is similar for men and women. Given the average age of participants in this study was approximately 54 years and ranged from 45 to 79, the results indicate that the impact of the negative aging-related stereotype exists in middle adulthood, and adversely effects functioning equally with increasing age. Additionally, the extent to which aging causes pain beliefs impact a woman or man's pain-related interference did not yield a significant difference. While it was hypothesized that age and gender would moderate the relationship between aging-related pain beliefs and pain interference, the null finding replicates a study by Thomas and colleagues (2004) that found no moderating impact of age and sex on pain interference in a general population sample. Therefore, it appears that

men and women across middle and older adulthood may both be adversely affected by citing aging as a primary reason for pain. This highlights the importance of screening pain beliefs in individuals that present in primary care settings as a means to take preventative measures to address potential health decline (this will be discussed further below).

Hypothesis 2a was not supported as individuals who endorsed chronic pain reported significantly more health conditions (e.g., heart problems, stroke, diabetes, etc.) than individuals who did not endorse chronic pain. However, while individuals with higher ACI-P scores reported more health conditions for the chronic pain versus non-chronic pain groups, the difference was not statistically different. Overall, the number of health conditions reported was relatively low (ranging from an average of 0.85 to 1.96) across groups). This could result in decreased variance to obtain necessary power to observe the main effects. However, the range of reported health conditions is comparative to prevalence studies that have shown a more than 70% of middle-age to older adults report no or one health condition (Vogell, et al., 2007; Schneider, O'Donnell, & Dean, 2009). The finding from this study is also consistent with recent research that has compared clinical to non-clinical chronic pain samples in health profiles. Specifically, Baird & Haslam (2013) found that individuals who did not endorse chronic pain reported significantly better general health. Further, aging-related beliefs regarding health has been associated with the low control mindset that may contribute to a reduced sense of responsibility for one's health and lowered motivation to engage in healthy behavior change to mitigate health condition onset (Roesch & Weiner, 2001; Stewart,

Chipperfield, Perry, & Hamm, 2016). Future studies should investigate this further as the belief that aging causes pain may contribute to overlooking aches and pains associated with health condition symptoms and reduce the likelihood an individual will seek treatment and address emergent or ongoing health conditions.

Consistent with the pain interference finding above, hypothesis 2b was supported. This study showed that individuals who endorsed chronic pain reported significantly higher functional impairment due to pain than individuals that did not endorse chronic pain, and that individuals with high aging-related pain beliefs reported significantly higher functional impairment due to pain than individuals with low aging-related pain beliefs. Looking closer at how pain coping strategies impact the relationship between the belief that aging causes pain and pain interference, hypothesis 2c was partially supported as pain catastrophizing was the only pain coping strategy to be associated with aging-related pain beliefs. Further, pain catastrophizing significantly mediated the relationship between aging-related pain beliefs and pain interference. Hypothesis 2c was not fully supported, because the other pain coping strategies (i.e., distraction, distancing, ignoring, praying, and positive self-statements) were not associated with the attribution "aging causes pain."

Pain catastrophizing, a negative cognitive-affective response to anticipated or actual pain that is associated with magnification, rumination or helplessness in the face of the threat of pain sensations (Chaves & Brown, 1987; Spanos et al., 1979), has been consistently linked with negative pain outcomes in both non-chronic pain and chronic pain samples (Quartana, Campbell, & Edwards, 2009; Craner, Gilliam, & Sperry, 2016).

The finding that aging-related pain beliefs are mediated by pain catastrophizing expands the literature by illustrating how the stereotypic aging-related attribution fits into the cognitive portion of the fear-avoidance model and vicious cycles of chronic pain. Specifically, to believe that aging causes pain appears to fall into a broader negative cognitive style (as higher Affective Distress subscale of the WHYMPI for chronic pain endorsing individuals with high ACI scores compared to low ACI scores may suggest) associated with pain that may inhibit adaptive pain coping and promote a threatening evaluation of the pain experience. The literature supports this finding as organic causal beliefs (i.e., pain is caused primarily by a biological factor that includes age) and pain catastrophizing are related to lower levels of perceived personal control (Baird & Haslam, 2013; Hagger & Orbell, 2003), and in turn lower functional pain-related impairments (Turner, Jensen, & Romano, 2000; Baird & Sheffield, 2016). To this author's knowledge, this is the first study to look at both aging as a causal pain belief and catastrophizing concurrently. The sense of uncontrollability of pain may increase a heightened perceived threat of pain which contributes to a sense of learned helplessness. This may serve as a barrier for adaptive behaviors that may attenuate pain interference. The association between aging-related beliefs, catastrophizing, and pain should be evaluated with further research.

Hypothesis 3a and 3b were not supported as there were no significant differences in daily physical activity (total, vigorous, and moderate) and sedentary time among individuals with high and low aging-related pain beliefs or among individuals who endorsed or did not endorse chronic pain. While other results within this study found that

an individual's perceived functional impairment, affective distress, and life control (i.e., cognitive barriers to the pain experience) were affected by either ACI-P or chronic pain status, individuals were able to engage in similar levels of physical activity and sedentary time (i.e., measures of specific behavior) across groups. Dogra and Stathokostas (2012) found that physical activity and sedentary time independently predict successful aging in middle-aged and older adults. The negative aging-related belief that aging causes pain may not be an indicator of successful aging, and therefore, further research investigating the relationship between the belief that to be old is to be in pain and successful aging is warranted. While research has linked associations of decreased physical activity and sedentary time to pain outcomes (Layne & Nelson, 1999; Jessup et al., 2003; Hagen et al., 2002; Nelson et al., 2007), individuals in this study reported an average daily physical activity of 33.81minutes. Further, this is above the American College of Sports Medicine's Physical Activity recommendations for individuals above the age of 45 of 30 minutes of moderate to vigorous physical activity per day (Pescatello, Arena, Riebe, & Thompson, 2014). Further, the average sedentary time as indicated by the response to the question "How much time do you usually spend sitting or reclining on a typical day?" was much lower (this study's average was 62 minutes) than would be expected from population research that suggests average sedentary times ranges from 6 to 8 hours per day (Owen, Healy, Matthews & Dunstan, 2010). Therefore, the null findings could be associated with an active, relatively healthy (as noted by the relatively low number of health conditions endorsed) sample and/or an over-reporting of physical activity / under-reporting of sedentary time.

Further, it may be that the total activity endorsed by participants in this study falls into the optimal middle ground with regard to pain interference and physical activity. Heneweer, Vanhees, and Picavet (2009) found that physical activity acts as a preventative factor for chronic pain, but excessive physical activity raised the risk for chronic pain conditions in middle-age and older adults. This finding was replicated by Landmark and colleagues (2011) who showed that bouts of exercise around 30 minutes in duration were predictive of negative pain outcomes. While researchers are still attempting to understand the u-shaped relationship of physical activity and pain outcomes, it is assumed that physical deconditioning and a lack of pacing accounts for the negative pain outcomes (Landmark et al., 2011). The findings with regard to physical activity and sedentary time provides further evidence that the impact of the belief "aging causes illness" is closely associated with the cognitive aspects of chronic pain as opposed to the physical/behavioral aspects. Further research is needed to objectively measure physical activity and sedentary time to confidently evaluate the connection between aging-related causal pain beliefs, physical activity, and pain outcomes.

Lastly, exploratory analyses revealed nuanced relationships between aging-related pain beliefs, chronic pain, and pain acceptance (i.e., the process of giving up the struggle with pain and live a meaningful life despite pain, McCracken, Vowles, Eccleston, 2004). The results showed that individuals who endorsed chronic pain reported greater pain acceptance (consisting of two factors: pain willingness and engagement in valued activities despite pain) but the extent to which an individual believes that aging causes pain did not significantly predict total pain acceptance. Looking closer at pain

acceptance, individuals with chronic pain reported significantly higher pain willingness, and those with high aging causes pain beliefs reported greater pain willingness. One way to account for this finding is that individuals with chronic pain have already lived and experience pain for some time and are more willing to recognize that avoidance and control of pain are often unworkable methods of adapting to pain. This aligns with the null findings from hypotheses 3a and 3b, as individuals may experience chronic pain but still be willing to engage in necessary activities to complete their day. Interestingly, individuals with low aging causes pain beliefs and chronic pain were more likely to pursue life activities regardless of pain compared to individuals with high aging causes pain beliefs that endorsed chronic pain, individuals with high aging causes pain beliefs without chronic pain, and individuals with low aging causes pain beliefs without chronic pain. This is in line with past research that suggests aging attributions about pain may offer greater tolerance for uncertainty and acceptance of the lack of "easy fixes" associated with the chronic pain experience (Le, 2008; Williamson, 2000). Further research is needed to elucidate the relationship between aging attributions, pain acceptance, and pain interference.

The results of this study demonstrate that individuals hold aging-related causal beliefs about pain to a varying degree, and it appears individuals who endorse chronic pain hold this belief to a greater extent than those who do not endorse chronic pain. Further, the belief that aging causes pain is associated with pain interference, and this relationship is mediated by pain catastrophizing. There is also evidence that the relationship between aging-related pain beliefs and pain interference is not affected by

age or sex. The impact of holding the belief that aging causes pain appears to be associated with the cognitive aspects of the fear-avoidance model/vicious cycle of pain as the belief that aging causes pain was not associated with physical activity or sedentary time. While the maladaptive aspects of this belief are established, there is some evidence that this belief may reinforce pain tolerance and willingness that may buffer from psychological distress.

Study Implications

Persistent pain affects over 100 million Americans each year (U.S. Department of Health and Human Services, 2006; Institute of Medicine, 2011) and imparts a substantial societal and economic burden. This burden manifests as an incremental cost of health care and lost productivity attributable to chronic pain is between $560 and 635 billion dollars per year (Institute of Medicine, 2011). At the individual level, it is estimated that only 40 to 60% of patients with chronic pain adequately manage their pain as indicated by comorbid medical concerns, work status, and maintaining activities despite persistent pain (Breivik et al., 2006; Breivik, 2012). However, the subspecialty of pain management is relatively new in the grand scope of psychology and medicine, and the supply of pain management specialists is greatly outnumbered by the demand (Dubois & Follett, 2014). As such, chronic pain and pain complaints are often addressed in primary care settings with accessible, continued, and coordinated care between healthcare providers (Mills, Torrance, & Smith, 2016). Yet, the assessment and management of chronic pain is challenging in primary care settings due to the complex multidimensional nature of pain

and the rapid pace and volume of patients seen in the primary care setting. Thus, efficient and effective screening of chronic pain is needed.

The current study demonstrates the preliminary psychometrics regarding the ACI-P, a brief, new measure that clinicians can use as a screener to differentiate between individuals who hold aging attributions with chronic pain and those who do not. Additionally, while the relationship between chronological age and pain should not be overlooked, the data gathered with a brief clinical interview and ACI-P findings offers an opportunity for the clinician to 1) discuss the role of aging on the pain experience and 2) reduce the emphasis placed upon aging-related pain attributions. For example, clinicians could discuss the cumulative impact of physical and mental stress on the body throughout the lifespan, and emphasize the role of health behaviors (e.g., paced physical activity, sleep hygiene, stress management) on the chronic pain experience as a way to highlight the barriers that aging-related pain attributions may aggravate. This causality-focused intervention (Stewart et al., 2016) would aim to modify the patient's perception about the nature of aging (i.e., physical declines and pain associated with aging can be attenuated by one's engagement in health behaviors, and not that aging is a series of inevitable, immutable physical declines) and offer a realistic prescription of lifestyle attributions that impact the pain experience (i.e., chronic pain is caused by a variety of factors, not only biological ones). This type of intervention includes a psychoeducation component that defines biological and chronological age (Fries, 1980; Roizen, 1999), and promotes a discussion about common misconceptions regarding aging. The message of this intervention is that biological age (physiological, biological and social experience

associated with aging) is modifiable, and chronological age (the days, months, and years in which an individual has been alive) differs from biological age (Stewart et al., 2016).

Along with the causality-focused intervention that could serve as an adjunctive to primary care hospital visits, this study also has clinical implications for outpatient and pain subspecialty settings. Specifically, the findings of this study can be integrated into CBT, and ACT for chronic pain (Williams, Eccleston & Morley, 2012).

Randomized controlled trials and outcome studies of CBT for chronic pain have shown that CBT for chronic pain is effective at improving pain interference and decreasing pain catastrophizing (Smeets, Vlaeyen, Kester & Knotterus, 2006; Turner, Hollzmann, & Manel, 2007; Ehde, Dillworth, & Turner, 2014). A large component of CBT is to establish the connection between negative cognitions about pain and for clinicians to teach the patient that many of these negative thoughts are automatic. The clinician's goal includes having the patient monitor automatic negative thoughts, eventually challenging negative cognitions about pain (i.e., cognitive restructuring) with coping statements such as "This pain will pass." This study demonstrates that many individuals who endorse chronic pain may hold thoughts about aging and pain, and these thoughts impact their lives negatively. As such, clinicians should elicit the patient's thoughts regarding their pain and the aging process, and offer appropriate education and challenging of the thought that aging in and of itself causes pain. Based on the mediational findings from this study, challenging these thoughts could address the impairment caused by pain catastrophizing. As this is the first studies to examine aging causal beliefs and catastrophizing, future research should aim to elucidate the relationship

between the belief that aging causes pain and pain catastrophizing further and test the efficacy of this proposed addition to CBT for chronic pain.

ACT for chronic pain focuses on decreasing the struggle with pain facilitating acceptance, and highlighting that attempts to reduce or eliminate the pain entirely has resulted in decreased involvement in valued activities (McCracken & Vowles, 2014). The results of this study indicate that while the belief that aging causes pain appears to buffer pain willingness for individuals who endorsed chronic pain, this belief interferes with engagement in valued activities such as work, hobbies, chores, and social involvement. Integrating this finding with an ACT model, clinicians may validate that a patient's pain sensations may occur in frequency with age, but highlight how aging-related beliefs about pain is associated with the emotional aspects of the pain experiences (e.g., thoughts and expectations regarding pain and not the pain sensation itself). The ACT clinician should help the patient identify values and valued activities that may have been lost due to pain and the aging process, and encourage experiential exercises (e.g., mindfulness exercises, metaphors, engagement in valued activities) to demonstrate that the patient may "take their life back" from pain. In summary, the results of this study have implications that can be efficiently implemented in primary care and outpatient pain management settings that address the diversity factor of aging in the context of the pain experience.

Limitations and Future Directions

There are several notable limitations to this study despite offering findings that lay a foundation for understanding aging-related causal attributions about chronic pain.

First, the cross-sectional design of this study does not allow for any conclusions about the temporal relationships among variables and claims regarding causality cannot be made. Additionally, this study utilized a non-clinical convenience sample recruitment, and participants were placed into chronic pain or non-chronic pain groups based on self-report of experiencing pain (or not experiencing pain) lasting longer than one month at some point in their life. It could be that some of the participant's pain difficulties have resolved prior to participation. Further, the non-chronic pain participants were not a pure control comparison (i.e., these participants were not randomly selected participants that were verified to not have chronic pain). Moreover, while the sample size is relatively large and effects were similar across subsamples, full statistical power may have been compromised due to utilizing two separate samples recruited in two different ways, one via online recruitment. While studies have shown that MTurk appears to be a source of high quality and inexpensive data, effect sizes obtained in laboratory social science research are comparable to those obtained on MTurk (Buhrmester, Kwang, & Gosling, 2011; Necka, Cacioppo, Norman, & Cacioppo, 2016), and reliability checks were utilized throughout the online questionnaires, other confounding variables (e.g., self-selection to a study titled "Health, Pain, and Aging") may have not been accounted for. There could also be self-selecting or ability factors associated with the *Aging in Idaho* sample that impacted those who returned mailed out questionnaires compared to those who did not respond to the mail out.

Another limitation is related to how functional impairment was measured (i.e., self-reported disability or inability to perform physical activity on the WHYMPI). It may

be pertinent to ask additional questions to assess functional impairment due to chronic pain or other physical limitations such adding an additional item inquiring whether or not a medical provider has asked the participant to limit any physical activity. While significant effects were found for Hypotheses 2a and 2b, a loss of effect size, loss of power, or distortion of effects may have occurred when evaluating Hypotheses 3a and 3b due to the dichotomization of the ACI-P score to create high and low groups for analysis. For future studies it may be warranted to evaluate whether dichotomizing the groups based on clinical utility via mean of the chronic pain reference group or maintaining the ACI-P as a continuous and evaluating group differences using regression models.

There has been very little research focused on the impact of attributions about health conditions over time. To address these issues, future research should utilize longitudinal methods with participants randomly selected from clinical settings using medical records to verify the presence of chronic pain. This would help establish the relationship between aging-related beliefs about pain and pain interference among those with and without chronic pain over time. Both longitudinal and sequential designs would offer a clearer understanding of 1) how holding aging-related beliefs about pain impact the individual across periods of adulthood, and 2) how aging-related beliefs might change across the lifespan. To determine if these beliefs differ among age cohorts, participants representative of different age groups such as decade cohorts or young, middle, and older adult cohorts should recruited in future studies and investigated further.

This was the first study to utilize a multi-item measure to investigate the impact of attributing pain to aging, and as such, further assessment of the ACI-P's psychometric

properties is needed. With regard to reliability, the ACI-P should be evaluated for split-half reliability, or given to individuals over time to determine the test-retest reliability. For convergent and discriminant validity, future research should compare ACI-P scores to established measures of aging-related beliefs (e.g., Self-Perceptions of Aging; Levy, Slade, Kunkel, & Kasl, 2002; Expectations Regarding Aging; Sarkisian, 2005), pain-related beliefs (Pain Beliefs Questionnaire; Edwards, Pearce, Turner-Stokes, & Jones, 1992), and controllability over health behaviors (Multidimensional Health Locus of Control; Wallston et al., 1978). Moreover, the ACI-P should be compared to measures of personality and other psychosocial outcomes (e.g., optimism, social support, depressive symptoms) to determine other moderating and mediating variables that may be implicated in the relationship between aging-related beliefs about pain and pain interference. Another limitation for this study was the lack of ACI-P comparison to objective indictors of health and wellness. Future research should investigate the relationship between ACI-P and recorded number of physician visits or hospitalization, monitored physical activity (via electronic fitness tracker or actigraphy), and biomarkers of stress.

While it appears that holding the belief that aging causes pain negatively impacts pain outcomes, it is unclear whether it is a general causal aging attribution that drives the negative aspects of this belief or if internalizing this belief is the larger problem (i.e., "aging is causing *my* pain."). Additional research could address the potential logical fallacy of claiming that the "aging causes pain" belief is a self-directed stereotype by modifying the wording and evaluating differences in how wording impacts the

relationship between ACI-P and pain interference. This could be accomplished by personalizing the causal attribution of age within the ACI-P (e.g., changing "Most people are in pain because of old age" to "I am in pain because of old age" or "Old age is the main cause of pain in North America" to "Old age is the main cause of my pain"). If differences were not to be found between this study's ACI-P and a reworded, internalized ACI-P with regard to pain outcomes, assumptions regarding the ACI-P measuring self-directed stereotypes about age would be more robust. However, the more internalized stereotype version of the ACI-P could be a more accurate measure of the self-directed stereotype "aging causes pain." Future research should evaluate this further.

Further, just because individuals rate high levels of aging-related attributions about pain does not mean they did not also report high levels of other attributions. Rather than causal attributions being a single unidimensional construct individuals appear to adhere to multidimensional ideas about their pain and its origins, consistent with a biopsychosocial conceptualization of chronic pain (Gatchel et al., 2007). Therefore, future research should investigate how different attributions about pain interact or modify relationships with pain interference.

Unmeasured constructs such as pain self-efficacy (Nicholas, 1994) may also play a role in the relationships examined in this study. Research has established the connection between lower perceived control and lower pain self-efficacy (i.e., an individual's appraisal of their ability to cope with pain) leading to greater pain interference (Turner, Ersek, & Kemp, 2005). It may be possible that ACI-P is associated with decreased pain self-efficacy. Future studies should examine this relationship. Finally, as noted in the

study implications section above, the impact of this belief should be evaluated within the context of psychoeducation or therapeutic interventions to elucidate its connection to the fear-avoidance model/vicious cycle of pain.

Conclusion

In summary, this is the first study to demonstrate preliminary evidence of the psychometric properties and utility of a new measure, the *Aging Causes Illness – Pain* (ACI-P), which evaluates the extent to which individuals believe that aging causes pain. In addition, individuals who endorse chronic pain report that they hold this belief to a greater extent than individuals who do not endorse chronic pain. Moreover, this belief is associated with pain interference and is mediated by maladaptive cognitive aspects of the pain interference, namely pain catastrophizing. The current study did not establish the connection between aging-related pain beliefs and physical activity or sedentary time. Given that the findings held across two separate subsamples, the initial investigation of this belief demonstrates a robustness and potential that ACI-P may serve as a predictor of other psychological factors and functional impairments associated with pain. Consequently, future controlled studies should seek to expand these findings within clinical samples to determine the ecological utility of the ACI-P and determine its clinical significance in chronic pain outcomes.

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Appendix A: Tables & Figures of the Results

Table 1

Participant Demographic Information

	MTurk Sample	*Aging in Idaho* Sample	Significance	Total
N	294	76		370
Age	52.95 (7.05)	59.58 (7.23)	$p<.001$	54.26 (7.54)
% Women	54.4%	57.3%	$p= .59$	55.0%
% Employed	73.5%	61.3%	$p<.01$	70.8%
Ethnicity				
% White/Caucasian	81% (238)	97.3% (74)	$p<.001$	84.1%
% African American	8.2% (24)			
% Hispanic/Latino	3.7% (11)	2.7% (2)		
% Asian American	4.4% (13)			
% Native American/ American Indian	1.7% (5)			
% Asian/Pacific Islander	1.0% (3)			
Relationship Status				
% Married	51.0% (150)	80.0% (61)	$p<.001$	56.8%
% Single	21.8% (64)	3.9% (3)		
% Divorced or Separated	18.3% (54)	6.6% (5)		
% Dating or in Domestic/Partnership	5.4% (16)	5.3% (4)		
% Widowed	3.4% (10)	4.0% (3)		
Highest Education Completed				
% Completed 4-Year College	36.7% (108)	26.7% (20)	$p= .12$	34.6%
% Completed Graduate School	17.3% (51)	10.7% (8)		
% Completed Some College	16.0% (47)	19.7% (15)		
% Completed 2-Year College	15.6% (46)	13.3% (10)		
% Graduated High School/GED	9.9% (29)	16.0% (12)		
% Some Graduate School	4.4% (13)	13.3% (10)		
% Less than High School		1.3% (1)		
% Served in Military	9.5%	10.5%	$p= .69$	9.7%
Endorsed Chronic Pain	172 (58.5%)	46 (60.5%)	$p= .75$	259 (58.9%)
Pain Rating (0 to 6)	1.60 (1.38)	1.68 (1.43)	$p = .63$	1.62 (1.39)

Note: An independent samples *t*-test was used to compare significant differences among continuous variables, while Pearson Chi-Square was used to compare significant differences among categorical variables.

Table 2

Inter-correlations among study variables for total sample

	ACI-P	MPI PI	MPI GA	MPI LC	MPI AD	CPAQ Total	CPAQ PW	CPAQ AE	CSQ DRT	CSQ C	CSQ I	CSQ DTC	CSQ CSS	CSQ P	Phys. Act.	Sed. Time	# H. Co.	SDS 17
ACI-P	---																	
MPI PI	.29**	---																
MPI GA	.10*	-.10*	---															
MPI LC	-.06	-.39**	.36**	---														
MPI AD	.15**	.53**	-.22**	-.63**	---													
CPAQ Total	.12*	.12*	.27**	.26**	-.05	---												
CPAQ PW	.26**	.51**	-.02	-.22**	.28**	.60**	---											
CPAQ AE	-.07	-.29**	.36**	.51**	-.30**	.74**	-.11*	---										
CSQ DRT	-.01	.08	.21**	.12*	-.02	.35**	.28**	.20**	---									
CSQ C	.20**	.56**	-.13**	-.45**	.48**	.04	.54**	-.41**	.16**	---								
CSQ I	-.01	-.14**	.14**	.29**	-.19**	.26**	-.07	.38**	.38**	-.22**	---							
CSQ DTC	.09	.16**	.12*	.04	.05	.21**	.27**	.04	.56**	.23**	.45**	---						
CSQ CSS	.01	-.05	.25**	.30**	-.14**	.45**	.07	.50**	.47**	-.14**	.57**	.34**	---					
CSQ P	.02	.25**	.03	-.10	.14**	.18**	.30**	-.03	.32**	.36**	-.02	.24**	.11	---				
Physical Activity (mins/day)	.12*	.06	.14**	.02	-.02	.07	-.02	.10*	.12*	.01	.16**	.17**	.09	.06	---			
Sedentary Time	.04	.14**	-.19**	-.20**	.16**	-.04	.13**	-.15**	-.05	.08	-.08	-.07	-.03	-.10*	-.18**	---		
# Health Conditions	.18**	.46**	-.11*	-.23**	.30**	.01	.23**	-.19**	.02	.28**	-.09	.02	.01	.17**	.02	.16**	---	
SDS-17	.02	.04	.04	.03	.01	.08	.03	.07	.02	.02	-.06	-.03	.02	.08	.01	.06	.01	---

Note: ACI-P = Aging Causes Illness-Pain total score, MPI PI = West Haven-Yale Multidimensional Pain Inventory Pain Interference Subscale, MPI GA = West Haven-Yale Multidimensional Pain Inventory Pain General Activities Subscale, MPI LC = West Haven-Yale Multidimensional Pain Inventory Pain Life-Control Subscale, MPI Affective Distress = West Haven-Yale Multidimensional Pain Inventory Pain Affective Distress Subscale, CPAQ = Chronic Pain Acceptance Questionnaire Total Score, CPAQ PW = Chronic Pain Acceptance Questionnaire Pain Willingness Subscale, CPAQ AE = Chronic Pain Acceptance Questionnaire Activity Engagement Subscale, CSQ-R-DRT = Coping Strategies Questionnaire-Revised Distraction Subscale, CSQ-R-C = Coping Strategies Questionnaire-Revised Catastrophizing Subscale, CSQ-R-I = Coping Strategies Questionnaire-Revised Ignore Subscale, CSQ-R-DTC = Coping Strategies Questionnaire-Revised Distancing from

Pain Subscale, CSQ-R-CSS = Coping Strategies Questionnaire-Revised Coping Self Statements Subscale, CSQ-R-P = Coping Strategies Questionnaire-Revised Praying Subscale, and SDS-17 = Social Desirability Scale. * $p<.05$. ** $p<.01$.

Table 3

Descriptive Statistics for the MTurk Sample, Aging in Idaho Sample, and Total Sample

	MTurk Sample		*Aging in Idaho* Sample		Total Sample	
	NCP (n = 122)	CP (n = 172)	NCP (n = 30)	CP (n = 46)	NCP (n = 152)	CP (n = 218)
ACI-P	20.41 (4.88)[a]	23.60 (4.90)[b]	19.80 (3.86)[a]	22.19 (5.15)[b]	20.29 (4.69)[a]	23.18 (4.97)[b]
WHYMPI PI	6.93 (8.52)[a]	20.70 (11.79)[b]	4.58 (8.11)[a]	18.73 (12.76)[b]	6.47 (8.47)[a]	20.29 (12.00)[b]
WHYMPI GA	2.83 (.92)	2.94 (.91)	2.97 (.91)	3.07 (.89)	2.85 (.92)	2.97 (.90)
WHYMPI LC	4.47 (1.40)[a]	4.03 (1.22)[b]	4.92 (1.22)[a]	4.39 (1.29)[b]	4.56 (1.37)[a]	4.11 (1.25)[b]
WHYMPI AD	1.46 (1.03)[a]	2.32 (1.29)[b]	1.32 (1.09)[a]	2.04 (1.26)[b]	1.43 (1.04)[a]	2.26 (1.28)[b]
CPAQ Total	60.30 (18.66)[a]	66.21 (12.20)[b]	56.45 (19.61)[a]	62.43 (11.72)[b]	59.54 (18.85)[a]	65.41 (12.17)[b]
CPAQ PW	19.39 (10.53)[a1]	24.25 (9.65)[b1]	15.33 (10.12)[a2]	18.20 (8.73)[b2]	18.59 (10.54)[a]	22.97 (9.77)[b]
CPAQ AE	40.91 (13.77)	41.96 (11.03)	41.11 (14.53)	44.22 (9.34)	40.95 (13.87)	42.44 (10.72)
CSQ-R-DRT	13.87 (7.75)[a]	13.60 (6.65)[b]	11.87 (6.90)[a]	11.86 (6.36)[b]	13.47 (7.61)[a]	13.23 (6.61)[b]
CSQ-R-C	8.25 (7.78)[a1]	9.97 (7.42)[b1]	4.68 (4.43)[a2]	7.77 (6.47)[b2]	7.54 (7.37)[a]	9.51 (7.27)[b]
CSQ-R-I	13.98 (7.60)	12.72 (6.72)	13.87 (4.86)	14.97 (5.83)	13.96 (7.13)	13.19 (6.59)
CSQ-R-DTC	7.03 (5.93)[1]	6.56 (5.53)[1]	4.30 (4.90)[2]	5.00 (4.98)[2]	6.49 (5.83)	6.23 (5.44)
CSQ-R-CSS	14.99 (5.48)	15.44 (4.92)	14.20 (4.94)	14.76 (5.22)	14.84 (5.37)	15.29 (4.98)
CSQ-R-P	6.21 (6.09)	5.89 (5.50)	6.57 (5.95)	6.24 (5.45)	6.28 (6.05)	5.96 (5.48)
Physical Activity minutes/day	30.27 (45.96)	35.38 (55.47)	20.76 (23.15)	27.97 (30.02)	28.39 (42.55)	33.81 (51.19)
Sedentary Time minutes/day	61.36 (34.41)[a1]	65.38 (34.90)[b1]	37.36 (24.33)[a2]	53.95 (30.19)[b2]	56.62 (33.97)[a]	62.96 (34.21)[a]
# Health	0.93 (1.83)[a]	1.83 (1.55)[b]	1.20 (1.30)[a]	2.03 (1.73)[b]	0.99 (1.73)[a]	1.87 (1.59)[b]

Conditions						
SDS-17	8.75 (1.66)	9.12 (1.79)	9.47 (1.76)	9.07 (1.70)	8.89 (1.70)	9.11 (1.77)

Note: Refer to Table 2 Note for explanation of all measure abbreviations. Superscript letters [a] and [b] indicate significant group differences ($p<.05$) between individuals that endorsed chronic pain (CP) or did not endorse chronic pain (NCP). Superscript numbers [1] and [2] indicate significant group differences ($p<.05$) between the MTurk and *Aging in Idaho* samples.

Table 4

Aging Causes Illness – Pain (ACI-P) scale items and factor loading

#	Item Wording	Factor 1 Loading	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6	Factor 7
1.	Pain is an unavoidable part of getting old.	.73						
2.	Old age is the main causes of pain in North America.	.79						
3.	Many types of pain are caused by age.	.63						
4.	It is impossible to escape being in pain when you are old.	.80						
5.	Most people are in pain because of old age.	.75						
6.	Although there are other causes of pain, old age is the main cause.	.81						
7.	You can do some things to stay healthy, but in the end old age will result in pain.	.80						
	Eigenvalue	4.05	.967	.641	.409	.382	.315	.237
	% of Variance	57.85	13.81	9.16	5.84	5.46	4.50	3.38
	α	.88						

Table 5

Inter-item correlations for the Aging Causes Illness-Pain (ACI-P) measure

	Item 1	Item 2	Item 3	Item 4	Item 5	Item 6	Item 7
Item 1	----						
Item 2	.440	----					
Item 3	.491	.412	----				
Item 4	.601	.487	.426	----			
Item 5	.392	.613	.293	.497	----		
Item 6	.393	.698	.393	.534	.716	----	
Item 7	.610	.500	.442	.676	.445	.544	----

Note: All correlations are $p<.001$.

Table 6

Hypothesis 1a: Chronic Pain Status predicting ACI-P Total Score for each sample

	Total Sample			MTurk Sample			*Aging In Idaho Sample*		
	B	SE *B*	β	*B*	SE *B*	β	*B*	SE *B*	β
Constant	17.28	.85		17.22	.96		17.40	1.85	
Chronic Pain Group	3.01	.51	.29**	3.19	.579	.31**	2.40	1.10	.25*

Note: R^2 = .09 for total sample (p<.001), R^2 = .09 for MTurk Sample (p<.001), R^2 = .06 for *Aging in Idaho* sample (p<.05). * p <.05. **p<.001.

Table 7

Regression analysis of aging-related beliefs (ACI-P) predicting pain coping strategy (CSQ-R subscales)

	CSQ-R Distraction			CSQ-R Catastrophizing			CSQ-R Coping Self-Statements			CSQ-R Ignore			CSQ-R Distance		
	B	SE *B*	*β*	*B*	SE *B*	*β*	*B*	SE *B*	*β*	*B*	SE *B*	*β*	*B*	SE *B*	*β*
ACI-P	-.03	.07	-.02	.27	.07	.19***	-.001	.05	-.001	-.04	.07	-.03	.07	.06	.06

	CSQ-R Pray		
	B	SE *B*	*β*
ACI-P	-.02	.06	-.02

Note: ***significant predictor at $p<.001$

For CSQ-R Distraction $R^2 = .00$, $p = .691$
For CSQ-R Catastrophizing $R^2 = .04$, $p < .001$
For CSQ-R Coping Self-Statements $R^2 = .00$, $p = .980$
For CSQ-R Ignore $R^2 = .001$, $p = .577$
For CSQ-R Distance $R^2 = .004$, $p = .229$
For CSQ-R Pray $R^2 = .00$, $p = .773$

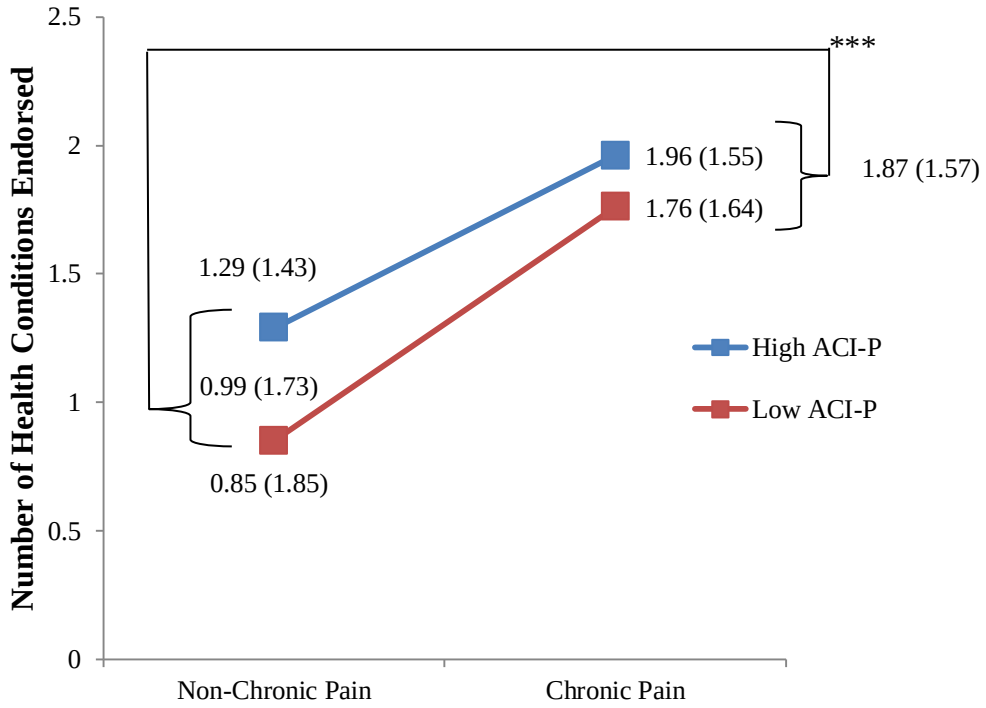


Figure 7. Group differences in reported number of health conditions among individuals with high and low aging-related pain beliefs and pain status. *** Significant difference between endorsers of chronic pain and those who did not endorse chronic pain, $p<.001$.

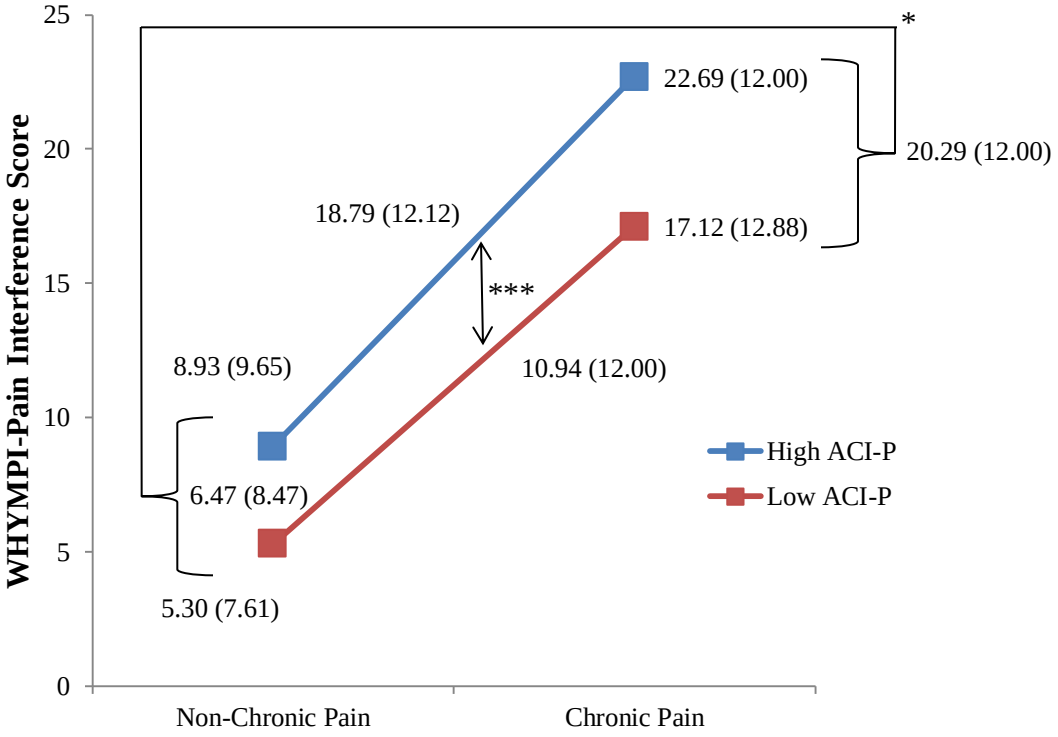


Figure 8. Group differences in pain interference among high/low aging-related beliefs and chronic pain status. ***Significant difference between endorsers of chronic pain and those who did not endorse chronic pain. There was also a significant difference between High and Low ACI-P on pain interference, $p<.001$.

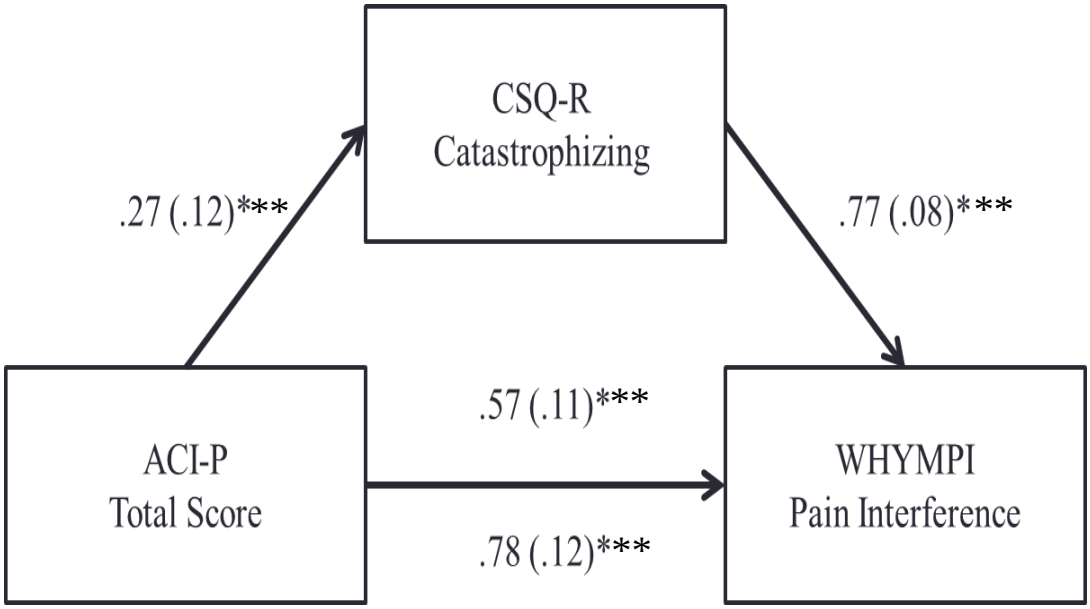


Figure 9. Regression coefficients and standard errors for the relationship between aging-related pain beliefs and pain interference as mediated by pain catastrophizing. ***p<.001.

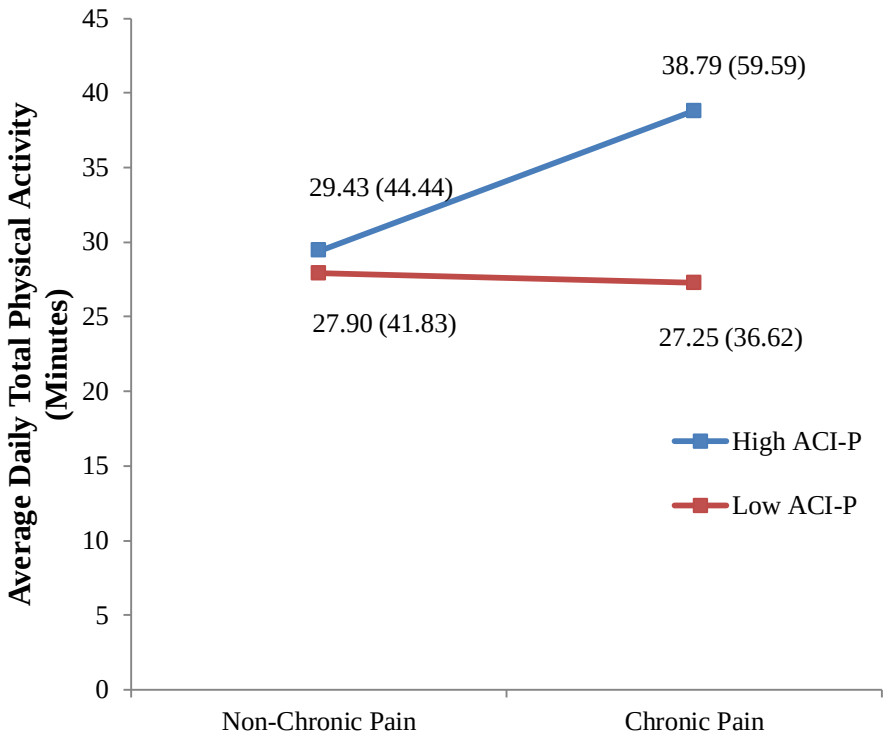


Figure 10. There were no significant group differences among high/low aging-related pain beliefs and chronic pain status on average daily total physical activity (vigorous and moderate).

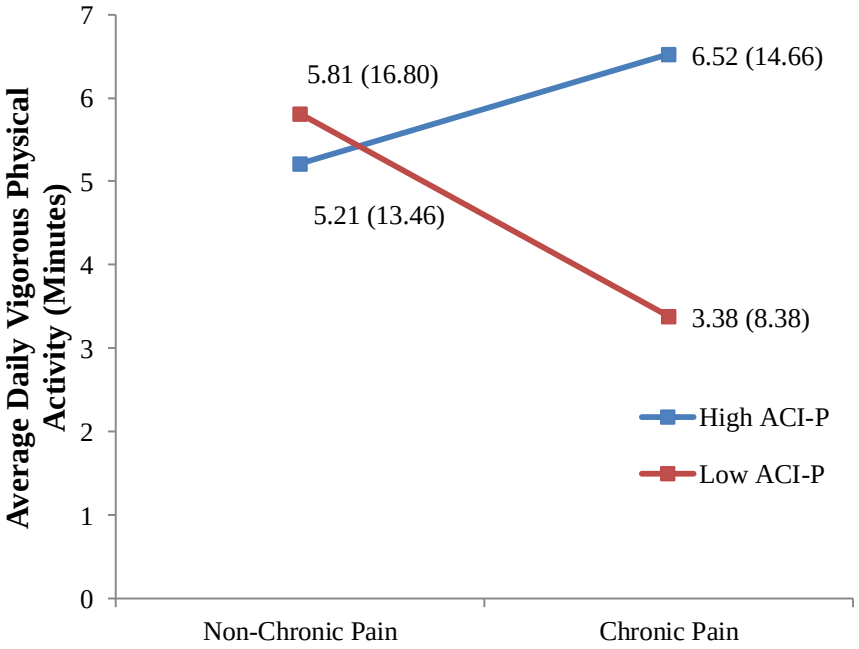


Figure 11. There were no significant group differences among high/low aging-related pain beliefs and chronic pain status on average daily vigorous physical activity.

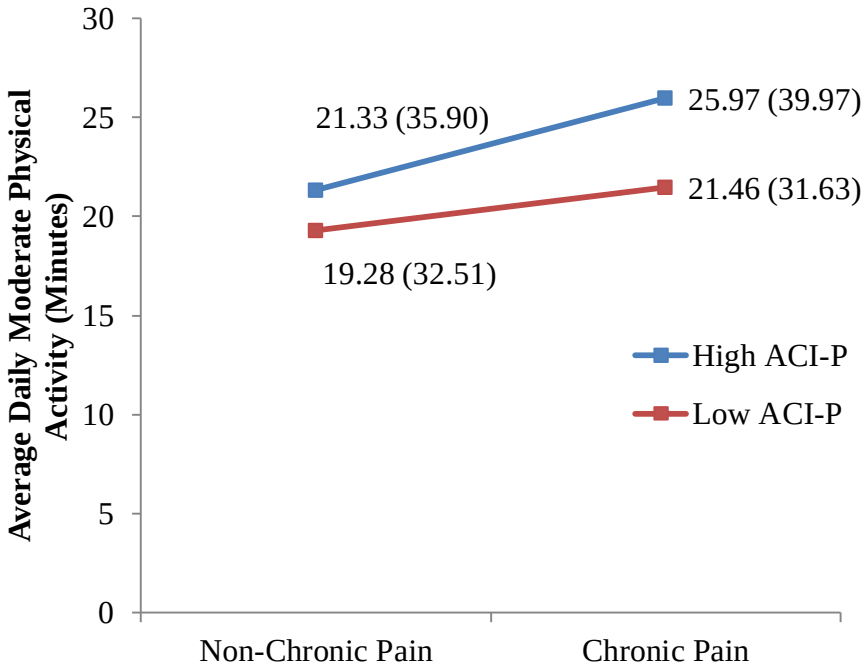


Figure 12. There were no significant group differences among high/low aging-related pain beliefs and chronic pain status on average daily moderate physical activity.

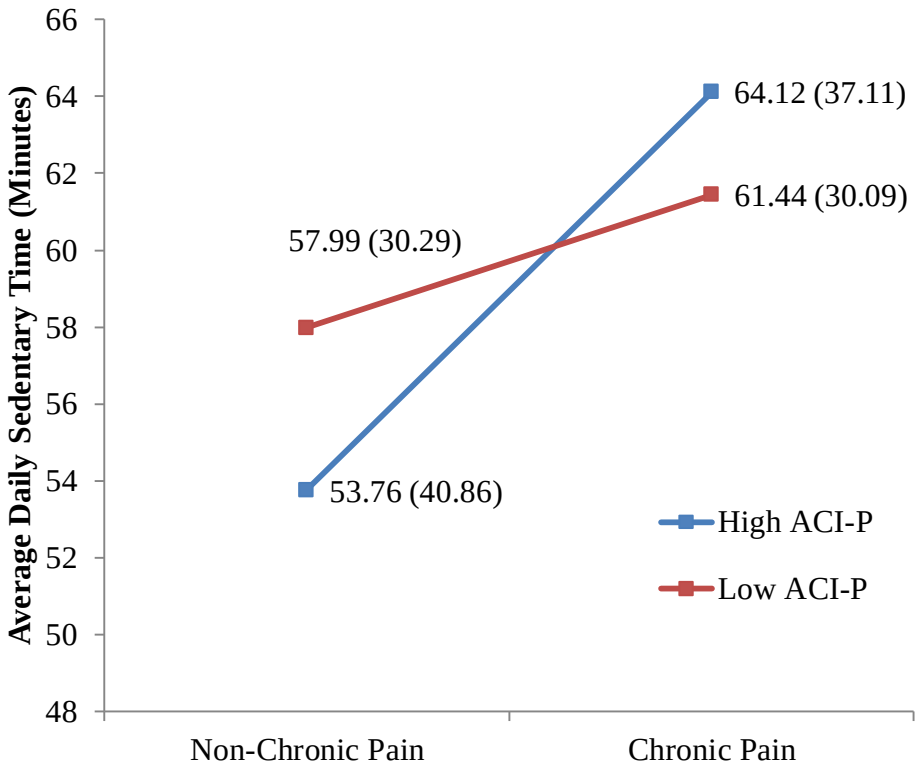


Figure 13. There were no significant group differences among high/low aging-related pain beliefs and chronic pain status on average daily sedentary time.

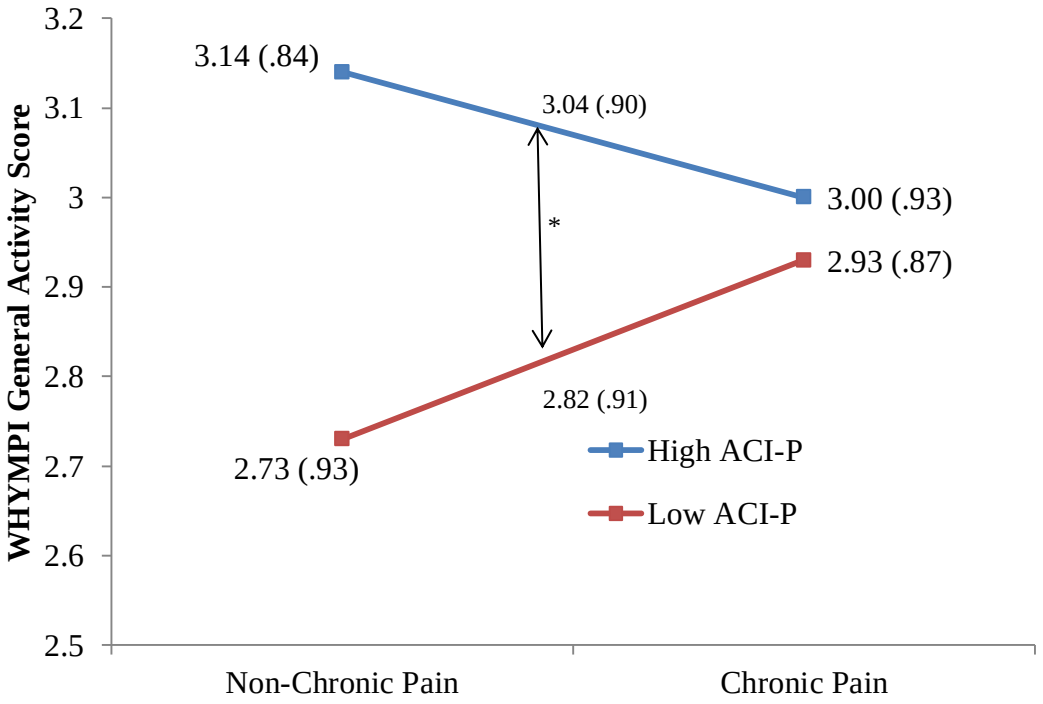


Figure 14. Group differences among high/low aging-related pain beliefs and chronic pain status on activities impacted by pain. *Significant main effect of ACI-P Beliefs, $p<.05$.

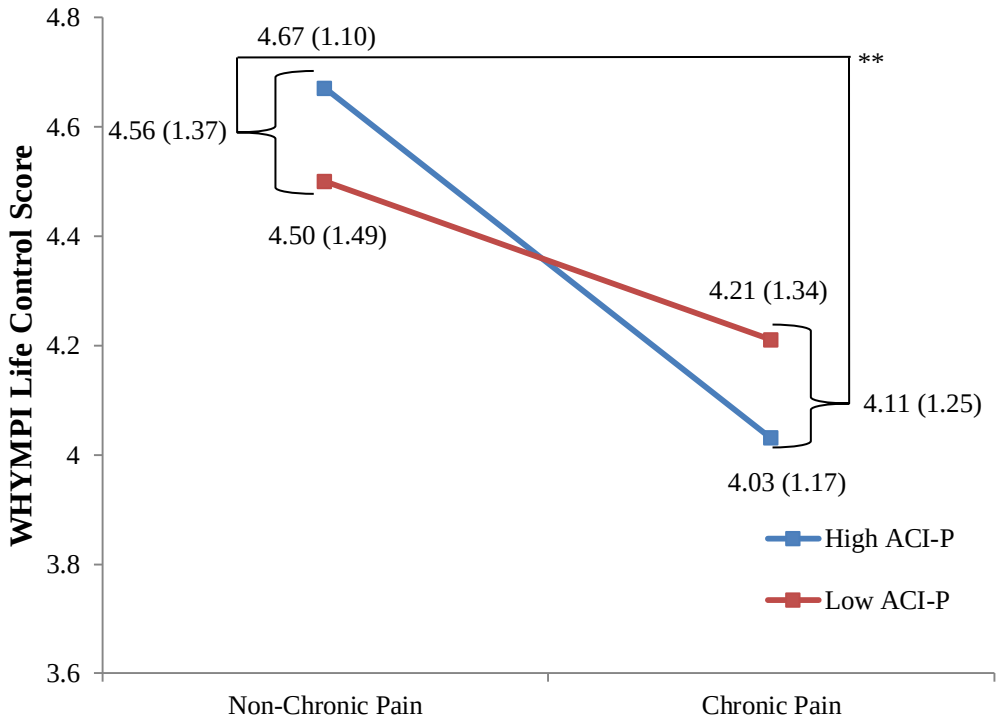


Figure 15. Group differences among high/low aging-related pain beliefs and chronic pain status on perceived life control due to pain. **Significant main effect of pain status, $p<.01$.

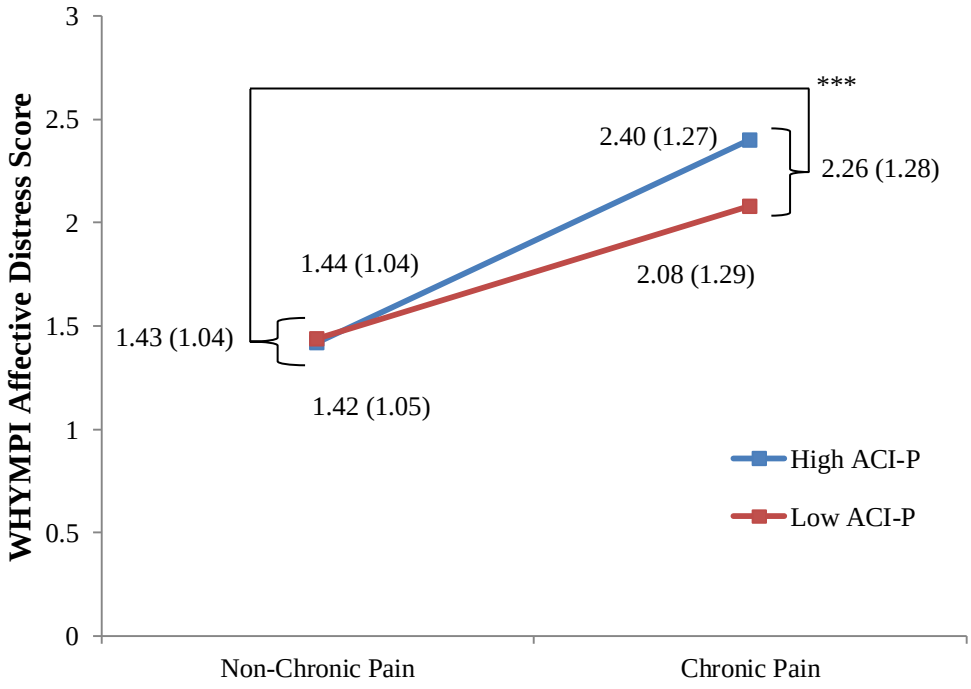


Figure 16. Group differences among high/low aging-related pain beliefs and chronic pain status on affective distress due to pain. ***Significant main effect of pain status, $p<.001$.

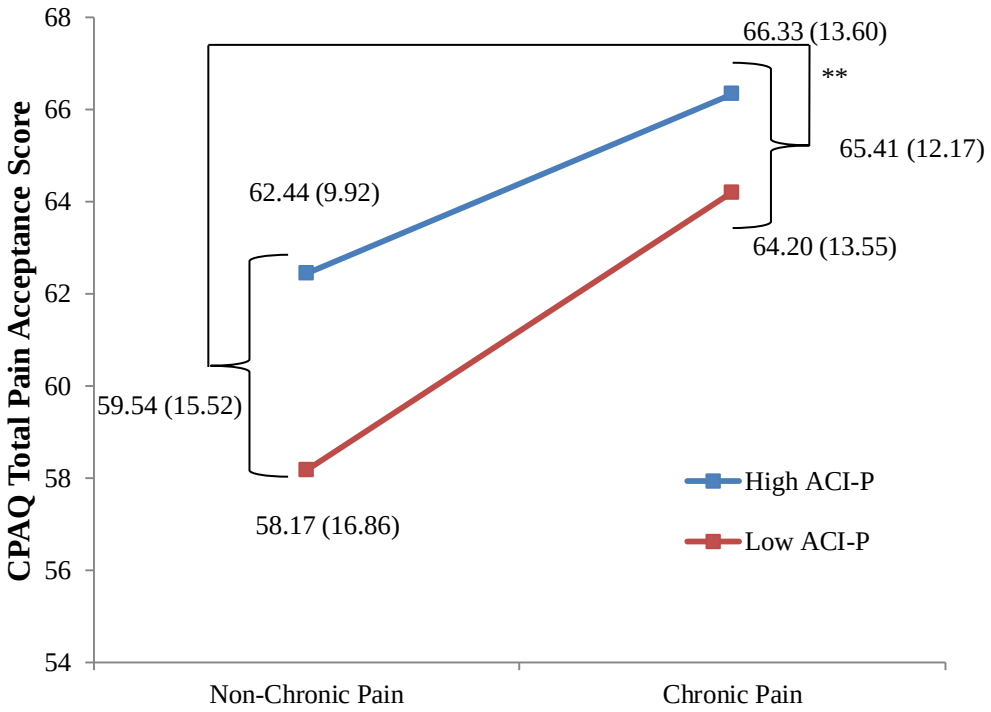


Figure 17. Group differences among high/low aging-related pain beliefs and chronic pain status on pain acceptance. **Significant main effect of pain status, $p<.01$.

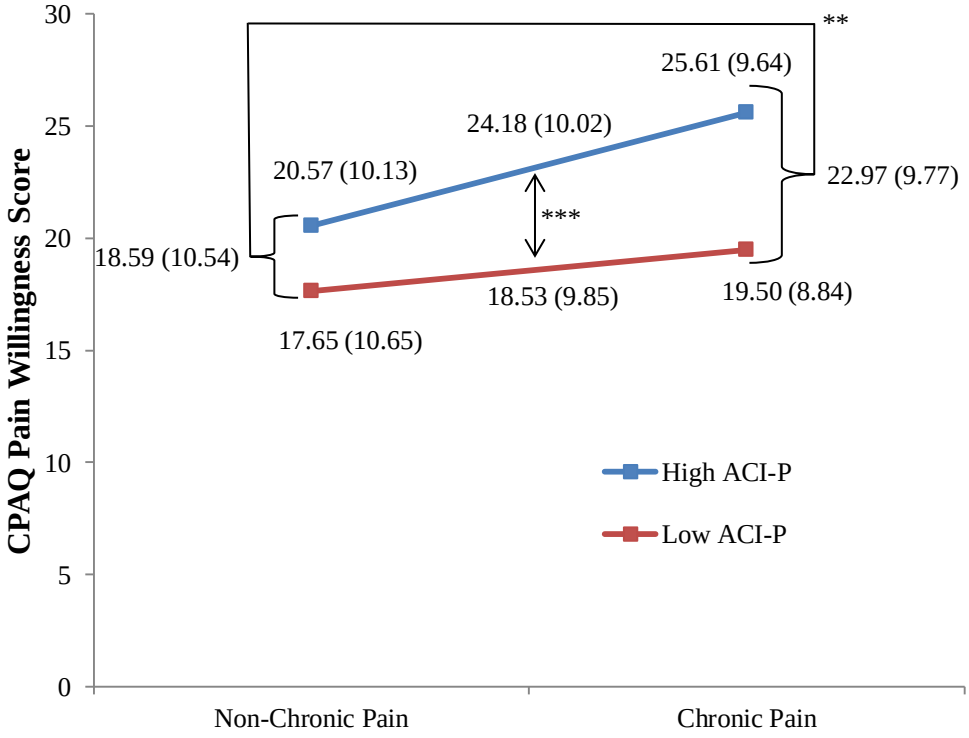


Figure 18. Group differences among high/low aging-related pain beliefs and chronic pain status on pain willingness. **Significant main effect of pain status, $p<.01$. ***Significant main effect of aging-related pain beliefs, $p<.001$.

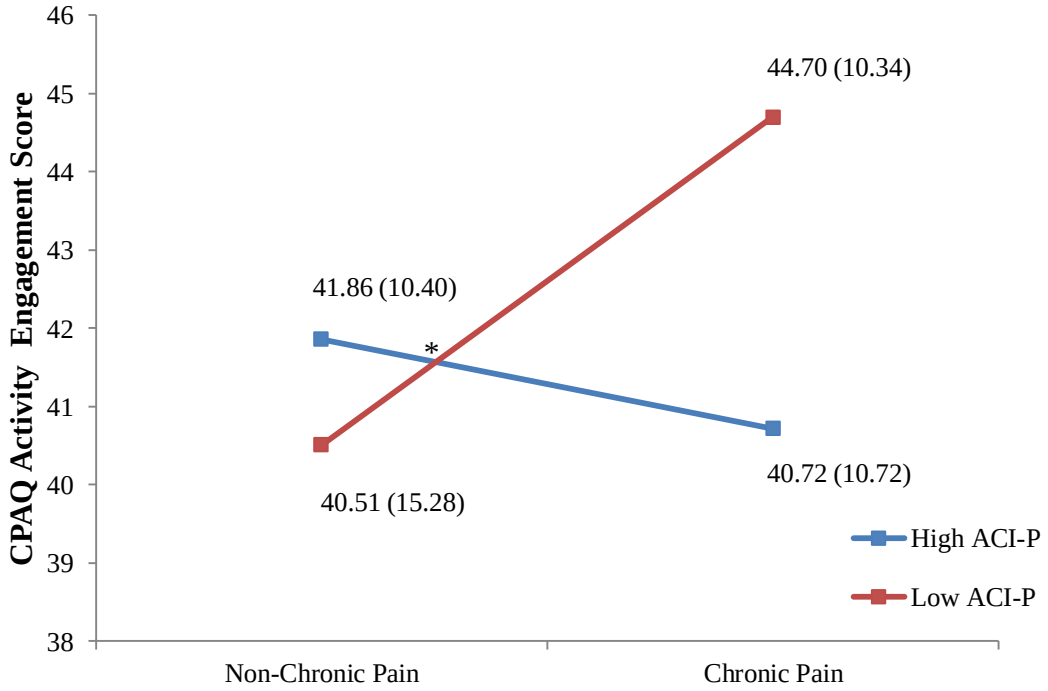


Figure 19. Group differences among high/low aging-related pain beliefs and chronic pain status on the pursuit of life activities regardless of pain. *Significant interaction effect between aging related pain beliefs and pain status, $p<.05$.

Appendix B

Beliefs about Your Pain (ACI-P)

For each statement below, circle a number to indicate the extent to which you agree or disagree.

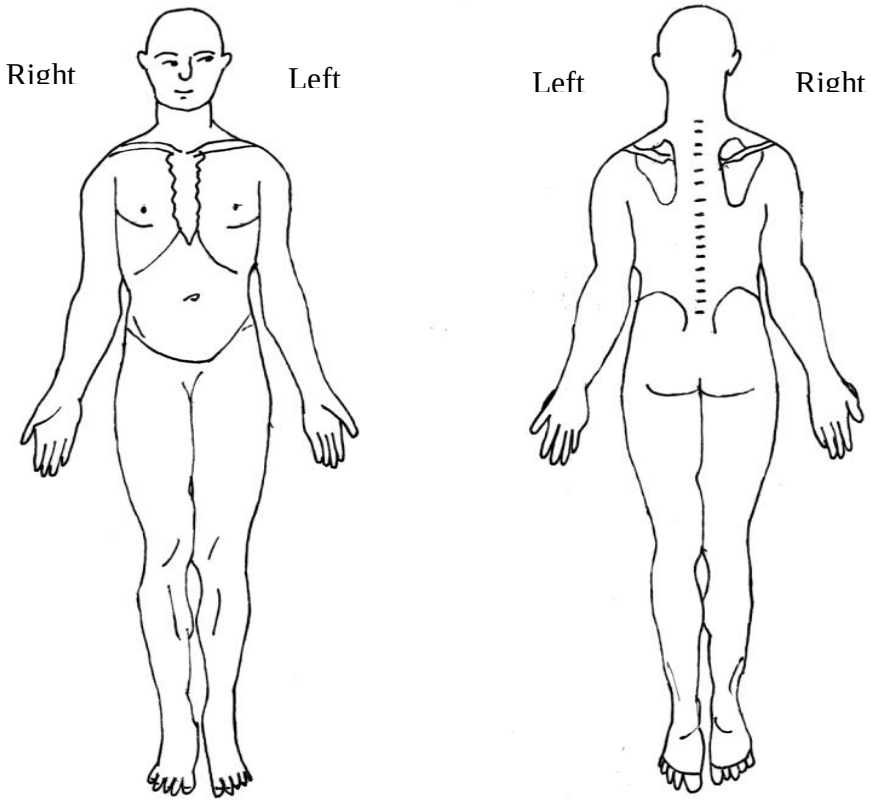
	Strongly Disagree ▼	Disagree ▼	Neutral ▼	Agree ▼	Strongly Agree ▼
Pain is an unavoidable part of getting old.	1	2	3	4	5
Old age is the main cause of pain in North America.	1	2	3	4	5
Many types of pain are caused by age.	1	2	3	4	5
It is impossible to escape being in pain when you are old.	1	2	3	4	5
Most people are in pain because of old age.	1	2	3	4	5
Although there are other causes of pain, old age is the main cause.	1	2	3	4	5
You can do some things to stay healthy, but in the end old age will result in pain.	1	2	3	4	5

Please indicate whether you have experienced pain that has persisted for longer than three months following an injury or due to an unknown cause.

_____Yes

_____No

If you responded yes, where do you **primarily** experience this pain? Either list below or place an X on the area that hurts the most.



How long have you experienced pain in this area?

_____: _____
Years Months

Appendix C

Coping Strategies (CSQ-R)

Individuals who experience pain have developed a number of ways to cope with, or deal with, their pain. These include saying things to themselves when they experience pain, or engaging in different activities. Below is a list of things that individuals have reported doing when they feel pain. For each activity, indicate, using the scale below, how when you engage in that activity when you feel pain, where a 0 indicates you never do that when you experience pain, a 3 indicates you sometimes do that when you experience pain, and a 6 indicates you always do it when you are experiencing pain. Remember, you can use any point along the scale.

When I feel pain....	Never True ▼	▼	▼	Sometimes True ▼	▼	▼	Always True ▼
I try to feel distant from the pain almost as if the pain was in somebody else's body.	0	1	2	3	4	5	6
I try to think of something pleasant.	0	1	2	3	4	5	6
It's terrible and I feel it's never going to get any better.	0	1	2	3	4	5	6
I tell myself to be brave and carry on despite the pain.	0	1	2	3	4	5	6
I tell myself that I can overcome the pain.	0	1	2	3	4	5	6
It's awful and I feel that it overwhelms me.	0	1	2	3	4	5	6
I feel my life isn't worth living.	0	1	2	3	4	5	6
I pray to God it won't last long.	0	1	2	3	4	5	6
I try not to think of it as my body, but rather as something separate from me.	0	1	2	3	4	5	6
I don't think about the pain.	0	1	2	3	4	5	6
I tell myself I can't let the pain stand in the way of what I have to do.	0	1	2	3	4	5	6
I don't pay any attention to it.	0	1	2	3	4	5	6
I pretend it's not there.	0	1	2	3	4	5	6
I worry all the time about whether it will end.	0	1	2	3	4	5	6
I replay in my mind pleasant experiences in the past.	0	1	2	3	4	5	6
I think of people I enjoy doing things with.	0	1	2	3	4	5	6
I pray for the pain to stop.	0	1	2	3	4	5	6
I imagine that the pain is outside of my body.	0	1	2	3	4	5	6
I just go on as if nothing happened.	0	1	2	3	4	5	6
Although it hurts, I just keep on going.	0	1	2	3	4	5	6
I feel I can't stand it anymore.	0	1	2	3	4	5	6
I ignore it.	0	1	2	3	4	5	6
I rely on my faith in God.	0	1	2	3	4	5	6
I feel like I can't go on.	0	1	2	3	4	5	6
I think of things I enjoy doing.	0	1	2	3	4	5	6
I do something I enjoy, such as watching TV or listening to music.	0	1	2	3	4	5	6
I pretend it's not a part of me.	0	1	2	3	4	5	6

Appendix D

WHYMPI

In the following 20 questions, you will be asked to describe your pain and how it affects your life. Under each question is a scale to record your answer. Read each question carefully and then circle a number on the scale under that question to indicate how that specific question applies to you.

1. Rate the level of your pain at the present moment.

0 1 2 3 4 5 6

No pain Very intense pain

2. In general, how much does your pain problem interfere with your day to day activities?

0 1 2 3 4 5 6

No interference Extreme interference

3. Since the time you developed a pain problem, how much has your pain changed your ability to work?

0 1 2 3 4 5 6

No change Extreme change

___ Check here, if you have retired for reasons other than your pain problem

4. How much has your pain changed the amount of satisfaction or enjoyment you get from participating in social and recreational activities?

0 1 2 3 4 5 6

No change Extreme change

5. How supportive or helpful is your spouse (significant other) to you in relation to your pain?

0 1 2 3 4 5 6

Not at all supportive Extremely supportive

6. Rate your overall mood during the past week.

0 1 2 3 4 5 6

Extremely low mood Extremely high mood

7. On the average, how severe has your pain been during the last week?

0 1 2 3 4 5 6

Not at all severe Extremely severe

8. How much has your pain changed your ability to participate in recreational and other social activities?

0 1 2 3 4 5 6
No change Extreme change

9. How much has your pain changed the amount of satisfaction you get from family-related activities?

0 1 2 3 4 5 6
No change Extreme change

10. How worried is your spouse (significant other) about you in relation to your pain problem?

0 1 2 3 4 5 6
Not at all worried Extremely worried

11. During the past week, how much control do you feel that you have had over your life?

0 1 2 3 4 5 6
Not at all in control Extremely in control

12. How much suffering do you experience because of your pain?

0 1 2 3 4 5 6
No suffering Extreme suffering

13. How much has your pain changed your marriage and other family relationships?

0 1 2 3 4 5 6
No change Extreme change

14. How much has your pain changed the amount of satisfaction or enjoyment you get from work?

0 1 2 3 4 5 6
No change Extreme change
__ Check here, if you are not presently working.

15. How attentive is your spouse (significant other) to your pain problem?

0 1 2 3 4 5 6
Not at all attentive Extremely attentive

16. During the past week, how much do you feel that you've been able to deal with your problems?

0 1 2 3 4 5 6
Not at all Extremely well

17. How much has your pain changed your ability to do household chores?

0 1 2 3 4 5 6
No change Extreme change

18. During the past week, how irritable have you been?
0 1 2 3 4 5 6
Not at all irritable Extremely irritable

19. How much has your pain changed your friendships with people other than your family?
0 1 2 3 4 5 6
No change Extreme change

20. During the past week, how tense or anxious have you been?
0 1 2 3 4 5 6
Not at all tense or anxious Extremely tense or anxious

Listed below are 18 common daily activities. Please indicate how often you do each of these activities by circling a number on the scale listed below each activity. Please complete all 18 questions.

___ Check here if you are physically disabled or physically unable to perform exercise.

1. Wash dishes.
0 1 2 3 4 5 6
Never Very Often
2. Mow the lawn.
0 1 2 3 4 5 6
Never Very Often
3. Go out to eat.
0 1 2 3 4 5 6
Never Very Often
4. Play cards or other games.
0 1 2 3 4 5 6
Never Very Often
5. Go grocery shopping.
0 1 2 3 4 5 6
Never Very Often
6. Work in the garden.
0 1 2 3 4 5 6
Never Very Often
7. Go to a movie.
0 1 2 3 4 5 6
Never Very Often

8. Visit friends.

0	1	2	3	4	5	6
Never						Very Often

9. Help with the house cleaning.

0	1	2	3	4	5	6
Never						Very Often

10. Work on the car.

0	1	2	3	4	5	6
Never						Very Often

11. Take a ride in a car.

0	1	2	3	4	5	6
Never						Very Often

12. Visit relatives.

0	1	2	3	4	5	6
Never						Very Often

13. Prepare a meal.

0	1	2	3	4	5	6
Never						Very Often

14. Wash the car.

0	1	2	3	4	5	6
Never						Very Often

15. Take a trip.

0	1	2	3	4	5	6
Never						Very Often

16. Go to a park or beach.

0	1	2	3	4	5	6
Never						Very Often

17. Do a load of laundry.

0	1	2	3	4	5	6
Never						Very Often

18. Work on a needed house repair.

0	1	2	3	4	5	6
Never						Very Often

Appendix E

Your Life and Pain (CPAQ)

Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following rating scale to make your choices.

	Never True ▼	Very Rarely True ▼	Seldom True ▼	Sometimes True ▼	Often True ▼	Almost Always True ▼	Always True ▼
I am getting on with the business of living no matter what my level of pain is.	0	1	2	3	4	5	6
My life is going well, even though I have chronic pain.	0	1	2	3	4	5	6
It's OK to experience pain.	0	1	2	3	4	5	6
I would gladly sacrifice important things in my life to control this pain better.	0	1	2	3	4	5	6
It's not necessary for me to control my pain in order to handle my life well.	0	1	2	3	4	5	6
Although things have changed, I am living a normal life despite my chronic pain.	0	1	2	3	4	5	6
I need to concentrate on getting rid of my pain.	0	1	2	3	4	5	6
There are many activities I do when I feel pain.	0	1	2	3	4	5	6
I lead a full life even though I have chronic pain	0	1	2	3	4	5	6
Controlling my pain is less important than any other goals in my life.	0	1	2	3	4	5	6
My thoughts and feelings about pain must change before I can take important steps in my life.	0	1	2	3	4	5	6
Despite the pain, I am now sticking to a certain course in my life.	0	1	2	3	4	5	6

	Never True ▼	Very Rarely True ▼	Seldom True ▼	Sometimes True ▼	Often True ▼	Almost Always True ▼	Always True ▼
Keeping my pain level under control takes first priority whenever I'm doing something.	0	1	2	3	4	5	6
Before I can make any serious plans, I have to get some control over my pain.	0	1	2	3	4	5	6
When my pain increases, I can still take care of my responsibilities.	0	1	2	3	4	5	6
I will have better control over my life if I can control my negative thoughts about pain.	0	1	2	3	4	5	6
I avoid putting myself in situations where my pain might increase.	0	1	2	3	4	5	6
My worries and fears about what pain will do to me are true.	0	1	2	3	4	5	6
It's a great relief to realize that I don't have to change my pain to get on with life.	0	1	2	3	4	5	6
I have to struggle to do things when I have pain.	0	1	2	3	4	5	6

Appendix F

Global Physical Activity Questionnaire (GPAQ)

Please answer these questions even if you do not consider yourself to be a physically active person. Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting, seeking employment. In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate.

Activity at work

1. Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like carrying or lifting heavy loads, digging or construction work for at least 10 minutes continuously?

VIGOROUS Intensity Activities

Make you breathe <u>much</u> harder than normal

Examples for vigorous activities at WORK

Other examples for VIGOROUS activities at WORK

- Forestry (cutting, chopping, carrying wood)
- Sawing hardwood
- Ploughing
- Cutting crops (sugar cane)
- Gardening (digging)
- Grinding (with pestle)
- Laboring (shoveling sand)
- Loading furniture (stoves, fridge)
- Instructing spinning (fitness)
- Instructing sports aerobics
- Sorting postal parcels (fast pace)
- Cycle rickshaw driving

_____Yes

_____ No (If No, go to question 4)

2. In a typical week, on how many days do you do vigorous-intensity activities as part of your work?

Number of days _____

3. How much time do you spend doing vigorous-intensity activities at work on a typical day? Hours : minutes _____: _____

hrs mins

4. Does your work involve moderate-intensity activity that causes small increases in breathing or heart rate such as brisk walking [or carrying light loads] for at least 10 minutes continuously?

MODERATE Intensity Activities

Make you breathe somewhat harder than normal

Examples MODERATE activities at work




Other examples for MODERATE activities at WORK

- Cleaning (vacuuming, mopping, polishing, scrubbing, sweeping, ironing)
- Washing (beating and brushing carpets, wringing clothes (by hand)
- Gardening
- Milking cows (by hand)
- Planting and harvesting crops
- Digging dry soil (with spade)
- Weaving
- Woodwork (chiseling, sawing softwood)
- Mixing cement (with shovel)
- Laboring (pushing loaded wheelbarrow, operating jackhammer)
- Walking with load on head
- Drawing water
- Tending animals

_____ Yes

_____ No (If No, go to question 7.)

5. In a typical week, on how many days do you do moderate-intensity activities as part of your work?

Number of days _____

6. How much time do you spend doing moderate-intensity activities at work on a typical day?

Hours : minutes _____: _____
hrs mins

Travel to and from places

The next questions exclude the physical activities at work that you have already mentioned.

Now I would like to ask you about the usual way you travel to and from places (e.g., to work, for shopping, or to your place of worship).

7. Do you walk or use a bicycle (pedal cycle) for at least 10 minutes continuously to get to and from places?

_____Yes

_____No (If No, go to question 10)

8. In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places?

Number of days _____

9. How much time do you spend walking or bicycling for travel on a typical day?

Hours : minutes _____: _____
hrs mins

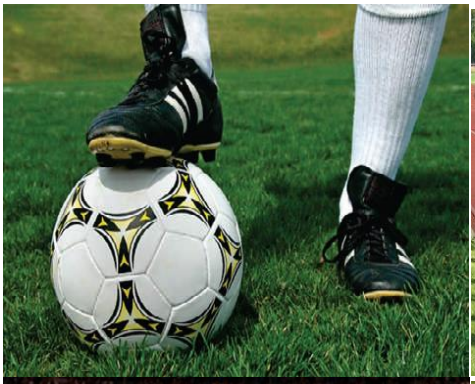
Recreational activities

The next questions exclude the work and transport activities that you have already mentioned.
Now I would like to ask you about sports, fitness and recreational activities (leisure).

10. Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like running or football for at least 10 minutes continuously?

VIGOROUS Intensity Activities During Leisure Time
Make you breathe <u>much</u> harder than normal

Examples for VIGOROUS activities during LEISURE TIME



Other examples for VIGOROUS activities during LEISURE TIME

- Soccer
- Rugby
- Tennis
- High-impact aerobics
- Aqua aerobics
- Ballet dancing
- Fast swimming

_____Yes

_____No (If No, go to question 13)

11. In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (leisure) activities?

Number of days _____

12. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?

Hours : minutes _____: _____
hrs mins

13 Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that causes a small increase in breathing or heart rate such as brisk walking, cycling, swimming, volleyball for at least 10 minutes continuously?

MODERATE Intensity Activities
Make you breathe somewhat harder than normal

Examples for MODERATE activities during LEISURE TIME

Other examples for MODERATE activities at WORK

- Cycling
- Jogging
- Dancing
- Horse-riding
- Tai chi
- Yoga
- Pilates
- Low-impact aerobics

_____Yes

_____No (If No, go to question 16)

14. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (leisure) activities?

Number of days _____

15. How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?

Hours : minutes _____:_____
hrs mins

Sedentary Behavior

The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent [sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television], but do not include time spent sleeping.

16. How much time do you usually spend sitting or reclining on a typical day?

Hours : minutes _____:_____
hrs mins

Appendix G

Demographics Questionnaire

1. Age: _____

2. Sex:
   Woman
   Man
   Other (please specify): _____

3. Education:
___ Sixth grade or less
___ Completed 8th grade
___ Some high school
___ Completed high school
___ GED
___ Technical degree
___ some college
___ 2 year college degree
___ 4 year college degree
___ some graduate school
___ completed a graduate program

4. Employment Status:
   ___ full-time
   ___ part-time
   ___ occasional
   ___ disability/SSI
   ___ retired
   ___ no income

5. Marital/Relationship Status:
   ___ single
   ___ divorced
   ___ widowed
   ___ married
   ___ living with partner
   ___ dating, but not living with partner

6. Ethnicity (check all that apply):
   ___ African American/Black
   ___ Caribbean / Haitian
   ___ African
   ___ Asian American
   ___ Asian / Pacific-Islander
   ___ White / European American / Caucasian
   ___ European
   ___ Hispanic American / Hispanic

___ Native American / American Indian
___ Other _____

7. Are you are current member of the military or veteran?
____ Yes
____ No

8. Household Income:
___ Under $10,000
___ $10,000 - $20,000
___ $20,000 - $40,000
___ $40,000 - $60,000
___ $60,000 - $80,000
___ $80,000 - $100,000
___ Over $100,000
___ Prefer not to disclose

Appendix H

General Health Questionnaire
Your responses to these questions are CONFIDENTIAL and will not be shared with anyone.

1. What is your height? _____ feet, _____ inches

2. What is your weight in pounds? _____lbs.

3. With regard to your weight, do you consider yourself (please check ONE):
___ Underweight
___ Normal Weight
___ Overweight
___ Obese

4. Have you smoked at least 100 cigarettes in your entire life?
___ No
___ Yes

5. How often do you currently smoke cigarettes?
___ Not at all
___ Some days
___ Most days
___ Everyday

6. How often do you drink alcohol?
___ Not at all
___ Some days
___ Most days
___ Everyday

7. How many alcoholic drinks (drink = one 12oz. can of beer, 5 ounces of wine, or 1.5/one shot of distilled spirits) do you have in a typical week? _____

8. Below is a list of health problems that people may have. Please circle a response to indicate (Yes/No) if you have ever had any of these health problems.

	NO ▼	YES ▼	**If YES, is this condition current?** NO	YES
Heart condition or circulation problems (hardening of the arteries, heart disease, heart failure	N	Y	N	Y
High blood pressure (hypertension)	N	Y	N	Y
High cholesterol	N	Y	N	Y
Have had a heart attack	N	Y	N	Y
Have had a stroke	N	Y	N	Y
Anemia or other blood diseases	N	Y	N	Y
Arthritis or rheumatism	N	Y	N	Y
Fibromyalgia	N	Y	N	Y
Stomach trouble (upper and lower gastro-intestinal problems)	N	Y	N	Y
Emotional Difficulties (depression, anxiety, etc.)	N	Y	N	Y
Schizophrenia or schizoaffective disorder	N	Y	N	Y
Liver disease (cirrhosis, hepatitis)	N	Y	N	Y
Osteoporosis	N	Y	N	Y
Diabetes	N	Y	N	Y
Multiple sclerosis (MS)	N	Y	N	Y
HIV, AIDS	N	Y	N	Y

Appendix I

SDS-17

Instructions. Below you will find a list of statements. Please read each statement carefully and decide if that statement describes you or not. If it describes you, select the word "true"; if not, select the word "false".

1. I sometimes litter	True	False
2. I always admit my mistakes openly and face the potential negative consequences.	True	False
3. In traffic I am always polite and considerate of others.	True	False
4. I always accept others' opinions, even when they don't agree with my own.	True	False
5. I take out my bad moods on others now and then.	True	False
6. There has been an occasion when I took advantage of someone else.	True	False
7. In conversations I always listen attentively and let others finish their sentences.	True	False
8. I never hesitate to help someone in case of emergency.	True	False
9. When I have made a promise, I keep it – no ifs, ands, or buts.	True	False
10. I occasionally speak badly of others behind their back.	True	False
11. I would never live off at other people's expense.	True	False
12. I always stay friendly and courteous with other people, even when I am stressed out.	True	False
13. During arguments I always stay objective and matter-of-fact.	True	False
14. There has been at least one occasion when I failed to return an item that I borrowed.	True	False
15. I always eat a healthy diet.	True	False
16. Sometimes I only help because I expect something in return.	True	False

Note. One item was removed from the final version of the SDS-17.