Coping with Chronic Illness: Do Strategies Differ by Illness Type?

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Coping with Chronic Illness: Do Strategies Differ by Illness Type?

by

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in partial fulfillment of the requirements for the degree of

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Dean of the Graduate School

8/5/11
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Abstract

While coping varies with individuals and is a product of complex, interrelated factors, the primary interest of this study was determining how coping behaviors manifest according to these chronic illness types: those of known pathology (e.g., asthma, osteoarthritis, lupus) and those that lack a clear, structural pathology (i.e., functional somatic syndromes and medically unexplained symptoms). Data for this study were gathered from a series of four comprehensive online surveys, which included measures of coping (Brief COPE), pain and health outcomes (SF-36), negative affect (I-PANAS-SF) and depression (PHQ-8). The analyzed sample was comprised of 148 participants (119 Female, 28 Male and 1 Not Answered) with a mean age of 43.34 ($SD = 13.69$), all of whom experienced at least three months of chronic physical symptoms. Based on diagnosis, the participants were grouped into that of conventional disease (CD), functional somatic syndromes (FSS) or medically unexplained symptoms (MUS). Even while controlling for significant covariates (e.g., depression, negative affect, pain perception), multivariate analyses revealed no significant differences in coping strategies by illness group, Wilks’s Lambda $= .96$, $F(4, 222) = 1.05$, $p = .38$.

The results suggest that the use of coping strategies does not differ by illness type, but can be predicted by other, health-related factors, notably stress, $\beta = -.21$, $t(120) = -2.09$, $p = .04$; symptoms, $\beta = .32$, $t(120) = 2.82$, $p = .01$; personal control, $\beta = .19$, $t(120) = 2.16$, $p = .03$, and negative affect, $\beta = .34$, $t(121) = 2.81$, $p = .01$. It appears that patient experience with chronic illness can be more informative regarding appropriate treatments and therapeutic interventions than just illness type itself.
Coping with Chronic Illness: Do Strategies Differ by Illness Type?

According to recent estimates, chronic illnesses are widely prevalent; in 2005, a reported 133 million Americans, or nearly 1 out of every 2 adults had at least one chronic illness (Centers for Disease Control, 2010; Kung, Hoyert, Xu, & Murphy, 2008). The leading cause of death and disability, chronic illness is distressing and costly, purportedly costing the U.S. economy more than $1 trillion annually, a figure that represents both the explicit costs of health care as well as the indirect costs of productivity losses (Centers for Disease Control, 2010; DeVol, Bedroussian, & Charuworn, 2007). Due to the sustained nature of chronic illnesses and their pervasive, potentially severe impact, it is necessary to identify ways in which illnesses can be effectively prevented, treated or managed. This requires articulation of the possible factors at work in the etiopathogenesis and perpetuation of certain chronic illness experiences, particularly those that lack distinct, uniformly presenting and universally accepted diagnostic criteria, those that occur at the interface of pathophysiology and psychopathology (Henningsen, Zipfel, & Herzog, 2007; McFarlane, Ellis, Barton, Browne, & Van Hooff, 2008). Coping, integral to illness and symptom management, is a function of these multiple interacting factors as they affect cognitive appraisal, behavioral response and adjustment. Determining whether different coping patterns are associated with certain illness experiences can provide useful information regarding illness treatment and patient outcomes.

**Stress and Coping**

Living with chronic illness requires the ability to adapt to living with the stressors of unremitting symptoms, such as pain, fatigue, depression and anxiety. While recovery is the desired outcome of illness, for those with chronic illnesses, the more attainable
outcome may be that of maintained psychological and physiological well-being in the face of these chronic demands on fitness, a process of “stability through change” known as *allostasis* (McEwen, 2007; Sterling, 2004; Sterling & Eyer, 1988 in Stewart et al., 1989). The use of effective coping strategies, mechanisms by which people respond to and manage stress, is essential to achieving this adaptive dynamic, particularly when illness course and outcomes are uncertain (Cairns & Hotopf, 2005; Carver, Scheier & Weintraub, 1989; Lazarus & Folkman, 1984).

**Appraisal.** The transactional model of stress and coping by Lazarus and Folkman (1984) regards coping as a function of stress appraisal; stress is defined in their model as perceived threats to well-being or of demands that are perceived to be taxing of individual internal and/or external resources (Folkman & Moskowitz, 2004; Lazarus & Folkman, 1984). Essential to their model is the idea that stress and coping is a *transaction* mediated by the person’s appraisal of both the perceived threat and the perceived resources available to respond to the threat (Carver et al., 1989; Lazarus & Folkman, 1984; Glanz, Rimer, & Lewis, 2002, p.215). As a result, coping is a highly perceptually-driven process that yields strategies befitting individual needs across a variety of situations (Carver et al., 1989; Lazarus & Folkman, 1984; Schreurs & de Ridder, 1997; Walker, Jackson, & Littlejohn, 2004).

**Coping response.** Stressors provoke coping responses that fall into two general categories: *problem-focused coping*, which involves directing action to resolve or alter the threat and *emotion-focused coping*, which involves management of the emotions elicited by the threat (Carver et al., 1989; Folkman & Lazarus, 1980). Problem-focused coping is oriented to approaching the source of stress, encompassing coping strategies
such as planning, problem-solving, restraint, seeking information/instrumental advice and cognitive reappraisal (Carver et al., 1989; Feder, Nestler, Westphal, & Charney, 2010; Thoits, 1995). Emotion-focused coping is managing and/or mitigating problematic emotional responses to stress, such as fear and distress, by using strategies like avoidance, denial, venting, disengagement, acceptance, positive reappraisal and emotional support (Carver et al., 1989; Thoits, 1995). Typically, people employ both forms of coping to some degree when managing stress, adjusting their strategies as context and individual preferences dictate (Folkman & Lazarus, 1980).

**Stress regulation and neural correlates.** Stress appraisal and coping responses are largely associated with the construct of executive functioning, the interrelated and effortful cognitive processes involved in attention, perception, problem solving, strategizing, task-switching, emotion regulation and behavioral modification (Hall, Crossley, & D’Arcy, 2010; Nes, Roach, & Segerstrom, 2009; Williams, Suchy, & Rau, 2009; Williams & Thayer, 2009). Regardless of the type or combination of strategies used, all coping requires executive function to exert self-control over cognitive, emotional, and behavioral responses to stress (Nes et al., 2009). The process of conscious cognitive self-management, or self-regulation, is particularly important as it can direct stress appraisal and reactivity (Maes & Karoly, 2005; Williams et al., 2009; Williams & Thayer, 2009). While self-regulation is the act of self-management, the perception of capability or effective performance is typically regarded as self-efficacy; this theoretical concept, interwoven with factors like attribution, motivation and expectation, influences coping strategy selection (Bandura, 1997 in Glanz et al., 2002, p.215; Deary, Chalder, & Sharpe, 2007). For example, an individual that perceives greater controllability or sense
of mastery regarding a stressor is more likely to use the more *active* problem-focused and emotion-focused coping strategies, like problem solving and positive reappraisal; conversely, one that perceives less controllability is more likely to use the more *passive* and *avoidant* coping strategies, like avoidance, denial and escapism, as the stressor is seen as something not able to be changed, but ‘endured’ (Carver et al., 1989; Folkman & Lazarus, 1980; Thoits, 1995).

Findings from brain imaging and behavioral studies of those with frontal lobe damage suggest that executive functions are orchestrated by the prefrontal cortex (PFC) and connections along the frontoparietal network (Nes et al., 2009; Williams et al., 2009; Williams & Thayer, 2009). Associated brain structures are the amygdala, thalamus and hippocampus, structures that feature in the limbic system, which is implicated in memory and top-down regulatory processing (Nes et al., 2009; Williams et al., 2009; Williams & Thayer, 2009). Damage to the PFC is associated with poorer executive functioning, which can manifest as disinhibition, perseveration, inflexibility, organizational and attention deficits and impairments in task switching and problem solving (Nes et al., 2009; Rossi, Pessoa, Desimone, & Ungerleider, 2009; Williams et al, 2009). Factors that inhibit or disrupt the executive operations of the brain, such as lesions to the PFC, dysfunctional network communication with components of the brainstem (which feature in arousal and evolutionarily-wired, autonomic responses) or other functional neuroconnectivity and processing issues, can negatively influence or interfere with the cognitive operations of the stress response (Sterling, 2004; Williams et al., 2009; Williams & Thayer, 2009).

**Stress regulation in chronic illness.** Chronic illness poses a significant challenge
to successful coping because chronic illness induces stress on an already vulnerable individual; the demands of illness compounded by the accumulative effects of stress may impose severe limitations on a sufferer’s ability to self-regulate to manage stress (Heijmans et al., 2004). There is empirical evidence that self-regulatory ability is subject to fatigue and that as an internal resource, it is subject to limits that vary among individuals and can be depleted (Nes et al., 2009; Williams & Thayer, 2009). Stressors can drain self-regulatory strength, inhibiting perceptual-sensitive processes like coping.

As it appears that it is a person’s subjective experience with stressors that yields variability of coping response, alterations in the perceptual process of stress appraisal could make illness symptoms and stressors virtually ‘intolerable’ as sensations are amplified and tolerance is reduced (Bombardier, D'Amico, & Jordan, 1990; Leventhal et al., 1997; Maes et al., 1996 in Heijmans et al., 2004; Heijmans et al., 2004; Tak & Rosmalen, 2010; Van Houdenhove, Van Den Eede, & Luyten, 2009; Yunus, 2007). Research on pain, a common symptom and stressor in chronic illness, illustrates that intensity of sensation can be modulated by emotional regulation and cognitive processes, like attention and expectation (Wiech, Ploner, & Tracey, 2008). In addition to amplifying symptom perception, perceptual alterations associated with chronic stress can exaggerate the sensations of peripheral, irrelevant stimuli, interfering with healthy functioning as threat perception and hyper-reflection or hypervigilance for threats increase (Deary et al., 2007). This cognitive bias to detect negative or fear-related stimuli, in conjunction with enhanced bodily sensitization, sustains arousal and stress activation (Deary et al., 2007). As the attentional system is automatically oriented to threats, hypervigilance for threats can restrict available cognitive resources required for
information processing and self-regulation (Putman & Roelofs, 2011; Ursin, 2005 in Deary et al., 2007).

**A Model of Stress Dysregulation**

**Chronic activation of the stress response.** The stress of chronic illness can heighten threat detection and initiate greater stress reactivity, prompting repeated mobilization of physiological and psychological responses. Stress triggers cascades of neuroendocrine effectors, such as the secretion of hypothalamic corticotrophin-releasing hormone (CRH) and arginine vasopressin, as well as the glucocorticoid cortisol by the adrenal gland and the brainstem-derived catecholamines norepinephrine and epinephrine. These effectors activate the “fight-or-flight” response of the sympathetic nervous system (e.g., increased arousal, accelerated heart and metabolic rate, suppression of the “rest and digest” activities of the parasympathetic nervous system) and executive application of coping mechanisms, mediated by activation of the hippocampus and amygdala, to address the source of stress or to manage arousal and distress (Chrousos, 2009; McFarlane et al., 2008; Nes et al, 2009; Olff, Langeland, & Gersons, 2005; Williams et al., 2009; Williams & Thayer, 1999). The framework behind *allostatic load* suggests that repeated stress response activation leads to greater “wear and tear” of the stress system, which is characterized by physiological indices of stress *dysregulation* or dysfunctional operation of the processes of the neuroendocrine and immune systems (Feder et al., 2010; Glei, Goldman, Chuang, & Weinstein, 2007). The hypothalamic-pituitary-axis (HPA) axis of the PFC, the locus ceruleus-norepinephrine (LC-NE) system, the mood-associated serotonergic system, and the reward-mechanisms of the dopaminergic system are neurobiological components of the stress system; dysregulation of one or more of these
components, reflected in hypo or hyperactivity of neuroendocrine effector secretion, suggests malfunctioning of the stress system (Feder et al., 2010; Haglund, Nestadt, Cooper, Southwick, & Charney, 2007; Van Houdenhove et al., 2009).

**A dysregulated system.** An implication of dysregulation is that once arrived, it persists. As stressors trigger inadequate or inappropriate stress responses, executive functions necessary to self-regulate and “override” are impaired by the neurobiological effects of dysregulation, thus maintaining, or even intensifying, the cycle of stress system dysfunction (Chrousos, 2009; Van Houdenhove & Luyten, 2008; Van Houdenhove et al., 2009). Irregular activity of the stress system is associated with structural changes in the hippocampus and amygdala; changes in these memory and emotion-associated structures are implicated in maladaptive executive functions, like hypervigilance and rumination (McEwen & Milner, 2007 *in* Feder et al., 2010). Hori et al. (2010) tested the relationship between irregular stress activity and executive functions in 121 healthy adults, dividing them into three groups based on their cortisol secretion suppression pattern (blunted or suppressed patterns are indices for stress system irregularities). According to self-report of psychological distress and coping styles, in comparison to the other groups, the enhanced suppressors of cortisol reported higher levels of anxiety, obsessive-compulsiveness and greater uses of avoidant coping strategies (Hori et al., 2010). While this study was conducted with non-clinical adults with differing cortisol patterns, it shows that stress dysregulation is associated with poorer “top-down” executive functioning and maladaptive behaviors.

**Illnesses associated with stress dysregulation.** Functional somatic syndromes (FSS), which include fibromyalgia, chronic fatigue syndrome (CFS) and
irritable bowel syndrome (IBS), are syndromes that lack a clearly established
pathophysiology (e.g., tissue damage, structural abnormality) and are characterized by the
suffering of a constellation of chronic physical symptoms, like muscle aches, nausea,
palpitations and sleeping difficulties (Barsky & Borus, 1999; Sharpe & Carson, 2001;
Yunus, 2007). Often, the distinction between FSS and medically unexplained symptoms
(MUS) is the official diagnosis for FSS by a physician; in some studies, illnesses of
unknown etiology, like CFS and IBS, are considered MUS because of their biomedically
“unexplained” nature (Deary et al., 2007; McFarlane et al., 2008). The multiple
functional somatic syndromes and MUS share considerable overlap of symptoms (e.g.,
many fibromyalgia patients report severe fatigue and many chronic fatigue patients report
chronic pain); as a result, speculation remains as to whether the many syndromes are
really just different bodily manifestations of the same underlying disorder (Henningsen et
al., 2007; Yunus, 2007). Researchers operating from the stress dysregulation hypothesis
argue that the presence of persistent physical symptoms of unknown etiology, as in FSS
and MUS, may result from and be perpetuated by stress response abnormalities; there
may be nominal and diagnostic differences, but the disorders may essentially be the same
(Chrousos, 2009; Heim, 2009; McFarlane et al., 2008; Van Houdenhove & Egle, 2004;
Van Houdenhove et al., 2009; Yunus, 2007).

Because of the frequent comorbidity of depression and/or anxiety with FSS and
MUS, it was previously hypothesized that FSS/MUS symptom experience resulted from
somatization or even malingering by the depressed or chronically anxious patient. To
examine relationships between FSS/MUS, anxiety and depression, Henningsen,
Zimmermann, and Sattel (2003) conducted a meta-analysis of 244 observational studies,
which encompassed 18,690 participants, focusing on comparisons between FSS (i.e., irritable bowel syndrome, nonulcer dyspepsia, fibromyalgia and chronic fatigue syndrome) and healthy controls (Henningsen et al., 2003). While they found that the association between FSS with depression and anxiety was significantly higher than that of controls, the studied syndromes were shown to be independent of anxiety and depression, dispelling the notion that the somatic symptoms of these syndromes arose from depression and anxiety (Henningsen et al., 2003; McFarlane et al., 2008).

Physiological indices suggestive of stress system dysregulation have been found in those with FSS. In several studies, including a meta-analysis of 14 studies on subjects with FSS and a population-based study of FSS in 744 adults, lower baseline cardiac vagal activity (derived from heart rate variability, a proxy for increased arousal and reduced parasympathetic activity) was associated with FSS (Tak & Rosmalen, 2010). There was no evidence that this cardiac vagal activity differed significantly among the syndromes of chronic fatigue syndrome, fibromyalgia and irritable bowel syndrome (Tak & Rosmalen, 2010). While these results suggest that there are alterations of the autonomic nervous system in those with FSS, other studies suggest a general correlation between FSS and irregular HPA axis activity in the central nervous system (McFarlane et al., 2008; Tak & Rosmalen, 2010). Lower cortisol levels and a flattened diurnal rhythm of cortisol (i.e., in a diurnal pattern of enhanced suppression: lower levels upon awakening in the morning and higher levels in the evening), considered indices of irregular HPA activity, have been found in those with chronic fatigue syndrome, fibromyalgia and post-traumatic stress disorder (PTSD) (McFarlane et al., 2008; Tak & Rosmalen, 2010). Immunology research also suggests a link between deficient HPA axis functioning and neuroimmune
activation; chronic activation by release of inflammatory cytokines can induce a systemic sickness response that includes symptoms that resemble that of FSS: fatigue, flu-like symptoms, mood disturbance, cognitive deficits and hyperalgesia (Chrousos, 2009; Van Houdenhove et al., 2009).

The sickness symptoms experienced by those with FSS and MUS are prevalent in the general, non-clinical population. Yet, it is those with FSS and MUS that experience these symptoms to a degree that can be disabling (Barsky & Borus, 1999). Through the lens of the integrative model of stress system dysregulation, these symptom experiences may result from heightened central and peripheral neuronal sensitivity to stimuli proposed to manifest from and/or exist concurrently with a complex interaction of factors, such as prior trauma, biological predisposition, psychosocial vulnerability and lifestyle characteristics, like obesity, medication use or physical inactivity (Tak & Rosmalen, 2010; Van Houdenhove & Luyten, 2008; Van Houdenhove et al., 2009; Yunus, 2007). While it is not fully clear whether this heightened sensitivity precipitates stress dysregulation or if these overlapping “sensitivity” syndromes emerge as products of dysregulation, the relationship appears to be bidirectional (Van Houdenhove & Luyten, 2008; Yunus, 2007). As regulatory anomalies perpetuate the disease experience, amplifying symptoms of pain, fatigue and stress, chronic activation of the stress system in response to illness symptoms can yield physiological and maladaptive alterations of the stress system (Chrousos, 2009; Henningsen et al., 2007; Nes et al., 2009; Sanders & Korf, 2007; Tak & Rosmalen, 2010; Van Houdenhove et al., 2009).

**Impact of Stress Dysregulation on Appraisal and Coping**
In the context of chronic illness, coping strategies are not only dependent on how illness symptoms and illness consequences are appraised, but emerge from a combined influence of factors along with illness, which include age, gender, available resources, affect, perceived social support, symptom duration and intensity and perceived impact on what the individual values (e.g., employment, romantic relationship, lifestyle; Folkman & Moskowitz, 2004; Schreurs & de Ridder, 1997). As a result of this complexity, teasing apart whether coping strategies are associated with certain illness experiences independent from other relevant factors can be difficult.

**Gender and affect confounds.** There is empirical support for coping variation by individual differences like gender and negative affect (Carver et al., 1989; Folkman & Moskowitz, 2004). Women have reported greater use of active and passive emotion-focused coping strategies, while negative affect has shown association with greater passive strategies like avoidance (Carver et al., 1989; Folkman & Moskowitz, 2004; Matud, 2004). This gender distinction is important to consider because of the “preponderance” of women reporting the symptoms of FSS and epidemiological data that indicate that women report higher rates of depression and pain severity (Henningsen et al., 2007; Munce & Stewart, 2007). How gender features in the incidence of FSS, whether psychosocially in gender roles as they impact stress appraisal and/or as possible neuroendocrine correlates of affective/perceptual differences, is relevant because of the tendency for FSS patients to be women (Fantoni et al., 2007; Matud, 2004).

Still, gender alone does not predict coping, even in FSS and MUS. A study by Nater and colleagues (2006) of chronic fatigue symptoms in the general population of
Wichita, Kansas evaluated the coping strategies of a sample of 164 men and women in which 43 were diagnosed with chronic fatigue syndrome, 61 with chronic unexplained fatigue and 60 were healthy controls (Nater et al., 2006). Though the men-to-women ratio of the sample was approximately 1:4, each group approximated the same ratio. They found that the CFS and chronic unexplained fatigue group did not significantly differ from one another, but that both groups used significantly more escape-avoidance strategies than controls (Nater et al., 2006). The difference in strategies was not associated with gender, but with chronic illness.

**Appraisal and attentional biases.** More research findings have emerged to suggest that patterns of coping in chronic illnesses could be illness or illness-cluster associated, particularly if the illness is stress dysfunction-related. People suffering from illnesses characterized by heightened stress-associated perceptual sensitivity, as in FSS and MUS, would theoretically be less equipped to tolerate and manage stress, especially as internal resources are depleted, allostatic load is reached, and executive functions required to self-regulate are inhibited or compromised (Nes et al., 2009; Van Houdenhove et al., 2009; Yunus, 2007). Perceptual sensitivity yields a chronically reactive stress system as the individual interprets even peripheral, insignificant stimuli as stressful or threatening, triggering the neuroendocrine responses that can further sensitize the individual (Olff et al., 2005). Olff and colleagues (2005) regard this appraisal process as the “crucial first step” that can lead to symptom experience. Furthermore, in their review of stress and trauma studies, they determined that “conscious threat perception is necessary” for stress dysregulation to occur (Olff et al., 2005).
**Distress avoidance and regulatory fatigue.** The association of FSS and MUS with passive coping strategies is well documented, even if the mechanisms of this association are not understood (Barsky & Borus, 1999; Nater et al., 2006; Van Houdenhove et al., 2009; Zautra, Hamilton, & Burke, 1999). Studies of chronic fatigue syndrome, chronic pain conditions (including fibromyalgia, temporomandibular disorder and irritable bowel syndrome) illustrate that those with such conditions engage in more passive coping strategies, like avoidance, disengagement, inactivity, escapism and reliance on external sources for support (Feder et al., 2010; Heim, 2009; Nater et al., 2006; Nes et al., 2009, Zautra et al., 1999). One consideration for this relationship is that FSS and MUS consist of unexplained illnesses with few, if any, treatment options. Medication to mitigate symptoms, sensory gating and cognitive-behavioral therapy are treatment options that show some promise; however, these are treatments that help manage, not eliminate, illness (McFarlane et al., 2008; Van Houdenhove et al., 2009). Because of this inherent lack of active strategies in treating these illnesses, it could be that the greater reliance on passive coping strategies reflects this lack of available options.

Another consideration is that the passive strategies may arise from impairments resulting from illness. In their review of chronic pain studies, Nes and colleagues (2009) speculate that those with chronic pain conditions experience self-regulatory fatigue or deficits in executive functioning, requiring them to use passive coping strategies as those are only ones for which they have enough strength (Nes et al., 2009). Therefore, it is not surprising that those suffering from FSS or MUS may exhibit functional limitations in coping that would lead them to engage in more passive strategies.
While there is nothing inherently maladaptive about passive coping strategies, many studies suggest that passive coping strategies, like avoidance and escapism, contribute to higher distress levels and poorer outcomes; whereas active coping strategies, such as problem-focused strategies and the seeking of social support for instrumental reasons, are associated with greater well-being, capacity for stress and positive psychological adjustment (Ax, Gregg, & Jones, 2001; Carver et al., 1989; Feder et al., 2010; Folkman & Moskowitz, 2004; Hori et al., 2010; McFarlane et al., 2008; Pellissier, Dantzer, Canini, Mathieu, & Bonaz, 2010; Taylor & Stanton, 2007; Walker et al., 2004; Zautra et al., 1999). Passive coping strategies may be beneficial in the short-term; for example, mental or behavioral disengagement strategies, like watching TV or daydreaming, can distract someone from the demands of a stressor, providing temporary relief. However, as passive coping strategies do not directly address the stressor, whether through behavioral or cognitive means, over the long term they may just prolong and intensify stressful experience (Carver et al., 1989).

**Coping Behaviors by Chronic Illness Groups**

The purpose of this study was to examine the coping behaviors of those with chronic illnesses of known and unknown structural pathology. Illnesses were grouped by illness pathology type into that of “conventional disease” (CD), FSS or MUS. Predictions of coping behaviors of these groups, particularly the FSS and MUS groups, were based on the model of stress system dysregulation and how dysregulation is postulated to affect stress appraisal and coping response. The CD group served as a reference group.

**Hypotheses.** In terms of appraisal, when compared to the CD group, it was expected that the FSS and MUS groups would report higher levels of symptom
experience, intensity and severity, higher levels of stress and more serious consequences of their illnesses. The FSS group was also expected to report more negative appraisals (e.g., more symptom distress, more negative feelings) than the MUS group. Perceived controllability and coping strategies, as categorized by active or passive/avoidant, were expected to differ between the CD and the FSS/MUS groups. Those in the FSS and MUS groups were expected to report greater use of passive/avoidant coping strategies.

**Method**

Data were collected from participant responses to a series of four comprehensive online surveys designed to capture chronic illness experiences. Collectively known as the VOICE (Verification of Illness, Coping and Experience) study, the surveys examined four different aspects of illness experience: personal views of physical symptoms; coping with symptoms; relationships and support; and impact of illness. The surveys were open to all consenting adults that reported at least three months of recurrent physical symptoms. The final data used for analysis excluded participants with primary diagnoses of psychiatric or affective disorders (e.g., depression, generalized anxiety disorder, somatization disorder) and participants that did not complete all four surveys.

**Participants**

**Illnesses and group categorization.** Participants fulfilling the study criteria represented 45 different chronic illness and symptom conditions (see Table 1 and Table 2 for more details). Reflective of this breadth, 29 participants alone represented 29 different illnesses. Of them all, fibromyalgia (N = 36) was the most frequently reported (see Figure 1 for the top 5 diagnoses). Based on categorizations found in the literature (Henningen et al., 2007; Yunus, 2007), medical convention and diagnostic criteria
reported by the Centers for Disease Control (2010), illnesses were assigned to one of three illness groups: conventional disease (CD), functional somatic syndrome (FSS) or medically unexplained symptoms (MUS). Illnesses characterized by distinct structural pathology and etiology as well as established criteria formed the CD group. Illnesses characterized by indistinct or unknown structural pathology and etiology as well as emerging and/or conflicting diagnostic criteria formed the FSS group. Patients that reported no diagnosis for their symptoms and/or selected the diagnosis of “Medically Unexplained Symptoms” in the surveys formed the MUS group (N = 23). While not a traditional control group, the intent for this study was for the CD group to serve as a comparison group of those with “typical”, medically accepted, chronic diseases.

<table>
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<th>Illness Name</th>
<th>Count</th>
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<td>Graves’ Disease</td>
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<td>High Blood Pressure</td>
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<td>Ehlers Danlos Syndrome</td>
<td>2</td>
<td>Stiff Person Syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Endometriosis</td>
<td>1</td>
<td>Systemic Lupus Erythematosus</td>
<td>1</td>
</tr>
</tbody>
</table>

\[^a\]Chronic Obstructive Pulmonary Disease
Table 2. *Functional Somatic Syndromes*

<table>
<thead>
<tr>
<th>Illness Name</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atypical Facial Pain</td>
<td>1</td>
</tr>
<tr>
<td>Chronic Fatigue Syndrome/CFIDS</td>
<td>12</td>
</tr>
<tr>
<td>Chronic Low Back Pain</td>
<td>3</td>
</tr>
<tr>
<td>Chronic Pain</td>
<td>1</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>36</td>
</tr>
<tr>
<td>Insomnia</td>
<td>1</td>
</tr>
<tr>
<td>Irritable Bowel Syndrome</td>
<td>3</td>
</tr>
<tr>
<td>Morgellons Disease</td>
<td>2</td>
</tr>
<tr>
<td>Multiple Chemical Sensitivity</td>
<td>2</td>
</tr>
<tr>
<td>Myofascial Pain Syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Pseudoseizures</td>
<td>1</td>
</tr>
<tr>
<td>Restless Legs Syndrome</td>
<td>12</td>
</tr>
<tr>
<td>Sick Building Syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Temporomandibular Joint Disorder</td>
<td>3</td>
</tr>
</tbody>
</table>

Figure 1. *Top 5 Diagnoses of Participants*
Demographics. The sample consisted of 148 participants (119 female, 28 male and 1 not answered). The mean age of participants was 43.34 ($SD = 13.69$) and 85.80% of participants reported experiencing symptoms for over one year. While the groups were not evenly divided (greatest amount was in the FSS group), their demographic characteristics were similar. Affirming the similarities, cross tabulations using chi-square tests showed no significant differences by group at the .05 level. See Tables 3 and 4 for further details on the sample and group demographics.

Table 3. Sample Demographics

<table>
<thead>
<tr>
<th>Number of Participants</th>
<th>148</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>119 Female, 28 Male, 1 Not Answered</td>
</tr>
<tr>
<td>Mean Age</td>
<td>43.34 ($SD = 13.69$)</td>
</tr>
<tr>
<td>Illness Chronicity</td>
<td>85.8% symptoms for over 1 year</td>
</tr>
<tr>
<td>Annual Income</td>
<td>49.0% in $20,000 - $50,000 bracket</td>
</tr>
<tr>
<td>Relationship Status</td>
<td>68.9% in a relationship/married/cohabitating</td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td>91.2% White/Caucasian</td>
</tr>
</tbody>
</table>

Table 4. Demographics by Illness Group

<table>
<thead>
<tr>
<th></th>
<th>CD</th>
<th>FSS</th>
<th>MUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participants</td>
<td>46</td>
<td>79</td>
<td>23</td>
</tr>
<tr>
<td>Gender</td>
<td>36 F, 10 M</td>
<td>67 F, 11 M, 1 Not Answered</td>
<td>16 F, 7 M</td>
</tr>
<tr>
<td>Mean Age</td>
<td>42.07 ($SD = 1.11$)</td>
<td>45.58 ($SD = 12.80$)</td>
<td>37.52 ($SD = 15.63$)</td>
</tr>
<tr>
<td>Illness Chronicity$^{ab}$</td>
<td>80.43%</td>
<td>95.0%</td>
<td>66.3%</td>
</tr>
<tr>
<td>Annual Income</td>
<td>37.0% in $20,000 - $50,000 bracket</td>
<td>33.0% in $20,000 - $50,000 bracket</td>
<td>39% in less than $20,000 bracket</td>
</tr>
<tr>
<td>In a Relationship$^{b}$</td>
<td>71.8%</td>
<td>72.2%</td>
<td>52.1%</td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td>87.0% White/Caucasian</td>
<td>92.4% White/Caucasian</td>
<td>95.7% White/Caucasian</td>
</tr>
</tbody>
</table>

$^{a}$Symptoms lasting for over 1 year. $^{b}$Group chronicity and relationship proportions did not differ significantly at the .05 level.
Procedure

Recruitment. Participant recruitment was accomplished through in-person visits to health clinics in the Jacksonville area and through online postings to discussion boards of illness support groups. Brochures left at the clinics (per clinic permission) and the web recruitment notices (adhering to particular web community guidelines) contained information describing the VOICE study and directed interested participants to the online surveys. To ensure as representative a sample as possible, clinics of the medical spectrum, from primary care physicians and specialists to massage therapists and alternative healers, on all sides of town were visited. Online recruitment followed this same inclusive strategy. Recurrent postings were made on discussion boards for a wide variety of chronic physical conditions as well as those devoted to unexplained symptoms. At the time of analysis, 72 clinics had been visited and 42 websites/discussion boards had VOICE postings. Recruitment efforts succeeded in engaging the participation of hundreds of people (as many as 572 for one survey); however, attrition and other exclusionary factors resulted in the smaller, final data set.

Measures. While the four surveys encompassed multiple measures, only the ones relevant to this analysis will be discussed here. Of primary interest for this particular study were measures that assessed symptom experience, stress appraisal, perceived controllability and coping strategies. Additionally, measures that captured health-related factors that could be of impact, such as depression, anxiety and affect were also included.

Symptom experience and stress appraisal. Symptom experience was measured using the Revised Illness Representations Questionnaire (IPQ-R) with modifications to evaluate the severity of somatic symptoms (PHQ-15) and the treatment effects for
somatoform disorders (SOMS-7). Revised from the Illness Perception Questionnaire developed by Weinman and colleagues (1996) to measure the five components of Leventhal’s self-regulatory model of illness representations (i.e., identity, consequences, timeline, control/cure and cause), the IPQ-R by Moss-Morris and colleagues (2002) expanded the widely adopted IPQ to include additional subscales (Moss-Morris et al., 2002). Factor analyses of both the IPQ and the IPQ-R indicated sound validity and reliability (Cronbach’s alphas for study items range from .80 to .84); thus, they were appropriate measures in determining patient perception of illness (Hagger & Orbell, 2005; Moss-Morris et al., 2002).

For the questions regarding symptom experience, participants were presented with a list of symptoms, like nausea, fatigue, breathlessness and sore throat, to which they responded yes or no to whether they have been bothered and just how bothered (e.g. “bothered a little”, “bothered a lot”) by the symptom(s) during the past four weeks. If they answered in the affirmative, a follow-up question was asked to determine if the particular symptom was believed to be related to their chronic condition. From these responses, scales for illness identity and illness severity were calculated. For the VOICE study, the IPQ-R was adapted to include symptom questions from the PHQ-15 and the SOMS-7 for a total of 63 symptoms explicitly assessed and one open-ended question to assess any other symptom(s) not included in the list.

Pain was assessed through the IPQ-R as well as through responses to specific pain questions from the SF-36 Health Survey Manual and Interpretation Guide (Ware, Kosinski, & Gandek, 2004). Participants responded to the SF-36 questions through selection of one of two (i.e., yes/no), three, five or six responses. For the pain questions,
participants rated their bodily pain by responding *none, very mild, mild, moderate, severe or very severe* to the question, “How much bodily pain have you had during the past 4 weeks?” Participants rated interference by pain by responding “not at all, a little bit, moderately, quite a bit or extremely” to the question, “During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?” Internal consistency reliability estimates for the SF-36 scales are above .70, which are acceptable measures of reliability (Gandek, Sinclair, Kosinski, & Ware, 2004).

To assess current levels of overall stress, participants were asked to rate their stress level on a 5-point scale (with 1 indicating “low stress” and 5 “high stress”). This measure, the Self-Rated Stress Score (SRSS), was shown to be positively correlated with psychological distress (Lim, Williams, & Hagen, 2005). The addition of the SF-36 and SRSS measures allowed a more composite picture to emerge of symptom appraisal and experience.

**Perceived controllability and consequences.** IPQ-R items that formed the subscales of personal control, treatment control and consequences contained questions which included “There is a lot which I can do to control my symptoms” and “My illness is a serious condition”. Participants responded to each question on a 5-point Likert scale that ranged from *strongly disagree, disagree, neither agree nor disagree, agree* to *strongly agree*. The average score of the questions within each subscale was used to determine the extent to which a participant experienced the given construct (e.g. higher scores for personal control reflected a greater sense of mastery over illness).

**Negative affect, depression and anxiety.** Negative affect was measured using five
items from the International Positive and Negative Affect Schedule Short Form (I-PANAS-SF) developed by E.R. Thompson (Thompson, 2007). The I-PANAS-SF items used in the study were those that measured negative affect; a Cronbach’s alpha of .76 is estimated for this particular subscale (Thompson, 2007). Responses to these items were on a 6-point Likert scale from 0 (never) to 6 (always); participants were asked to rate how often they normally felt upset, hostile, ashamed, nervous and afraid. To assess depression, the Patient Health Questionnaire (PHQ-8) by Kroenke and colleagues was used (Kroenke et al., 2009). The PHQ-8 consists of eight items that assessed how frequently participants were bothered by problems that included “feeling down, depressed, or hopeless” and “trouble concentrating on things, such as reading the newspaper or watching television”. Responses were in the format of 4-point Likert scale that ranged from 0 (not at all) to 3 (nearly every day). To assess anxiety, the anxiety scale from the Hopkins Symptom Checklist (HSCL) developed by Derogatis and colleagues was used (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). The six items of the anxiety scale asked to what extent participants felt “nervousness or shakiness inside” and “suddenly scared for no reason” during the past 4 weeks; participants responded on a 4-point Likert scale that ranged from 1 (not at all) to 4 (extremely).

**Coping strategies.** Coping strategies were assessed using the Brief-COPE (1997), a 28-item measure adapted by Carver from the longer COPE protocol (Carver, 1997; Carver et al., 1989). The Brief-COPE consists of 14 subscales of two items each of various forms of coping: active coping, planning, positive reframing, acceptance, humor, religion, using emotional support, using instrumental support, self-distraction, denial, venting, substance use, behavioral disengagement and self-blame. Participants responded
on a 4-point Likert scale (from I haven’t been doing this at all to I’ve been doing this a lot) to 28 statements (e.g., “I’ve been taking action to try to make the situation better” and “I’ve been making jokes about it”) that made up the 14 coping dimensions. Like its predecessor, the Brief-COPE is regarded as a valid, reliable (subscale Cronbach’s alphas range from .50 to .90 with ten at .65 or greater) instrument; it is a widely used measure in many health-related studies (Carver, 1997; Muller & Spitz, 2003).

**Results**

Statistical analyses were conducted, as appropriate for the item(s) being analyzed, using multivariate tests, correlational coefficients and linear regression. Alpha levels for all analyses were set at .05 and the nature of significant differences in groups was determined by Tukey’s HSD post-hoc tests. Levene’s test for equal variances was used to test the homogeneity of variances assumption; unless otherwise noted, Levene’s tests for the multivariate analyses were not significant (all ps > .05). Because of missing data for particular items, the number of participant responses for some items is less than the total participant count of 148.

**Symptom Experience and Stress Appraisal**

Multivariate analyses (MANOVAs) were run on the adapted IPQ-R items for symptoms and the SF-36 pain questions to determine if there were illness group differences in symptom experience. Significant differences by illness group were found, Wilks’s Lambda = .84, $F(10, 272) = 2.40, p = .01$, partial $\eta^2 = .08$. Results from the post-hoc tests determined that the FSS group reported significantly more symptoms, Wilks’s Lambda = .84, $F(2, 140) = 3.74, p = .03$, partial $\eta^2 = .05$, and amount of symptoms bothered by, Wilks’s Lambda = .84, $F(2, 140) = 3.81, p = .02$, partial $\eta^2 = \ldots$
.05, when compared to the MUS group; no such differences existed between the FSS and CD groups nor the MUS and CD groups. The groups showed no significant differences in illness identity (number of symptoms believed to be related to illness) or illness severity (number of symptoms bothered by that are believed to be related to illness). However, significant group differences were found in pain perception, Wilks’s Lambda = .84, \( F(2, 140) = 7.03, p < .001, \) partial \( \eta^2 = .10. \) Post-hoc tests revealed that the FSS group reported significantly higher levels of pain (\( M = 8.17, SD = 2.02 \)) than both the CD (\( M = 7.02, SD = 2.58 \)) and MUS (\( M = 6.14, SD = 2.61 \)) groups; no significant differences in pain ratings existed between the CD and MUS groups. In terms of overall stress (SRSS), illness groups showed no significant differences, Wilks’s Lambda = .84, \( F(2, 140) = 1.12, p = .33. \)

Because of the significant differences by pain, the groups were split by their bodily pain severity (e.g., CD and CD with severe pain) to see if differences would emerge along this dimension. Even when these groups were further subdivided by pain, they showed no significant differences in symptoms bothered by, illness identity, illness severity nor overall stress: for illness groups with moderate to severe pain, Wilks’s Lambda = 0.84, \( F(2, 59) = 1.00, p = .45 \) and for illness groups with none/little to mild pain, Wilks’s Lambda = 0.87, \( F(2, 77) = 1.07, p = .39. \) When illness group delineation was not taken into account and responses were compared strictly by pain severity, there were significant differences, Wilks’s Lambda = 0.73, \( F(1, 140) = 10.16, p < .001, \) partial \( \eta^2 = 0.27. \) Participants with more severe pain, irrespective of illness group, reported significantly higher levels of symptoms, symptom distress and overall stress than those with little to mild pain. The partial \( \eta^2 \) indicates that pain severity accounted for 27% of
the differences in these two groups (including error variance). See Table 5 for more details.

### Table 5. *Mean Symptom Experience by Pain Severity*

<table>
<thead>
<tr>
<th>Items</th>
<th>None/Little to Mild Pain (N = 84)</th>
<th>Moderate to Severe Pain (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptom Count</strong></td>
<td>23.25 (SD = 11.62)</td>
<td>35.03 (SD = 11.44)</td>
</tr>
<tr>
<td><strong>Symptoms Bothered By</strong></td>
<td>32.25 (SD = 18.29)</td>
<td>54.13 (SD = 19.95)</td>
</tr>
<tr>
<td><strong>Illness Identity</strong></td>
<td>16.29 (SD = 11.04)</td>
<td>28.66 (SD = 14.27)</td>
</tr>
<tr>
<td><strong>Illness Severity</strong></td>
<td>23.44 (SD = 17.85)</td>
<td>44.13 (SD = 22.35)</td>
</tr>
<tr>
<td><strong>Self-Rated Stress Score</strong></td>
<td>3.24 (SD = 1.11)</td>
<td>3.68 (SD = 1.25)</td>
</tr>
</tbody>
</table>

*Note.* Levene’s test of equality of error variances was significant (p < .05) for both illness identity and illness severity.

* Difference significant at the .05 level. ** Difference significant at the .01 level.

**Perceived Controllability and Consequences**

Multivariate analyses (MANOVAs) were conducted on the IPQ-R subscales of consequences, personal control and treatment control. Significant illness group differences were found for consequences and treatment control, Wilks’s Lambda = .79, $F(2, 142) = 5.69, p < .001$, partial $\eta^2 = .11$. It is important to note that for these analyses, Levene’s test indicated significant differences in error variances, $p < .001$ for both subscales. With this in mind, the results showed that the MUS group ($M = 3.38, SD = 1.28$) exhibited significantly lower perceived consequences than both FSS ($M = 4.34, SD = 0.62$) and CD ($M = 4.12, SD = 0.76$). Regarding treatment control, the only significant difference was between FSS ($M = 2.37, SD = 0.93$) and CD ($M = 2.85, SD = 0.92$); this difference suggests that the CD group reports a greater sense of control concerning illness treatment.

**Negative Affect, Depression and Anxiety**

Multivariate analyses (MANOVAs) testing illness group differences in negative
affect, depression and anxiety revealed only significant group differences in depression, Wilks’s Lambda = .86, \(F(2, 145) = 10.28, p < .001\), partial eta\(^2\) = .12. Post-hoc tests indicated significantly higher levels of depression in the FSS group (\(M = 20.89, SD = 5.72\)) than the CD (\(M = 18.26, SD = 6.13\)) and MUS (\(M = 14.83, SD = 5.74\)) groups. The CD and MUS groups did not significantly differ from one another in any of these tests. For a comparison of illness groups by all these items, see Table 6.

<table>
<thead>
<tr>
<th>Item</th>
<th>CD</th>
<th>FSS ((SD = 12.16))</th>
<th>MUS ((SD = 14.16))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptom Count</td>
<td>27.18 ((SD = 12.85))</td>
<td>30.57* ((SD = 12.16))</td>
<td>22.45 ((SD = 14.16))</td>
</tr>
<tr>
<td>Bothered(^a)</td>
<td>39.61 ((SD = 20.30))</td>
<td>45.60* ((SD = 21.44))</td>
<td>31.82 ((SD = 23.73))</td>
</tr>
<tr>
<td>Illness Identity</td>
<td>20.80 ((SD = 13.73))</td>
<td>23.12 ((SD = 14.13))</td>
<td>17.91 ((SD = 13.40))</td>
</tr>
<tr>
<td>Illness Severity</td>
<td>30.93 ((SD = 21.51))</td>
<td>34.78 ((SD = 22.55))</td>
<td>26.68 ((SD = 23.00))</td>
</tr>
<tr>
<td>Pain</td>
<td>7.02 ((SD = 2.58))</td>
<td>8.17* ((SD = 2.02))</td>
<td>6.14 ((SD = 2.61))</td>
</tr>
<tr>
<td>Stress Level</td>
<td>3.43 ((SD = 1.18))</td>
<td>3.52 ((SD = 1.21))</td>
<td>3.09 ((SD = 1.11))</td>
</tr>
<tr>
<td>Consequences</td>
<td>4.12 ((SD = 0.76))</td>
<td>4.34 ((SD = 0.62))</td>
<td>3.38* ((SD = 1.28))</td>
</tr>
<tr>
<td>Pers. Control(^b)</td>
<td>3.07 ((SD = 0.90))</td>
<td>3.02 ((SD = 1.09))</td>
<td>2.81 ((SD = 1.32))</td>
</tr>
<tr>
<td>Treat. Control(^c)</td>
<td>2.85 ((SD = 0.92))</td>
<td>2.37* ((SD = 0.93))</td>
<td>2.52 ((SD = 1.45))</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>14.74 ((SD = 7.02))</td>
<td>16.29 ((SD = 6.58))</td>
<td>13.35 ((SD = 6.26))</td>
</tr>
<tr>
<td>Depression</td>
<td>18.26 ((SD = 6.13))</td>
<td>20.89* ((SD = 5.72))</td>
<td>14.83 ((SD = 5.74))</td>
</tr>
<tr>
<td>Anxiety</td>
<td>10.26 ((SD = 3.96))</td>
<td>10.90 ((SD = 3.84))</td>
<td>8.74 ((SD = 3.51))</td>
</tr>
</tbody>
</table>

\(^a\)Bothered is symptoms bothered by. \(^b\)Personal Control, \(^c\)Treatment Control.

* Mean difference significant at the .05 level

**Coping Strategies**

Each coping subscale from the Brief-COPE was analyzed for illness group differences. A MANOVA revealed no significant differences in coping strategies by illness groups, Wilks’s Lambda = .71, \(F(2, 122) = 1.45, p = .08\). Coping strategies were also assessed to see if they varied by whether they were active or passive/avoidant. To accomplish this, strategies were grouped into the categories of active or passive/avoidant based on recommendations in the literature (Carver et al., 1989), which were further supported by evidence that logically related strategies were positively correlated (examples of strategies that were significantly correlated at the .01 level include active...
coping and planning, \( r(140) = .77 \); using emotional support and using instrumental support, \( r(141) = .51 \); self-blame and denial, \( r(140) = .33 \); denial and behavioral disengagement, \( r(137) = .32 \). Two subscales, humor and religion, were excluded from the active and passive/avoidant groups. These two subscales were excluded because they were significantly correlated with both active and passive/avoidant coping style. This was not an unexpected finding as Carver and colleagues (1989) remarked that the multifaceted uses of humor and religion can manifest as seemingly contradictory forms of coping (i.e., humor can be a form of instrumental coping as well as a form of venting; Carver et al., 1989). Table 7 illustrates the grouping of the coping subscales.

<table>
<thead>
<tr>
<th>Active Coping</th>
<th>Passive/Avoidant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Using Instrumental Support</td>
<td>Self-Distraction</td>
</tr>
<tr>
<td>Using Emotional Support</td>
<td>Behavioral Disengagement</td>
</tr>
<tr>
<td>Planning</td>
<td>Venting</td>
</tr>
<tr>
<td>Positive Reframing</td>
<td>Substance Use</td>
</tr>
<tr>
<td>Acceptance</td>
<td>Self-Blame</td>
</tr>
</tbody>
</table>

*Note.* The two scales were created by summing the relevant subscales. Pearson’s correlation coefficient indicated that the two styles were not significantly correlated, \( r(124) = .10, p = .28 \).

Following the creation of the coping style groups, multivariate analyses (MANOVAs) were conducted to determine if coping styles varied by illness group. The results showed no significant differences in coping styles by illness groups, Wilks’s Lambda = .96, \( F(4, 244) = 1.25, p = .29 \).
Pearson’s correlation coefficients ($r$) were calculated to assess the degree of co-relationships among items measuring symptom experience, personal controllability, negative affect, depression, anxiety, stress, pain and coping styles. Because the four IPQ-R items of symptom experience (i.e., count, bothered by, identity and severity) were highly and significantly intercorrelated (ranging from $r$ of .82 to .96), they were summed to create a new scale variable (“Symptoms”) so as to simplify analysis. For personal controllability, significant correlations were found between that item and active coping, $r(131) = .17$; anxiety, $r(145) = -.19$; depression, $r(145) = -.23$ and symptoms, $r(140) = -.17$; all correlations for that item were significant at the .05 level, except depression which was significant at the .01 level. Significant results for the remainder of the items are summarized in Table 8.
Table 8. Significant Correlations Among Items

<table>
<thead>
<tr>
<th></th>
<th>Negative Affect</th>
<th>Depression</th>
<th>Anxiety</th>
<th>Stress</th>
<th>Pain</th>
<th>P/A\textsuperscript{c} Coping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neg. Affect</td>
<td>--</td>
<td>.66\textsuperscript{**}</td>
<td>.71\textsuperscript{**}</td>
<td>.56\textsuperscript{**}</td>
<td>.36\textsuperscript{**}</td>
<td>.56\textsuperscript{**}</td>
</tr>
<tr>
<td>Depression</td>
<td>.66\textsuperscript{**}</td>
<td>--</td>
<td>.64\textsuperscript{**}</td>
<td>.39\textsuperscript{**}</td>
<td>.58\textsuperscript{**}</td>
<td>.48\textsuperscript{**}</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.71\textsuperscript{**}</td>
<td>.64\textsuperscript{**}</td>
<td>--</td>
<td>.41\textsuperscript{**}</td>
<td>.36\textsuperscript{**}</td>
<td>.49\textsuperscript{**}</td>
</tr>
<tr>
<td>Stress\textsuperscript{a}</td>
<td>.56\textsuperscript{**}</td>
<td>.39\textsuperscript{**}</td>
<td>.41\textsuperscript{**}</td>
<td>--</td>
<td>.21\textsuperscript{**}</td>
<td>.34\textsuperscript{**}</td>
</tr>
<tr>
<td>Pain</td>
<td>.36\textsuperscript{**}</td>
<td>.58\textsuperscript{**}</td>
<td>.36\textsuperscript{**}</td>
<td>.21\textsuperscript{**}</td>
<td>--</td>
<td>.34\textsuperscript{**}</td>
</tr>
<tr>
<td>Symptoms\textsuperscript{b}</td>
<td>.30\textsuperscript{**}</td>
<td>.48\textsuperscript{**}</td>
<td>.46\textsuperscript{**}</td>
<td>.16\textsuperscript{*}</td>
<td>.58\textsuperscript{**}</td>
<td>.31\textsuperscript{**}</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Stress was negatively correlated with active coping, \(r(132) = -.21, p = .02\).
\textsuperscript{b}Symptoms was positively correlated with active coping, \(r(128) = .21, p = .02\).
\textsuperscript{c}Passive/Avoidant.
*Correlation is significant at the .05 level.
**Unless otherwise noted, all correlations significant at the .01 level.

As tests continued to show that the factors of negative affect, depression, anxiety, stress, pain, symptom experience and personal control showed significant relationships with coping styles (particularly passive/avoidant coping), a MANCOVA was conducted to see if coping styles differed by illness groups when the significant factors were included as covariates. Even while controlling for the significant covariates, there was no indication of any significant differences in coping styles by illness group, Wilks’s Lambda = .96, \(F(4, 222) = 1.05, p = .38\).

Exploratory Analyses

To explore the relationships between the aforementioned significant factors and coping styles, multiple linear regressions were conducted to see if coping styles could be predicted by those factors. Both regressions proved to be significant for this predictive model: for active coping, \(R^2 = .16, F(7, 121) = 3.34, p = .003\) and for passive/avoidant coping, \(R^2 = .35, F(7, 122) = 9.45, p < .001\). Multicollinearity tests for both showed no tolerance values of less than .05, indicating no problems with multicollinearity. For both models, the seven factors explained a significant proportion of variance in coping styles.
The results indicated that stress, $\beta = -.21$, $t(120) = -2.09$, $p = .04$; symptoms, $\beta = .32$, $t(120) = 2.82$, $p = .01$; and personal control, $\beta = .19$, $t(120) = 2.16$, $p = .03$, significantly predicted active coping strategies; negative affect significantly predicted passive/avoidant coping strategies, $\beta = .34$, $t(121) = 2.81$, $p = .01$. Table 9 provides more details on the regression predictors.

Table 9. Regression Predictors for Coping Styles

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Active (N = 128)</th>
<th></th>
<th></th>
<th></th>
<th>Passive/Avoidant (N = 129)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>$t$</td>
<td>$p$</td>
<td>$\beta$</td>
<td>$t$</td>
<td>$p$</td>
<td></td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.05</td>
<td>0.38</td>
<td>.71</td>
<td>.34</td>
<td>2.81</td>
<td>.01*</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>-.20</td>
<td>-1.49</td>
<td>.14</td>
<td>.13</td>
<td>1.09</td>
<td>.28</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>.01</td>
<td>0.06</td>
<td>.95</td>
<td>.10</td>
<td>0.88</td>
<td>.38</td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td>-.21</td>
<td>-2.09</td>
<td>.04*</td>
<td>.04</td>
<td>0.41</td>
<td>.69</td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>.05</td>
<td>0.45</td>
<td>.66</td>
<td>.06</td>
<td>0.61</td>
<td>.55</td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td>.32</td>
<td>2.82</td>
<td>.01*</td>
<td>.05</td>
<td>0.51</td>
<td>.61</td>
<td></td>
</tr>
<tr>
<td>Personal Control</td>
<td>.19</td>
<td>2.16</td>
<td>.03*</td>
<td>-.02</td>
<td>-0.31</td>
<td>.76</td>
<td></td>
</tr>
</tbody>
</table>

*Predictor is significant at the .05 level

Discussion

**Tests of the hypotheses.** The results of this study revealed that coping strategies did not differ by illness group, but as expected, it was the mechanisms of appraisal that contributed to coping variation. Coping style was predicted by health-related factors that influence appraisal; in particular, stress, symptoms and personal control were significant contributors for active coping prediction, whereas negative affect was significant for passive/avoidant coping. It was interesting to see that while illness groups largely associated with stress, symptom appraisals and negative affect (i.e., FSS and MUS) did not exhibit relationships with any specific coping strategies, it was these defining characteristics of FSS and MUS that did.

The findings on negative affect reflect that of previous research, such as that of Kraaij and associates (2009) who found that the passive/avoidant coping strategies of
self-blame and disengagement were significant in their regression model for negative affect as well as findings by Theadom and Humphrey (2007) that negative affect was significantly and inversely correlated with active coping and positive reinterpretation in patients with fibromyalgia (Kraaij, Garnefski, & Schroevers, 2009; Theadom & Humphrey, 2007). When Ben-Zur (2009) examined coping-affect relationships in adolescents, undergraduate students and a sample of the general community, she found that avoidance coping showed a positive association with negative affect, $\beta = .31, p < .001$, and a negative association with positive affect, $\beta = -.24, p < .001$ (Ben-Zur, 2009). Her results suggest that while directionality of the avoidant coping-negative affect relationship is ambiguous, the two may mutually reinforce one another (Ben-Zur, 2009).

Negative affect embodies sensitivity to aversive cues, like symptoms and stress. Those with negative affect may have greater difficulty in using active coping strategies, resulting in more frequent use of passive/avoidant coping strategies that, by definition, do not directly address stressors or involve conscious regulation of the emotional responses to stress. Through passive/avoidant coping, the stressors themselves are not changed or mitigated; a person with greater proclivity toward negative appraisals may keep assessing and avoiding the aversive cues, maintaining this avoidant coping-negative affect relationship, which is associated with poorer outcomes (Ben-Zur, 2009; Felton & Revenson, 1984).

As for active coping, levels of personal control predicted this dimension of coping, which is consistent with the idea that those that believe they can control health outcomes will be engaged in action-oriented strategies to cope with illness. A more counterintuitive finding in the regression model was of symptoms and active coping. It
would seem that with increased symptoms and presumably, increased levels of discomfort, distress, impairment and functional limitations, the predicted coping strategies would be passive/avoidant, as opposed to active. An explanation for this may be that as a result of their long duration of illness experiences, participants have learned how to accommodate for their symptoms. Brown and colleagues (2010) suggest that when living with chronic illness over a long period of time, coping skills improve from what they were in the early stages (Brown, M., Brown, A., & Jason, 2010). As participants of this study report long illness duration (85.8% for over one year), perhaps they have adapted to their conditions and are in a position to be able to actively manage symptoms.

Though the data do not support coping differences by illness types, the FSS group did report greater pain and symptom experience than the other groups, data consistent with the stress system dysregulation theory. Also consistent is the notion that for there to be greater appraisal and symptom perceptual differences, there should be greater differentiation in stress. For this study, the mean levels of stress were in the moderate range (from 3.09 for MUS to 3.52 for FSS) and did not significantly differ per group; the stress similarity could help explain why the groups showed few differences in appraisal and none in coping. The results of this study support other findings on stress system dysfunction and coping: a significant, positive correlation between passive/avoidant coping style and stress (Chrousos, 2009; Hori et al., 2010). Furthermore, in the regression model, higher ratings of stress predicted less use of active coping strategies. Stress sensitivities and disorders are not limited to any particular illnesses or illness groups, even if FSS and MUS are associated with stress dysregulation. These findings illustrate
that the relationship between stress and coping is not mediated by a type of illness, even if the illness is associated with appraisal sensitivity. It is the individual, subjective experience of stress that predicts coping.

In terms of illness experience and related factors, the MUS group was expected to behave similarly to the FSS group. However, analyses showed that the MUS group was an entity all its own in terms of lower perceived consequences, lower counts of symptoms and symptom severity, as well as lower levels of stress, pain, depression, anxiety and negative affect. Though these means were not significantly lower than that of the FSS and CD groups (except that of consequences), the quantitative differences suggest that having medically unexplained symptoms may be a qualitatively different experience than having a functional somatic syndrome or conventional disease.

The reliability and validity of these data are corroborated by the results of other studies. Approximating this study’s FSS group (N = 79), the sample for a study that investigated sensory amplification in 38 patients with fibromyalgia and/or chronic fatigue syndrome consisted of primarily women (73.6%) with a mean age of 42.0 (SD = 8.8) that reported pain for over one year and an average amount of 19.2 (SD = 9.7) physical symptoms (Geisser et al., 2008). The scales of physical symptoms differed (51 items for the Geisser study and 63 for this study), but both studies yielded a substantial amount of physical symptoms for analogous participants (Geisser et al., 2008). In a study of 206 patients with MUS considered to be high-utilizers of primary care (an average of 13.6 visits the year prior to the study), the psychosomatic symptom mean was 23.0 (SD = 15.2); in comparison, the mean symptom count for MUS in this study was nearly identical at 22.5 (SD = 14.2) (Smith et al., 2005). The typicality of this sample and these
results provide further evidence for the relationship between FSS/MUS and appraisal sensitivity.

**The sample.** It is important to consider the participants of this study and sample characteristics that may have influenced the results. The sample was 80% female; since the participants were mainly women, any gender differences would be difficult to find. The significant predictors for coping style were factors that some studies have shown are more associated with women. For example, in a Canadian study of 131,535 individuals, with an equal proportion of men and women and a mean age of 42.1 years, 38.4% of women versus 27.1% of men reported at least one chronic pain condition (i.e., fibromyalgia, arthritis/rheumatism, back problems and/or migraine headaches); excluding back problems, all the conditions were more prevalent in women and women reported depression at twice the rate of men (Munce & Stewart, 2007). It is hard to tease apart effects by illness group if effects are largely associated with confounds like gender and the patient profile is typically female.

Another thing to consider is that the sample participants may be somewhat different from the greater population of those with chronic illnesses. Perhaps these participants were more willing to communicate their illness experiences or uniquely persistent; perhaps these participants possessed characteristics that aligned more with the active coping style, which would thwart expected illness differentiation in coping. Survey attrition excluded the use of respondent data that may have provided more comprehensive understanding.

**Limitations.** There were some limitations to this study. The surveys were online instruments, so internet access was essential for participation. The surveys were also
quite lengthy, so attrition was somewhat unavoidable. Attrition was a major problem and even follow-up communication with consenting respondents did not necessarily secure participation.

Though illness group assignment was done through careful consideration of available diagnostic information, many of these illnesses have fuzzy epidemiological boundaries. For example, depending on the source, fibromyalgia can belong to FSS or MUS or CD; most current research supports it as a functional somatic syndrome, which is why it is categorized as such (Barsky & Borus, 1999; Sharpe & Carson, 2001; Yunus, 2007). Analyses may have been impacted by group assignment; examination of the experiences of the illnesses separately would be an excellent future avenue of research.

These limitations do not eclipse the study’s strengths: the surveys’ multiple measures, including those not featured in this study (e.g., relationships and other supports, stigma, emotional representations, the use of complementary and alternative health treatments, causal attributions); the wealth of information provided by the participants that completed all four surveys; the diversity of illness and symptom experiences that were recruited through online postings, which may not have been possible solely through clinic recruitment in northeast Florida; the online nature of the surveys, which is appropriate and convenient as the internet penetrates everyday life (266.2 million people in North America alone use the internet; Internet World Stats, 2010). The greatest success of this study was that it recruited so many participants of a demographic that is not easily accessible. While improvements could be made for future applications of the study, such as incentives to reduce attrition, additions of measures and
translation into other languages to capture non-English speakers, that the study elicited such a sizeable sample is quite an achievement.

**Future directions.** Other future directions of research include analyses of chronic illnesses by pain perception; analyses exploring the connection between negative affect and passive/avoidant coping; coping comparisons that include healthy controls along with the illness groups; and differences in symptom scanning or hypervigilance across chronic illnesses. Table 5 provides a glimpse of the impact of pain perception and the group differences in pain expression; a study that focused on examining pain would be a natural follow-up to this study. Another natural follow-up, similar to the suggestion of exploration by illnesses, would be further investigation into the functional somatic syndromes. The FSS illnesses, while sharing diagnostic criteria, exhibited within-group heterogeneity that most likely affected group analyses (see Table 10).

<table>
<thead>
<tr>
<th></th>
<th>Depression</th>
<th>Anxiety</th>
<th>Pain</th>
<th>P/A&lt;sup&gt;c&lt;/sup&gt; Coping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibromyalgia</td>
<td>22.48</td>
<td>11.35</td>
<td><strong>9.23</strong></td>
<td>10.16</td>
</tr>
<tr>
<td></td>
<td>(SD = 5.78)</td>
<td>(SD = 4.14)</td>
<td>(SD = 1.28)</td>
<td>(SD = 4.04)</td>
</tr>
<tr>
<td>CFS&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17.82</td>
<td>8.91</td>
<td>7.73</td>
<td>7.91</td>
</tr>
<tr>
<td></td>
<td>(SD = 2.48)</td>
<td>(SD = 1.87)</td>
<td>(SD = 1.68)</td>
<td>(SD = 2.77)</td>
</tr>
<tr>
<td>RLS&lt;sup&gt;b&lt;/sup&gt;</td>
<td>18.50</td>
<td>9.75</td>
<td>6.50</td>
<td>7.50</td>
</tr>
<tr>
<td></td>
<td>(SD = 7.07)</td>
<td>(SD = 3.92)</td>
<td>(SD = 2.73)</td>
<td>(SD = 6.87)</td>
</tr>
<tr>
<td>FSS Remaining</td>
<td>20.53</td>
<td>12.47</td>
<td>6.93</td>
<td>10.80</td>
</tr>
<tr>
<td></td>
<td>(SD = 6.91)</td>
<td>(SD = 4.26)</td>
<td>(SD = 2.89)</td>
<td>(SD = 6.29)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>20.75</td>
<td>11.00</td>
<td>8.11</td>
<td>9.60</td>
</tr>
<tr>
<td></td>
<td>(SD = 6.01)</td>
<td>(SD = 3.97)</td>
<td>(SD = 2.27)</td>
<td>(SD = 4.92)</td>
</tr>
</tbody>
</table>

<sup>a</sup>CFS is Chronic Fatigue Syndrome. <sup>b</sup>RLS is Restless Legs Syndrome. <sup>c</sup>Passive/Avoidant * Mean difference significant at the .05 level.
Overall, the results from this study suggest that coping style may be a function of how respondents experience their illnesses or unexplained symptoms, irrespective of the actual illness or illness type. Since responses to certain items (i.e., personal control, stress, symptoms and negative affect) were strong indices for coping style, the knowledge that a patient may be more prone to maladaptive coping behaviors would be a valuable tool in determining what treatment interventions to introduce and support. Such screening could yield better health outcomes; even brief, self-administered questionnaires of depression, anxiety and somatoform disorders can be helpful in quickly assessing health-relevant information, particularly for those patients in primary care with persistent symptoms that do not respond to standard treatments (Kroenke, Spitzer, Williams, & Löwe, 2010). Instead of an emphasis on diagnosis to determine prognosis and treatments, assessing the whole patient and his/her particular experience with illness, which includes appraisal, controllability, outcome beliefs and methods of coping, appears to be a more effective approach that responds to the individual needs of the illness sufferer. Identifying and addressing how those with illness cope can facilitate wellness. Ensuring that they are equipped with the strategies and support to cope empowers them to take a proactive role in managing life with illness.
References


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