May 2014

Longitudinal Associations Between Anxiety, Pain Catastrophizing, and Treatment Outcomes in Complex Pediatric Chronic Pain

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LONGITUDINAL ASSOCIATIONS BETWEEN ANXIETY, PAIN CATASTROPHIZING, AND TREATMENT OUTCOMES IN COMPLEX PEDIATRIC CHRONIC PAIN

by

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A Dissertation Submitted in

Partial Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

in Psychology

at

The University of Wisconsin-Milwaukee

May 2014

ABSTRACT
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The University of Wisconsin-Milwaukee, 2014
Under the Supervision of Professor W. Hobart Davies

Anxiety and pain catastrophizing have been identified as factors that may predispose an individual to developing chronic pain and influence functional outcomes. The purpose of this study was to investigate the longitudinal associations of anxiety and pain catastrophizing with functional outcomes in a sample of youth seeking treatment for chronic pain. The current study aimed to expand upon recent literature by examining the relative contributions of both anxiety and pain catastrophizing to important functional outcomes (pain, functional disability, and health-related quality of life [HRQOL]) in a longitudinal design.

Participants included 725 youth (69% females, 75% Caucasian) ranging in age from 8 to 18 who received services at an outpatient interdisciplinary pain clinic for complex chronic pain. Data were gathered from a retrospective chart review of questionnaires completed by families at their intake appointment at the pain clinic (baseline), one month, and three month follow-up. Families were mailed a packet including questionnaires for the youth with chronic pain and their parents/guardians. Youth self-reported questionnaires regarding anxiety, pain catastrophizing, functional disability, and HRQOL were examined.

Higher anxiety and pain catastrophizing were related to 1) higher pain intensity, 2) higher functional disability, and 3) lower HRQOL at baseline after controlling for demographic characteristics and pain characteristics. Changes in anxiety and pain catastrophizing contributed unique variance in longitudinally predicting later pain, functional disability, and HRQOL. The results of this study support targeting anxiety and pain catastrophizing in treatment of pediatric chronic pain. It will be important for future research to consider models including mediators of these relationships and moderators of functional outcomes.

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ACKNOWLEDGEMENTS

I would like to thank my committee, Dr. W. Hobart Davies, Dr. Keri Hainsworth, Dr. Bonnie Klein-Tasman, Dr. Katie Mosack, and Dr. Robyn Ridley, for their support and guidance in developing my dissertation. I would also like to thank Chris Tran for his ongoing support.

Introduction

Pediatric chronic pain is a relatively common condition among youth that can last for years and is often associated with poor functional outcomes. It is important to view chronic pain from a biopsychosocial perspective and take psychological and sociocultural factors into account that may play a role in the perpetuation of pain. Anxiety and pain catastrophizing have been identified as factors that may predispose an individual to developing chronic pain, interact with pain symptoms, and influence functional outcomes. These factors have been found to be closely related, but distinct, in adult populations. No research has been done to examine the distinct roles they play in influencing functional outcomes in pediatric chronic pain. The current study aimed to expand upon recent literature by examining the relative contributions of anxiety and pain catastrophizing to important functional outcomes over time in a pediatric chronic pain population. As improving functional disability and health-related quality of life (HRQOL) are primary treatment goals for pediatric chronic pain, these constructs along with pain intensity will be used as outcome variables.

This study addressed the following questions: What are the relationships between anxiety, pain catastrophizing, functional disability, HRQOL, and pain intensity? How well do anxiety and pain catastrophizing predict outcomes? What is the unique predictive ability of anxiety and pain catastrophizing to predict outcomes? Do changes in anxiety and pain catastrophizing predict later pain or functioning? In this literature review, the current literature on pediatric chronic pain and models of chronic pain are discussed. Next, anxiety and pain catastrophizing are defined and the literature in regards to comorbidity with chronic pain is reviewed. Then, the overlap between anxiety and

pain catastrophizing is discussed and the importance of identifying mediators of treatment outcomes and mechanisms of change are reviewed. Finally, the statistical relationships between the variables examined are defined.

Definition of Pain

The International Association for the Study of Pain defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey et al., 1979, p. 250). Pain is commonly described as an aversive sensation most often caused by injury to tissue containing nerve fibers and activation of specialized sensory receptors called nociceptors (Rosenzweig, Breedlove, & Leiman, 2002; Woolf & Mannion, 1999). Acute pain is a protective response to actual or potential damage; the experience elicits reflexive and behavioral responses to avoid further harm. The activation of different types of nociceptors results in different qualities of pain. Aching pain is caused by stimulation of nociceptors in muscle nerves; sharp, prickling pain (paraesthesia) is caused by stimulation of large, myelinated cutaneous Aδ nociceptors; and burning or dull pain results from stimulation of thin, unmyelinated cutaneous C nociceptors (Rosenzweig et al., 2002; Woolf & Mannion, 1999). Spontaneous activity in these C fibers is also thought to be responsible for the sensitization of dorsal horn neurons (Woolf & Mannion, 1999).

Nociceptive pain occurs when nociceptors in peripheral tissue are activated and signal potential or actual tissue damage. Sensitization of nociceptors can be altered by chemical stimuli released by either the surrounding tissue or nerve endings (Zieglgänsberger, Berthele, & Tölle, 2005). Nociceptive pain can be either acute or chronic (Harden, 2005). Neuropathic pain is caused by damage to the peripheral nerve or

the central nervous system (Zieglgänsberger et al., 2005). Neuropathic pain is either stimulus-independent spontaneous pain, or elicited by a stimulus and characterized by pain hypersensitivity. This pain hypersensitivity is caused by damage or loss of sensory neurons (Woolf & Mannion, 1999). The chemicals in the environment surrounding the neuron determine the baseline sensitivity of nociceptor receptors. Changes in the chemical environment, such as an increased number of cytokines, and inflammation of the nerve or ganglion can affect how the neurons function (Woolf & Mannion, 1999). Neuropathic pain also involves spontaneous production of action potentials signaling pain that would have otherwise produced innocuous sensations. It is often characterized by a complex combination of sensory deficits (Zieglgänsberger et al., 2005).

Chronic and recurrent pain is longer in duration than acute pain. It lasts longer than would be expected due to the nature of bodily injury and is not accompanied by the usual signs of sympathetic nervous system arousal (American Pain Society, 2012). Persistent pain symptoms offer no biological advantage, as physical healing has often occurred and the immediate threat has passed. Oftentimes the pain results from damage to (Woolf & Mannion, 1999) or sensitization of the central nervous system (American Pain Society, 2012). A commonly accepted definition of chronic pain is "any prolonged pain that lasts a minimum of three months or any pain that recurs throughout a minimum period of three months" (McGrath, 1999, pg. 81).

Pediatric Chronic Pain

Pain lasting three months or longer is commonly considered to be chronic (McGrath, 1999); however, individual diagnoses in children may be influenced by developmental factors (American Pain Society, 2012). Chronic pain is a common and

pervasive problem in children (Goodman & McGrath, 1991; Perquin et al., 2000; Perquin et al., 2003; Roth-Isigkeit, Thyen, Stöven, Schwarzenberger, & Schmucker, 2005). Estimated prevalence rates of pediatric chronic pain vary greatly. This may be due to any number of factors including: inconsistent psychometric properties (e.g., reliability and validity) of pain measures, sampling issues, discrepant definitions of chronic or recurrent pain (Goodman & McGrath, 1991; King et al., 2011), or general difficulty asking children for retrospective reports of pain episodes over extended periods of time (von Baeyer, 2011). The field of pediatric chronic pain has benefitted from contributions from many countries. Admittedly, social and cultural factors may affect the experience of chronic pain across countries and cultures, but since there is little research on these differences we will consider the experience of chronic pain to be equivalent regardless of country of origin. It is commonly cited that the prevalence of chronic pain among school-aged children is estimated to be between 15 and 30% (Goodman & McGrath, 1991). A current review of 32 epidemiological studies over the past 20 years found estimates around 25% of school age children report chronic and recurrent pain with median prevalence rates for many different types of chronic pain conditions between 11-36% (King et al., 2011).

Pediatric chronic pain is more prevalent in females than in males, particularly into and across adolescence (King et al., 2011; Hoftun, Romundstad, Zwart, & Rygg, 2011; Perquin et al., 2000; Perquin et al., 2003). Female patients are generally more common than males in pediatric chronic pain samples (Lynch, Kashikar-Zuck, Goldschneider, & Jones, 2007; Martin, McGrath, Brown, & Katz, 2007a). Among Dutch school children, females were more likely to report chronic pain than males in all age groups between 4 to

18 years, with a considerable increase in occurrences between 12 and 14 years (Perquin et al., 2000). A study of community children in Norway found that 54% of females aged 13-18 reported pain not due to injury or disease at least once a week for three months compared to 34% of males (Hoftun et al., 2011). Girls are more likely to report several chronic pain conditions compared to boys, including headache, abdominal pain, musculoskeletal pain (limb pain), and multiple pains (King et al., 2011). Among children with chronic pain, females have reported more frequent and more intense pain than males (Hunfeld et al., 2001; Perquin et al., 2000). Females are also at higher risk for continued experience of chronic pain; females reported more pain three years after being treated at a pediatric pain clinic compared to males (Martin et al., 2007a).

Children and adolescents with chronic pain are at risk for long-term disability. In a community sample of children and adolescents with chronic pain, nearly half (48%) reported continued pain one year after intake at a chronic pain clinic, and 30% were still experiencing pain after two years (Perquin et al., 2003). In a treatment sample, 62% of patients continued to experience pain three years after being discharged from a pediatric pain clinic (Martin et al., 2007a). Children who experience chronic pain are more likely to experience chronic pain in adulthood (Marugán, Fernández-Castaño, del Carmen Torres, & del Carmen de Fuentes, 2008). The development and maintenance of chronic pain may be a cycle, worsening and deepening over time (Asmundson, 1999; Vlaeyen & Linton, 2000). Pain intensity among children with chronic pain is related to decreased physical and psychological functioning, satisfaction with health and life, as well as increased functional disability (Hunfeld et al., 2001; Merlijn et al., 2006; Palermo, 2000). Considering the duration of chronic pain and the potential impact of pain and disability in

adulthood, it is important to investigate factors that contribute to chronic pain beginning in childhood.

Assessment of Pain

Pain is a private and subjective experience; therefore, it is difficult to measure objectively. There are a number of validated measures used to assess pain. However, the number of tools and variety of types of measures highlight the need for continued research into a perfect assessment tool (Breivik et al., 2008). Since self-report of pain is the "gold standard" in pain assessment, challenges occur in assessing pain in younger children and individuals with cognitive impairment. Different measures are used for assessing pain in infants, children, adults, and individuals who are cognitively impaired. Assessment of pain typically includes the location, quality, duration, frequency, and intensity of the pain (Breivik et al., 2008). Pain assessment can also include other descriptions regarding psychological and sociocultural factors and functioning, depending on the referral question (Austin & Henderson, 2011).

There are several simple and commonly used measures to assess pain intensity in children. A scale with six faces with expressions varying from happy to unhappy can be used with younger children beginning at age four. Older children can use the visual analogue scale (VAS) and the numeric rating scale (NRS) as well. The VAS asks patients to mark on a line where their pain intensity is at between "no pain" and "worst pain imaginable"; the NRS asks patients to rate their pain intensity on a scale of 0 (no pain) to 10 (worst pain imaginable). These dimensional assessments of pain intensity are more sensitive and reliable than the four-point verbal categorical rating scale (none, mild, moderate, or severe pain). These scales are appropriate to use to rate usual, highest, and

lowest intensity of current pain or pain in the recent past (up to one week; Breivik et al., 2008). In order to reduce the bias inherent in recalling past pain, some research projects elect to use prospective pain monitoring and pain diaries. Daily recording of pain intensity as used in Hunfeld et al. (2001) asked children to record their pain on a VAS at three specific times per day. These recordings were significantly lower than later recall of pain intensity, suggesting that immediate assessment of pain may yield different results than measures involving recall. Further complicating pain assessment is the fact that pain intensity varies throughout the day. A sample of adults with arthritis was asked to rate their pain several times throughout the day and researchers found that pain scores varied an average of 35 points (on a scale of 1-100) throughout the day (Allen, Coffman, Golightly, Stechuchak, & Keffe, 2009).

The assessment of chronic pain is complex. Even more so than acute pain, chronic pain may have a major impact on several areas of an individual's life (Breivik et al., 2008). A full biopsychosocial approach to assessment of chronic pain is recommended to take the reciprocal relationships between these factors into account (American Pain Society, 2012). This assessment usually includes full documentation of pain symptoms and history and a physical examination, followed by any recommended clinical diagnostic testing to rule out other comorbidity that should be addressed. Next, the patient's premorbid and current functioning (personal care, social, occupational, psychological, etc.) should be addressed to assess the functional impact of the pain. The environmental, emotional, and psychological context of the pain should be assessed as well to determine in what ways these factors may be interacting with the pain and the overall effect on quality of life (Austin & Henderson, 2011; Breivik et al., 2008). There

are many instruments related to the assessment of various aspects of functioning in youth. These areas include physical, emotional, psychosocial, academic functioning and coping. Most of these measures were developed using other populations first, such as adults or acute pain populations, before being applied to pediatric chronic pain populations (Eccleston, Jordan, & Crombez, 2006).

The Biopsychosocial Model of Pain

Chronic pain may begin as the result of an illness or injury, but the pain fails to follow the expected course of healing. It may persist and recur for other reasons (American Pain Society, 2012). From a biopsychosocial perspective, chronic pain is the result of the integration and interaction between biological, psychological, sociocultural, and developmental factors (American Pain Society, 2012; Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Current conceptualizations define pain as a sensory, cognitive, and affective experience. Understanding these factors is key to understanding what perpetuates and exacerbates pain, and how to best be able to treat it (Gatchel et al., 2007).

The roots of the biopsychosocial model of chronic pain can be traced back to the gate control theory of pain (Melzack &Wall, 1965). The gate control theory proposes that individual, situational, and environmental factors (such as experience, emotion, and attention) in addition to pure sensory information, contribute to the perception of pain. The gate control theory was able to account for pain that occurred disproportionately to - both greater than and less than - the amount of nerve stimulation (Melzack &Wall, 1965). Prior to the development of the gate control theory, contemporary theories conceptualized pain experiences to be directly proportionate to the degree of sensory input or damage done to tissue. This theory has helped moved the field away from classifying pain as

either “functional” or “organic”, and instead towards considering the integration of many factors contributing to pain (Novy, Nelson, Francis, & Turk, 1995).

Although the hallmark of the biopsychosocial model is in integration of physiological, psychological and sociocultural factors, the emergence of this model has also allowed for the expansion of the number of biological factors under consideration for contributing to pain. In addition to disease, injury, and exposure to noxious stimuli, an individual’s stress response and genetic makeup can also make them more vulnerable to the development of chronic pain (Flor & Hermann, 2004; Gatchel et al., 2007). Research supports a view of chronic pain including physiological, cognitive, behavioral, and sociocultural factors, whereas perspectives on chronic pain which account for only the physiological, social, or psychological factors are not supported by empirical evidence or clinical experience (Novy et al., 1995). The biopsychosocial model led to the development of holistic approaches to treating chronic pain (Gatchel et al., 2007). As discussed in more detail below, multimodal treatments are most effective for the treatment of chronic pain, lending more evidence for a comprehensive model of chronic pain.

The Fear-Avoidance Model of Chronic Pain

Some individuals may be more likely to develop and maintain chronic pain due to emotional and behavioral responses to pain. The fear-avoidance model of chronic pain demonstrates how negative expectancies regarding pain can play an important role in maintaining pain over time. The fear-avoidance model is built on the behavioral theories proposed to explain chronic pain by Fordyce, Shelton, and Dunmore (1982). Two behavioral principles were applied to the experience of pain: reinforcement and

avoidance learning. Positive reinforcement can occur when a person receives special attention or a treat after expressing pain, and negative reinforcement can occur when a person is relieved of responsibility because of pain. An individual also learns that by avoiding certain activities, such as limping to avoid using an injured leg, one can avoid a potentially painful experience (Fordyce et al., 1982). Individuals with negative expectations regarding pain are more likely to avoid potentially painful experiences.

This model of exaggerated pain perception was originally proposed by Lethem and colleagues (1983). The term "fear-avoidance" refers to avoidance of movement or activity due to fear of pain and the consequences of pain. Negative expectancies such as fear of pain, anxiety sensitivity, and pain catastrophizing are central to this model (Vlaeyen & Linton, 2000). Fear is an emotional reaction characterized by sympathetic arousal, and it motivates an individual to escape or engage in defensive behaviors (Leeuw et al., 2007). Anxiety sensitivity refers to the fear of physical symptoms that often accompany the experience of anxiety for fear of negative somatic, social, or psychological consequences (Reiss & McNally, 1985). Pain catastrophizing is an exaggerated cognitive appraisal of pain and perception of the consequences of pain (Sullivan, Bishop, & Pivik, 1995).

The intensity and interpretation of these negative expectancies can result in two outcomes for an individual: confrontation or avoidance. When pain is interpreted as non-threatening, an individual is likely to engage in their regular daily activities, learn that the pain signal was not dangerous, and promote recovery through functioning (Asmundson, 1999; Lethem et al., 1983; Vlaeyen & Linton, 2000). Individuals who have high negative expectancies about pain and the consequences of pain are more likely to avoid pain and

engage in pain behaviors such as reduced activity (Asmundson, 1999). These pain-related behaviors are adaptive in the case of acute pain, but can be counter-productive for chronic pain (Leeuw et al., 2007). By using avoidance in anticipation of pain a person is rewarded by avoiding a painful experience and does not learn that not all similar experiences are painful (Vlaeyen & Linton, 2000).

Escape and avoidance behaviors contribute to the cycle of chronic pain. An individual who escapes from daily activities due to expectations of pain is likely to experience a significant increase in functional disability over time (Vlaeyen & Linton, 2000). Furthermore, exaggerated avoidance behavior may lead to deconditioning of one's body. For example, disuse may increase sensitivity to pain and muscle atrophy, and avoiding active behaviors may increase weight gain. These consequences of avoidance may actually increase pain experiences, negative expectancies of activity, and avoidance behaviors. This cycle of increasing negative expectancies and avoidance behaviors is likely to contribute to maintaining chronic pain and decrease in functioning over time (Asmundson, 1999; Lethem et al., 1983; Vlaeyen & Linton, 2000).

The development and first applications of this model were intended to explain disability in chronic lower back pain patients (Leeuw et al., 2007; Vlaeyen & Linton, 2000). Since then, studies have demonstrated evidence for this model outside of chronic pain and for application to broader chronic pain populations. A population study of adults in Sweden found that fear-avoidance beliefs and pain catastrophizing exist in the general population, with higher pain catastrophizing beliefs associated with higher pain, and higher fear-avoidance beliefs associated with higher functional disability. These findings suggest that fear-avoidance beliefs and pain catastrophizing play a role in the

development and maintenance of chronic pain in the general population (Buer & Linton, 2002).

As for broader applications within chronic pain, associations between fear-avoidance beliefs and disability have been found in adults with leg ulcers (Roaldsen, Elfving, Stanghelle, Talme, & Mattsson, 2009), and in adults with lower back pain (Elfving, Anderson, & Grooten, 2007; Sions, & Hicks, 2011). Among adults with acute lower back pain, both fear of pain and pain catastrophizing are more strongly related to functional disability than pain intensity (Swinkels-Meewisse, Roelofs, Oostendorp, Verbeek, & Vlaeyen, 2006). Within a cross-sectional study of adults with whiplash, fear of movement and pain catastrophizing were predictive of disability. Moreover, fear of movement mediated the relationship between pain catastrophizing and disability (Nieto, Miró, & Huguet, 2009). There is preliminary evidence for application of this model to pediatric populations (Heinze, 2010; Simons & Kaczynski, 2012); however more research in this area is needed. Research has implicated negative expectancies such as fear of pain, anxiety, and pain catastrophizing in playing important roles in pediatric chronic pain. The literature on these constructs and their relationships to important treatment outcomes are discussed next.

Anxiety

Although fear and anxiety are related and the terminology is often used interchangeably, they are distinct constructs and have distinct behavioral consequences regarding pain. Fear refers to the brain's response to acute threat present in one's environment; it is an emotional reaction characterized by sympathetic arousal and it motivates an individual to escape or engage in defensive behaviors. Anxiety, on the other

hand, refers to the state of the brain when sustained cues in the environment predict threat more ambiguously; it is a future oriented state that is associated with avoidance (Leeuw et al., 2007; Pine, Helfinstein, Bar-Haim, Nelson, & Fox, 2009). An anxious state is characterized by physical and cognitive symptoms (Guite & Kazak, 2010). Research across species indicates that anxiety is an adaptive response in many situations to perceived threat in one's environment (Pine et al., 2009). However, anxiety becomes maladaptive when symptoms are distressing to an individual or interfere with an individual's everyday functioning (American Psychiatric Association, 2000). Still, individuals with sub-clinical levels of anxiety might suffer from a similar number of adverse consequences compared to those with clinical levels of anxiety (Wittchen, Nelson, & Lachner, 1998).

Anxiety disorders may be the most common mental disorder in children and adolescents. Prevalence rates of any anxiety disorder for children and adolescents are likely between 15-20%. Considering the longevity of such disorders, the 1- and 6-month rates are not significantly lower (Beesdo, Knappe, & Pine, 2009b). However, in terms of stability, anxiety disorders tend to have a fluid trajectory, with symptoms changing over time, especially in youth (Wittchen, Lieb, Pfister, & Schuster, 2000). Anxiety disorders occur more frequently in females than males, with differences between groups increasing through adolescence to ratios of 2:1 to 3:1 (Beesdo et al., 2009b; Wittchen et al., 1998). Adolescents with anxiety disorders report significant impairment in work, school, leisure, and social activities even when their symptoms are not at their peak (Wittchen et al., 1998). Therefore, it is important to address symptoms of anxiety even when they are not at clinical levels, especially in populations already at risk for functional impairment.

Youth who have a chronic illness are at increased risk for developing an anxiety disorder compared to healthy peers (Pao & Bosk, 2011). For example, youth with asthma and other chronic diseases experienced more panic attacks compared to a healthy comparison group (Rietveld, van Beest, & Prins, 2005). Additionally, anxiety may exacerbate physical symptoms. Among youth with asthma, those with symptoms of anxiety experienced more asthma symptoms, both in frequency and number (Richardson et al., 2006). The relationship between physical illness and anxiety may be cyclical: chronic worry and anxiety lead to decreased immune functioning and increased physical illness (Dhabhar, 2008), while physical symptoms of illness may lead to more triggers for panic attacks and anxiety (Chavira, Garland, Daley, & Hough, 2008; Richardson et al., 2006). Symptoms of several classes of medical conditions, including cardiac, pulmonary, neurological, endocrine, and metabolic disorders resemble symptoms of anxiety (Guite & Kazak, 2010), leaving room for misinterpretation of symptoms by youth. The connection between chronic pain in particular and anxiety may be circular: pain (another physical symptom) may trigger anxious worrying, and the muscle tension associated with anxiety may cause pain (Beesdo et al., 2009a).

Anxiety and Pediatric Chronic Pain

The connection between pediatric chronic pain and anxiety has been well documented. Among youth with recurrent abdominal pain, between 50-79% are reported to meet criteria for a current anxiety disorder (Campo et al., 2004; Dorn et al., 2003); 57.5% of those with juvenile primary fibromyalgia syndrome met criteria for an anxiety disorder (Kashikar-Zuck et al., 2008). Compared to healthy control groups, youth with recurrent abdominal pain (Campo et al., 2004; Galli et al., 2007), headaches (Galli et al.,

2007; Mazzone, Vitiello, Incorpora, & Mazzone, 2006; Smith, Martin-Herz, Marsigan, & Womack, 2003), and non-specific musculoskeletal pain (O'Sullivan, Beales, Jensen, Murray, & Myers, 2011) reported higher anxious and internalizing symptoms.

Directionality between chronic pain and anxiety disorders cannot yet be determined. Ramchandani and colleagues (2007) found that children who had recurrent abdominal pain at age six were more likely to have an anxiety disorder at age seven than other children. However, in another study of youth with recurrent abdominal pain and anxiety disorders, 79% of them reported that the onset of the anxiety disorder preceded the onset of recurrent abdominal pain. The onset of anxiety disorders was significantly more likely to precede the onset of chronic abdominal pain while the onset of depression was more variable (Campo et al., 2004). One research team has speculated that since anxiety often precedes depression in the general population, it is possible that children with chronic abdominal pain may develop depression later (Dorn et al., 2003). More studies in this area are needed before conclusions can be made about temporal relationships between these symptoms.

Additionally, several studies have found that children with chronic pain have higher levels of anxiety sensitivity than children without chronic pain (Lipsitz et al., 2004; Martin, McGrath, Brown, & Katz, 2007b; Tsao et al., 2009; Tsao, Meldrum, Kim, & Zeltzer, 2007). Anxiety sensitivity is a factor often considered a risk factor for development of an anxiety disorder (Reiss & McNally, 1985). This finding suggests that a general predisposition to anxiety may be related to heightened experiences of pain. The rates of these comorbid concerns underscore the importance of understanding underlying psychosocial factors in children who may be at higher risk for chronic pain.

Pain Catastrophizing

Catastrophizing is one of the core irrational beliefs listed by Albert Ellis (1958) and a thinking pattern commonly targeted in cognitive-behavioral therapy (CBT). Irrational beliefs are illogical, not empirically formed and maintained, and inflexible (Brown, Dowd, & Freeman, 2010). Catastrophizing is a negative cognition characterized by the general belief that "when things go wrong it is *awful* and *terrible*" (Brown et al., 2010, pg. 149). Catastrophic worrying is characterized by the inclination to exaggerate and ruminate over negative aspects across many areas of one's life (and even positive experiences), and a tendency to focus one's catastrophizing on personal weaknesses.

Pain catastrophizing is a cognitive risk factor for chronic pain (Michael & Burns, 2004; Muris et al., 2007; Weissman-Fogel, Sprecher, & Pud, 2008) characterized by a negative orientation toward noxious or painful stimuli (Sullivan et al., 1995; Jones, Rollman, White, Hill, & Brooke, 2003). Early definitions of pain catastrophizing varied; however, definitions shared the idea of exaggerated negative appraisals of pain (Sullivan et al., 1995; Martorella, Côté, & Choinière, 2008). Pain catastrophizing is a cognitive distortion directly related to negative thoughts and feelings regarding pain (Michael & Burns, 2004; Drahovzal, Stewart, & Sullivan, 2006). Factor analysis of various definitions has defined three major aspects of pain catastrophizing: rumination, magnification, and helplessness (Sullivan et al., 1995). Pain catastrophizing includes enhanced vigilance to painful stimuli (Muris et al., 2007), extreme focus on painful stimuli (Sullivan et al., 1995; Buer & Linton, 2002; Michael & Burns, 2004) and rumination of painful experiences (Buenaver, Edwards, Smith, Gramling, & Haythornthwaite, 2008). Pain catastrophizing also involves negative expectations about

the painful experience, and exaggerated worry about the consequences of pain (Sullivan et al., 1995). Additionally, pain catastrophizing includes feelings of helplessness (Buenaver et al., 2008), perceived lack of control over symptoms (Jones et al., 2003), and an inability to cope with pain (Sullivan et al., 1995; Jones et al., 2003). Taken together, pain catastrophizing is characterized by negative cognitive and affective tendencies and coping mechanisms specifically in terms of experiencing pain. The result of pain catastrophizing is avoiding situations that may be painful (Muris et al., 2007). These negative perceptions of pain can exacerbate the experience of pain when sensitivity to painful stimuli is high and consequences of pain are perceived to be extremely negative.

Pain Catastrophizing as a Predisposing Factor to Chronic Pain

Pain catastrophizing is believed to be related to stable psychological and biological traits; therefore some individuals are predisposed to developing chronic pain. Buenaver and colleagues (2008) have proposed a variation of the diathesis-stress model, in which a predisposition to catastrophizing painful experiences exacerbates the experience of pain, if and when pain is present. They support their theory with their findings that generally healthy individuals from the general population with problem headaches did not differ from other generally healthy individuals from the general population in terms of reporting pain catastrophizing or their ability to cope with pain. However, within the group with problem headaches, higher pain catastrophizing was related to higher severity headaches, higher pain-related disability, and more symptoms of depression (Buenaver et al., 2008). The finding that higher pain catastrophizing is related to higher levels of pain sensitivity to multiple types of pain among healthy individuals has been replicated elsewhere (Weissman-Fogel et al., 2008). Furthermore,

although gender has often been hypothesized to be a factor in predicting an individual's ability to control pain, pain catastrophizing was found to be a better predictor of pain tolerance than gender (Weissman-Fogel et al., 2008). These findings suggest that pain catastrophizing occurs in the general population, and the experience of pain or a pain condition may be necessary before the negative effects of pain catastrophizing are realized (Buenaver et al., 2008).

Pain catastrophizing is also related to stable temperament and biological traits that are related to sensitivity in processing painful stimuli and pain modulation. The behavioral inhibition system is sensitive to danger and threats in the environment, and increases avoidance behavior. Perceptual sensitivity is the ability to sense mild stimuli, such as pain. Within a population of young adolescents, sensitivity of the behavioral inhibition system, fear, anger-frustration, and perceptual sensitivity were all positively correlated with pain catastrophizing (Muris et al., 2007). Since pain catastrophizing is related to enhanced vigilance, sensitivity, and fear of pain, it follows that a combination of behavioral inhibition system sensitivity, fear, and high perceptual sensitivity would predispose an individual to catastrophizing painful experiences (Muris et al., 2007). Pain catastrophizing is also related to the body's pain modulation system. Pain catastrophizing was found to be negatively correlated with the diffuse noxious inhibitory control effect. This is when pain-signaling neurons in the spinal dorsal horn are weakened in response to presentation of other noxious stimuli. Decreased diffuse noxious inhibitory control results in experiencing greater pain. Higher pain catastrophizing and decreased diffuse noxious inhibitory control are both risk factors for development of chronic pain (Weissman-Fogel et al. 2008). Pain catastrophizing is

related to temperament and biological traits and is likely a predisposing trait for the development of chronic pain and/or experiencing more complications during painful experiences.

Pain Catastrophizing and Pediatric Chronic Pain

Children with pain have commonly been found to experience higher levels of pain catastrophizing than children without chronic pain (Hermann, Hohmeister, Zohsel, Ebinger, & Flor, 2007; Tsao, 2009). In populations of both school children and children with chronic pain, pain catastrophizing has been shown to be a significant predictor of pain intensity above and beyond gender, age, and negative affectivity (Crombez et al., 2003; Vervoort, Goubert, Eccleston, Bijttebier, & Crombez, 2006). Furthermore, pain catastrophizing has been shown to be positively correlated with pain intensity and frequency whereas positive coping such as problem-solving abilities and positive pain-related self-statements were not related to pain intensity and frequency (Hermann et al., 2007). Even in terms of long term effects, pain catastrophizing independently predicts pain intensity six months later, even when controlling for pain intensity and level of disability (Vervoort, Eccleston, Goubert, Buysse, & Crombez, 2010).

Pain catastrophizing has also been described as an ineffective coping strategy among youth with chronic pain. Children with chronic headaches endorsed more pain catastrophizing, whereas children without chronic pain endorsed more positive pain-coping self statements (Hermann et al., 2007). Pain catastrophizing is related to poor effectiveness of coping with pain, especially among females (Lynch et al., 2007). Use of emotion-focused coping strategies (such as catastrophizing) is related to decreased

physical, psychological, and social functioning, and satisfaction with life and health (Merlijn et al., 2006).

Overlap in Anxiety and Catastrophizing as Constructs

Anxiety and catastrophizing are similar in that they are both related to sensitivity to and negative expectancies about somatic symptoms such as pain and their consequences. It is possible that these constructs share an underlying factor of negative affectivity (MacDonald, Linton, & Jansson-Fröjmark, 2008; Noël, Francis, Williams-Outerbridge, & Fung, 2012; Vervoort et al., 2006; Vlaeyen & Linton, 2000). Especially in adolescence, catastrophizing is more closely related to the underlying negative affectivity shared between anxiety and depression than it is more closely aligned with either anxiety or depression alone. In fact, catastrophizing is often observed in anxiety, and rumination—a component of catastrophizing—in particular is a depressogenic cognitive style (Noël et al., 2012). Consequently, there has been speculation regarding whether anxiety and pain catastrophizing are too similar to distinguish (Vlaeyen & Linton, 2000; Asmundson, 1999). While research has demonstrated the two to be distinct constructs, little research has been done to compare the two in children and adolescents or in terms of unique associations with pain-related outcomes variables (Jastrowski Mano et al, 2012). Complicating the picture is the fact that different studies have measured different variations of these constructs (general catastrophizing or pain catastrophizing; anxiety sensitivity, anxiety, or pain anxiety) depending on the measure used.

Variations of these two constructs have been found to be correlated in several studies: anxiety sensitivity and catastrophizing in non-clinical children (Tsao et al. 2009); pain anxiety and catastrophizing in adults with chronic pain (Vowels, McCracken, &

Eccleston, 2008); anxiety sensitivity and pain catastrophizing in non-clinical undergraduates (Drahovzal, et al., 2006); and trait anxiety and pain catastrophizing in undergraduates (Sullivan et al., 1995), adults with migraine (Holroyd, Drew, Cottrell, Romanek, & Heh, 2007), and children both with (Jastrowski Mano et al., 2012) and without chronic pain (Hermann et al., 2007). Aside from pain populations, children (Legerstee, Garnefski, Jellesma, Verhulst, & Utens, 2010) and adolescents with anxiety disorders (Legerstee, Garnefski, Verhulst, & Utens, 2011) reported higher catastrophizing than their non-anxious peers. Furthermore, Langer and colleagues (2009) found that catastrophizing explained a significant amount of variance in anxiety above and beyond demographic and symptom variables in youth with chronic abdominal pain.

While anxiety and catastrophizing are related, there is support for them being different constructs. Drahovzal and colleagues (2006) determined that anxiety sensitivity and pain catastrophizing are separate constructs through principal components analysis; each of the items on the Anxiety Sensitivity Inventory and the Pain Catastrophizing Scale loaded most significantly on their respective factors (Drahovzal, et al., 2006). Furthermore, anxiety and catastrophizing contribute unique variance to important outcomes such as pain and functioning. Pain catastrophizing predicted unique variance in pain ratings over trait anxiety (Sullivan et al., 1995), and in functioning and quality of life controlling for anxiety in adults with migraines (Holroyd et al., 2007). However, in a study of adults with lower back pain, Moix and colleagues (2011) found that catastrophizing no longer predicted disability once trait anxiety was entered into the model. In children with pain, anxiety sensitivity, but not pain catastrophizing was significantly related to current pain (Tsao et al., 2009). The discrepancies in findings

demonstrate the lack of a clear understanding of how these variables interact, and highlight that there is a paucity of research of these constructs in pediatric chronic pain.

Researchers in the field have noted the strong correlations between these two constructs and called for further research working to distinguish the role of each (Hermann et al., 2007). Particularly in research with youth with chronic pain, there is a need to see if anxiety and pain catastrophizing are separate constructs and which better predicts treatment outcomes (Jastrowski Mano et al., 2012). The clinical utility of either of these constructs may be clarified by the extent to which each is related to functional outcomes and can be distinguished from one another (Drahovzal, et al., 2006; Tsao et al., 2009).

The goal of this study is to extend current research in this area and examine the unique predictive abilities of pain catastrophizing and anxiety for functional outcomes in pediatric chronic pain. A measure of anxiety was chosen for this study because of the high prevalence rate of anxiety disorders in the pediatric pain population (Campo et al., 2004; Kashikar-Zuck et al., 2008) and because generalized anxiety symptoms, not just those that are pain specific, can contribute to pain and functional disability (Beesdo et al., 2009b; Wittchen et al., 1998). A measure of pain catastrophizing was chosen because pain catastrophizing is a well-studied construct in pediatric chronic pain, a reliable predictor of functional disability and HRQOL (Merlijn et al., 2006; Vervoort et al., 2010), as well as a feasible target of treatment.

Chronic Pain, HRQOL, and Functional Disability

Many youth with chronic pain experience negative consequences of pain in several domains of their daily lives. The effects of a chronic condition on one's life are

commonly measured by two constructs: functional disability and HRQOL. Functional disability is defined as limitations in daily functioning in home, school, recreation, and social interactions; HRQOL is defined as one's perception of how his or her health status influences physical, psychological, social, and emotional functioning (Long, Krishnamurthy, & Palermo, 2008; Spilker, 1996). Among children with chronic pain, longer history of pain was associated with lower psychological health and overall life satisfaction (Merlijn et al., 2006). Additionally, pain intensity is related to decreased physical and psychological functioning, satisfaction with health and life, as well as increased functional disability (Hunfeld et al., 2001; Merlijn et al., 2006; Palermo, 2000). It is commonly accepted that chronic pain also interferes with a child or adolescent's ability to sleep and eat (Bennett, Huntsman, & Lilley, 2000; Palermo, 2000; Roth-Isigkeit et al., 2005) which perpetuates lower HRQOL and higher functional disability (Long et al., 2008). However, recent research using actigraphy technology has shown that while youth with chronic pain may get similar sleep during the night as their healthy peers, they sleep more during the day which interferes with several domains of functioning (Law, Dufton, & Palermo, 2011).

Physical functioning is negatively affected by having chronic pain, not only in terms of sports involvement (Bennett et al., 2000; Palermo, 2000), but in completing activities of daily living (Bennett et al., 2000; Roth-Isigkeit et al., 2005) and vigorous social activities (Palermo, 2000). Families have reported several social activities needing to be limited due to pain including sports, social get-togethers, family get-togethers, and employment (Bennett et al., 2000). This means that time interacting with peers decreases (Palermo, 2000). Almost half of youth with chronic pain have difficulties meeting

friends (Roth-Isigkeit et al., 2005). It is unknown what is cause and what is effect, but adolescents with chronic pain perceive themselves to be behind their peers in terms of social functioning (Eccleston, Wastell, Crombez, & Jordan, 2008; Roth-Isigkeit et al., 2005). Increased pain intensity has negative effects for adolescents' independence, emotional adjustment, and identity formation; however, strong peer relationships play a protective role and aid social development (Eccleston et al., 2008).

Several studies have noted the problem of decreased school attendance in youth with chronic pain (Bennett et al., 2000; Palermo, 2000; Roth-Isigkeit et al., 2005). In a small sample of children from a tertiary care center for chronic pain, a quarter of youth reported missing over a month of school in the past year (Bennett et al., 2000); a larger sample reported about 40% of youth with chronic pain missing 10 or more days in the last three months (Logan, Simons, & Carpino, 2012). School stress, such as being singled out by teachers and peers and academic pressure, is associated with frequent headaches and stomachaches (Hjern, Alfven, & Östberg, 2008). Children with pain may perceive themselves to have low academic competence and experience high stress at school (Palermo, 2000). Parents also reported that youth with chronic pain have difficulty completing homework (Bennett et al., 2000). Sato and colleagues (2007) discussed psychosocial factors related to school avoidance and common school avoidance behaviors. Notably, children's anxiety moderates the effect of pain on school attendance (Cohen, Vowels, & Eccleston, 2010), and parents' own catastrophizing about their child's pain and protective behaviors predict their child's school attendance and global school functioning (Logan et al., 2012).

Many children with chronic pain also dedicate significant amounts of time to seeking care from professionals, many attending multiple appointments per month. Families may consult many different providers for their child's pain including family doctors, neurologists, rheumatologists, and other specialists (Bennett et al., 2000). This results in high health care costs (Martin et al., 2007a; Palermo, 2000; Roth-Isigkeit et al., 2005), costs associated with taking time off of work, and lost personal time to attend medical appointments (Palermo, 2000). Furthermore, children with chronic pain are at higher risk for having chronic pain during adulthood (e.g. Marugán et al., 2008), which is associated with further limitations and costs. Long-term disability due to chronic pain incurs great costs to society as a whole and has increasingly negative effects on one's quality of life and psychological well-being.

It is well established that pain is inversely related to HRQOL in children with chronic pain (Hunfeld et al., 2001; Merlijn et al., 2006; Palermo, 2000); however, recent research has demonstrated that psychosocial factors are more predictive of HRQOL and functional disability beyond pain variables alone. In fact, in a study of adults with various chronic pain diagnoses, psychological factors including anxiety, depression, and pain catastrophizing were strongly related to HRQOL and functional disability in contrast to smaller correlations with pain intensity, functional disability and HRQOL (Börsbo, Peolsson, & Gerdle, 2009). In adolescents with chronic pain, psychosocial factors explained an additional 29% of variance of psychological functioning above and beyond pain characteristics alone, 23% social functioning, 15% physical functioning, and 30% overall life satisfaction (Merlijn et al., 2006).

Anxiety, HRQOL, and Functional Disability

As noted earlier, symptoms of anxiety are common among youth with chronic pain (Campo et al., 2004; Kashikar-Zuck et al., 2008). These symptoms may negatively influence a child's functioning and HRQOL. Even when an individual with chronic pain is not experiencing pain, he or she may anticipate having pain during an activity, worry about questions from peers about their pain, or worry about their loss of functioning due to pain (Powers, Gilman, & Hershey, 2006). Gauntlett-Gilbert and Eccleston (2007) found that anxiety and functional disability were correlated in a highly disabled sample of adolescents with chronic pain. Similarly, anxiety and functional disability were correlated in youth with recurrent abdominal pain (Walker & Greene, 1991). Within a sample of youth with juvenile fibromyalgia syndrome, those with an anxiety disorder were functioning more poorly than those without an anxiety disorder (Kashikar-Zuck et al., 2008). In youth with various chronic pain diagnoses, Tsao and colleagues (2007) found anxiety sensitivity to explain unique variance in psychological and social functioning, but not physical functioning, above and beyond demographic and pain specific variables.

We know that pain symptoms also influence functioning, and may interact with anxiety to affect functioning. Some research teams have found that anxiety may moderate the relationship between pain and functioning in youth with chronic pain. Cohen and colleagues found that there was an interaction effect between pain and anxiety such that at low levels of anxiety, both pain and anxiety are related to functioning, whereas, at high levels of anxiety, only anxiety and not pain was related to functioning. Results indicated that decreased physical functioning, increased school absences, and

frequent medical visits were reported when anxiety was high regardless of pain intensity. Therefore they suggest that anxiety, and not pain intensity, may be the driving force behind disability (Cohen et al., 2010). Interestingly, Wendland and colleagues (2010) found a different moderation effect in a sample of youth with chronic abdominal pain. For youth with low symptoms of anxiety, as pain increased, functional disability stayed low; but for those with high symptoms of anxiety, as pain increased functional disability significantly increased. So unlike Cohen et al.'s sample, youth with high symptoms of anxiety were not uniformly disabled, rather only those with high pain and high anxiety had high functional disability (Wendland, Jackson, & Stokes, 2010).

Pain Catastrophizing, HRQOL, and Functional Disability

Individuals with chronic pain who have higher pain catastrophizing are more likely to feel disabled by their pain and suffer more psychological distress than those who have lower pain catastrophizing (Severeijns, Vlaeyen, van den Hout, & Weber, 2001). This has been well documented in adults, and there is support in pediatric chronic pain as well. Crombez et al. (2003) found that pain catastrophizing significantly predicted functional disability in children with chronic or recurrent pain above and beyond gender, age, and pain intensity. Similarly, Vervoort and colleagues (2006) found that pain catastrophizing better predicted disability beyond gender, age, and negative affectivity in both community and clinical samples of children. In pediatric fibromyalgia, higher pain catastrophizing predicted decreased HRQOL in children and adolescents with fibromyalgia (Libby & Glenwick, 2010).

Pain catastrophizing moderates the relationship between pain and HRQOL, as well as predicts functional disability six months later. A community sample of children

with chronic pain demonstrated that emotion-focused avoidance coping, including pain catastrophizing, strengthens the relationship between pain and HRQOL such that at high levels of emotion-focused avoidance coping, greater pain was related to lower HRQOL (Merlijn et al., 2006). Vervoort and colleagues (2010) found that pain catastrophizing predicts decreased functional disability six months later, even after controlling for baseline pain and initial disability levels. In this study, trait anxiety was also measured and did not predict pain intensity or functional disability six months later (Vervoort et al., 2010).

Treatment of Pediatric Chronic Pain

Due to the complex relationship between pediatric chronic pain and functioning, the primary goal of treating pediatric chronic pain should be to improve functioning in all affected areas and to improve quality of life (American Pain Society, 2012). A multidisciplinary approach for the treatment of pediatric chronic pain based on the biopsychosocial model is supported by research. In this way, an individual's pain symptoms are treated in addition to targeting the individual's ability to cope with chronic pain and functional disability (Kashikar-Zuck, 2006). The most beneficial treatment for chronic pain includes CBT, physical and occupational rehabilitation, and standard medical care (American Pain Society, 2012).

Treatment research has shown CBT to be an important part of a pediatric pain intervention. In fact, without treatment targeting these symptoms, psychological symptoms may diminish the effect that medication is intended to have. Sullivan and colleagues (2008) showed that increased pain catastrophizing is related to decreased responding to analgesic medication. Their study examined the effects of catastrophizing

in adults with neuropathic pain receiving either medication or a placebo. Individuals with high pain catastrophizing in the placebo group had greater pain reduction, while those with high pain catastrophizing in the medication group had less pain reduction (Sullivan, Lynch, Clark, Mankovsky, & Sawynok, 2008). These findings demonstrate a need for treatment of chronic pain to target both physical and psychological symptoms.

A combination of pharmacological and psychological treatment options is indicated particularly for individuals with anxiety (Beesdo et al., 2009a) and for youth with a tendency to catastrophize (Merlijn et al., 2006). If anxiety does moderate the effect between pain and functioning as found in Cohen et al. 2010, then it might be expected that youth with high anxiety would experience decreased functioning regardless of their pain level. Therefore, anxiety would need to be a primary target of treatment in order to improve functioning. According to Cohen and colleagues' findings (2010), when anxiety is brought down to a manageable level, then the relationship between pain and functioning will be stronger.

Generally speaking, CBT can help youth with pain identify the relationships between their thoughts, feelings, and behavior (American Pain Society, 2012). Pain-specific CBT helps individuals learn how to cope with pain; this may include relaxation training, activity pacing, progressive muscle relaxation, and guided imagery. CBT also targets pain-related thoughts and anxiety, and fostering problem solving (Kashikar-Zuck, 2006). The process of changing thinking patterns around pain may be necessary in order to decrease fear of pain and avoidance of activities in anticipation of pain. Furthermore, treatments which combine cognitive treatments with graded in-vivo exposure have been shown to be more effective than education, physical therapy, or traditional CBT alone

due to the ability to confront feared activities and disconfirm the fear of pain (Lohnberg, 2007). Working with parents and schools is also critical to care as they will be key players in helping the child increase his or her functioning (Kashikar-Zuck, 2006).

As outlined above, there are numerous areas of psychosocial functioning intertwined in the experience of chronic pain, and targeting these constructs in treatment may be necessary to alleviate chronic pain. One meta-analysis of psychological treatments for pediatric chronic pain found that CBT was effective in reducing the frequency and severity of pain in children and adolescents (Eccleston, Morley, Williams, Yorke, & Mastroyannopoulou, 2002). Recent research has demonstrated that adding CBT to standard medical care is more effective in treating chronic pain than standard medical care alone. Specifically, in youth with fibromyalgia, coping skills training was related to greater confidence in coping with pain compared to self-monitoring (Kashikar-Zuck, Swain, Jones, & Graham, 2005). Additionally, Robins and colleagues (2005) found that five sessions of family CBT in addition to standard medical care was more effective in reducing recurrent abdominal pain symptoms immediately after treatment and six to twelve months later compared to standard medical care alone (Robins, Smith, Glutting, & Bishop, 2005).

While the field has advanced in knowledge of what treatments are effective in reducing pain symptoms in youth with chronic pain, few studies have included outcome measurements other than pain severity (Eccleston et al., 2006). The limited outcome data are misleading since a primary goal in treating pediatric chronic pain is to improve functioning and HRQOL (American Pain Society, 2012). This also limits what is known about mechanisms of change in symptom improvement (Eccleston et al., 2006). In future

studies it will be important to include functional outcomes such as functional disability and HRQOL in addition to pain symptoms. Additionally, measurement of constructs believed to cause changes in outcome variables will be beneficial. Identification of intervening variables and better understanding of relationships between variables will help clinicians to know what constructs to target in treatment, thereby shortening treatment and strengthening outcomes.

Current Research Questions

The purpose of this study was to investigate the longitudinal associations of pain catastrophizing and anxiety with treatment outcomes in a sample of youth seeking treatment for chronic pain. This study addressed the following questions: What are the longitudinal relationships between anxiety, pain catastrophizing, functional disability, HRQOL, and pain intensity? How well do anxiety and pain catastrophizing predict functioning outcomes? What is the unique predictive ability of anxiety and pain catastrophizing to predict outcomes? Do changes in anxiety and pain catastrophizing predict later pain and/or functioning?

What are the longitudinal relationships between anxiety, pain catastrophizing, functional disability, HRQOL, and pain intensity? Based on previous research demonstrating that anxiety is related to pain (Campo et al., 2004; Kashikar-Zuck et al., 2008), functional disability (Cohen et al., 2010; Wendland et al., 2010) and HRQOL (Tsao et al., 2007) and that pain catastrophizing is related to pain (Crombez et al., 2003; Vervoort et al., 2006), functional disability (Vervoort et al., 2010), and HRQOL (Libby & Glenwick, 2010) the first hypothesis was that *both higher anxiety*

and pain catastrophizing were related to higher pain scores and functional disability, and lower HRQOL at baseline.

How well do anxiety and pain catastrophizing predict outcomes in youth seeking multidisciplinary treatment? Based on previous research (Cohen et al., 2010; Vervoort et al., 2010; Wendland et al., 2010), the second hypothesis was that *the relationships between anxiety, pain catastrophizing, and functioning outcomes would continue to be significant after controlling for demographic variables, and in the cases of functional disability and HRQOL for pain characteristics*.

What is the unique predictive ability of anxiety and pain catastrophizing to predict changes in these outcome variables? The third hypothesis was that *anxiety and pain catastrophizing would each contribute unique variability in predicting pain scores, functional disability, and HRQOL* (Holroyd et al., 2007; Moix et al., 2011; Sullivan et al., 1995; Tsao et al., 2009).

Do changes in anxiety and pain catastrophizing predict later pain or functioning? There is a lack of longitudinal studies examining relationships between variables over time in pediatric chronic pain. The final goal of this study was to examine changes in outcome variables over time and variables associated with change. The fourth hypothesis was that *anxiety, pain catastrophizing, pain, and functional disability would decrease over time, and that HRQOL would increase over time*. Clinically, it is important to know if changes in variables representing the targets of treatment (anxiety symptoms and pain catastrophizing) are associated with desired effects in treatment outcome variables (pain, functional disability, and HRQOL). Subsequently, the fifth hypothesis was that *changes in anxiety and pain catastrophizing from baseline to one*

month follow-up predicted functioning at the one month follow-up, and that changes in anxiety and pain catastrophizing from one month to three month follow-up predicted functioning at three month follow-up. Exploratory analyses examined an alternative fifth hypothesis examining independent variables as static variables (rather than change scores) predicting later functioning: *anxiety and pain catastrophizing at baseline predicted functioning at the one month follow-up, and anxiety and pain catastrophizing at one month predicted functioning at three month follow-up*.

Methods

Participants

Participants included 725 youth and their parents who received services at the Jane B. Pettit Pain and Palliative Care Center, an outpatient interdisciplinary pain clinic at Children's Hospital of Wisconsin for complex chronic pain. Youth were referred to the pain clinic by a physician or specialized tertiary care clinic recommending further treatment for pain. Inclusion criteria included: referral to the pain clinic for pain management, completion of the measures listed below, and age between 8 and 18. Exclusion criteria include history of significant developmental delay and non-English speaking families; these families were not given questionnaires to complete, and therefore were not included in the study database.

Participants included 500 females (69.0%) and 225 males (31.0%). The sample was 74.8% Caucasian, ranging in age from 8 to 18 at intake (M = 14.11 years, SD = 2.45). The distribution of sex and ethnicity of this sample is similar to other pediatric chronic pain clinics (Simons, Sieberg, & Claar, 2012). The most frequently reported primary pain location was head (37%), followed by abdomen (15%), lower extremity (15%), and back (14%). Seventeen percent of the sample reported a second pain location, and 4% reported a third pain location.

Each participant attended an intake appointment and was given a treatment plan by the medical team and mental health professional. After this intake appointment, the level of treatment that each participant received from the medical team, mental health team, and from other outside providers is variable and the details for each family are unknown. Some families are followed closely by the pain team and are seen every week;

at the other extreme, some families come into an intake appointment for consultation and are followed elsewhere. Patients are not classified as "drop-outs" if they are not followed immediately by the pain clinic as many patients return for treatment later after seeming not to have engaged. Patients completed longitudinal questionnaires at home and mailed back questionnaires, so they need not be actively engaged with the treatment team in order to gather data from these families. Furthermore, the extent to which PC and anxiety were targeted in treatment is unknown. Therefore, the results of this study should be considered as longitudinal naturalistic observation of relationships between variables over time among families seeking treatment, rather than as a treatment outcome study.

Participants were asked to complete questionnaires at three time points: at their intake appointment, one month post-intake, and three months post-intake. Of the 725 youth who completed measures at their intake appointment and are therefore eligible for the study, 533 completed the required measures at the intake appointment only, 79 completed the intake and one month questionnaires, 21 completed the intake and three month questionnaires, and 92 completed the measures at all three time points. The 92 youth who completed measures at all three time points did not differ significantly from those who did not complete all measures in terms of age, ethnicity, pain location, duration, frequency, or severity, SCARED, PCS-C, CALQ, or PedsQL. There was a statistically significant difference in the gender distribution of the sample that completed measures at all three time points compared to those who did not complete measures at all three time points, with female participants more likely to complete all three surveys than male (smaller sample total $n = 92$, 19 males [21%], 73 females [79%], $\chi^2 = 5.31$, $p < .05$); however, there was a small effect size for this difference (phi = .09).

Procedure

Data were gathered from a retrospective chart review of questionnaires completed by families at their intake appointment at the pain clinic (baseline), one month, and three month follow-up. Families were mailed a packet including questionnaires for the youth with chronic pain and their parents/guardians. Families were asked to complete these questionnaires before their first visit to the pain clinic and family members were asked to complete the questionnaires independently of one another. Families were then mailed questionnaires and a stamped and addressed envelope approximately one and three months after their intake appointment at the pain clinic. Questionnaire data were compiled into a de-identified database combined with data from the physicians and mental health professionals. The Institutional Review Boards at the Children's Hospital of Wisconsin and the University of Wisconsin-Milwaukee approved this retrospective data review.

Measures

The data used for this study are part of a larger clinic database. Only the measures containing the variables of interest were examined in this investigation.

Demographic information. At the intake appointment at the pain clinic, parents provided basic demographic information (e.g., patient's age and gender, family ethnicity) on the New Patient Questionnaire form.

Pain ratings. As a part of the questionnaire packet, youth recorded duration of their pain, their usual and worst pain intensity rating on a scale of 0 (no pain) to 10 (most pain possible), and pain the frequency of their pain (number of days per two week period, 0-14).

Anxiety. The *Screen for Child Anxiety Related Disorders* (SCARED; Birmaher et al., 1999) is a 41-item validated child report of anxiety symptoms with good reliability and validity able to discriminate between anxiety disorders and depression, and anxiety and disruptive disorders, as well as within different anxiety disorders in children and adolescents (Birmaher et al., 1999). This measure has recently demonstrated equivalent psychometric properties as a screening tool in an outpatient sample of non-Hispanic White and African American youth (Gonzalez, Weersing, Warnick, Scahill, & Woolston, 2012), as well as good psychometric properties in youth with chronic pain ($\alpha = 0.93$; Jastrowski Mano et al., 2012). The SCARED yields a total anxiety score and several subscales: panic or somatic symptoms, generalized anxiety, separation anxiety, social anxiety, and school avoidance. The total score ranges from 0 to 82 with a clinical cutoff score of 25, which optimizes the sensitivity and specificity when discriminating anxiety disorders from other groups (Birmaher et al., 1999). For this investigation, the total score was used for all analyses.

Pain catastrophizing. The *Pain Catastrophizing Scale for Children* (PCS-C; Crombez et al., 2003) is a child self-report of pain catastrophizing symptoms based on the Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) self-report for adults. The child adaptation includes 13 rephrased questions so that they are easily read at the fourth grade level and a relabeled five-point Likert scale ranging from 0 (Not at all) to 4 (Extremely). The PCS-C has been validated in children ages 8 to 16 in both community and clinical populations. The validation of the PCS-C confirmed use of three subscales of catastrophizing: *rumination*, *magnification*, and *helplessness* ($\alpha = 0.73$, 0.68, 0.79 respectively, and $\alpha = 0.87$ for the total score; Crombez et al., 2003). *Rumination*

describes the tendency to dwell on thoughts and worries while being unable to stop. *Magnification* reflects the exaggeration of the negative sensations of pain and negative expectancies of pain. The final subscale, *helplessness*, describes the inability to cope with painful stimuli (Sullivan et al., 1995). For this investigation, the total score was used for all analyses.

Functional disability. The *Child Activity Limitations Questionnaire* (CALQ*;* Hainsworth, Davies, Khan, & Weisman, 2007*)* is a 21-item child self-report measure of his or her own functional disability. The respondent answers how difficult 21 activities are due to pain on a Likert scale from 0 (Not at all difficult) to 5 (Extremely difficult), for a possible total score ranging from 0 to 105. It is a written version of the Child Activity Limitations Interview (Palermo, Witherspoon, Valenzuela, & Drotar, 2004). The CALQ has demonstrated good internal consistency ($\alpha = 0.91$), construct and discriminant validity as a self-report written questionnaire for children ages 8 to 18 (Hainsworth et al., 2007).

HRQOL. The *Pediatric Quality of Life Inventory* (PedsQL 4.0; Varni, Seid, & Rode, 1999) is a child self-report measure of HRQOL. The PedsQL 4.0 is a 23-item measure and has been validated for use with children ages 8 to 18 in both community and pediatric settings. This measure has demonstrated good internal consistency as a whole measure ($\alpha = 0.88$), and has four supported reliable and valid subscales: Physical, Emotional, Social, and School (Varni, Seid, & Kurtin, 2001). The total score ranges from 0 to 100, with higher scores indicating better HRQOL. For this investigation, the total score was used for all analyses.

Data Analyses Plan

Descriptive statistics of the study variables were computed and an analysis of the internal consistency of each measure was conducted. The range, mean, and standard deviation of scores on each measure were computed. Following this, demographic characteristics (age, gender, pain duration) were compared with each of the variables under study to determine the need for covariates. See Table 1 for an overview of all data analyses.

Hypothesis 1: Zero order correlations at baseline. In order to test the first hypothesis, *that both higher anxiety and pain catastrophizing were related to higher pain scores and functional disability, and lower HRQOL at baseline,* Pearson correlations were conducted between scores on the SCARED, PCS-C, pain intensity, CALQ, and PedsQL collected at baseline.

Hypothesis 2: Predicting functioning variables from anxiety and pain catastrophizing at baseline. In order to test the second hypothesis, *that both higher anxiety and pain catastrophizing were related to higher pain scores and functional disability, and lower HRQOL at baseline after controlling for demographic variables, and, in the cases of functional disability and HRQOL, for pain characteristics*, hierarchical multiple regression models were conducted predicting treatment outcomes. Six separate regression analyses were conducted to examine relationships between variables at baseline using either total SCARED scores or total PCS-C scores to predict the three outcome variables: pain intensity, CALQ scores, and PedsQL scores. Demographic variables found to be related to SCARED scores, PCS-C scores, pain intensity, CALQ scores, and PedsQL scores were entered first, followed by pain

variables, in the cases of predicting CALQ and PedsQL scores, and finally either SCARED total scores or PCS-C total scores were entered into the final step of the regression.

Hypothesis 3: Finding unique variance in predicting functioning variables from anxiety and pain catastrophizing at baseline. In order to test the third hypothesis, which is *that anxiety and pain catastrophizing would contribute unique variability in predicting pain scores, functional disability, and HRQOL,* hierarchical multiple regression models were conducted predicting treatment outcomes. Three regression analyses were conducted to determine if PCS-C scores add additional variance to models predicting pain intensity, CALQ, and PedsQL scores above and beyond SCARED scores. Demographic variables related to SCARED, PCS-C, pain, CALQ, and PedsQL scores were entered first, then in the cases of predicting CALQ and PedsQL scores, pain variables were entered, then SCARED total scores were entered, followed by PCS-C total scores.

Three additional regression analyses were conducted to determine if SCARED scores add additional variance to models predicting pain intensity, CALQ, and PedsQL scores above and beyond pain catastrophizing. Demographic variables related to SCARED, PCS-C, pain, CALQ, and PedsQL scores were entered first, then in the cases of predicting CALQ and PedsQL scores, pain variables were entered, next PCS-C total scores were entered, followed by total SCARED scores.

Hypothesis 4: Examining changes in anxiety, pain catastrophizing, pain, functional disability, and HRQOL over time. The next set of analyses examined changes in the variables under study over time. In order to test the fourth hypothesis, *that*

anxiety, pain catastrophizing, pain, and functional disability would decrease over time, and that HRQOL would increase over time, repeated measures t-tests were conducted assessing differences between baseline and one month follow-up, and one month and three month follow-up using SCARED, PCS-C, pain intensity scores, CALQ, and PedsQL scores.

Hypothesis 5: Predicting functioning variables at one month from change in anxiety and pain catastrophizing from baseline to one month, and predicting functioning variables at three months from change in anxiety and pain catastrophizing from one month to three months. In order to test the final hypothesis, which is *that changes in anxiety and pain catastrophizing from baseline to one month follow-up predicted functioning outcomes at the one month follow-up, and that changes in anxiety and pain catastrophizing from one month to three month follow-up predicted functioning outcomes at three month follow-up*, hierarchical multiple regression models predicting functioning were conducted. Separate regression analyses were completed for each outcome variable at one and three month follow-up. Demographic variables found to be related to SCARED, PCS-C, and outcome variable scores were entered into the first step, followed by pain variables at baseline (one month), the outcome variable at baseline (one month), and finally the SCARED and PCS-C change scores from baseline (one month) to one month (three month) follow-up.

Exploratory Analyses: Predicting functioning variables at one month from anxiety and pain catastrophizing from baseline and predicting functioning variables at three months from anxiety and pain catastrophizing from one month. In order to test the alternative fifth hypothesis, which is *that anxiety and pain catastrophizing at*

baseline predict functioning at the one month follow-up, *and that anxiety and pain catastrophizing at one month predict functioning at three month follow-up*, hierarchical linear regression models predicting functioning were conducted. Separate regression analyses were completed for each outcome variable at one and three month follow-up. Demographic variables found to be related to the SCARED, PCS-C, and the outcome variable were entered first, followed by pain variables at baseline (one month), the outcome variable at baseline (one month), and finally the SCARED total score and PCS-C total score from baseline (one month).

Results

Descriptive Statistics

Pain ratings. Youth reported a mean duration of pain problem of 25.00 months (*SD* = 29.42, range = 1-204 months, median 12.00 months). Over a two-week period, youth reported having pain an average of 10.82 days (*SD* = 4.04, range = 0-14 days). On a scale of 0-10, 10 being the most intense pain, youth reported their usual pain to be an average of 6.40 (*SD* = 2.04, range = 0-10), and average worst pain as 8.21 (*SD* = 1.97, range = 0-10).

SCARED. Total child reported SCARED scores at baseline (*n* = 709) ranged from 0 to 69 (*M* = 19.55, *SD* = 14.00) out of a possible range of 0 to 82, and the scale demonstrated excellent internal consistency (α = .94). At one month follow-up (*n* = 171), scores ranged from 0 to 64 (*M* = 19.88, *SD* = 16.26), and the scale demonstrated excellent internal consistency (α = .96). At three month follow-up (*n* = 113), scores ranged from 0 to 72 (*M* = 16.84, *SD* = 15.25), and the scale demonstrated excellent internal consistency (α = .96).

PCS-C. Total PCS-C (child version) scores at baseline (*n* = 711) ranged from 0 to 52 (*M* = 25.38, *SD* = 12.74) out of a possible range of 0 to 52, and the scale demonstrated excellent internal consistency (α = .94). At one month follow-up (*n* = 171), scores ranged from 0 to 52 (*M* = 22.86, *SD* = 13.99), and the scale demonstrated excellent internal consistency (α = .96). At three month follow-up (*n* = 112), scores ranged from 0 to 52 (*M* = 19.55, *SD* = 14.38), and the scale demonstrated excellent internal consistency (α = .96).

CALQ. Total child reported CALQ scores at baseline (n = 717) ranged from 0 to 105 (M = 40.31, SD = 22.84) out of a possible range of 0 to 105, and the scale demonstrated excellent internal consistency (α = .95). At one month follow-up (n = 170), scores ranged from 0 to 85 (M = 33.76, SD = 23.32), and the scale demonstrated excellent internal consistency (α = .96). At three month follow-up (n = 113), scores ranged from 0 to 95 (M = 29.39, SD = 24.26), and the scale demonstrated excellent internal consistency (α = .96).

PedsQL. Total child reported PedsQL scores at baseline (n = 720) ranged from 1.39 to 100.00 (M = 58.47, SD = 17.59) out of a possible range of 0 to 100, and the scale demonstrated excellent internal consistency (α = .90). At one month follow-up (n = 173), scores ranged from 20.24 to 100.00 (M = 62.26, SD = 18.55), and the scale demonstrated excellent internal consistency (α = .92). At three month follow-up (n = 113), scores ranged from 25.00 to 98.91 (M = 66.43, SD = 19.53), and the scale demonstrated excellent internal consistency (α = .94).

Examination of Potential Covariates

Gender, age, and pain characteristics were examined in relation to SCARED, PCS-C, CALQ, and PedsQL scores at all three time points. Older age was related to higher usual and worst pain intensity (r = .66, p < .001, and r = .12, p < .01 respectively), higher CALQ scores (r = .13, p < .01), and lower PedsQL scores at baseline (r = -.09, p < .05). Female participants scored higher on the SCARED at baseline (t[707] = -4.07, p < .001), higher on the PCS-C at baseline (t[709] = -2.88, p < .01), higher on the CALQ at baseline (t[715] = -3.02, p < .01), higher on the CALQ at one month (t[168] = -2.48, p <

.05), lower on the PedsQL at baseline (t[718] = 2.06, $p < .05$), and lower on the PedsQL at one month follow-up (t[171] = 2.60, $p < .05$) compared to males.

Females reported more frequent pain than males (t[695] = -3.90, $p < .001$), more intense usual pain (t[696] = -3.45, $p < .01$), and worse pain (t[700] = -3.74, $p < .001$) compared to males. Longer pain duration was related to lower CALQ scores at baseline (r = -.13, $p < .01$) and higher PedsQL scores at baseline (r = .10, $p < .05$). Pain frequency was related to PCS-C, CALQ, and PedsQL scores; usual and worst pain intensity were related to SCARED, PCS-C, CALQ, and PedsQL scores and correlation values are included in Table 2.

SCARED scores and PCS-C scores are the independent variables used in this study. These scores were moderately correlated at each time point: r = .53, r = .61, r = .57, at baseline, one month follow-up, and three month follow-up respectively, all $p <$ .001.

Hypothesis 1: Zero Order Correlations at Baseline

Pearson correlations were conducted to test the first hypothesis, that *both higher anxiety and pain catastrophizing were related to higher pain scores and functional disability, and lower HRQOL at baseline*. SCARED scores were significantly correlated with usual and worst pain intensity (r = .17, $p < .001$ and r = .11, $p < .01$ respectively), CALQ (r = .30, $p < .001$), and PedsQL scores (r = -.51, $p < .001$). PCS-C scores were significantly correlated with usual and worst pain intensity (r = .38, $p < .001$ and r = .36, $p < .001$ respectively), CALQ (r = .48, $p < .001$), and PedsQL scores (r = -.50, $p < .001$). Results supported the first hypothesis.

Hypothesis 2: Predicting Functioning Variables from Anxiety and Pain Catastrophizing at Baseline

Hierarchical multiple regressions were conducted in order to test the second hypothesis, that *both higher anxiety and pain catastrophizing were related to higher pain scores and functional disability, and lower HRQOL at baseline after controlling for demographic variables, and, in the cases of functional disability and HRQOL, for pain characteristics*. Three linear regression models were conducted using the SCARED to predict pain intensity, CALQ scores, and PedsQL scores; three linear regression models were conducted using PCS-C scores to predict pain intensity, CALQ scores, and PedsQL scores. Gender was controlled for in predicting pain intensity; age, gender, pain duration, frequency, and intensity were controlled for in predicting CALQ and PedsQL scores.

Both models predicting pain with SCARED and PCS-C scores were significant ($R = .20$, $p < .001$ and $R = .39$, $p < .001$ respectively) adding a significant amount of variance (2% and 13% respectively) to models predicting pain intensity over gender (see Table 3). Similarly, both models predicting CALQ scores with SCARED and PCS-C scores were significant ($R = .58$, $p < .001$ and $R = .61$, $p < .001$ respectively) adding a significant amount of variance (4% and 8% respectively) to models predicting CALQ scores over age, gender, and pain characteristics (see Table 4); both models predicting PedsQL scores with SCARED and PCS-C scores were also significant ($R = .65$, $p < .001$ and $R = .58$, $p < .001$ respectively) adding a significant amount of variance (19% and 12% respectively) to models predicting HRQOL scores over age, gender, and pain characteristics (see Table 5). Results supported the second hypothesis.

Hypothesis 3: Finding Unique Variance in Predicting Functioning Variables from Anxiety and Pain Catastrophizing at Baseline

Hierarchical multiple regressions were conducted to test the third hypothesis, that *the SCARED and PCS-C would contribute unique variability in predicting pain scores, functional disability, and HRQOL.* Three regression analyses were conducted to determine if PCS-C scores add additional variance to the models predicting pain intensity, CALQ, and PedsQL scores above and beyond SCARED scores. PCS-C scores added a significant amount of variance (11%) to the model predicting pain intensity (model R = .39, $p < .001$, see Table 3). PCS-C scores also added a significant amount of variance (4%) to the model predicting CALQ (model R = .61, $p < .001$, see Table 4) and PedsQL scores (added 2%; model R = .66, $p < .001$ see Table 5).

Three additional regression analyses were conducted to determine if SCARED scores added additional variance to the models predicting pain intensity, CALQ, and PedsQL scores above and beyond PCS-C scores. SCARED scores did not add a significant amount of variance to the model predicting pain intensity (model R = .39, $p < .001$; $\beta = -.04$, $p > .05$, see Table 3). SCARED scores did, however, contribute a significant amount of variance (1%) in predicting CALQ scores (model R = .61, $p < .001$, see Table 4); and PedsQL scores (added 10% variance; model R = .66, $p < .001$, see Table 5). Results partially supported the third hypothesis.

Summary of Results for Hypotheses 2 and 3

The following is a summary of the results from hypotheses two and three together. For predicting pain at baseline, SCARED scores were significant predictors in the model without PCS-C scores and accounted for 15% of unique variance in pain

intensity; PCS-C scores were significant predictors in the model without SCARED scores and accounted for 37% of unique variance in pain intensity. In the model with both SCARED and PCS-C scores, SCARED scores accounted for 3% of variance (ns) and PCS-C scores accounted for 33% (Table 3).

For predicting CALQ scores at baseline, SCARED scores were significant predictors in the model without PCS-C scores and accounted for 21% of unique variance in CALQ scores; PCS-C scores were significant predictors in the model without SCARED scores and accounted for 29% of the unique variance in CALQ scores. In the model with both predictors, both the SCARED and PCS-C were significant; SCARED scores accounted for 8% of variance and PCS-C scores accounted for 21% (Table 4).

For predicting PedsQL scores at baseline, SCARED scores were significant in the model without the PCS-C and accounted for 44% of unique variance in PedsQL scores; PCS-C scores were significant in the model without SCARED scores and accounted for 35% of the unique variance in PedsQL scores. In the model with both predictors, both SCARED and PCS-C scores were significant; SCARED scores accounted for 31% of variance and PCS-C scores accounted for 14% (Table 5).

Hypothesis 4: Examining Changes in Anxiety, Pain Catastrophizing, Pain, Functional Disability, and HRQOL over Time

Repeated measure t-tests were conducted in order to examine test the fourth hypothesis, *that anxiety, pain catastrophizing, pain, and functional disability would decrease over time, and that HRQOL would increase over time*. Differences between SCARED total, PCS-C total, pain intensity, CALQ total, and PedsQL total scores at baseline and one month follow-up, and one month and three month follow-up were

examined. Exploratory analyses examined differences between baseline and three month follow-up. SCARED scores did not differ between the three time points. PCS-C, CALQ, and PedsQL scores were significantly different in the expected direction at each time point. Pain significantly decreased between baseline and one month, and baseline and three month follow-up (Table 6). Results partially supported the fourth hypothesis.

Hypothesis 5: Predicting Functioning Variables at One Month from Change in Anxiety and Pain Catastrophizing from Baseline to One Month and Predicting Functioning Variables at Three Months from Change in Anxiety and Pain Catastrophizing from One Month to Three Months

Hierarchical multiple regression models predicting functioning were conducted to test the final hypothesis, that *changes in anxiety and pain catastrophizing from baseline to one month follow-up predicted functioning at the one month follow-up, and that changes in anxiety and pain catastrophizing from one month to three month follow-up predicted functioning at three month follow-up*. Separate regression analyses were completed for each outcome variable at one and three month follow-up. First, demographic variables found to be related to anxiety, pain catastrophizing, and the outcome variable at baseline (one month) were entered, followed by pain variables at baseline (one month), the outcome variable at baseline (one month), and finally the change scores from baseline (one month) to one month (three month) follow-up of the SCARED total score and PCS-C total score were entered into the final step of the regression.

Predicting pain at one month follow-up. The model with gender, pain intensity at baseline, and change scores for both the SCARED and PCS-C from baseline to one

month follow-up was significant (R = .66, $p < .001$). Adding in the SCARED and PCS-C change scores added 10% of the variance in pain ($p < .001$), and both the SCARED change score coefficient and the PCS-C change score coefficient were significant in the final model (Table 7).

Predicting pain at three month follow-up. The model with gender, pain intensity at one month, and both SCARED and PCS-C change scores from one month to three month follow-up was significant (R = .77, $p < .001$). Adding in the SCARED and PCS-C change scores added 1% variance explained (p = ns), and neither of the change score coefficients was significant in the final model (Table 8).

Exploratory analysis: Predicting pain at three month follow-up from baseline. The model with gender, pain intensity at baseline, and both SCARED and PCS-C change scores from baseline to three month follow-up was significant (R = .55, $p < .001$). Adding in the SCARED and PCS-C change scores added 8% variance explained ($p < .01$). The SCARED change score coefficient was not significant in the final model, however the PCS-C change score was significant (Table 9).

Taken together, changes in the SCARED and PCS-C from baseline to one month predicted pain intensity at one month, and changes in the PCS-C from baseline to three months predicted pain intensity at three months. Each accounted for between 13% and 19% unique variance (Table 7 and 9). Changes in the SCARED and PCS-C from one month to three months were not predictive of the functioning variable at three months.

Predicting CALQ at one month follow-up. The model with demographic variables, pain variables at baseline, CALQ at baseline, and change scores for both the SCARED and PCS-C from baseline to one month follow-up was significant (R = .79, $p <$

.001). Adding in the SCARED and PCS-C change scores added 10% ($p < .001$) variance. The SCARED change score coefficient was not significant while the PCS-C change score coefficient was significant (Table 10).

Predicting CALQ at three month follow-up. The model with demographic variables, pain variables at one month, CALQ at one month, and both SCARED and PCS-C change scores from one month to three month follow-up was significant ($R = .91$, $p < .001$). Adding in the SCARED and PCS-C change scores added < .01% of variance and neither change score coefficient was significant (Table 11).

Exploratory analysis: Predicting CALQ at three month follow-up from baseline. The model with demographic variables, pain variables at baseline, CALQ at baseline, and both SCARED and PCS-C change scores from baseline to three month follow-up was significant ($R = .72$, $p < .001$). Adding in the SCARED and PCS-C change scores added 14% explained variance and both SCARED and PCS-C change score coefficients were significant (Table 12).

Taken together, changes in the PCS-C from baseline to one month predicted the CALQ at one month, and changes in the SCARED and PCS-C from baseline to three months predicted the CALQ at three months. Each accounted for between 17% and 24% unique variance (Table 10 and 12). Changes in the SCARED and PCS-C from one month to three month were not predictive of the functioning variable at three months.

Predicting PedsQL at one month follow-up. The model with demographic variables, pain variables at baseline, PedsQL at baseline, and both SCARED and PCS-C change scores from baseline to one month follow-up was significant ($R = .84$, $p < .001$). Adding in the SCARED and PCS-C change scores added 18% explained variance ($p <$

.001), and both the SCARED change score coefficient and the PCS-C change score coefficient were significant in the final model (Table 13).

Predicting PedsQL at three month follow-up. The model with demographic variables, pain variables at one month, PedsQL at one month, and both SCARED and PCS-C change scores from one month to three month follow-up was significant ($R = .88$, $p < .001$). Adding in the SCARED and PCS-C at baseline added 9% explained variance ($p < .001$), and both the SCARED change score coefficient and the PCS-C change score coefficient were significant in the final model (Table 14).

Exploratory analysis: Predicting PedsQL at three month follow-up from baseline. The model with demographic variables, pain variables at baseline, PedsQL at baseline, and both SCARED and PCS-C change scores from baseline to three month follow-up was significant ($R = .82$, $p < .001$). Adding in the SCARED and PCS-C change score from baseline to three month follow-up added 22% explained variance ($p < .001$), and both the SCARED change score coefficient and the PCS-C change score coefficient were significant in the final model (Table 15).

Taken together, changes in the SCARED and PCS-C from baseline to one month predicted the PedsQL at one month; changes in the SCARED and PCS-C from one month to three months predicted the PedsQL at three months; and changes in the SCARED and PCS-C from baseline to three month predicted the PedsQL at three months. Each accounted for between 13% and 29% unique variance, with the semi-partial correlation for the PCS-C consistently higher than the semi-partial correlation for the SCARED (Table 13, 14, and 15).

Exploratory Analyses: Predicting Functioning Variables at One Month from Anxiety and Pain Catastrophizing from Baseline, and Predicting Functioning Variables at Three Months from Anxiety and Pain Catastrophizing from One Month

Hierarchical multiple regressions were conducted to test the final component of the research plan, that *anxiety and pain catastrophizing at baseline (rather than change scores) predict functioning at the one month follow-up, and that anxiety and pain catastrophizing at one month predict functioning at three month follow-up*. Results are presented in the Appendix and Tables A1 through A9. In summary, the SCARED and PCS-C did not predict later pain intensity. The SCARED predicted later scores on the CALQ at two time points, but the PCS-C did not. The SCARED predicted a later score on the PedsQL at one time point, but the PCS did not.

Discussion

The results of this study are evidence for relationships between the independent variables anxiety and pain catastrophizing and the dependent variables functional disability and health-related quality of life (HRQOL) at baseline and over time. Higher anxiety and pain catastrophizing were related to 1) higher pain intensity, 2) higher functional disability, and 3) lower HRQOL, after controlling for demographic characteristics and, in the case of functional disability and HRQOL, for pain characteristics. Pain decreased after intake; pain catastrophizing and functional disability decreased over three months; and HRQOL increased over three months. Anxiety did not change significantly over time. Changes in anxiety and pain catastrophizing predicted later pain, functional disability, and HRQOL. Anxiety predicted later functional disability and HRQOL. These results are reviewed in detail in the context of the individual hypotheses below.

Results of Hypothesis 1

The findings supported the first hypothesis: **higher anxiety and pain catastrophizing were related to higher pain intensity and functional disability, and lower HRQOL at baseline**. Findings were consistent with previous research in which anxiety was related to presence of chronic pain (Campo et al., 2004; Kashikar-Zuck et al., 2008), functional disability (Cohen et al., 2010; Wendland et al., 2010) and HRQOL (Tsao et al., 2007) and pain catastrophizing was related to pain severity (Crombez et al., 2003; Vervoort et al., 2006), functional disability (Vervoort et al., 2010), and HRQOL (Libby & Glenwick, 2010). Notably there was a significant correlation between higher levels of anxiety and more intense pain. This advances research in pediatric chronic pain

a step further – past research cited that youth with chronic pain have increased symptoms of anxiety, but little has been said about a relationship between symptoms. Pain frequency and duration were also related to functional disability and HRQOL. Pain catastrophizing was more strongly correlated with pain and functional disability, while anxiety and pain catastrophizing had equally strong correlations with HRQOL. However, these analyses were based on cross-sectional data, and shared variance had not yet been accounted for.

Results of Hypothesis 2

The findings supported the second hypothesis: **higher anxiety and pain catastrophizing were related to higher pain intensity and functional disability, and lower HRQOL** after controlling for demographic characteristics and in the case of functional disability and HRQOL, for pain characteristics. Findings were consistent with previous research in which anxiety contributed unique variance to predicting functional disability and HRQOL (Cohen et al., 2010; Wendland et al., 2010); and PC contributed unique variance to predicting pain (Vervoort et al., 2006) and functional disability (Vervoort et al., 2010) above and beyond pain and demographic characteristics.

Results of Hypothesis 3

The next step in analysis (hypothesis 3) was to determine if anxiety and pain catastrophizing contributed unique variability in predicting pain scores, functional disability, and HRQOL. The findings partially supported the third hypothesis. **Pain catastrophizing added a significant amount of unique variance to models predicting pain, functional disability, and HRQOL. Anxiety added a significant amount of**

unique variance to models predicting functional disability and HRQOL, but not pain.

In the model predicting pain at baseline, pain catastrophizing was a significant predictor in the final model, while anxiety was not. In separate models, both anxiety and pain catastrophizing predicted pain; however, in the model with both predictors, anxiety was no longer a significant predictor, while pain catastrophizing accounted for a significant portion of unique variance in pain intensity. It appears that the shared variance between pain catastrophizing and anxiety accounted for more of the relationship between anxiety and pain than the unique contribution of anxiety. This is consistent with past research in which pain catastrophizing predicted unique variance in pain intensity over anxiety (Sullivan et al., 1995); however, these findings are different from findings in youth with pain in which anxiety sensitivity was more predictive of presence of pain than pain catastrophizing (Tsao et al., 2009). The findings from the current study and those from Tsao and colleagues (2009) cannot be directly compared; first, Tsao and colleagues were predicting presence of current pain complaint, while the outcome in this study was pain intensity. Secondly, the independent variables, anxiety and anxiety sensitivity, are related, but distinguishable constructs (Reiss, Peterson, Gursky, & McNally, 1986). Pain catastrophizing is more specific to pain-related negative affectivity regarding pain than overall anxiety, which is likely why it contributed more unique variance to the model.

In the models predicting functional disability and HRQOL at baseline, anxiety and pain catastrophizing were both significant predictors in separate models, and in a model together. In the model together predicting functional disability, however, pain catastrophizing accounted for more variance in functional disability than anxiety after

accounting for shared variance. Further, in the model together predicting HRQOL, anxiety accounted for more variance in HRQOL than pain catastrophizing after accounting for shared variance. Findings were similar to those in adults in which pain catastrophizing uniquely predicted functional disability (Holroyd et al., 2007); however, findings were not consistent with other studies in which pain catastrophizing uniquely predicted quality of life over anxiety (Holroyd et al, 2007) and pain catastrophizing no longer predicted disability once the variance accounted for by anxiety was examined (Moix et al., 2011). The findings of the current study extend current research limited to adult populations by addressing application of these models in pediatric chronic pain. Both anxiety and pain catastrophizing remained significant predictors in the final models predicting functional disability and HRQOL at baseline. Notably, pain catastrophizing accounted for more unique variance in predicting functional disability while anxiety accounted for more unique variance in predicting HRQOL.

It is clear from examining cross-sectional data that both pain catastrophizing and anxiety are important to functional outcomes, and it is important to consider both factors. The next step in analysis was to examine the nature of these relationships over time.

Results of Hypothesis 4

Results partially supported the fourth hypothesis, which examined changes in anxiety, pain catastrophizing, pain, functional disability, and HRQOL over time. **Pain catastrophizing and functional disability significantly decreased, and HRQOL significantly increased at each time point. Pain significantly decreased between baseline and one-month, and baseline and three-month follow-up. Anxiety did not change significantly over time.** Therefore, it is possible to see change in PC, functional

disability, HRQOL, and pain over a short period of time. Further, this allowed for examining variables associated with change and effects of change on functional variables. Clinically, it is promising to see positive movement in these areas shortly after presenting for treatment.

Anxiety did not change significantly over time. Although some research teams have found that anxiety symptoms in community youth may change over time (Wittchen, Lieb, Pfister, & Schuster, 2000), other teams found that rates of anxiety in youth are not significantly different over six months (Beesdo et al., 2009b). The findings of the current study are in line with those of the latter, however, there were a variety of responses to anxious symptoms over time. Although there were no group differences in anxiety over time, there were individual changes in both directions. Furthermore, youth were not getting treatment targeting general anxiety, so we would not expect anxiety to decrease overall. No conclusions can be made here about how these results might look different if treatment explicitly included treatment of anxiety symptoms, or how well the SCARED can capture potential change based on the results of this study.

Results of Hypothesis 5 and Exploratory Analyses

The fifth hypothesis, which proposed to examine the predictive ability of changes in anxiety and pain catastrophizing for later treatment outcomes, was partially supported. **Changes in anxiety and pain catastrophizing from baseline to one-month, and in pain catastrophizing from baseline to three-months predicted later pain intensity. In predicting functional disability, changes in pain catastrophizing from baseline to one-month, and changes in anxiety and pain catastrophizing from baseline to three-months predicted later functional disability. In predicting HRQOL, changes in both**

anxiety and pain catastrophizing from baseline to one-month, from one-month to three-months, and from baseline to three-month predicted later HRQOL. See Figures 1 through 3 for models that synthesize these results regarding the relationships between anxiety and pain catastrophizing, and pain, functional disability, and HRQOL over time. These models give us an example of how these relationships will be able to be explored in terms of mediators and moderators as the statistical power of the database is increased. Exploratory analyses examined the predictive ability of static anxiety and pain catastrophizing (in contrast to change scores) for later functioning. Neither anxiety nor pain catastrophizing predicted later pain intensity. Anxiety predicted functional disability and HRQOL in some analyses, and pain catastrophizing did not.

Anxiety and Pain Catastrophizing Predicting Pain

As discussed above, pain catastrophizing was more predictive of pain at baseline than anxiety after taking shared variance into account. The shared variance between pain catastrophizing and anxiety accounted for more of the relationship between anxiety and pain than the unique contribution of anxiety at baseline, and changes in pain catastrophizing predicted later pain intensity in more analyses than changes in anxiety. Both anxiety and pain catastrophizing are related to pain, however pain catastrophizing is a more specific aspect of negative affectivity regarding pain than overall anxiety. Catastrophizing and anxiety are both related to negative affectivity (MacDonald et al., 2008; Noël et al., 2012; Vervoort et al., 2006; Vlaeyen & Linton, 2000), in fact catastrophizing may be a shared factor between anxiety and depression; catastrophizing is often observed in anxiety, and rumination is a depressogenic cognitive style (Noël et al., 2012). Pain catastrophizing involves negative expectations about the painful experience,

and exaggerated worry about the consequences of pain (Sullivan et al., 1995); it is a very specific aspect of negative affectivity pertaining to pain. Neuroticism lowers the threshold at which pain is perceived as threatening (pain catastrophizing; Goubert, Crombez, & Van Damme, 2004). In other words, overall negative affectivity is related or predisposes one to pain catastrophizing, which is more closely related to more intense pain than overall negative affectivity, including general anxiety. Therefore, while the negative orientation shared by anxiety and pain catastrophizing was observed in significant relationships between both independent variables and pain, after accounting for shared variance, pain catastrophizing explained more unique variance in models predicting pain intensity compared to anxiety.

Neither anxiety nor pain catastrophizing predicted later pain intensity; however, changes in anxiety and pain catastrophizing predicted later pain intensity. Therefore, changes in anxiety and pain catastrophizing are more predictive of later pain intensity than the level of anxiety or pain catastrophizing. Reducing one's anxiety or catastrophizing regarding pain predicts lower pain intensity, whereas individuals with consistently high pain catastrophizing experience higher pain intensity. One explanation may be that there is a correlation between change scores of pain and change scores of anxiety and pain catastrophizing. Therefore, decreases in pain catastrophizing and anxiety are related to lower pain intensity. Pain reports do not always change significantly, especially in the first few months of treatment. Shifts in rating pain intensity may demonstrate flexible thinking regarding pain; youth are able to identify gradations in their pain rather than all or nothing thinking regarding pain. These change variables may in fact measure openness to change and flexible thinking patterns as much

as they assess true change in anxiety and pain catastrophizing. If youth are able to shift the way they think about pain and the effect on their functioning, they may be more likely to experience decreased pain intensity.

Anxiety Predicting Functional Disability and HRQOL

Anxiety at baseline and changes in anxiety over three months predicted later functional disability and HRQOL. Functional disability and HRQOL are related constructs that are important treatment outcomes for chronic pain. Functional disability is limited ability to participate in daily activities such as at home, school, and social situations. While functional disability contributes to one's HRQOL, HRQOL is a broader multidimensional construct including perceptions of how one's health influences physical, occupational, psychological, emotional, and social functioning (Long et al., 2008; Spilker, 1996). Lower anxiety and decreases in anxiety over three months predicted lower functional disability and higher HRQOL.

Symptoms of anxiety include thoughts, feelings, and somatic symptoms that may affect functional disability and HRQOL. It is difficult to participate in many activities with high anxiety, and with somatic symptoms, including pain, which make regular functioning uncomfortable. Worry and fear of pain increase the probability that an individual will avoid daily activities. Additionally, one aspect of HRQOL is emotional functioning; therefore if a person is experiencing high anxiety, they are likely experiencing difficulties in emotional functioning resulting in lower overall quality of life.

Notably, decreases in anxiety significantly predicted lower functional disability and higher HRQOL despite group means of anxiety not changing significantly over the

course of this study. General anxiety was not a systematic target of treatment and there was not the same degree of change over time in anxiety that there was in other study variables. Other populations may experience a greater change in anxiety that would be even more predictive of functional outcomes than found in this study.

Pain Catastrophizing Predicting Functional Disability and HRQOL

Pain catastrophizing at baseline and changes in pain catastrophizing over three months predicted later functional disability and HRQOL. Consistent with the fear-avoidance model of chronic pain (Vlaeyen & Linton, 2000), catastrophizing may interfere with an individual's ability to participate in daily activities. Pain catastrophizing is a cognitive pattern regarding perceived negative consequences of pain that interferes with an individual's ability to face pain and function in daily life. The underlying negative affectivity and resulting functional disability of pain catastrophizing likely contributes to poor HRQOL. The processes through which anxiety and pain catastrophizing are related to functional disability and HRQOL have yet to be determined, however the results of this study found that each component is important, and that there are unique relationships between variables.

Anxiety and pain catastrophizing both contribute unique variance to models predicting pain, functional disability, and HRQOL. This is an important contribution to the study of pediatric chronic pain; no past research has examined the unique contributions of anxiety and pain catastrophizing to pain and functional outcomes over time in youth with chronic pain. In contrast to past research examining pain catastrophizing and anxiety in adults with chronic pain (Holroyd et al., 2007; Moix et al., 2011; Sullivan et al., 1995) and research examining pain catastrophizing and anxiety

sensitivity in youth with chronic pain (Tsao et al., 2009), in the majority of models, *both* pain catastrophizing and anxiety contributed unique variance to predicting current and later pain, functional disability, and HRQOL.

Clinical Implications

The results of this study support the inclusion of CBT targeting anxiety and pain catastrophizing in the treatment of pediatric chronic pain. This is consistent with the current recommendations for treatment of chronic pain (American Pain Society, 2012; Beesdo et al., 2009a), which involve treatment from a multidisciplinary approach. The findings of this study support treatment in a multidisciplinary setting where anxiety and pain catastrophizing are emphasized as targets of treatment as much as reducing pain symptoms is addressed. Pain-specific CBT can target anxiety and pain catastrophizing with relaxation training and challenging pain-related thoughts (Kashikar-Zuck, 2006). Using CBT in conjunction with medical treatment is effective in treating pediatric chronic pain (Eccleston et al., 2002) likely because it decreases anxiety and pain catastrophizing, which was found to be related to decreased pain, better functioning and HRQOL in this study.

Recently, there has been a call for routine screening for anxiety in youth with specific types of chronic pain (Cunningham, Lynch-Jordan, Mezoff, Farrell, Cohen, & Kashikar-Zuck, 2013). Results of this study support the recommendation for screening for anxiety in all youth with chronic pain as well as screening for pain catastrophizing. Both constructs predicted unique variance in important functional outcomes and likely affect treatment. Further, anxiety has received attention as a potential moderator between pain and treatment outcomes (Cohen et al., 2010; Wendland et al., 2010). Given the

finding that anxiety plays an independent role in predicting treatment outcomes, it may also be beneficial to target general anxiety, not only anxiety related to pain, in treatment (Moix et al., 2011).

Limitations

Although 725 youth were eligible for the study at their intake appointment, only 92 completed the requested measures at all three time points. Despite the smaller sample not being statistically different from the larger sample in terms of the study variables at baseline, results of the study must be interpreted with caution. It is impossible to know the functional status of youth who did not complete follow-up measures. While longitudinal studies nearly always suffer from decreased participation over time, it would be beneficial to gather more information about why participants might drop out (e.g., changing medical providers, dissatisfaction with treatment, spontaneous recovery). We also do not have direct information about each family's on-going relationship with the pain clinic, which does not classify cases as "drop-outs" or other labels that may seem pejorative, recognizing that many patients return for treatment after seeming not to have engaged. Furthermore, although we know that the smaller sample did not differ from the larger sample in terms of pain or other study variables, it is possible that the strength of the relationships (e.g., correlations) between these variables would be shown to be different if data were available from the entire sample.

Although longitudinal studies are better able to address questions of causality than cross-sectional studies, more research needs to be done before drawing conclusions about causality between anxiety, pain catastrophizing, and pain. It is not known how much anxiety and pain catastrophizing youth had prior to the beginning of their pain problems.

Prospective studies using large, representative samples are needed to make more solid conclusions regarding risk factors for development of chronic pain.

Additionally, the level of treatment that each participant received from the medical team, mental health team, and from other outside providers was not considered as a variable in this study. Some families are able to and choose to be followed closely by the pain team and are seen every week, some families come into an intake appointment for consultation and are followed elsewhere. Specifically, the extent to which PC and anxiety were targeted in treatment is unknown. This was not a treatment study, and psychological treatments conducted in the pain clinic or at an outside clinic were not evaluated for specifically targeting anxiety or pain catastrophizing. The results of this study should be considered as naturalistic observation of relationships between variables over time among youth seeking treatment rather than as a treatment outcome study. Finally, the sample in this study included a heterogeneous sample of pain problems. Future studies might benefit from consideration of different types of pain problems and medical history variables as potential moderators.

Future Directions

Considering that this was the first examination of anxiety and pain catastrophizing predicting functional outcomes over time in pediatric chronic pain, there are many directions for future research in this area. Although the families in this sample sought treatment for chronic pain, treatment variables were not included in this study. Closer examination of treatment variables such as modalities of treatment used, target of psychological treatment, and medication trials could offer useful information for building future models of trajectory of treatment and outcomes. It is important for treatment

research to know if targeting anxiety and pain catastrophizing decreases anxiety and pain catastrophizing, and if symptom reduction has the same desired effects on functional disability and HRQOL found in this study. Further, randomized controlled treatment trials targeting general anxiety, pain related anxiety, and catastrophic thinking regarding pain are needed to differentiate effects based on treatments received.

Future studies would also benefit from consideration of more sophisticated statistical analyses allowing for analyses of potential mediator and moderator relationships. Specifically, samples with more power, particularly longitudinal samples, might benefit from structural equation modeling to consider relationships between variables. Type of pain problem and medical history, as well as other demographic variables, may be moderators of some relationships under study. Females consistently scored higher than males on anxiety, pain catastrophizing, pain, and functional disability, and lower on HRQOL. Although gender was controlled for in this study, future research may consider other models accounting for differences that female and male youth experience chronic pain and related psychosocial functioning. Additionally, future studies should address the research questions from this study incorporating a developmental perspective. Children and adolescents ages 8 to 18 were included in this study, and a cognitive variable such as pain catastrophizing changes over the course of childhood and adolescence (Noël et al., 2012), therefore it will be important to consider developmental level as a factor in building future models.

Conclusions

Anxiety and pain catastrophizing both contribute unique variance to models predicting pain, functional disability, and HRQOL at baseline and over time. This is an

important contribution to the study of pediatric chronic pain; no past research has examined the unique contributions of anxiety and pain catastrophizing to pain and functional outcomes over time in youth with chronic pain.

Pain catastrophizing and anxiety share an underlying factor of negative affectivity which is related to pain intensity; however, pain catastrophizing is more specific to pain, and explained unique variance in pain intensity beyond anxiety. Results supported the fear-avoidance model of chronic pain (Vlaeyen & Linton, 2000). Pain catastrophizing and anxiety interfere with daily functioning and well-being, resulting in functional disability and poor HRQOL. Specifically, it was found that decreases in anxiety and pain catastrophizing over three months uniquely predicted lower functional disability and higher HRQOL.

Reductions in anxiety and pain catastrophizing over three months are associated with favorable outcomes. The results of this study support including CBT targeting anxiety and pain catastrophizing in treatment of pediatric chronic pain. Overall anxiety, not just pain specific anxiety, was related to functional outcomes assessed in treatment. Therefore, it may be beneficial to target the full breadth of anxiety in treatment, rather than limiting treatment to pain-related anxiety. Furthermore, this study supports recent calls for routine screening for anxiety in youth, and also suggests that screening for pain catastrophizing would be beneficial as well.

Future research should expand upon this initial examination of the relationships between anxiety, pain catastrophizing, pain, functional disability, and HRQOL. Longitudinal treatment studies are needed to study and differentiate effects based on treatment received. Further, it will be important to apply a developmental framework to

future models and consider mediators of these relationships, and moderators of treatment success. There is still model building to be done, but it is clear that both anxiety and pain catastrophizing have unique predictive ability regarding important functional outcomes.

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Table 1

Table of Data Analyses

Hypothesis	Statistical Analyses	*n*	Outcome Variables	Predictors
1	Pearson correlations	725		T0 SCARED T0 PCS-C T0 Pain intensity T0 CALQ T0 PedsQL
2	Multiple Regression	725	T0 Pain intensity	T0 demographic variables T0 SCARED
	Multiple Regression		T0 CALQ	T0 demographic variables T0 pain variables T0 SCARED
	Multiple Regression		T0 PedsQL	T0 demographic variables T0 pain variables T0 SCARED
	Multiple Regression		T0 Pain intensity	T0 demographic variables T0 PCS-C
	Multiple Regression		T0 CALQ	T0 demographic variables T0 pain variables T0 PCS-C
	Multiple Regression		T0 PedsQL	T0 demographic variables T0 pain variables

				T0 PCS-C
3	Multiple Regression	725	T0 Pain intensity	T0 demographic variables T0 SCARED T0 PCS-C
	Multiple Regression		T0 CALQ	T0 demographic variables T0 pain variables T0 SCARED T0 PCS-C
	Multiple Regression		T0 PedsQL	T0 demographic variables T0 pain variables T0 SCARED T0 PCS-C
	Multiple Regression		T0 Pain intensity	T0 demographic variables T0 PCS-C T0 SCARED
	Multiple Regression		T0 CALQ	T0 demographic variables T0 pain variables T0 PCS-C T0 SCARED
	Multiple Regression		T0 PedsQL	T0 demographic variables T0 pain variables T0 PCS-C T0 SCARED

4	Repeated measures T-test	171	1 mo. SCARED	T0 SCARED
	Repeated measures T-test		1 mo. PCS-C	T0 PCS-C
	Repeated measures T-test		1 mo. pain intensity	T0 pain intensity
	Repeated measures T-test		1 mo. CALQ	T0 CALQ
	Repeated measures T-test		1 mo. PedsQL	T0 PedsQL
	Repeated measures T-test	92	3 mo. SCARED	1 mo. SCARED
	Repeated measures T-test		3 mo. PCS-C	1 mo. PCS-C
	Repeated measures T-test		3 mo. pain intensity	1 mo. pain intensity
	Repeated measures T-test		3 mo. CALQ	1 mo. CALQ
	Repeated measures T-test		3 mo. PedsQL	1 mo. PedsQL
5	Multiple regression	171	1 mo. Pain intensity	T0 demographic variables
				T0 Pain intensity
				Δ 1 mo. -T0 SCARED
				Δ 1 mo. -T0 PCS-C
	Multiple Regression		1 mo. CALQ	T0 demographic variables
				T0 pain variables
				T0 CALQ
				Δ 1 mo. -T0 SCARED
				Δ 1 mo. -T0 PCS-C
	Multiple Regression		1 mo. PedsQL	T0 demographic variables
				T0 pain variables
				T0 PedsQL
				Δ 1 mo. -T0 SCARED

	Multiple Regression	92	3 mo. Pain intensity	Δ 1 mo. -T0 PCS-C total T0 demographic variables 1 mo. Pain intensity Δ 3 mo. -1 mo. SCARED Δ 3 mo. -1 mo. PCS-C
	Multiple Regression		3 mo. CALQ-C total	T0 demographic variables 1 mo. pain variables 1 mo. CALQ Δ 3 mo. -1 mo. SCARED Δ 3 mo. -1 mo. PCS-C
	Multiple Regression		3 mo. PedsQL total	T0 demographic variables 1 mo. pain variables 1 mo. PedsQL Δ 3 mo. -1 mo. SCARED Δ 3 mo. -1 mo. PCS-C
Exploratory	Multiple Regression	171	1 mo. Pain intensity	T0 demographic variables T0 Pain intensity T0 SCARED total T0 PCS-C total
	Multiple Regression		1 mo. CALQ-C total	T0 demographic variables T0 pain variables T0 CALQ-C total T0 SCARED total

				T0 PCS-C total
	Multiple Regression		1 mo. PedsQL total	T0 demographic variables
				T0 pain variables
				T0 PedsQL total
				T0 SCARED total
				T0 PCS-C total
	Multiple Regression	92	3 mo. Pain intensity	T0 demographic variables
				1 mo. Pain intensity
				1 mo. SCARED total
				1 mo. PCS-C total
	Multiple Regression		3 mo. CALQ-C total	T0 demographic variables
				1 mo. pain variables
				1 mo. CALQ-C total
				1 mo. SCARED total
				1 mo. PCS-C total
	Multiple Regression		3 mo. PedsQL total	T0 demographic variables
				1 mo. pain variables
				1 mo. PedsQL total
				1 mo. SCARED total
				1 mo. PCS-C total

Note. T0 denotes baseline measure.

Table 2

Correlations between Pain Intensity and Frequency, and Anxiety, Pain Catastrophizing, Functional Disability, and HRQOL

	Usual Pain	Worst Pain	Pain Frequency
	r (*n*)	*r* (*n*)	*r* (*n*)
SCARED Baseline	.17***	.11**	.04
	(692)	(696)	(692)
SCARED 1 month	.13	.12	.02
	(163)	(164)	(163)
SCARED 3 month	.11	.13	-.01
	(109)	(110)	(108)
PCS-C Baseline	.38***	.36***	.18***
	(687)	(691)	(687)
PCS-C 1 month	.31***	.21**	.03
	(163)	(164)	(163)
PCS-C 3 month	.28**	.26**	.03
	(108)	(109)	(107)
CALQ Baseline	.41***	.47***	.40***
	(693)	(697)	(691)
CALQ 1 month	.31***	.28**	.34***
	(163)	(164)	(163)
CALQ 3 month	.25**	.22*	.27**
	(109)	(110)	(108)

PedsQL Baseline	-.37***	-.42***	-.36***
	(696)	(699)	(694)
PedsQL 1 month	-.23**	-.19*	-.24
	(165)	(166)	(165)
PedsQL 3 month	-.24*	-.18	-.21
	(109)	(110)	(108)

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

Table 3

Predicting Pain Intensity with the SCARED, PCS-C, and Both at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.02**
Gender	0.54	0.17	.12**	.12	
STEP TWO					.04***
Gender	0.44	0.17	.10**	.10	
SCARED	0.02	0.01	.16***	.15	
STEP ONE					.02***
Gender	0.61	0.17	.14***	.14	
STEP TWO					.15***
Gender	0.43	0.16	.10**	.10	
PCS-C	0.06	0.01	.37***	.37	
STEP THREE COMBINED MODEL					.15***
Gender	0.44	0.16	.10**	.10	
SCARED	-0.01	0.01	-.04	-.03	
PCS-C	0.06	0.01	.39***	.33	

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

Table 4

Predicting CALQ with the SCARED, PCS-C, and Both at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.30***
Gender	0.30	1.71	.01	.01	
Age	0.57	0.32	.06	.06	
Pain duration	-0.06	0.03	-.08*	-.08	
Pain frequency	1.33	0.22	.24***	.20	
Usual pain	2.27	0.50	.21***	.16	
Worst pain	2.45	0.60	.21***	.14	
STEP TWO					.34***
Gender	-1.14	1.67	-.02	-.02	
Age	0.72	0.31	.08*	.08	
Pain duration	-0.06	0.03	-.07*	-.07	
Pain frequency	1.39	0.22	.25***	.21	
Usual pain	1.89	0.49	.17***	.13	
Worst pain	2.41	0.58	.20***	.14	
SCARED	0.34	0.06	.21***	.21	
Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.29***
Gender	0.51	1.72	.01	.01	

Age	0.53	0.32	.06	.06	
Pain duration	-0.06	0.03	-.08*	-.08	
Pain frequency	1.33	0.22	.24***	.20	
Usual pain	2.24	0.50	.20***	.15	
Worst pain	2.46	0.60	.21***	.14	
STEP TWO					.37***
Gender	0.14	1.62	.003	.003	
Age	0.70	0.30	.08*	.07	
Pain duration	-0.05	0.03	-.07*	-.06	
Pain frequency	1.33	0.21	.24***	.20	
Usual pain	1.38	0.48	.13***	.09	
Worst pain	1.80	0.57	.15***	.10	
PCS-C	0.55	0.06	.31***	.29	
Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP THREE COMBINED MODEL					.38***
Gender	-0.43	1.63	-.01	-.01	
Age	0.75	0.30	.08*	.08	
Pain duration	-0.05	0.03	-.07*	-.06	
Pain frequency	1.35	0.21	.24***	.21	
Usual pain	1.38	0.48	.13**	.09	
Worst pain	1.85	0.57	.16**	.11	
SCARED	0.15	0.06	.09*	.08	

PCS-C	0.46	0.07	.26***	.21

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

Table 5

Predicting PedsQL with the SCARED, PCS-C, and Both at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.22***
Gender	0.26	1.39	.01	.01	
Age	-0.18	0.26	-.03	-.03	
Pain duration	0.03	0.02	.05	.05	
Pain frequency	-0.83	0.18	-.19***	-.17	
Usual pain	-1.46	0.41	-.17***	-.13	
Worst pain	-1.88	0.48	-.21***	-.14	
STEP TWO					.42***
Gender	2.77	1.22	.07*	.07	
Age	-0.43	0.23	-.06	-.06	
Pain duration	0.03	0.02	.05	.05	
Pain frequency	-0.95	0.16	-.22***	-.19	
Usual pain	-0.84	0.36	-.10*	-.07	
Worst pain	-1.79	0.42	-.20***	-.13	
SCARED	-0.57	0.04	-.45***	-.44	

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.22***
Gender	0.43	1.40	.01	.01	

Age	-0.17	0.26	-.02	-.02	
Pain duration	0.04	0.02	.06	.06	
Pain frequency	-0.83	0.18	-.19***	-.16	
Usual pain	-1.43	0.41	-.17**	-.13	
Worst pain	-1.92	0.49	-.21***	-.14	
STEP TWO					.34***
Gender	0.85	1.29	.02	.02	
Age	-0.34	0.24	-.05	-.05	
Pain duration	0.03	0.02	.04	.04	
Pain frequency	-0.83	0.17	-.19***	-.16	
Usual pain	-0.63	0.39	-.07	-.05	
Worst pain	-1.31	0.45	-.14**	-.10	
PCS-C	-0.52	0.05	-.38***	-.35	

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP THREE COMBINED MODEL					.44***
Gender	2.50	1.21	.07*	.06	
Age	-0.47	0.23	-.07*	-.06	
Pain duration	0.03	0.02	.04	.04	
Pain frequency	-0.93	0.16	-.21***	-.18	
Usual pain	-0.56	0.36	-.07	-.05	
Worst pain	-1.53	0.42	-.17***	-.11	
SCARED	-0.47	0.05	-.37***	-.31	

PCS-C	-0.25	0.05	-.18***	-.14

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

Table 6

Differences in SCARED. PCS-C, Pain Intensity, CALQ, and PedsQL Scores over Time

	N	*M (SD) T_1*	*M (SD) T_2*	*t (df)*	*d*
SCARED Baseline-1 mo.	167	20.84 (13.84)	19.75 (16.10)	1.60 (166)	.07
SCARED 1 mo.-3 mo.	92	18.87 (15.05)	17.87 (16.02)	.903 (91)	.06
SCARED Baseline-1 mo.	110	18.60 (12.00)	16.78 (15.32)	1.59 (109)	.12
PCS-C Baseline-1 mo.	170	25.23 (12.31)	22.99 (13.92)	2.65 (169)**	.17
PCS-C 1 mo.-3 mo.	91	22.81 (14.26)	20.54 (14.44)	2.09 (90)*	.16
PCS-C Baseline-1 mo.	112	24.61 (12.51)	19.55 (14.38)	4.22 (111)***	.37
Pain Baseline-1 mo.	161	6.66 (1.86)	5.91 (2.16)	5.04 (160)***	.36
Pain 1 mo.-3 mo.	86	6.12 (2.62)	5.95 (2.33)	.926 (85)	.07
Pain Baseline-1 mo.	105	6.75 (1.85)	5.68 (2.32)	4.95 (104)***	.47
CALQ Baseline-1 mo.	170	42.58 (22.64)	33.76 (23.32)	6.41 (169)***	.38
CALQ 1 mo.-3 mo.	91	34.51 (23.61)	30.33 (24.70)	3.08 (90)**	.18
CALQ Baseline-1 mo.	113	41.27 (24.37)	29.39 (24.26)	5.70 (112)***	.50
PedsQL Baseline-1 mo.	173	58.21 (16.84)	62.26 (18.55)	-4.01 (172)***	.22
PedsQL 1 mo.-3 mo.	92	61.99 (17.84)	65.46 (19.24)	-2.67 (91)**	.18
PedsQL Baseline-1 mo.	113	59.87 (16.49)	66.43 (19.53)	-4.50 (112)***	.35

Note. *$p < .05$, **$p < .01$, ***$p < .001$. Cohen's d calculated using equation in Cumming, 2012.

Table 7

Predicting Pain Intensity at One Month Follow-Up with Change in SCARED and PCS-C between Baseline and One Month

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.34***
Gender	0.49	0.33	.10	.10	
Pain intensity$_{T0}$	0.65	0.08	.57***	.57	
STEP TWO					.44***
Gender	0.35	0.31	.07	.07	
Pain intensity$_{T0}$	0.65	0.07	.57***	.57	
ΔSCARED $_{T0\text{-}1\ mo.}$	-0.05	0.02	-.22**	-.19	
ΔPCS-C $_{T0\text{-}1\ mo.}$	-0.03	0.01	-.14*	-.13	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure. Negative coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to less pain.

Table 8

Predicting Pain Intensity at Three Month Follow-Up with Change in SCARED and PCS-C between One and Three Month Follow-up

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.58***
Gender	0.13	0.42	.02	.02	
Pain intensity$_{1\ mo.}$	0.78	0.07	.76***	.75	
STEP TWO					.60***
Gender	0.21	0.43	.04	.04	
Pain intensity$_{1\ mo.}$	0.79	0.08	.76***	75	
ΔSCARED $_{1\ mo.-3\ mo.}$	-0.02	0.02	-.10	-.10	
ΔPCS-C $_{1\ mo.-3\ mo.}$	-0.01	0.02	-.03	-.03	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. Negative coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to less pain.

Table 9

Predicting Pain Intensity at One Month Follow-Up with Change in SCARED and PCS-C between Baseline and Three Month Follow-Up

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.22***
Gender	0.42	0.50	.07	.07	
Pain intensity$_{T0}$	0.56	0.11	.44***	.44	
STEP TWO					.30***
Gender	0.38	0.49	.07	.07	
Pain intensity$_{T0}$	0.56	0.11	.45***	.44	
ΔSCARED $_{T0\text{-}3\ mo.}$	-0.03	0.02	-.16	-.15	
ΔPCS-C $_{T0\text{-}3\ mo.}$	-0.04	0.02	-.19*	-.18	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure. Negative coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to less pain.

Table 10

Predicting CALQ at One Month Follow-Up with Change in SCARED and PCS-C between Baseline and One Month

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.52***
Gender	2.32	3.57	.04	.04	
Age	1.37	0.65	.14*	.13	
Pain duration	0.09	0.06	.09	.09	
Pain frequency	0.82	0.47	.13	.11	
Usual pain	2.27	1.02	.19*	.14	
Worst pain	-2.01	1.12	-.16	-.11	
$CALQ_{To}$	0.68	0.07	.65***	.56	
STEP TWO					.62***
Gender	0.36	3.24	.01	.01	
Age	1.20	0.59	.12*	.11	
Pain duration	0.08	0.06	.09	.08	
Pain frequency	0.81	0.43	.13	.10	
Usual pain	1.64	0.94	.14	.10	
Worst pain	-1.26	1.04	-.10	-.07	
$CALQ_{T0}$	0.68	0.07	.65***	.56	
$\Delta SCARED_{T0\text{-}1\ mo.}$	-0.29	0.16	-.11	-.10	
$\Delta PCS\text{-}C_{T0\text{-}1\ mo.}$	-0.53	0.14	0.25***	-.22	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure. Negative coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to less functional disability.

Table 11

Predicting CALQ at Three Month Follow-Up with Change in SCARED and PCS-C between One and Three Month Follow-Up

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.83***
Gender	-2.92	3.21	-.05	-.05	
Age	-0.98	0.60	-.09	-.08	
Pain duration	0.01	0.06	.01	.01	
Pain frequency	-0.16	0.36	-.03	-.02	
Usual pain	-0.18	1.01	-.02*	-.01	
Worst pain	-0.19	1.08	-.02	-.01	
$CALQ_{1\ mo.}$	1.01	0.07	.94***	.78	
STEP TWO					.83***
Gender	-2.08	3.34	-.03	-.03	
Age	-1.01	0.62	-.09	-.08	
Pain duration	-0.01	0.06	-.01	-.01	
Pain frequency	-0.15	0.36	-.03	-.02	
Usual pain	-0.32	1.03	-.03	-.02	
Worst pain	-0.18	1.09	-.02	-.01	
$CALQ_{1\ mo.}$	1.01	0.07	.94***	.77	
$\Delta SCARED_{1\ mo.-3\ mo.}$	-0.04	0.12	-.02	-.02	
$\Delta PCS\text{-}C_{1\ mo.-3\ mo.}$	-0.14	0.15	-.05	-.05	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. Negative coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to less functional disability.

Table 12

Predicting CALQ at Three Month Follow-Up with Change in SCARED and PCS-C between Baseline and Three Month Follow-Up

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.37***
Gender	-4.28	5.50	-.07	-.07	
Age	1.18	0.92	.11	.11	
Pain duration	0.06	0.09	.06	.06	
Pain frequency	0.82	0.63	.14	.11	
Usual pain	2.30	1.54	.18	.13	
Worst pain	-1.47	1.68	-.11	-.08	
$CALQ_{T0}$	0.55	0.10	.55***	.46	
STEP TWO					.52***
Gender	-4.58	4.95	-.08	-.07	
Age	1.59	0.82	.15	.15	
Pain duration	0.05	0.09	.05	.05	
Pain frequency	0.89	0.57	.15	.12	
Usual pain	2.34	1.39	.18	.13	
Worst pain	-1.44	1.50	-.11	-.07	
$CALQ_{T0}$	0.53	0.09	.53***	.45	
$\Delta SCARED_{T0\text{-}3\ mo.}$	-0.54	0.18	-.27**	-.24	
$\Delta PCS\text{-}C_{T0\text{-}3\ mo.}$	-0.36	0.16	-.19*	-.17	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure. Negative coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to less functional disability.

Table 13

Predicting PedsQL at One Month Follow-Up with Change in SCARED and PCS-C

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.53***
Gender	-5.98	2.73	-.14*	-.13	
Age	-0.15	0.49	-.02	-.02	
Pain duration	-0.04	0.05	-.06	-.06	
Pain frequency	0.03	0.35	.01	.01	
Usual pain	-1.07	0.78	-.11	-.08	
Worst pain	0.17	0.84	.12	.08	
$PedsQL_{T0}$	0.79	0.07	.70***	.64	
STEP TWO					.71***
Gender	-3.90	2.18	-.09	-.09	
Age	0.14	0.39	.02	.02	
Pain duration	-0.04	0.04	-.05	-.05	
Pain frequency	0.04	0.28	.01	.01	
Usual pain	-0.42	0.62	-.04	-.03	
Worst pain	0.47	0.68	.05	.03	
$PedsQL_{T0}$	0.82	0.06	.73***	.66	
$\Delta SCARED_{T0\text{-}1\ mo.}$	0.31	0.11	.15**	.13	
$\Delta PCS\text{-}C_{T0\text{-}1\ mo.}$	0.56	0.09	.34***	.29	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure. Positive coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to higher HRQOL.

Table 14

Predicting PedsQL at Three Month Follow-Up with Change in SCARED and PCS-C between One and Three Month Follow-Up

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.68***
Gender	5.29	3.46	.11	.11	
Age	0.63	0.64	.07	.07	
Pain duration	-0.07	0.06	-.10	-.09	
Pain frequency	-0.16	0.36	-.04	-.03	
Usual pain	0.42	1.06	.05	.03	
Worst pain	-0.97	1.14	-.11	-.06	
$PedsQL_{1\ mo.}$	0.95	0.09	.81***	.73	
STEP TWO					.77***
Gender	2.30	3.08	.05	.04	
Age	0.64	0.57	.07	.07	
Pain duration	-0.06	0.05	-.08	-.07	
Pain frequency	-0.15	0.31	.03	-.03	
Usual pain	1.06	0.94	.12	.07	
Worst pain	-1.04	0.99	-.11	-.06	
$PedsQL_{1\ mo.}$	1.02	0.08	.86***	.77	
$\Delta SCARED_{1\ mo.-3\ mo.}$	0.25	0.11	.14*	.13	
$\Delta PCS\text{-}C_{1\ mo.-3\ mo.}$	0.54	0.14	.25***	.23	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. Positive coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to higher HRQOL.

Table 15

Predicting PedsQL at Three Month Follow-Up with Change in SCARED and PCS-C between Baseline and Three Month Follow-Up

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.45***
Gender	-0.74	4.19	-.02	-.01	
Age	0.33	0.71	.04	.04	
Pain duration	-0.12	0.07	-.14	-.13	
Pain frequency	-0.45	0.48	-.09	-.08	
Usual pain	-1.40	1.18	-.13	-.10	
Worst pain	1.48	1.30	.13	.09	
$PedsQL_{T0}$	0.78	0.11	.66***	.58	
STEP TWO					.67***
Gender	-1.06	3.32	-.02	-.02	
Age	0.03	0.56	.01	.01	
Pain duration	-0.11	0.06	-.13	-.12	
Pain frequency	-0.35	0.38	-.07	-.06	
Usual pain	-1.09	0.93	-.10	-.07	
Worst pain	1.52	1.02	.14	.09	
$PedsQL_{T0}$	0.88	0.09	.75***	.65	
$\Delta SCARED_{T0\text{-}3\ mo.}$	0.43	0.12	.25***	.23	
$\Delta PCS\text{-}C_{T0\text{-}3\ mo.}$	0.50	0.11	.32***	.29	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure. Positive coefficients for change score values indicate that larger decreases in anxiety and pain catastrophizing are related to higher HRQOL.

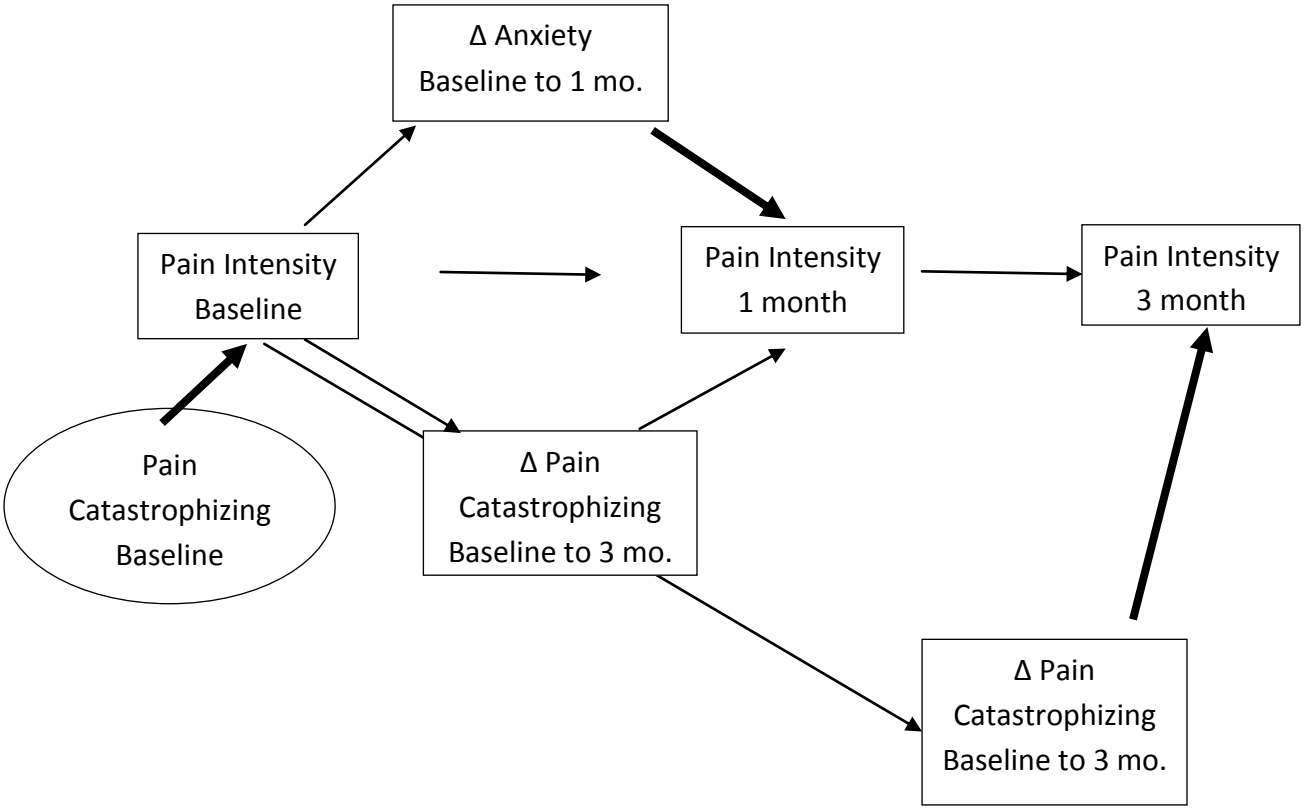

Figure 1. Predicting pain intensity over time. Model shows unique contributions of pain catastrophizing at baseline and changes in anxiety and pain catastrophizing predicting pain intensity over time. Bolded lines show strongest predictor of pain intensity from individual models.

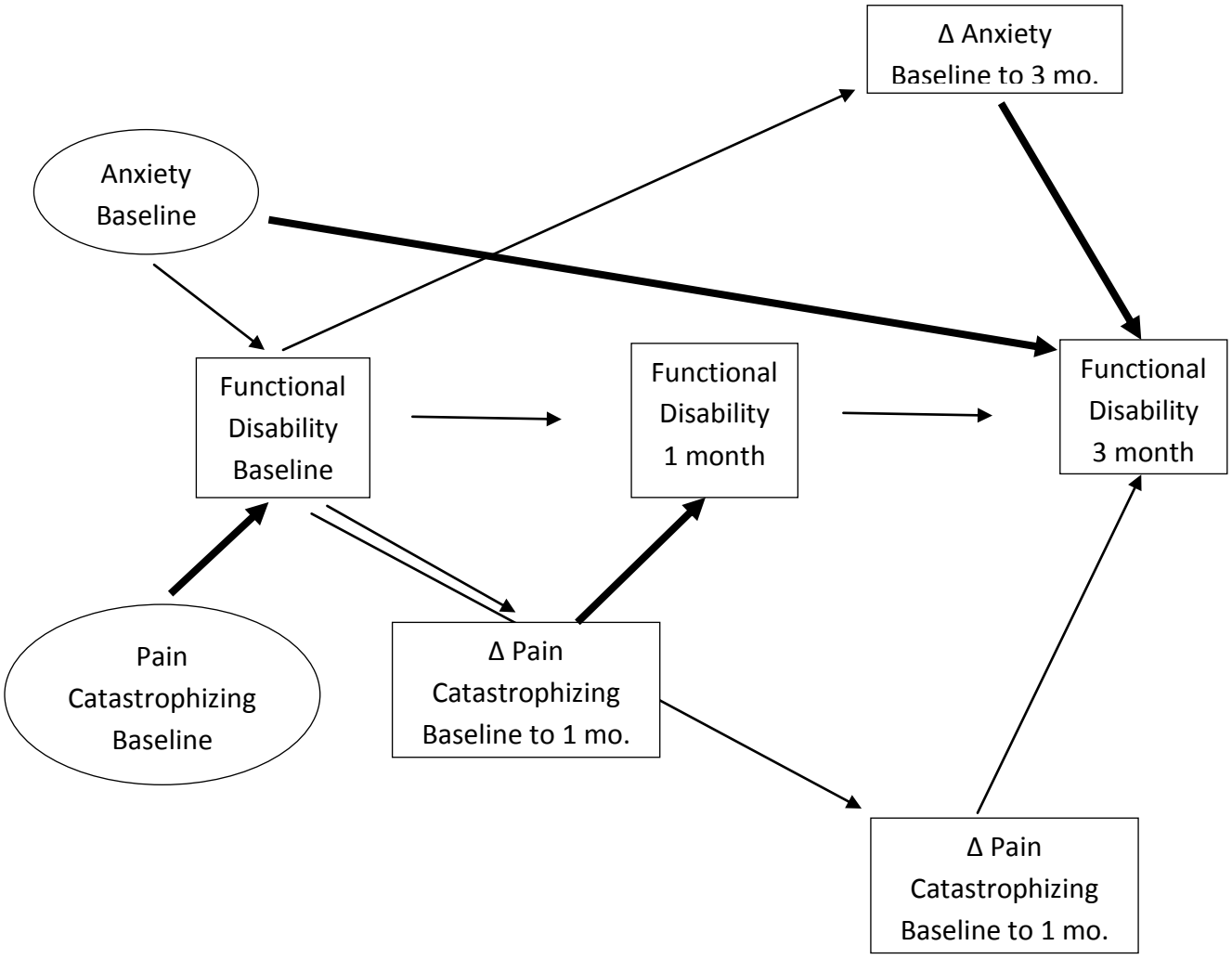

Figure 2. Predicting functional disability over time. Model shows unique contributions of anxiety and pain catastrophizing at baseline and changes in anxiety and pain catastrophizing predicting functional disability over time. Bolded lines show strongest predictor of functional disability from individual models.

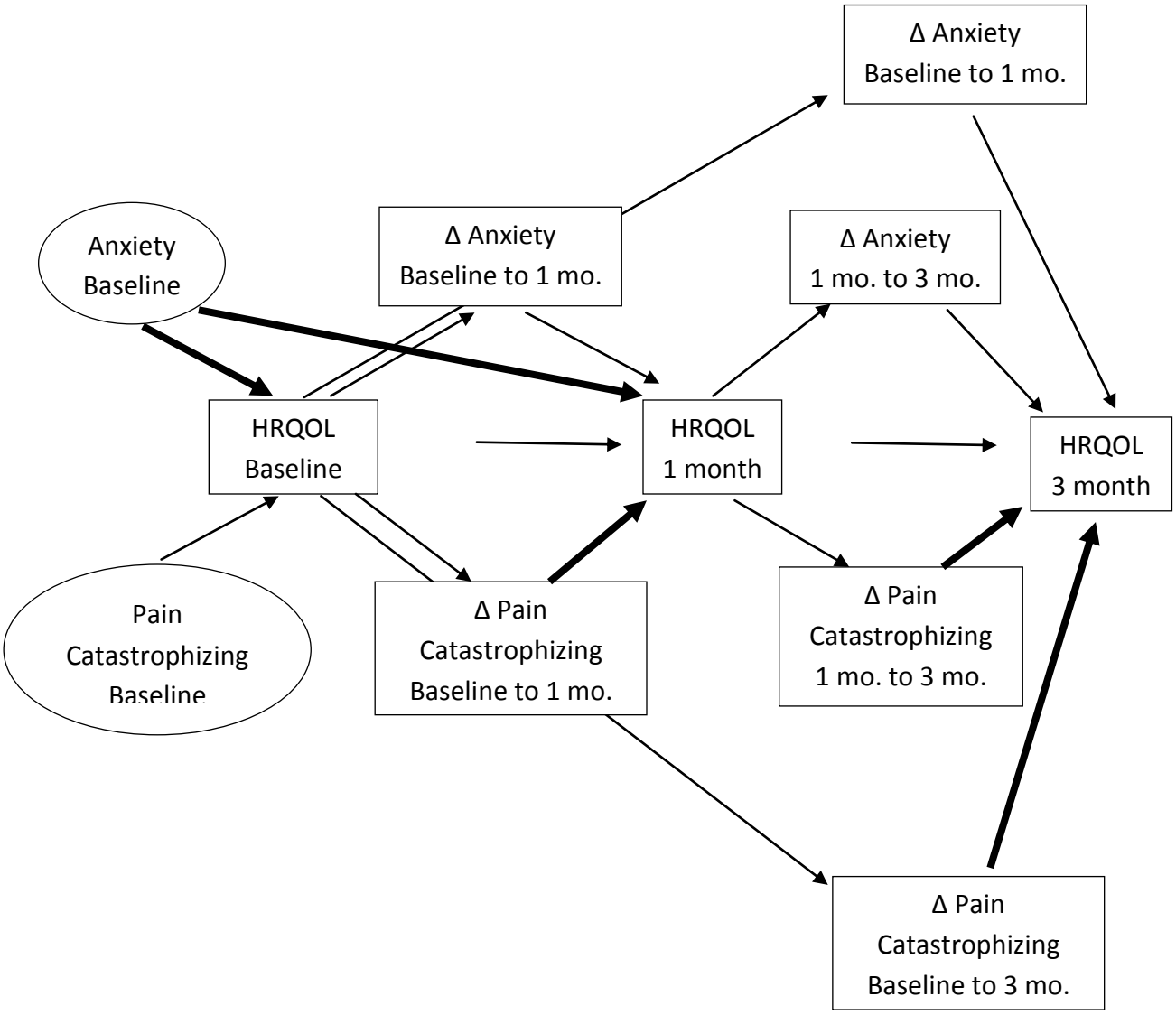

Figure 3. Predicting HRQOL over time. Model shows unique contributions of anxiety and pain catastrophizing at baseline and changes in anxiety and pain catastrophizing predicting HRQOL over time. Bolded lines show strongest predictor of HRQOL from individual models.

Appendix

Exploratory Analyses: Predicting Functioning Variables at One Month from Anxiety and Pain Catastrophizing from Baseline and Predicting Functioning Variables at Three Months from Anxiety and Pain Catastrophizing from One Month.

Hierarchical multiple regressions were conducted to test the final component of the research plan, that *anxiety and pain catastrophizing at baseline predict functioning at the one month follow-up, and that anxiety and pain catastrophizing at one month predict functioning at three month follow-up*. Separate regression analyses were completed for each outcome variable at one and three month follow-up. Demographic variables found to be related to the SCARED, PCS-C, and the outcome variable were entered first, followed by pain variables at baseline (one month), the outcome variable at baseline (one month), and finally SCARED total score and PCS-C total score from baseline (one month) were entered into the final step.

Predicting pain at one month follow-up. The model with demographic variables, pain intensity at baseline, and SCARED and PCS-C scores at baseline was significant ($R = .57$, $p < .001$). Adding in the SCARED and PCS-C scores at baseline added 1% (p = ns) of variance, and neither coefficient was significant in the final model (Table A1).

Predicting pain at three month follow-up. The model with demographic variables, pain intensity at one month, and SCARED and PCS-C scores at one month was significant ($R = .76$, $p < .001$). Adding in the SCARED and PCS-C scores at one month

added 2% of explained variance (p = ns), and neither coefficient was significant in the final model (Table A2).

Predicting pain at three month follow-up from baseline. The model with demographic variables, pain intensity, SCARED, and PCS-C scores at baseline was significant (R = .46, $p < .001$). Adding in the SCARED and PCS-C scores at baseline added <1% of explained variance (p = ns), and neither coefficient was significant in the final model (Table A3).

Predicting CALQ at one month follow-up. The model with demographic variables, pain variables at baseline, and CALQ, SCARED, and PCS-C scores at baseline was significant (R = .74, $p < .001$). Adding in the SCARED and PCS-C at baseline added 2% of variance explained ($p < .05$), however neither coefficient was significant in the final model (Table A4).

Predicting CALQ at three month follow-up. The model with demographic variables, pain variables at one month, and CALQ, SCARED, and PCS-C scores at one month was significant (R = .92, $p < .001$). Adding in SCARED and PCS-C scores at baseline added 2% of explained variance ($p < .05$). SCARED scores at one month were significant while PCS-C scores were not significant (Table A5).

Predicting CALQ at three month follow-up from baseline. The model with demographic variables, pain variables at baseline, and CALQ, SCARED, and PCS-C scores at baseline was significant (R = .66, $p < .001$). Adding in SCARED and PCS-C scores at baseline added 5% explained variance ($p < .05$). SCARED scores at baseline were significant while PCS-C scores were not significant (Table A6).

Predicting PedsQL at one month follow-up. The model with demographic variables, pain variables at baseline, and PedsQL, SCARED, and PCS-C scores at baseline was significant (R = .77, $p < .001$). Adding in the SCARED and PCS-C scores at baseline added 7% ($p < .001$) variance. SCARED scores were significant while PCS-C scores were not significant (Table A7).

Predicting PedsQL at three month follow-up. The model with demographic variables, pain variables at one month, and PedsQL, SCARED, and PCS-C scores at one month was significant (R = .82, $p < .001$). Adding in SCARED and PCS-C scores at one month added 2% of explained variance (p = ns), however neither coefficient was significant in the final model (Table A8).

Predicting PedsQL at three month follow-up from baseline. The model with demographic variables, pain variables at baseline, and PedsQL, SCARED, and PCS-C scores at baseline was significant (R = .69, $p < .001$). Adding in SCARED and PCS-C scores at baseline added 2% of explained variance (p = ns), however neither coefficient was significant in the final model (Table A9).

Table A1

Predicting Pain Intensity at One Month Follow-Up with SCARED and PCS-C at Baseline

Variable	*B*	*SE(B)*	*B*	*Semi-partial correlation*	R^2
STEP ONE					.32***
Gender	0.66	0.33	.13	.13	
Pain intensity$_{T0}$	0.63	0.08	.54***	.54	
STEP TWO					.33***
Gender	0.63	0.34	.12	.12	
Pain intensity$_{T0}$	0.60	0.08	.52***	.49	
SCARED $_{T0}$	0.01	0.01	.01	.01	
PCS-C $_{T0}$	0.01	0.02	.07	.06	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure.

Table A2

Predicting Pain Intensity at Three Month Follow-Up with SCARED and PCS-C at One Month

Variable	*B*	*SE(B)*	*B*	*Semi-partial correlation*	R^2
STEP ONE					.56***
Gender	0.33	0.42	.06	.06	
Pain intensity$_{1\ mo.}$	0.76	0.08	.74***	.73	
STEP TWO					.58***
Gender	0.27	0.42	.05	.05	
Pain intensity$_{1\ mo.}$	0.71	0.08	.69***	.64	
SCARED $_{1\ mo.}$	0.01	0.01	.02	.02	
PCS-C $_{1\ mo.}$	0.02	0.02	.14	.10	

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

Table A3

Predicting Pain Intensity at Three Month Follow-Up with SCARED and PCS-C at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.21***
Gender	0.51	0.50	.09	.09	
Pain intensity$_{T0}$	0.55	0.11	.44***	.43	
STEP TWO					.21***
Gender	0.49	0.50	.09	.09	
Pain intensity$_{T0}$	0.54	0.12	.43***	.40	
SCARED $_{T0}$	0.01	0.02	.05	.04	
PCS-C $_{T0}$	-0.01	0.02	-.01	-.01	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure.

Table A4

Predicting Functional Disability at One Month Follow-Up with SCARED and PCS-C at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.52***
Gender	2.52	3.55	.05	.04	
Age	1.32	0.65	.13*	.12	
Pain duration	0.09	0.06	.09	.09	
Pain frequency	0.85	0.46	.14	.11	
Usual pain	2.11	1.00	.18*	.13	
Worst pain	-1.84	1.11	-.15	-.10	
$CALQ_{T0}$	0.67	0.07	.65***	.56	
STEP TWO					.55***
Gender	2.29	3.50	.04	.04	
Age	1.33	0.67	.13	.12	
Pain duration	0.07	0.06	.07	.07	
Pain frequency	0.94	0.47	.15	.12	
Usual pain	1.84	0.99	.15	.11	
Worst pain	-1.78	1.14	-.14	-.09	
$CALQ_{T0}$	0.63	0.07	.61***	.51	
$SCARED_{T0}$	0.26	0.13	.14	.11	
$PCS\text{-}C_{T0}$	0.06	0.17	.03	.02	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure.

Table A5

Predicting CALQ at Three Month Follow-Up with SCARED and PCS-C at One Month

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.82***
Gender	-1.59	3.17	-.03	-.03	
Age	-0.85	0.61	-.08	-.07	
Pain duration	-0.01	0.06	-.01	-.01	
Pain frequency	-0.14	0.36	-.03	-.02	
Usual pain	-0.11	1.02	-.01	-.01	
Worst pain	-0.29	1.09	-.03	-.01	
$CALQ_{1\ mo.}$	0.99	0.07	.93***	.77	
STEP TWO					.84***
Gender	-2.05	3.02	-.03	-.03	
Age	-0.88	0.61	-.08	-.07	
Pain duration	-0.03	0.06	-.03	-.02	
Pain frequency	-0.07	0.35	-.01	-.01	
Usual pain	-0.06	0.98	-.01	-.01	
Worst pain	-0.49	1.05	-.04	-.02	
$CALQ_{1\ mo.}$	0.93	0.07	.87***	.67	
$SCARED_{1\ mo.}$	0.24	0.10	.15*	.11	
$PCS\text{-}C_{1\ mo.}$	0.03	0.13	.02	.01	

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

Table A6

Predicting CALQ at Three Month Follow-Up with SCARED and PCS-C at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.38***
Gender	-4.44	5.29	-.08	-.07	
Age	1.17	0.91	.11	.11	
Pain duration	0.06	0.09	.06	.06	
Pain frequency	0.83	0.63	.14	.11	
Usual pain	2.30	1.53	.18	.13	
Worst pain	-1.46	1.67	-.11	-.07	
$CALQ_{T0}$	0.55	0.10	.55***	.47	
STEP TWO					.43***
Gender	-5.40	5.14	-.09	-.09	
Age	0.63	0.92	.06	.06	
Pain duration	0.01	0.09	.01	.01	
Pain frequency	0.80	0.61	.13	.11	
Usual pain	2.26	1.49	.17	.12	
Worst pain	-1.09	1.66	-.08	-.05	
$CALQ_{T0}$	0.54	0.10	.54***	.44	
$SCARED_{T0}$	0.60	0.22	.30**	.23	
$PCS\text{-}C_{T0}$	-0.38	0.23	-.19	-.14	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure.

Table A7

Predicting PedsQL at One Month Follow-Up with SCARED and PCS-C at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.52***
Gender	-6.60	2.76	-.15*	-.14	
Age	-0.08	0.49	-.01	-.01	
Pain duration	-0.05	0.05	-.07	-.07	
Pain frequency	-0.09	0.35	-.02	-.02	
Usual pain	-0.77	0.78	-.08	-.06	
Worst pain	0.92	0.85	.09	.07	
$PedsQL_{T0}$	0.78	0.08	.69***	.63	
STEP TWO					.59***
Gender	-6.23	2.58	-.14*	-.13	
Age	-0.03	0.49	-.01	-.01	
Pain duration	-0.02	0.04	-.03	-.03	
*Pain frequency	-0.30	0.34	-.06	-.05	
Usual pain	-0.60	0.72	-.06	-.05	
Worst pain	0.64	0.82	.06	.04	
$PedsQL_{T0}$	0.59	0.08	.52***	.40	
$SCARED_{T0}$	-0.44	0.10	-.31***	-.24	
$PCS\text{-}C_{T0}$	0.01	0.12	.01	.01	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure.

Table A8

Predicting PedsQL at Three Month Follow-Up with SCARED and PCS-C at One Month

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
STEP ONE					.66***
Gender	3.20	3.45	.07	.07	
Age	0.45	0.66	.05	.05	
Pain duration	-0.07	0.06	-.09	-.08	
Pain frequency	-0.17	0.37	-.04	-.03	
Usual pain	0.33	1.10	.04	.02	
Worst pain	-0.82	1.18	-.09	-.05	
$PedsQL_{1\ mo.}$	0.90	0.09	.78***	.72	
STEP TWO					.67***
Gender	3.35	3.41	.07	.07	
Age	0.82	0.68	.09	.08	
Pain duration	-0.07	0.06	-.09	-.08	
Pain frequency	-0.06	0.38	-.01	-.01	
Usual pain	0.15	1.09	-.02	.01	
Worst pain	-0.93	1.18	-.10	-.06	
$PedsQL_{1\ mo.}$	0.92	0.13	.80***	.51	
$SCARED_{1\ mo.}$	-0.19	0.12	-.15	-.11	
$PCS\text{-}C_{1\ mo.}$	0.25	0.16	.17	.11	

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

Table A9

Predicting PedsQL at Three Month Follow-Up with SCARED and PCS-C at Baseline

Variable	*B*	*SE(B)*	*β*	*Semi-partial correlation*	R^2
***STEP ONE					.45***
Gender	-0.91	4.05	-.02	-.02	
Age	0.32	0.70	.04	.04	
Pain duration	-0.12	0.07	-.14	-.13	
Pain frequency	-0.45	0.47	-.09	-.08	
Usual pain	-1.40	1.17	-.13	-.10	
Worst pain	1.49	1.29	.13	.09	
$PedsQL_{T0}$	0.77	0.11	.66***	.56	
STEP TWO					.47***
Gender	-0.23	4.04	-.01	-.01	
Age	0.42	0.73	.05	.05	
Pain duration	-0.08	0.07	-.10	-.09	
Pain frequency	-0.53	0.48	-.11	-.09	
Usual pain	-1.44	1.17	-.14	-.10	
Worst pain	1.38	1.31	.12	.08	
$PedsQL_{T0}$	0.67	0.13	.57***	.42	
$SCARED_{T0}$	-0.31	0.18	-.19	-.13	
$PCS\text{-}C_{T0}$	0.06	0.18	.04	.03	

Note. *$p < .05$, **$p < .01$, ***$p < .001$. T0 denotes baseline measure.

Susan T. Tran

Curriculum Vitae

EDUCATION

2011 – Present
Doctoral Candidate, Department of Psychology
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Major: Clinical Psychology
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Dissertation Title: "Longitudinal Associations between Anxiety, Pain Catastrophizing, and Treatment Outcomes in Complex Pediatric Chronic Pain"
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2008 – 2010
Master of Science, Department of Psychology
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Major: Clinical Psychology
Thesis Title: "Applying the Fear-Avoidance Model to Pediatric Chronic Pain"
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2004 – 2008
Bachelor of Arts, *Magna Cum Laude*, Department of Psychology
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Major: Psychology
Minors: Spanish and Anthropology

HONORS AND AWARDS

2013	Society of Pediatric Psychology Graduate Student Spotlight Award
2013, 2011	University of Wisconsin-Milwaukee Graduate Student Travel Award
2012, 2010	American Psychological Association Student Travel Award
2011-2012	University of Wisconsin-Milwaukee Graduate School Fellowship
2008	Marquette University Psychology Department Outstanding Senior Award
2006-2008	Psi Chi, Psychology National Honors Society, Marquette University
2004-2008	Ignatius Scholarship, Marquette University, awarded annually
2004-2008	Dean's List, Marquette University, awarded each semester

CLINICAL EXPERIENCE

July 2013 – Present
Predoctoral Clinical Internship, *O'Grady Residency in Behavioral Medicine,* Cincinnati Children's Hospital Medical Center

Sept. 2012 – May 2013
Practicum in Clinical Supervision, *Constipation and Encopresis Clinic, Gastroenterology Center,* Children's Hospital of Wisconsin, Milwaukee

Sept. 2012 – May 2013 **Assessment Practicum Graduate Student Supervisor,** *Psychology Clinic,* University of Wisconsin-Milwaukee

Aug. 2012 – May 2013 **Advanced Assessment Practicum,** *Children's Hospital of Wisconsin Community Services,* Milwaukee

June 2012 – May 2013 **Advanced Therapy Practicum,** *Jane B. Pettit Pain and Palliative Care Clinic,* Children's Hospital of Wisconsin, Milwaukee

June 2012 – Aug. 2012 **Advanced Therapy Practicum,** *Consultation & Liaison Service,* Children's Hospital of Wisconsin, Milwaukee

June 2012 – Aug. 2012 **Advanced Therapy Practicum,** *High-Risk Asthma Clinic,* Children's Hospital of Wisconsin, Milwaukee

May 2012 – Aug. 2012 **Advanced Assessment Practicum,** *Moderate Traumatic Brain Injury Clinic,* Children's Hospital of Wisconsin, Milwaukee

March 2012 **Advanced Assessment Practicum,** *Child Development Center,* Children's Hospital of Wisconsin, Milwaukee

Sept. 2011 – May 2012 **Advanced Therapy Practicum,** *Gastroenterology Center,* Children's Hospital of Wisconsin, Milwaukee

Aug. 2010 – July 2011 **Therapy Practicum,** *Constipation and Encopresis Clinic, Gastroenterology Center,* Children's Hospital of Wisconsin, Milwaukee

Aug. 2010 – Dec. 2011 **Therapy Practicum,** *UWM Psychology Clinic,* University of Wisconsin-Milwaukee

June 2009 – May 2010 **Assessment Practicum,** *UWM Psychology Clinic,* University of Wisconsin-Milwaukee

April 2007 – May 2008 **Line Therapist,** *Wisconsin Early Autism Project,* Milwaukee, WI

OTHER TRAINING EXPERIENCES

2011-2013 **Didactic Training, Advanced Practicum,** *Children's Hospital of Wisconsin*
Met weekly with other practicum students and pediatric psychologists to discuss topics in pediatric psychology and give case presentations. Supervised by Alan H. Silverman, Ph.D., Kimberly Anderson Khan, Psy.D., & Sara E. Williams, Ph.D.

2012 **Trauma-Focused Cognitive Behavioral Therapy Training**
Completed web-based learning course offered through the Medical University of South Carolina on how to treat children and adolescents following a trauma.

2012 **EPIC Electronic Medical Record Training**
Trained to use EPIC system at the Children's Hospital of Wisconsin to locate medical records and write session notes.

RESEARCH EXPERIENCE

July 2010 – Present *Center for Empirical Ethics and Research Practices*
University of Wisconsin-Milwaukee, Milwaukee, WI

July 2011 – May 2013 *Biomechanical Evaluation of Yoga for Pediatric Obesity*
Children's Hospital of Wisconsin, Milwaukee, WI

Aug. 2008 – May 2013 *Pain and Palliative Care Center*
Children's Hospital of Wisconsin, Milwaukee, WI

Jan. 2009 – July 2011 *Parent Attitudes toward Multidisciplinary Pain Clinic*
Children's Hospital of Wisconsin, Milwaukee, WI

Oct. 2009 – Jan. 2011 *Parent Attitudes and Behaviors towards H1N1Vaccination*
University of Wisconsin-Milwaukee, Milwaukee, WI

Jan. 2009 – June 2010 *Sibling Relationships When a Sibling Has Chronic Pain*
Children's Hospital of Wisconsin, Milwaukee, WI

Sept. 2008 – March 2009 *Barriers to Optimal Pain Management*
Children's Hospital of Wisconsin, Milwaukee, WI

Aug. 2007 – May 2008 *Adherence to Diabetes Treatment Study*
Marquette University, Milwaukee, WI

May 2007 – May 2008 *ADHD Clinic*
Marquette University, Milwaukee, WI

Aug. 2006 – May 2008 *Constipation Quality of Life Study*
Marquette University, Milwaukee, WI

PUBLICATIONS

1. **Tran, S. T.,** Salamon, K. S., Hainsworth, K. R., Kichler, J. C., Davies, W. H., Alemzadeh, R., & Weisman, S. J. (in press). Pain reports in children and adolescents with type 1 diabetes mellitus. *Journal of Child Health Care*.

2. Gorodzinsky, A. Y., Davies, W. H., **Tran, S. T.**, Medrano, G. R., Bernacki, J. M., Burks, L. M., Anderson Khan, K., Hainsworth, K. R., & Weisman, S. J. (2013). Adolescents' perceptions of family dynamics when a sibling has chronic pain. *Children's Health Care, 42* (4).
3. Engel, J. M., Wilson, S., **Tran, S. T.,** Jensen, M. P. & Ciol, M. A. (2013). Pain catastrophizing in youths with physical disabilities and chronic pain. *Journal of Pediatric Psychology, 38,* 192-201. doi:10.1093/jpepsy/jss103.
4. Jastrowski Mano, K. E., Evans, J. R., **Tran, S. T.,** Anderson Khan, K., Weisman, S. J., & Hainsworth, K. R. (2012). The psychometric properties of the Screen for Child Anxiety Related Emotional Disorders (SCARED) in pediatric chronic pain. *Journal of Pediatric Psychology, 37* (9), 999-1011. doi:10.1093/jpepsy/jss069.
5. Gorodzinsky, A. Y., **Tran, S. T.**, Medrano, G. R., Fleischman, K. M., Anderson Khan, K., Ladwig, R. J., & Weisman, S. J. (2012). Parents' initial perceptions of multidisciplinary care for pediatric chronic pain. *Pain Research and Treatment, 2012*. doi:10.1155/2012/791061.
6. **Tran, S. T.,** Gorodzinsky, A. Y., & Davies, W. H. (2011). Predictors and barriers of H1N1 vaccination intention in healthy and high-risk children. *Children's Health Care, 40* (4), 269-281. doi: 10.1080/02739615.2011.617245.
7. Kaugars, A. S., Silverman, A., Kinservik, M., **Heinze, S**., Reinemann, L., Sander, M., Schneider, B., & Sood, M. (2010). Families' perspectives on how constipation and fecal incontinence impact quality of life. *Journal of Pediatric Gastroenterology and Nutrition, 51* (6), 747-752. doi: 10.1097/MPG.0b013e3181de0651.

PAPER PRESENTATIONS AT CONFERENCES

1. Feller, T. M., **Tran, S. T.,** & Davies, W. H. (2012 April). *Behavior problems and quality of life in a community sample of children with food allergies*. Paper presentation at the 2012 Midwest Regional Conference in Pediatric Psychology, Milwaukee, WI.
2. **Heinze, S. T.,** Khan, K. A., Hainsworth, K. R., Weisman, S. J. & Davies, W. H. (2011 February). *Elevated reports of anxiety symptoms among pediatric chronic pain patients: A need for routine screening?* Paper presentation at the 2011 Annual Pediatric Behavioral Health Research Conference in Milwaukee, WI.
3. Gorodzinsky, A. Y., **Heinze, S. T.,** Joseph, J. M., Medrano, G. R., Anderson Khan, K., Ladwig, R., Hainsworth, K. R., Weisman, S. J., & Davies, W. H. (2011 February). *Adolescents' perceptions of their familial relationship when a sibling has chronic pain*. Paper presentation at the 2011 Annual Pediatric Behavioral Health Research Conference in Milwaukee, WI.

PRESENTATIONS AT INTERNATIONAL MEETINGS

1. Jastrowski-Mano, K. E., Hainsworth, K. R., Medrano, G. R., **Tran, S. T.,** & Weisman, S. (2012 August). *Validation of the Pediatric Symptom Checklist in a chronic pain sample*. Poster presentation at the International Association for the Study of Pain 14th World Congress on Pain, Milan, Italy.

2. Gorodzinsky, A. Y., Davies, W. H., Medrano, G. R., **Tran, S. T.,** Joseph, J. M., Burks, L. M., Feller, T. M., Anderson Khan, K., Ladwig, R. J., Hainsworth, K. R. & Weisman, S. J. (2011 October). *Changes in familial relationships related to pediatric chronic pain: Perspectives from adolescent patients and their siblings*. Poster presentation at the International Forum on Pediatric Pain, White Point, Nova Scotia, Canada.
3. Anderson Khan, K., **Heinze, S. T.**, Ladwig, R. J., Jastrowski Mano, K., Hainsworth, K. R. (2011 April). *The effect of anxiety symptoms on school functioning in a pediatric chronic pain sample*. Poster presentation and at the International Forum on Pediatric Pain, White Point, Nova Scotia, Canada and at the 2011 National Conference in Pediatric Psychology, San Antonio, TX.

PRESENTATIONS AT NATIONAL MEETINGS

1. Liu, X. C., Hainsworth, K., Marquez, C., Simpson, P., Swartz, A., **Tran, S.**, Medrano, G., Lyon, R., & Weisman, S. (2013 May). *Application of the gait deviation index in quantifying overweight children's mobility.* Platform presentation to the Gait and Clinical Movement Analysis Society 2013 Annual Conference, Cincinnati, OH.
2. **Tran, S. T.,** Medrano, G. R., Anderson Khan, K., Ladwig, R. J., Weisman, S. J., Davies, W. H., & Hainsworth, K. R. (2013 April). *Differential utility of pain catastrophizing by reporter for predicting later functioning in chronic pain*. Poster presentation at the 2013 National Conference in Pediatric Psychology, New Orleans, LA.
3. Bauer, K., Karvounides, D., Burks, L. M., Tran, S. T., & Davies, W. H. (2013 April). *History of dismissed pain predicts psychosocial functioning in emerging adults*. Poster presentation at the 2013 National Conference in Pediatric Psychology, New Orleans, LA.
4. Karvounides, D., Jastrowski-Mano, K. E., **Tran, S. T.,** Weisman, S., Davies, W. H., & Hainsworth, K. R. (2013 April). *Initial validation of the Stress Numerical Rating Scale-11 (SNRS-11) with a community sample of parents with school-age children*. Poster presentation at the 2013 National Conference in Pediatric Psychology, New Orleans, LA.
5. Hainsworth, K. R., Simpson, P., Swartz, A., **Tran, S. T.**, Medrano, G. R., Mascarenhas, B. Weisman, S. J., & Liu, X. C. (2013 January). *Effects of yoga on gait performance in obese youth*. Orthopedic Research Society.
6. **Tran, S. T.,** Davies, W. H., Medrano, G. R., & Burks, L. M. (2012 August). *The relationship between provider satisfaction and likelihood of research participation*. Poster presentation at the 2012 Annual Convention of the American Psychological Association, Orlando, FL.
7. **Heinze, S. T.,** Majewski, A. J., Medrano, G. R., & Davies, W. H. (2011 April). *Parent intended participation and attitudes toward opt-in and opt-out recruitment methods*. Poster presentation at the 2011 National Conference in Pediatric Psychology, San Antonio, TX, and at the 2012 Annual Interdisciplinary Pediatric Behavioral Health Research Conference, Milwaukee, WI.
8. **Heinze, S. T.,** Majewski, A. J., Bennaton, E. C., & Davies, W. H. (2011 April). *Parent beliefs regarding acceptability of recruitment methods in pediatric research*.

Poster presentation at the 2011 National Conference in Pediatric Psychology, San Antonio, TX.

9. **Heinze, S. T.**, Anderson Khan, K., Ladwig, R., Hainsworth, K. R., Davies, W. H., Weisman, S. J. (2011 April). *Anxiety symptoms and pain catastrophizing in a pediatric population*. Poster presentation at the 2011 National Conference in Pediatric Psychology, San Antonio, TX.
10. Joseph, J. M., **Heinze, S. T.,** Holman, K. S., Hainsworth, K. R., & Davies, W. H. (2010 November). *Validation of the Pain Frequency-Severity-Duration Scale in community young adults*. Poster presentation at the 2010 Association for Behavioral and Cognitive Therapies Annual Convention, San Francisco, CA and the 2011 Annual Interdisciplinary Pediatric Behavioral Health Research Conference, Milwaukee, Wisconsin.
11. **Heinze, S. T.,** Elftman, J. L., & Davies, W. H. (2010 August). *Anxiety sensitivity and pain catastrophizing: Distinct factors in predicting pain*. Poster presentation at the 2010 American Psychological Association Annual Convention, San Diego, CA.
12. **Heinze, S. T.,** Gorodzinsky, A. Y., Drew, J. G., & Davies, W. H. (2010 August). *Parents' perceptions of H1N1 risk predicts child vaccinations.* Poster presentation at the 2010 American Psychological Association Annual Convention, San Diego, CA.
13. **Heinze, S. T.,** Medrano, G. R., Gorodizinsky, A. Y., Hainsworth, K. R., & Weisman, S. J. (2010 April). *Applying the fear-avoidance model of chronic pain to a pediatric population*. Poster presentation at the 2010 Annual Meeting & Scientific Sessions of the Society of Behavioral Medicine, Seattle, WA.
14. Medrano, G. R., **Heinze, S. T.,** Hainsworth, K. R., & Weisman, S. J. (2010 April). *Pediatric chronic pain and differences in parental health-related quality of life*. Poster presentation at the 2010 Annual Meeting & Scientific Sessions of the Society of Behavioral Medicine in Seattle, WA and the 2011 Annual Interdisciplinary Pediatric Behavioral Health Research Conference, Milwaukee, Wisconsin.
15. **Heinze, S. T.,** Medrano, G. R., Simon, K., Czarnecki, M., Turner, H., & Wrona, S. (2009 October). *Pediatric nurses' perceptions of optimal pain management across departments*. Poster presentation at the 2009 Annual Meeting of The Society for Developmental and Behavioral Pediatrics, Portland, OR.
16. Gorodzinsky, A. Y., Joseph, J. M., **Heinze, S. T.,** Simon, K., Drendel, A. L., & Davies, W. H. (2009 October). *Parents' perceptions of pharmacological and non-pharmacological techniques for pain control in children*. Poster presentation at the 2009 Annual Meeting of The Society for Developmental and Behavioral Pediatrics, Portland, OR.
17. Schneider, B. W., Gerdes, A. C., & **Heinze, S. T.** (2008 August). *Child factors that predict treatment dropout in parent training interventions in families of children with ADHD*. Poster presentation at the 2008 Conference of the American Psychological Association, Boston, MA.
18. Kaugars, A. S., **Heinze, S. T.,** Kinservik, M., Reinemann, L., Schneider, B., & Sood, M. (2008 April). *Emotional, social, family, and academic functioning in children with constipation and encopresis: How is quality of life affected?* Poster presentation at the 2008 National Conference on Child Health Psychology, Miami, FL.

PRESENTATIONS AT REGIONAL MEETINGS

1. Burks, L. M., **Tran, S. T.,** Medrano, G. M., & Davies, W. H. (2012 April). *Parent perceptions on the right to request destruction of data after study drop-out*. Poster presentation at the 2012 Midwest Regional Conference in Pediatric Psychology, Milwaukee, WI.
2. Evans, J., Jastrowski Mano, K., **Tran, S. T.,** Anderson Khan, K., Weisman, S. J, & Hainsworth, K. (2012 April). *Confirmatory factor analysis of the SCARED in a pediatric chronic pain sample: Implications for use*. Poster presentation at the 2012 Midwest Regional Conference in Pediatric Psychology, Milwaukee, WI.
3. Silverman, A., Begotka, A., **Tran, S. T.,** Holman, K., & Sood, M. (2012 April). *Constipation and soiling disorders: Effects on quality of life in a pediatric population*. Poster presentation at the 2012 Midwest Regional Conference in Pediatric Psychology, Milwaukee, WI.
4. **Tran, S. T**., Hainsworth, K. R., Anderson Khan, K., Ladwig, R., Rusy, L., Varadarajan, J., Guastello, A. D., Davies, W. H., & Weisman, S. J. (2012 April). *Stability of anxiety ratings and parent-child concordance in a pediatric chronic pain population*. Poster presentation at the 2012 Midwest Regional Conference in Pediatric Psychology, Milwaukee, WI.
5. **Tran, S. T.** & Williams, S. (2012 April). *Longitudinal outcomes for pediatric functional abdominal pain patients*. Poster presentation at the 2012 Midwest Regional Conference in Pediatric Psychology, Milwaukee, WI.
6. Feller, T. M., **Tran, S. T**., Bernacki, J. M., Hainsworth, K. R., Davies, W. H., & Weisman, S. J. (2012 February) *Readiness to adopt self management approaches in children with chronic pain*. Poster presentation at the 2012 Annual Interdisciplinary Pediatric Behavioral Health Research Conference, Milwaukee, WI.
7. Feller, T. M., Gorodzinsky A. Y., Medrano, G. R., Burks, L. M., **Heinze, S. T.,** Joseph, J. M., & Davies, W. H. (2011 April). *Chronic pain and how it changes family relationships*. Poster presentation at the Wisconsin Psychological Association Conference, Middleton, WI, and at the Association of Graduate Students in Psychology Research Symposium, Milwaukee, WI.
8. Gorodzinsky, A. Y., **Heinze, S. T.,** Joseph, J. M., Medrano, G. R., Elftman, J., Khan, K. A., Hainsworth, K. R., Weisman, S. J., & Davies, W. H. (2010 October). *Adolescents' perceptions of their relationship to a sibling with chronic pain.* Poster presentation at the 2010 Kansas Conference in Clinical Child and Adolescent Psychology, Lawrence, KS.
9. **Heinze, S. T.,** Khan, K. A., Hainsworth, K. R., & Weisman, S. J. (2010 October). *Elevated reports of anxiety symptoms among pediatric chronic pain patients: A need for routine screening?* Poster presentation at the 2010 Kansas Conference in Clinical Child and Adolescent Psychology, Lawrence, KS.
10. **Heinze, S. T.,** Gorodzinsky, A. Y., & Davies. W. H. (2010 March). *H1N1 vaccination behavior and attitudes not predicted by high-risk pediatric diagnosis*. Poster presentation at the 2010 Wisconsin Psychological Association Annual Convention, Middleton, WI.
11. **S**ayers, K, Salamon, K. S., **Heinze, S. T.** & Davies, W. H. (2010 March). *Perceptions of complementary and alternative medicine (CAM) therapies among*

those with anxiety sensitivity. Poster presentation at the 2010 Wisconsin Psychological Association Annual Convention, Middleton, WI.

12. Elftman, J. L., Gorodzinsky, A. Y., **Heinze, S. T.**, Joseph, J. M., Medrano, G. R., Salamon, K. S., Ladwig, R., Khan, K. A., Hainsworth, K., & Weisman, S. J. (2010 March). *Pain effect on siblings of adolescents with chronic pain*. Poster presentation at the 2010 Wisconsin Psychological Association Annual Convention, Middleton, WI.
13. Czarnecki, M., **Heinze, S. T.,** Medrano, G. R., Salamon, K. S., Turner, H., & Wrona, S. (2010 February). *Optimal pain management: What does it mean to pediatric nurses?* Poster presentation at the 2010 Annual Interdisciplinary Pediatric Behavioral Health Research Conference, Milwaukee, WI.
14. Gorodzinsky, A. Y., **Heinze, S. T.,** Joseph, J. M., Medrano, G. R., Salamon, K. S., Khan, K. A., & Hainsworth, K. R. (2010 February). *Adolescents' perceptions of sibling relationships when their sibling has chronic pain*. Poster presentation at the 2010 Annual Interdisciplinary Pediatric Behavioral Health Research Conference, Milwaukee, WI.
15. **Heinze, S. T.,** Medrano, G. R., Simon, K., Czarnecki, M., Turner, H., & Wrona, S. (2009 April). *Perceptions of optimal pain management among nurses who work with pediatric patients*. Poster presentation to the 2009 Midwest Conference on Pediatric Psychology, Kansas City, MO.
16. Medrano, G. R., **Heinze, S. T.,** Czarnecki, M., Simon, K., Turner, H., & Wrona, S. (2009 April). *Role of experience in nurses' perceptions of barriers to optimal pain management with pediatric patients*. Poster presentation to the 2009 Midwest Conference on Pediatric Psychology, Kansas City, MO.

GRANTS AWARDED

2012 Student Association Group Travel Grant, awarded $1520
Health Psychology Graduate Students Club, University of Wisconsin-Milwaukee

2010 Student Association Group Travel Grant, awarded $900
Health Psychology Graduate Students Club, University of Wisconsin-Milwaukee

2009 Student Association Group Travel Grant, awarded $700
Health Psychology Graduate Students Club, University of Wisconsin-Milwaukee

2008 Student Association Group Travel Grant, awarded $1529
Association of Graduate Students in Psychology, University of Wisconsin-Milwaukee

TEACHING EXPERIENCE

June 2012 – May 2013 **Teaching Assistant (Graduate Practicum),** Department of Psychology, UWM, Course: *Psychology 831:* 2nd Year Clinical Practicum: Assessment
Supervised by Bonita P. Klein-Tasman, Ph.D. and Han Joo Lee, Ph.D.

Jan. 2012 – May 2012 **Associate Lecturer,** Department of Psychology, UWM
Course: *Psychology 660:* Experimental Child Psychology (online)

Sept. 2011 – Dec. 2011	**Teaching Assistant,** Department of Psychology, UWM Course: *Psychology 325:* Research Methods in Psychology (online) Supervised by Katie E. Mosack, Ph.D.
Sept. 2009 – May 2011	**Teaching Assistant,** Department of Psychology, UWM Course: *Psychology 660/760:* Experimental Child Psychology (offline and online) Supervised by W. Hobart Davies, Ph.D.
Sept. 2008 – May 2009	**Teaching Assistant,** Department of Psychology, UWM Course: *Psychology 260:* Child Psychology Supervised by Richard Passman, Ph.D.
Aug. 2006 – May 2008	**Tutor,** *Freshmen Frontier Program,* Marquette University Courses: Introduction to Psychology, Spanish

GUEST LECTURES

June 2013	*Pediatric Chronic Pain,* Health Psychology, Marquette University
May 2013	*Group Treatment of Pediatric Encopresis,* Graduate Seminar: Introduction to Scientifically-Validated Treatments, UWM
Dec. 2012/May 2013	*Pediatric Chronic Pain,* Child Psychology, UWM
April 2012	*Physiology and Treatment of Encopresis,* Graduate Seminar: Introduction to Scientifically-Validated Treatments, UWM
March 2011	*Parenting Styles and Discipline*, Child Psychology, UWM
March 2010/2011	*Qualitative Methods of Research,* Experimental Child Psychology, UWM
May 2010	*Quality of Life in Pediatric Populations,* Experimental Child Psychology, UWM
June 2009	*Treatment of Pediatric Encopresis,* Experimental Child Psychology, UWM

PROFESSIONAL AFFILIATIONS AND LEADERSHIP POSITIONS

2008-Present	American Psychological Association, Student Affiliate
2009-Present	Society of Pediatric Psychology, Student Member
2010-Present	Society of Clinical Child & Adolescent Psychology, Student Member
2009-2013	Health Psychology Graduate Students Club, UWM President (2011-2013), Vice President (2010-2011), Secretary (2009-2010)

PROFESSIONAL SERVICE

2013	Ad-Hoc Reviewer for the *Journal of Child Health Care*
2011-2013	Society of Pediatric Psychology (APA Div. 54) Campus Representative

2011 Member of the Graduate Student Planning Committee for the 2012 Regional Society of Pediatric Psychology (APA Div. 54) Conference

Ad Hoc Co-Reviewer for *Journal of Pediatric Psychology*

2008- present Supervised by W. Hobart Davies, Ph.D.
2009- present Supervised by Deirdre Logan, Ph.D. (JPP mentoring program)
2012 Supervised by Alan H. Silverman, Ph.D.

Ad Hoc Co-Reviewer for *The Clinical Journal of Pain*

2012 Supervised by Keri R. Hainsworth, Ph.D.

REFERENCES

W. Hobart Davies, Ph.D. (Major Professor/Advisor)
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