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THE ROLE OF PAIN-RELATED CATASTROPHIZING IN OUTCOMES AND RECOVERY FROM MINIMALLY INVASIVE AND SURGICAL PROCEDURES FOR TREATING TEMPOROMANDIBULAR DISORDERS

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A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University

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Abstract

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The current study examined the ability of pain-related catastrophizing to predict outcomes following non-surgical and surgical intervention for temporomandibular disorders (TMDs). The interpersonal context of pain-related catastrophizing, referred to as the communal coping model, was also examined to determine if patient perceptions of punishing and solicitous responses from significant others would moderate or mediate relations between pain catastrophizing and outcomes. The role of pain duration as a moderator of the relation between pain-related

catastrophizing and perceived significant other responding was also examined. A total of 94 patients were identified for which 65 had follow-up outcomes that could be examined. Patient follow-up data were obtained at approximately two to three weeks, two to three months, and six months post-intervention. Results showed that pain-related catastrophizing was predictive of greater pain severity at all three follow-up time points after controlling for baseline levels of pain severity, depressive symptoms, sleep disturbance, and pain duration. Pain-related catastrophizing was predictive of poorer range of motion (ROM) at the initial follow-up after controlling for baseline levels of ROM, gender, and form of intervention. Pain-related catastrophizing was not associated with ROM at the second and third post-intervention follow-ups. There was no interaction between pain-related catastrophizing and perceptions of either solicitous or punishing responses in predicting post-intervention pain severity or ROM and any time point. Perceptions of significant other responses also did not mediate the relation between pain-related catastrophizing and post-intervention outcomes at any time point. Additionally, the interaction between pain duration and pain-related catastrophizing in the prediction of post-intervention pain severity or ROM was not significant at any follow-up time point. The findings indicate that pain-related catastrophizing is an important predictor of pain severity following non-surgical and surgical interventions for TMDs both initially and in the long-term. Pain-related catastrophizing is related to ROM outcomes only in the short term. Perceptions of punishing and solicitous responses from significant others do not appear to play a role in these associations. The results suggest that patients with high levels of pre-intervention catastrophizing may benefit from adjunctive cognitive-behavioral intervention to attenuate post-intervention pain severity.

The Role of Pain-Related Catastrophizing in Outcomes and Recovery from Minimally Invasive and Surgical Procedures for Treating Temporomandibular Disorders

Chronic pain is a pervasive societal problem. It impacts the lives of more than 50 million people (Castillo, MacKenzie, Wegener, & Bosse, 2006). Pain-related symptoms account for nearly 80% of all physician visits (Gatchel & Turk, 1996). National surveys indicate that during a three-month period, nearly one-third of Americans experience some form of pain (Lethbridge-Cejku & Vickerie, 2005). It is estimated that the total annual cost of chronic pain is between $150 and $215 billion in the United States alone (National Research Council, 2001). Compared to the treatment costs of other chronic conditions, pain ranks as the most expensive condition to treat in the primary care setting, ahead of both heart disease and hypertension (Fishman, Von Korff, Lozano, & Hecht, 1997). Upwards of $80 billion are lost each year as a result of loss of work associated with pain-related disability.

The International Association for the Study of Pain (IASP) defines pain as " an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey, 1994). While this definition makes evident that pain provides an important protective and adaptive function, often prompting individuals to seek medical attention when necessary, it also emphasizes that pain is a subjective experience which, in part, owes to its emotional component. Although pain is often viewed as a symptom of an underlying disease process, prolonged pain can often lead to psychological distress and

depression in addition to functional disability (Gatchel, 2007; Turk & Melzack, 2011). When pain has reached a chronic and severe level it may be viewed as a disease in and of itself. As with adjustment to other chronic medical conditions, chronic pain has been associated with greater rates of psychopathology such as depression and anxiety, suggesting a heavy burden on the mental health and emotional wellbeing of those who suffer (Dersh, Polatin, & Gathcel, 2002). Broad psychological constructs such as anger (Burns, Quartana, & Bruehl, 2008) and demographic variables such as gender (Unruh, 1996), race and socioeconomic status (Day & Thorn, 2010; Fuentes, Hart-Johnson, & Green, 2007) have also been shown to be related to chronic pain.

The fact that psychosocial correlates of pain exist suggests that pain is not solely "skin deep." That is, pain is not a function of tissue damage alone. Indeed, the emotional experience of the IASP's definition should be interpreted as referring to not only an "outcome" of the pain experience, but also a factor that can influence the pain experience itself; the sensory aspects of pain can be greatly amplified or ameliorated by the beliefs and attitudes associated with pain (DeGood & Tait, 2001; Turk & Melzack, 2011). Several specific beliefs and attitudes have been identified which are related to increased pain severity. These have included beliefs about the cause of the pain (Spiegel et al., 1994), one's ability to control pain (Hanley, Raichle, Jensen, & Cardenas, 2008), fear of pain (Vlaeyen & Linton, 2006), viewing pain as an illness (Raichle, Hanley, Jensen, & Cardenas, 2007), and acceptance of pain (McCracken, 2005) to name a few.

Although many constructs have been examined, pain-related catastrophizing has seen the greatest amount of attention since the early 1990's (DeGood & Cook, 2011). Briefly, pain-related catastrophizing is a psychological variable characterized by hopelessness about the ability to alter pain, a ruminative style regarding the pain experience, and a tendency to magnify the

perceived damage associated with pain (Sullivan et al., 2001; Quartana, Campbell, & Edwards, 2009). Individuals who have a tendency towards greater levels of catastrophizing experience pain with greater intensity and emotional distress compared to their non-catastrophizing counterparts. This has been demonstrated in healthy participants undergoing laboratory pain procedures (Roelofs, Peters, van der Zijden, & Vlaeyen, 2004) as well as in numerous clinical medical conditions (Sullivan et al., 2001).

Temporomandibular disorders (TMDs) are one group of conditions that have seen a considerable amount of attention in the pain-related catastrophizing literature. TMDs are a heterogeneous collection of medical and dental conditions which can result in significant levels of oro-facial pain. The TMDs affect the temporomandibular joint (TMJ), the muscles of mastication and the surrounding tissues (National Institutes of Health [NIH], 1997). Moreover, there is no common etiology or biological explanation for these conditions because the symptoms that compose the TMD syndrome may result from such things as osteoarthritis, trauma (e.g., dislocation or fracture), developmental disorders, repetitive oral habits, as well as psychosocial factors such as stress or depression (Laskin, Green, & Hylander, 2006; Wright, 2005). As such, the symptoms associated with TMDs are similarly varied and may include earaches, headaches, limited jaw opening, and clicking and popping sounds in the jaw (NIH, 1997).

Despite the wide range of symptoms associated with TMDs, pain is often cited as the most common reason for seeking treatment (Wright, 2005). This pain can result in serious dysfunction and disability and it is estimated that roughly 10 million individuals in the United States suffer from TMDs (NIH, 1997). Approximately 75% of the entire population is thought to have at least one sign of dysfunction due to a TMD (i.e., joint noise, deviation on opening,

episodic lock) while 33% have at least one symptom of facial or jaw pain due to a TMD (McNeill, 1997). Median prevalence rates have been estimated at 29% for those with tenderness at the TMJ upon palpation (Macfarlane, Glenny & Worthington, 2001). Prevalence estimates of those in need of treatment range from 5% to 7%, with women seeking treatment more frequently than men at a rate of 4:1 to 9:1 (McNeill, 1997; Wright, 2005). The likelihood that symptoms will resolve is lower for women than men (Wanman, 1996). The obvious physical limitations associated with TMD pain give way to psychological disability as well. In patients with oro-facial pain, the prevalence of comorbid depression has been demonstrated to range anywhere from 35% in one dental clinic sample to 100% of patients with atypical facial pain in another study (Feinmann, 1983). The association between pain and psychological health is strong for this clinical condition. In one treatment outcome study for TMD pain, the only individuals to show marked psychological improvement were those who saw a reduction of pain intensity greater than 50% (Ohrbach & Dworkin, 1998). This included improvement on measures of depressive symptoms.

As with other chronic painful conditions, when the symptoms associated with TMDs persist individuals may seek out medical interventions characterized by varying degrees of invasiveness. They range from the less invasive use of dental splints to surgical interventions which vary in terms of invasiveness depending on the procedure employed. In addition to its unpleasant effects on the pain experience, greater pain-related catastrophizing has also been associated with poorer response to certain medical treatments, particularly surgical procedures, with patients continuing to experience significant levels of pain and dysfunction (Riddle, Wade, Jiranek, & Kong, 2010). Although longitudinal studies have been performed examining the impact of pain-related catastrophizing on surgical outcomes for many surgical procedures, none

have focused on similar effects in a TMD population (e.g., Lautenbacher et al., 2010). Knowledge regarding who will most benefit from various treatment options may aid in identifying who could benefit from adjunctive psychosocial interventions prior to, during or following TMD treatment procedures. This is of immense importance for a society whose demands already outstrip the financial and human resources of its health care system (Kaplan, 2007).

The current study attempts to fill this knowledge gap by examining pre-intervention levels of pain-related catastrophizing as a predictor of various TMD post-treatment outcomes. Additionally, variables relevant to the Communal Coping Model, a specific model proposed to account for catastrophizing's role in the pain experience were examined. This aspect of the investigation will focus on the perceived responses of significant others in the social environment. Additionally, given the shared variance between depressive symptoms and pain-related catastrophizing and the impact that sleep disruption has on the pain experience, these relevant aspects will also be examined.

In the following sections, a review of the literature will be presented wherein a history of psychology's role in understanding the pain experience will be detailed. This will include a description of the behavioral and cognitive-behavioral theories of chronic pain which will serve as a preface to the role that psychosocial variables, namely pain-related catastrophizing, play in the pain experience. A detailed review of catastrophizing will be presented and its role in surgical recovery and outcomes will be highlighted. Various treatment options for TMDs, including surgical procedures, will be described followed by a description of the Communal Coping Model. The role of responses perceived to be made by significant others to those suffering from pain conditions will be elucidated as an important process in the catastrophizing-

intervention outcomes link. The roles of sleep disturbance and depression will also be elaborated on because both are likely to play a role in response to treatment given their relation to chronic pain. Last, the hypotheses for the proposed study will be presented.

Review of the Literature

A History of Psychology's Role in Understanding Chronic Pain

Early Stages. Initial concepts of pain formulated by Descartes in the 17th century greatly influenced early research into the physiological underpinnings of pain and directed how those suffering from pain were treated for centuries (Melzack, 1993). He argued that those attempting to study and understand bodily processes should approach the body as if it were a machine, separate from the mind. Pain was no exception. His theory on the specificity of pain contended that an injury would activate pain receptors and fibers at the site of harm which would then send impulses along a direct route via a spinal pathway to a pain center in the brain. This tendency to view the body as machine-like, meant that the pain experience was believed to be in proportion to the level of pathology or tissue damage. Descartes' dualism, his separation of mind from body, led to a discounting of the contextual and psychological variables that play a role in how pain is experienced. Instead, research focused on locating specific pathways and a neurological pain center. Ultimately, this had the effect of perpetuating a disease-only model with regard to the treatment of pain, limiting the options available to chronic pain sufferers. The general approach in this instance involved attempting to identify underlying pathology associated with pain and treat the pain by attacking that underlying pathology, often through neurosurgical lesions (Fordyce, 1976).

The limitations of a strictly biomedical model of pain led to natural frustration among practitioners with patients who failed to respond to traditional medical interventions. It became increasingly apparent that the pain experience was a subjective one which would inevitably vary from one person to the next. This conclusion was strongly supported by the seminal work of Henry Beecher (Hardin, 2004). While working in Italy during World War II and at

Massachusetts General Hospital following the war, he noticed that soldiers, severely injured during combat, requested fewer analgesic medications than did patients recovering from surgical procedures. He reasoned that soldiers behaved as if they were attaching a very different meaning to the painful sensations (e.g., a ticket home), compared to post-surgical patients. To account for this and other similar anecdotal examples a more complex theory, the Gate-Control theory, was eventually introduced in 1965 (Melzack & Wall) and allowed for the inclusion of a dynamic brain which could modulate pain sensory inputs. The theory proposes that nerve impulses travel from the site of injury to a spinal gating mechanism in the dorsal horn of the spinal cord. This gating mechanism acts to modulate the pain signals based on the relative amount of input from inhibitory and excitatory fibers. The gating mechanism also receives nerve impulses which descend from the brain. Cognitive and emotional factors influence the modulating processes that occur in the gating mechanism to "open" the pain-gate allowing the perception of greater pain or "close" the pain-gate reducing an individual's overall experience of pain sensations. For example, ruminating about one's pain, feeling depressed, and stress all tend to lead to the pain gate being "open" and a greater intensity in pain sensations. By including the active brain in the schematic of pain, cognitive variables could now be considered in pain research and treatment. As Melzack writes, "Psychological factors, which were previously dismissed as 'reactions to pain' were now seen to be an integral part of pain processing…" (p. 619, 1993; Flor & Turk, 2006). Although the gate control model continues to be revised as research reveals more details about the nature of the nervous system, its heuristic value remains largely intact. Moreover, the contribution of incorporating psychosocial variables has revealed opportunities for researchers and clinicians to apply psychological principles to the understanding and treatment of pain (Melzack, 1996).

Fordyce's Operant Conceptualization of Chronic Pain. Throughout the 1960s and 1970s significant developments were also being made in treating pain in terms of operant behavior (McCracken & Vowles, 2006). During this time pain began to be conceptualized as a behavioral problem that was the result of environmental contingencies. Fordyce (1976) was one of the first to apply operant theories to managing chronic pain, publishing a seminal manual on its management using behavioral methods. Just as Descartes had heavily influenced the proceeding search for nerve pathways and brain based pain centers, Fordyce's contributions are still applied in treatment and research today. In many ways his theory was a step in the direction of understanding chronic pain in terms of social interactions.

Fordyce's (1976) first act was to conceptualize pain as a behavior. "Pain behaviors," as he labeled them, are observable responses to pain sensations that can often serve a communicative role, letting others in the social environment know that one is experiencing distress associated with these noxious sensations. These may come in the form of verbal behaviors such as groaning, indicating that one is in pain, or directly asking for assistance with difficult tasks. Other observable features of pain behaviors that do not involve verbal behavior include grimacing, moving gingerly or slowly, and taking pain medications. Pain behaviors encompass not only what the patient does, but those behaviors he or she may not display that are ordinarily part of the "well-behavior repertoire" (e.g., not cleaning; p. 44).

Like most behaviors, pain behaviors are capable of being reinforced or punished. That is, they will increase or decrease with frequency given the proper environmental contingencies. Fordyce (1976) contended that initial reactions to pain are respondent behaviors, elicited by antecedent stimuli (e.g., painful sensations) and typically reflexive in nature. Pain behaviors may go on to be learned, resulting in chronic pain, when those behaviors take on operant properties.

In other words, they become more influenced by the *consequences* of pain behavior via pain contingent reinforcement in the environment. Pain behaviors are most likely to be reinforced in an environment that involves others, particularly family members, who offer solicitous responses. Overt responding may come in the form of rubbing a spouse's back, providing pain medications, or simply telling the spouse in pain to avoid doing household chores. Even subtle environmental responses which provide indirect forms of attention, such as looking in the direction of the pain patient, may provide reinforcement for pain behaviors. Reinforcement of these behaviors can increase disability and suffering in the long-run by increasing attention to and perception of pain. Patients may even attempt to avoid this pain by engaging in socially approved pain behaviors, such as bed rest or "taking it easy." The ultimate result is physical deconditioning, making future physical activity all the more painful and debilitating.

The preceding examples demonstrate how pain behaviors might become positively reinforced by the social environment. Avoidance learning may also take place. That is, individuals suffering from pain may be negatively reinforced by engaging in behaviors which they use to avoid painful aversive consequences. Fordyce (1967) provides a classic example of how this might occur behaviorally. Early in the history of a pain problem a patient may develop a limp to temporarily alleviate or avoid pain. This may be an effective strategy initially, but over long periods of time will cause problems related to fatigue and restriction of activity. If the individual, realizing these consequences, attempts to walk naturally they are given an opportunity to find they no longer experience the pain and can return to normal functioning. However, if this distorted gait is observed by concerned family members who automatically provide solicitous responses, positive reinforcement of this avoidance behavior may develop. These solicitous responses can come in the form of social reinforcers, such as affection, or

achieve instrumental goals such as advice to take time out from household chores. In turn, the chronic pain sufferer may never reach the point where they engage in normal functioning and are able to experience those activities without the aversive pain. It is at this point in the history of a pain problem that the pain itself is not necessary for the pain behavior (e.g., limping) to continue.

Punishment of behaviors that involve healthy activity will also occur in the interpersonal arena (Fordyce, 1976). For instance, a spouse may admonish the husband or wife who engages in well behaviors by telling him or her to "take it easy" and even respond critically if he or she does not comply. Fordyce also points out that medical professionals who treat pain may inadvertently reinforce pain behaviors in similar ways by encouraging an excessive amount of bed rest, leading to disuse and ultimately exacerbating the pain condition. This prescription for bed rest is often accompanied by the suggestion that a patient let the amount of pain they are experiencing act as a guide for the level of activity in which they should engage. It is also possible that a caring physician provides positive reinforcement, such as nurturance and attention, which those pain patients with limited social supports may not have available to them.

By its very nature, the operant model of pain incorporates the social environment as an important variable in answering the question, "What is maintaining the pain behavior?" Fordyce's answer was simple: "…the actions families [and friends] take to help are often not only ineffective, but actually serve to make things worse" (p. 2, 1976). In treatment planning, Fordyce encourages a solid understanding of how the family system responds to a patient's pain behaviors. Understanding how the environment reinforces pain behaviors or, even worse, punishes alternative well behaviors, makes the importance of the interpersonal environment apparent. Just like other learned behaviors, Fordyce contended that the conditioning effects of reinforcement were temporary. This means that withholding reinforcement will ultimately reduce

the pain behaviors and indirectly result in a return to functioning and less pain. The examples provided thus far may give the impression that this is a relatively simple problem. However, the process of reinforcing those behaviors which exacerbate painful conditions is often a subtle one, so much so that family members, physicians, and those living with chronic pain are unaware that it is occurring. The introduction of a cognitive component to the understand of the chronic pain experience will now be introduced.

Cognitive Revolution and the Cognitive-Behavioral View of Pain. Fordyce's (1976) strict application of Skinnerian principles meant that tackling the "black box" of the mind would be a task left up to other pain theorists (Fordyce, 1993; Fordyce, Roberts, & Sternbach, 1985). The cognitive revolution in psychology that was initiated by Beck (1963) and Ellis (1962) went beyond observable behavior and addressed how interpretations and meanings of certain situations could impact mental health and psychopathology. This shift into second wave behavioral therapies eventually made its way to the conceptualization and treatment of pain. The first to systematically propose that cognitive variables might also play a role in pain were Meichenbaum and Turk (1976; Turk, Meichenbaum, & Genest, 1983). The cognitive-behavioral model views pain in terms of a complex system that is influenced by physiological pathology, one's cognitions regarding the painful experience, the behavioral responses to these cognitions and the pain itself, and the affective responses related to both (Keefe, 1996). From this theoretical premise, individuals are not passive recipients of information, but actively process information from the environment. The cognitions that result from this process can modulate mood, impact physiological processes and act as an impetus for behavior. Cognitions and behaviors may also influence the environment, or conversely, both may be influenced by all these factors. Treatment implications are also apparent in that people can learn to be more

adaptive in their ways of thinking, feeling and behaving if they become active agents in changing their cognitions and learning new ways to cope with pain behaviorally (Eimer & Freeman, 1998; Flor & Turk, 2006; Turk, Rudy, & Tollison, 1989).

From this model, a person's beliefs regarding his or her illness influences how that illness (e.g., pain) is experienced. When pain is interpreted as a threat, as signifying ongoing tissue damage or a progressive disease, more suffering is likely to result (Turk, 2003). In many ways these assumptions depart sharply from Fordyce's (1976) strict Skinnerian perspective, where chronic pain patients were not seen as information processors, but were viewed more as behavioral automatons; simply responding to environmental reinforcers (Fordyce et al., 1985).

Prior learning history is often incorporated into the cognitive model as the basis for beliefs, appraisals and expectations related to the pain experience (Turk & Flor, 2006). That is, chronic pain patients will filter information through organized representations of knowledge they have already acquired and respond within this frame of reference (Turk & Salovey, 1985). This means that the behavioral responses enacted by chronic pain sufferers will often be derived from idiosyncratic interpretations about their pain experience and the environment around them. These behaviors may be used to elicit certain responses from spouses, friends, and health professionals. Those social responses may go on to reinforce adaptive and/or maladaptive patterns of thought, behavior or emotion in much the same way described by Fordyce (1976) earlier. In other words, cognitions associated with the pain experience shape how patients present their symptoms to significant others. For instance, patients who interpret their pain as beyond their control may overtly communicate this to others in the social environment who in turn might in some way offer assistance, confirming impressions about the uncontrollability of the pain and reinforcing that communicative style (Turk & Flor, 2006). Presentations may also focus on the seriousness

of the pain as a threat or the severity of the pain, both of which can be reinforced through solicitous responses of others. As Fordyce (1967) suggested, the social environment may perpetuate the pain experience by providing secondary gains, such as excusing the patient from his or her usual responsibilities, expressing sympathy, and encouraging dependence and passivity. It is obvious that the cognitive-behavioral model has built upon Fordyce's operant conditioning model in considering how external reinforcement operates within the framework of information processing (Turk & Flor, 2006).

It becomes apparent when considering a cognitive-behavioral view of the origin and perpetuation of chronic pain, that certain patterns of thinking with regard to one's pain can be more detrimental to the pain experience compared to others. Although a "maladaptive" strategy may be detrimental, in some instances that same strategy may serve a valuable role in a patient's life. Although rest may be harmful for coping with low back pain over months or years, it may enhance one's sense of control over the pain by providing relief or allow the individual to avoid stressful hassles around the home or within a family (Haythornthwaire, 2006). With regard to ineffective coping, much of the literature suggests that the most important factor is the presence of pain-related catastrophizing (Flor & Turk, 2006).

Pain-Related Catastrophizing

Definition and Role in the Pain Experience. Pain-related catastrophizing is one powerful and important psychological variable in the study of pain that has seen greater attention in the last two decades. Albert Ellis, the developer of rational-emotive behavior therapy, first coined the term catastrophizing. According to Ellis, catastrophizing is a consequence of *musterbation*, or the act of making absolutist statements about the outcomes and events in one's own life. Catastrophizing is the resulting, negative emotional and cognitive response that occurs

when unrealistic *must* statements are not fulfilled. Catastrophizing was later refined in terms of anxiety, as an overestimate of the probability of the most unpleasant outcome occurring (Beck, Emery & Greenberg, 1985). This also entailed an exaggeration of the potential ramifications of that outcome. Beck et al. (1985) described the vulnerability of the catastrophizer: "He sees each mistake, each slip, as a potential slide into oblivion" (p.68). Pain-related catastrophizing has been conceptualized and defined similarly as a negative cognitive–affective response to anticipated or actual pain and is characterized by hopelessness about one's pain, an inability to inhibit pain related thoughts (i.e., rumination), and a magnification of the threat that painful sensations pose (Quartana et al., 2009; Sullivan et al., 2001). In this way, pain-related catastrophizing is made up of three separate cognitions, each part of one unified construct (Sullivan, Bishop, & Pivik, 1995).

The importance of pain-related catastrophizing is made apparent when considering its association with numerous insalubrious effects on the pain experience. It accounts for 7 to 31% of the variance in pain ratings (Sullivan et al., 2001). Pain-related catastrophizing has been associated with increased pain experience on a variety of pain rating measures and across a variety of clinical populations including those experiencing pain due to whiplash (Sullivan, Stanish, Sullivan, & Tripp, 2002), spinal cord injuries (Turner, Jensen, Warms, & Cardenas, 2002; Wollaars, Post, van Asbeck, & Brand, 2007), persistent shoulder pain (George & Hirsh, 2009), phantom limb pain (Hill, Niven, & Knussen, 1995; Richardson, Glenn, Horgan, & Nurmikko, 2007), rheumatoid arthritis (Keefe, Brown, Wallston, & Caldwell, 1989), and dental hygiene treatment (Sullivan & Neish, 1999). This association with pain ratings has also been demonstrated for non-clinical samples undergoing experimental pain procedures (Sullivan, Tripp, & Santor, 2000; Roelofs, Peters, van der Zijden, & Vlaeyen, 2004).

Pain-related catastrophizing has been associated with longer time to return to work following whiplash injuries (Adams, Ellis, Stanish & Sullivan, 2007), psychological distress and pain related disability following spinal cord injury (Turner, Jensen, Warms, & Cardenas, 2002; Wollaars, Post, van Asbeck, & Brand, 2007), and pain interference related to phantom limb pain (Hanley et al., 2004). Pain-related catastrophizing also predicts physical disability over several months in rheumatoid arthritis patients (Keefe, Brown, Wallston, & Caldwell, 1989). These associations have also been seen in a non-treatment seeking sample of young adults with headache pain (Buenaver, Edwards, Smith, Gramling, & Haythornthwaite, 2008). Catastrophizing was related to greater headache pain and pain related interference. Moreover, catastrophizing has been linked to greater pain ratings in non-clinical populations including athletes (Sullivan, Tripp, Rodgers, & Stanish, 2000) and dancers (Paparizos, Tripp, Sullivan, & Rubenstein, 2005).

Catastrophizing in TMDs and Dental Populations. The painful conditions of TMDs are no exception to these associations. Despite not being associated with objective clinical examination findings, catastrophizing has been positively correlated with muscle and joint palpation pain severity, interference due to pain, masticatory and non-masticatory limitations, limitations in unassisted jaw opening, health care visits and depression scores (Turner, Brister, Huggins, et al., 2005; (Turner, Dworkin, Mancl, Huggins, & Truelove, 2001), as well as clinical pain ratings on visual analogue scales (Castrillon et al., 2008).

In one study, catastrophizing had a moderating effect on disclosure of anxiety regarding a dental procedure in terms of level of pain and emotional distress (Sullivan & Neish, 1999). This was such that catastrophizing and non-catastrophizing individuals who were given a chance to disclose did not differ on outcome measures, whereas catastrophizers in the control condition

experienced significantly greater pain and distress compared to their non-catastrophizing counterparts. This suggests that catastrophizers react with greater levels of stress and anxiety when faced with the possibility of a potentially uncomfortable routine dental hygiene procedure. It is important to keep in mind that this reflects the influence of catastrophizing in a healthy college sample. It is likely that a chronic pain sample undergoing a more invasive procedure may have greater levels of stress or feelings of hopelessness regarding this procedure and in turn could mean more intense pain or emotional distress with regard to outcomes. Moreover, of those individuals receiving cognitive-behavioral therapy (CBT) for dealing with chronic pain due to TMDs, individuals with greater levels of catastrophizing prior to treatment experienced higher activity interference at one year follow-up (Turner, Holtzman, &Mancl, 2007).

Catastrophizing's Impact on Surgical and Medical Treatment Outcomes. Although a burgeoning area of the literature focuses on the negative surgical outcomes associated with pain-related catastrophizing, the notion of examining the psychological correlates of poor surgical outcomes is not new and many psychosocial predictors for poorer pain outcomes or reduced functional recovery exist (Rosenberger, Jokl, & Ickovics, 2006). For example, chronic post-surgical pain has been predicted by depression in total knee replacement patients (Brander et al., 2003; Harden et al., 2003) and individuals receiving lumbar discectomy (Schade et al., 1999). Anxiety, stress and HPA axis reactivity have been shown to be predictive of poorer outcomes in back surgery (Geiss et al., 2005; Graver et al., 1995). Where the medical model may view pain related outcomes as a function of the difficulty of the surgical procedure, there is a clear need for a more holistic view when predicting recovery. It has been determined that psychosocial predictors, such as expectations prior to oral surgery, significantly predict post-operative symptom severity over and above medical factors (McCarthy et al., 2003). This predictive ability

remains even after controlling for anxiety, a finding that has been demonstrated immediately after surgery and at follow-up. Expectations also predicted the number of days it took to return to work following surgical intervention.

Pavlin and colleagues (2005) examined pain-related catastrophizing prior to surgery to repair the anterior cruciate ligament (ACL), as a predictor of post-operative pain. Results revealed a positive relation between pain-related catastrophizing and maximum pain ratings provided while in immediate recovery and at a seven day follow-up. Moreover, in a similar sample of athletes who had received surgery to repair an ACL injury, catastrophizing was negatively associated with confidence in one's ability to return to sports and actual return to activity (Tripp, Stanish, Ebel-Lam, Brewer, & Birchard, 2007). Similar associations have been found in mixed surgical samples of adolescents. Pain-related catastrophizing has been positively associated with average and high pain ratings over a 48 hours post-surgical time frame, as well as physical recovery (Logan & Rose, 2005; Bennett-Branson & Craig, 1993). Similar relations have also been found for patients undergoing ear, nose and throat surgery (Sommer et al., 2009), surgery to repair chest malformations (Lautenbachet et al., 2009), and breast cancer surgery (Jacobsen and Butler, 1996). In the latter study pain-related catastrophizing was also positively associated with post-operative analgesic use, in addition to acute post-operative pain intensity. Catastrophizing has also been demonstrated as a significant predictor of levels of post-operative disability following carpel tunnel surgery (Calderón, Paiva, & Ring, 2008).

Studies of recovery from total knee arthroplasty (replacement; TKA) have also examined psychosocial correlates of outcome, including catastrophizing. One study found that the number of days it took recovering patients to bend their leg at a 90 degree angle was positively associated with pain-related catastrophizing (Kendell, Saxby, Farrow, & Naisby, 2001). This

study utilized the catastrophizing subscale of the Coping Strategies Questionnaire (CSQ), which only taps into the helplessness dimension of catastrophizing. Another measure, the Pain Catastrophizing Scale (PCS), has been also been used for measuring this variable and has conceptualized catastrophizing as also including the dimensions of magnification and rumination of pain, in addition to helplessness (Sullivan et al., 1995). The relation between catastrophizing and recovery from TKA has also been demonstrated with this measure. One study found that total catastrophizing scores on this measure, as well as the rumination subscale, were predictive of which individuals still had pain at 24 months post-surgery (Forsyth, Dunbar, Henniger, Sullivan & Gross, 2008). Total PCS scores have also been found to be predictive of acute post-operative pain following TKA (Roth, Tripp, Harrison, Sullivan, & Carson, 2007). The ability of catastrophizing to predict post-operative pain for TKA has been replicated as far as 12 months post-surgery (Edwards, Haythornthwaite, Smith, Klick, & Katz, 2009). Another study found that PCS total score and all subscale domains were positively associated with levels of post-surgical pain, while magnification and helplessness were both positively associated with post-surgical pain. Moreover, pain-related catastrophizing was the only psychological variable (compared to depressive symptoms and fear of movement) to contribute significant unique variance in the prediction of post-surgical pain after controlling for age, sex, and pre-surgical pain (Sullivan, et al., 2009). One study examined the predictive ability of several specific psychological disorders and multiple health related beliefs, finding that catastrophizing was the only consistent predictor of pain outcomes following TKA (Riddle, Wade, Jiranek, & Kong, 2010).

In the cases of both ACL repair and TKA, the surgical procedure is undergone for the sake of treating pain or out of medical necessity. An association between heightened pain-related catastrophizing and poorer acute post-operative pain has also been found in pain free samples

having major elective thoracic surgery (Weissman-Fogel et al., 2009). Granot and Ferber (2005) also investigated this relationship in a sample of patients undergoing elective abdominal surgery. They found that pre-operative pain-related catastrophizing was predictive of post-operative pain, even after controlling for state anxiety. Similar relationships have been found in samples of women electing to have cesarean sections instead of vaginal deliveries (Strulov, Zimmer, Granot, Tamir, Jakobi, & Lowenstein, 2007). Preoperative levels of catastrophizing on the PCS predicted post-cesarean pain on the first and second days following surgery. In a mixed sample of patients undergoing elective ambulatory surgery, greater PCS scores were associated with an elevated postoperative pain risk on the third postoperative day (Gramke et al., 2009).

Pain-related catastrophizing has also been linked to pain and recovery from non-surgical medical procedures. In a sample of women giving birth for the first time, catastrophizers anticipated and experienced more pain during childbirth, and also had poorer outcomes in physical recovery and returning to daily activities compared to non-catastrophizers (Flink, Mroczek, Sullivan, & Linton, 2009). In patients receiving physical therapy for neck pain higher initial ratings of catastrophizing were associated with greater odds of poor outcome on a self-reported global change in neck pain at 6 weeks post treatment. This association was even greater at 6-months post-treatment (Hill, Lewis, Sim, Hay, & Dziedzic, 2007). It has also been demonstrated that pain outcomes for minimally invasive procedures such as radiofrequency lesioning and injection treatments for lower back pain have are influenced by catastrophizing, with greater levels of catastrophizing being associated with poorer outcomes (Samwel, Slappendel, Crul, & Voerman, 2000; van Wijk et al, 2008).

Treatment Options for TMDs

Despite evidence of the impact of pain-related catastrophizing on surgical outcomes, this important chronic pain variable has not been explored in TMD surgical samples. Some of the earliest recorded attempts to treat TMDs date back to 5th century B.C. and were documented by Hippocrates (Laskin, 2007; McNeil, 1997). Over time diagnostic approaches and treatment options have been influenced by changing conceptualizations about the source of pathology; as the muscles of mastication became a recognized source of the problem (in addition to the TMJ itself), approaches to management also took on a more biopsychosocial approach (Laskin, 2007). Currently the treatments for TMDs are as numerous as the symptoms themselves. These may include psychological interventions, such as stress-management programs or CBT, self-management and oral habit awareness, stretching exercises and physical therapy, and pharmacological management, typically via NSAIDS or tricyclic anti-depressants (Wright, 2005). Dental appliances and surgical approaches are two forms of treatment most germane to the current study and will be discussed in more detail below.

Dental appliances referred to as occlusal splints represent a more conservative intervention option for treating TMDs (Clark & Minakuchi, 2006; Tecco, Caputi, Tete, Orsini, & Festa, 2006). Appliances such as these have been in use for over 100 years (Dylina, 2001). The splints are carefully molded to fit patients' teeth and may be designed to achieve one of several goals. In some cases the device may serve as a behavior-changing device, making the patient aware of any parafunctional behavior, such as jaw clenching and teeth grinding (Clark & Minakuchi, 2006). Other times occlusal splints may be designed to hold the mandible in a desirable position to aid in proper alignment of the TMJ anatomy. Although evidence exists for the efficacy of occlusal splints, for many patients suffering from a TMD occlusal splints alone

may not provide sufficient relief (Forsell, Kalso, Koskela et al., 1999; Keiner, Betancor, & Clark, 2001; Suvinen, Hanes, & Reader, 1997; Turp, Komine, & Hugger, 2004; Wright, 2005).

Another common intervention for TMDs is physical therapy (PT). PT interventions include a variety of techniques and home exercises geared towards strengthening masticatory and cervical spine muscles and increasing mobility. This may involve passive manipulation or manual "hands on" therapy techniques (McNeely, Olivo, & Magee, 2006). Several studies have demonstrated effectiveness of PT for patients with a TMD (Furto, Cleland, Whiteman, & Olson, 2006; Medicott & Harris, 2006; Tuncer, Ergun, Tuncer, & Karahan, 2013;).

Surgical options are also available in treating TMDs and have existed since the late 1880's (Annandale, 1887; Laskin, 2007). Open surgery, in which an incision is often made just in front of the ear (e.g., standard preauricular) to expose the joint, is a more invasive approach. Open joint surgery is not a specific procedure itself, but rather is a process used to apply treatment procedures in the event of internal disc derangement. In these instances a mechanical disturbance in the articular disc, the fibrocartilage that allows the bones of the TMJ to slide easily upon opening and closing, requires that the disc be repaired or completely removed (discectomy; Stegenga & de Bont, 2006).

Less invasive surgical approaches to treating internal disc derangement include arthrocentesis and arthroscopy. Arthrocentesis involves placing two hypodermic needles into the upper joint space in order to irrigate the joint, removing adhesions and increasing range of motion (Laskin, 2007). By increasing this range of motion the force within the joint can be redistributed, allowing healing to take place. Arthroscopy is slightly more involved. While it too involves flushing the joint and removal of adhesions, it also entails the use of a scope in order to view the TMJ and perform additional surgical procedures (White, 2005). Numerous techniques

are used in conjunction with arthroscopic procedures (Laskin, 2006). For the purposes of the current study, it is important to recognize that although arthroscopy is considered less invasive than open surgical procedures, it offers greater opportunities for complications than arthrocentesis.

Communal Coping Model of Pain-Related Catastrophizing

Origins in Gender Differences. There is a great deal of variability in the levels of pain intensity reported by men and women, with women generally reporting greater pain intensity. This is due to discrepant rates of specific medical problems, but has also been observed under experimental circumstances using healthy participants (Unruh, 1996). Females also display more behaviors associated with pain (Keefe, Lefebvre, et al., 2000; Sullivina, Tripp & Santor, 2000). Several studies have demonstrated that pain-related catastrophizing is one psychological variable which seems to account for these differences. In one study, women reported more intense pain and scored higher on catastrophizing than men during a cold pressor task, but when levels of catastrophizing were statistically controlled, gender no longer predicted pain intensity (Sullivan, Tripp, & Santor, 2000). In an undergraduate sample, women reported more pain-related catastrophizing, more painful symptoms and lower pain tolerance and pain thresholds than did men. In this study, pain-related catastrophizing was found to mediate the relation between gender and recent daily pain (Edwards, Haythornthwaite, Sullivan, & Fillingim, 2004). In other words, when pain-related catastrophizing was statistically controlled, gender no longer predicted levels of reported pain intensity. Catastrophizing has also been found to mediate the relations between gender and pain ratings during experimental pain procedures for varsity college athletes (Sullivan, Tripp, Rodgers, & Stanish, 2000). Similar results have been obtained in studies of chronic pain populations. In one study of osteoarthritis patients, women reported significantly

higher levels of pain and physical disability than did men, but pain-related catastrophizing mediated the relationship between gender and pain outcomes (Keefe, et al., 2000). Moreover, another study found that diffuse noxious inhibitory control (DNIC), a proxy for measuring endogenous pain inhibition which involves experiencing more than one noxious stimulus at once, mediated the relation between in-vivo catastrophizing and pain scores (Goodin et al., 2009). However, this mediation was moderated by gender, such that there was strong mediation within women, extending the aforementioned research that has shown the deleterious effects of catastrophizing to be related to gender. Although the cause of this gender difference is unknown, it has been suggested that this may arise from variable socialization processes between genders (Sullivan, Tripp & Santor, 2000). It has also been suggested that women are more likely attend to pain because it may interfere with gender specific social roles, such as household and parental responsibilities (Unruh, 1996). Moreover, increased pain behaviors have long been thought of as serving a communicative function to others as a way to solicit assistance or empathy (Fordyce, 1976). Literature on how this communal approach to coping involves pain and catastrophizing is discussed next.

The Communal Coping Model. Several different theoretical models and mechanisms of action have been introduced to explain the construct of catastrophizing and its negative impact on pain experiences (Sullivan et al., 2001). One such model is the communal coping model. The communal coping model was initially proposed by Sullivan and colleagues (2000) to account for gender differences in levels of pain intensity and pain related behaviors in response to experimental pain procedures. This model holds that individuals differ in the degree to which they seek out social or relational goals when attempting to cope with pain (Sullivan et al., 2001). For instance, those who catastrophize about their pain are more likely to engage in pain

behaviors for the sake of communicating distress to others in order to solicit support or empathy from those in their social environment, making catastrophizing a form of coping which is interpersonally distinct from other forms of dealing with pain.

For catastrophizers, the pain behaviors used to direct their social resources may be more exaggerated which in turn increases attention to pain, making the pain experience more unpleasant and ultimately increasing suffering. When those in the social environment respond helpfully, catastrophizing may be reinforced despite the painful consequences of these behaviors (Fordyce, 1976). Moreover, the goal of catastrophizing may not be the reduction of pain or disability per se, but eliciting the expression of sympathy or other such responses. From a social learning or socialization perspective, this theory is highlighted by the aforementioned findings that women, on average, are more likely to catastrophize than men. Indeed, several areas of the literature demonstrate this with findings that women use more social support, are more relationally oriented in their efforts to cope (González-Morales, Peiró, Rodríguez, & Greenglass, 2006; Porter, Marco, Schwartz, Neale, Shiffman, & Stone, 2000; Rosario, Shinn, Mørch, & Huckabee, 1988;) and are more likely to be emotionally expressive (Brody, 1985; Bronstein, Briones, Brooks, & Cowan, 1996). One study found that under experimental pain conditions, women were more likely to engage in interpersonal interactions with an empathetic confederate than men (Jackson, Iezzi, Chen, Ebnet, & Eglitis, 2005). Moreover, interacting with an empathetic confederate negatively impacted the ability to cope with pain, producing lower levels of tolerance and greater pain-related catastrophizing.

Catastrophizing and Fordyce's Operant Conceptualization. Catastrophizing is the only pain related cognition for which a specific theory has been generated to account for the interpersonal nature of pain. This theory has been fraught with its own problems because operant

conceptualizations have been applied to the model without appropriately incorporating communicative or support seeking behaviors (Thorn, Ward, Sullivan, & Boothby, 2003).

Pain behaviors were an early focus of psychologists interested in treating and researching chronic pain (Fordyce, 1976). Researchers and clinicians who conceptualized pain in operant terms were primarily concerned with how social responses might maintain those behaviors that exacerbate pain. Initial pain behaviors that are a response to acute pain are thought to be maintained by the social responses they elicit from others. Later theorists described pain behaviors as also serving a communicative function. These views are not mutually exclusive and both signify the importance of pain behaviors in understanding the interpersonal pain experience. Additionally, research indicates that pain behaviors are positively correlated with self-reported ratings of pain intensity, suggesting pain behaviors represent a useful complement to traditional outcome assessments (Labus, Keefe, & Jensen, 2003). Various approaches to measuring pain behaviors exist (Keefe & Smith, 2002). The earliest measures included semi-structured interviews with chronic pain patients and their spouses and sometimes required patients to complete diaries. The goal of these approaches are to help identify the target behaviors in need of change, as well as the antecedents that led to the target behavior, and whatever consequences might be maintaining the behavior (e.g., solicitous responses from a spouse). However, the recognition of the overt nature of these phenomena, which allowed for them to be observed directly, led to the use of observation methods in research beginning in the early 1980's (Keefe & Block, 1982).

Early studies by Ramano and colleagues (1991) used an observational system whereby chronic pain patients and their spouses would be instructed to engage in a series of household activities such as sweeping a floor or folding laundry. This provided a context in which the

patient and spouse could work together for approximately 20 minutes. Based on coded observation of verbal and non-verbal pain behavior, they found that partner solicitous behavior both preceeded and followed non-verbal pain behaviors (Romano et al., 1992) and that these types of responses were associated with greater frequency of reported pain and disability (Jensen, Turner, Romano, & Strom, 1995), just as Fordyce (1973) predicted. Similar findings have been recently obtained using comparable procedures in patients with fibromyalgia (Thieme, Spies, Sinha, Turk, & Flor, 2005) and musculoskeletal pain (Romano, Jensen, Turner, Good, & Hops, 2000). However, using the same methods in osteoarthritis patients, Smith and colleagues (2004) found that spouse facilitative behavior and the expression of approval, agreement, or support, preceded and followed patient pain behavior significantly more often than spouse solicitous behavior. This suggests that different clinical populations may interact with their social networks differently when managing their pain through interpersonal means.

Laboratory pain studies have also been used in chronic pain and non-clinical populations to demonstrate aspects of the communal coping model. In an undergraduate sample undergoing laboratory pain procedures, catastrophizing has been related to inferences of pain and higher observer ratings of pain behaviors (Sullivan, Martel, Tripp, Savard, & Crombez, 2006) as well as longer durations of pain behavior displays when in the presence of an observer (Sullivan, Adams, & Sullivan, 2004). Additionally, high catastrophizing participants are less likely to engage in cognitive coping strategies when in the presence of an observer, suggesting they are more likely to engage in strategies that communicate their pain as opposed to effectively minimize it. However, one study found that a sample of osteoarthritis patients self-reported that they “hold-back” from discussing pain-related concerns despite engaging in higher levels of catastrophizing (Porter, Keefe, Wellington, & de Williams, 2008).

One study found that school children (mean age = 11 years) undergoing a pressure induced laboratory pain experience showed more pain expression if their parent, rather than a stranger, was in the room (Vervoort, Goubert et al., 2008). This however was only the case for children who had less catastrophic thoughts. Those who engaged in frequent catastrophizing engaged in greater pain behaviors overall regardless of who was in the room. Other research has demonstrated that catastrophizing in children is related to parental perceptions of pain behaviors in children with and without chronic pain conditions (Vervoort, Craig et al., 2008).

The act of catastrophizing has also been found to be related to an increase in the receipt of instrumental support from the partners of patients with cancer pain, as well as increased pain behaviors and increased partner perception of pain (Keefe et al., 2003). Other studies have not been so promising, finding that the facial expression of pain was *not* more exaggerated for those higher in catastrophizing undergoing experimental pain procedures using thermal stimuli (Kanz, 2008). It would seem that the type of pain behavior being examined may make a difference when considering the pain related cognitive variables being studied (Sullivan, Thibault et al., 2006). Thubault and colleagues (2008) examined the distinction between communicative and protective pain behaviors. Communicative pain behaviors include facial expressions such as grimacing or wincing, and verbal or paraverbal pain expressions that serve to indicate one is in pain. Protective pain behaviors however serve to insulate the area of pain from further insult and include movements such as guarding, holding, or rubbing of the painful area of the body. Findings indicated that while pain-related catastrophizing was associated with both forms of pain behaviors during a lifting task, fear of pain was only related to protective pain behaviors.

Sullivan and others (2006) demonstrated that in a healthy population under experimental pain conditions, 15% of the variance in observer's inferences of participants' pain levels was

accounted for by pain catastrophizing. This is consistent with other literature which has highlighted a positive relation between levels of catastrophizing and duration and variation of displays of pain behavior (Keefe, Lefebvre, et al., 2000; Sullivan, Tripp, & Santor, 2000; Spinhoven et al., 2004).

Perceptions of Partner Responses and the Communal Coping Model. Laboratory studies that assess pain behaviors may lack some of the external validity required to make inferences about the "real world" interpersonal pain experience. Moreover, the work that goes into coding in this line of research can be cumbersome and it may be difficult to obtain participation from both spouses (Keefe & Smith, 2002). One alternative that has been used to better understand operant processes is to assess the perceptions of the chronic pain patient regarding the responses of significant others via self-report. Currently, the most widely used measure for investigating chronic pain in this way is the West Haven-Yale Multidimensional Pain Inventory (WHYMPI; Kerns, Turk, & Rudy, 1985). The WHYMPI is a 52-item instrument designed to measure the relevant psychosocial, cognitive and behavioral aspects of responses to pain. Of the WHYMPI's three sections, one is dedicated to assessing the perceptions of a significant others responses to the pain behaviors of the chronic pain sufferer. All items are rated on a 7 point Likert-type scale anchored by descriptors indicating the frequency of occurrence (0 = "Never," 6 = "Very often"). Three types of partner responses are assessed. These include responses which are punishing (e.g., "Expresses frustration at me"), solicitous (e.g., "Takes over my jobs or duties"), or distracting (e.g., "Talks to me about something else to take my mind off the pain"). The measure also assesses typical pain related outcomes, such as level of activity interference due to pain, pain severity and affective distress. While the WHYMPI does not contain an objective measure of pain behavior, perceived solicitous responses measured by the

WHYMPI from the patient perspective have been found to be related to increased pain severity, lower patient activity, pain related activity interference and pain behaviors themselves (Flor, Breitenstein, Birbaumer, & Fairst, 1995; Flor, Kerns, & Turk, 1987; Flor, Turk, & Rudy, 1989). Additionally, daily diary studies have found that perceived punishing responses are related to average daily increases in pain (Grant, Long & Willms, 2002).

Several studies have examined the communal coping model using this measure. The relation of catastrophizing to perceptions of solicitous and punishing responses has been a particular focus. Catastrophizing has been positively associated with perceptions of solicitous responses (Raichle et al., 2007; Boothby, Thorn, Overduin, Charles, 2004; Cano, 2004). Among persons with spinal cord injury, the association between pain-related catastrophizing and the severity of pain has been moderated by the perception of solicitous responses from a partner, such that a stronger association existed between catastrophizing and affective dimensions of pain for those who perceived greater solicitousness (Giardino, Jense, Turner, Ehde, & Cardenas, 2003). Moreover, positive associations between catastrophizing and sensory pain reports were stronger for those living with a spouse or partner than for individuals who lived with someone else. These relationships remained even after controlling for depressive symptoms. Others have found that these variables are important for the long-term adjustment to pain due to limb amputation. Hanley and colleagues (Hanley et al., 2004) found that greater catastrophizing, greater social support, and less solicitous responding at one month post amputation were associated with greater decreases in pain interference at one year follow-up. The authors interpreted this finding as indicating that high catastrophizers elicit more support from their social environment, allowing for better adjustment. Consistent with Fordyce's operant prediction, low solicitous responses may have allowed amputees to learn to become sufficient in

daily activities without the assistance of others.

In contrast, others have found that catastrophizing is related to the perception of punishing responses from significant others, but not solicitous responses (Boothby, Thorn, Overduin, & Ward, 2004). That is, mixed chronic pain patients higher in catastrophizing perceived their partners to respond more with irritation, frustration and anger. This would seem to suggest that catastrophizing and perceived solicitous responses are independently associated with pain outcomes, such as severity and disability. Moreover, it may indicate that supportive responses from significant others do not reinforce catastrophic thinking. In another study which examined similar relationships in a sample of chronic pain patients with various conditions, only punishing responses partially mediated the relationship between catastrophizing and pain related disability and depression (Buenaver, Edwards, & Haythornthwaite, 2007). Boothby and colleagues (2004) argue that findings which do not implicate solicitous responding throws into question the operant nature of the communal coping model. Some have suggested that these findings may be the result of the some patients' ability to "successfully" catastrophize (MacDonald, 2004). That is, sometimes those who initially catastrophize will ultimately end up reducing their catastrophic thinking as they will effectively elicit solicitous responses from their social environment. This explanation runs counter to the aforementioned propositions of Fordyce (1976). More recently, a study which utilized electronic momentary diary data from over 7100 diaries demonstrated that catastrophizing is significantly related to both solicitous and punishing responses of spouses, with stronger positive associations for solicitous responses (Sorbi et al., 2006).

Other research would seem to support a more complex interpretation of these divergent findings. In one study, pain duration played a moderating role in the relationship between

perceived social responses, such that catastrophizing was related to more perceived solicitous spouse responses at shorter pain durations, but was significantly related to less perceived social support in patients with longer durations of pain (Cano, 2004). Other studies have presented findings which make similar suggestions about the role of pain duration in the perception of spousal responses (Buenaver et al., 2007). One interpretation of these findings is that the perceptions of spousal responses were accurate and the result of a pain patient who "wears down" their social supports as their pain becomes more prolonged. Alternatively, this perception may represent a distortion resulting from having to cope with a painful condition for a longer period of time. Lackner and Gurtman (2004) report findings that would seem to support the former interpretation. They found a positive relation between self-reported interpersonal relationship problems and levels of pain-related catastrophizing in a sample of patients with irritable bowel syndrome. This relation remained even after controlling for general emotional distress. This also lends support to the notion that catastrophizing may flourish in an interpersonal context. Additionally, increases in satisfaction with spousal responses have been shown to attenuate the relations between catastrophizing, pain and negative affect (Holtzman & Delongis, 2007). Solicitous responding has been found to be associated with worsening on measures of depression and pain interference in individuals adjusting to lower-limb amputation, while social support has been improvement (Hanley et al., 2004).

Additional Psychosocial Variables Important in the Pain Experience

Depression. As has already been alluded to, depressive symptoms may arise as part of the pain experience, and conversely depressive symptoms may in some way alter the pain experience --- often with unfavorable outcomes. This comorbidity and a bidirectional positive relation between pain and depression is well documented. Depending on the patient population

and the nature of the pain disorder, the prevalence of comorbid depression can range from 13% to 56% (Bair, Robinson, Katon, & Kroenke, 2003; Banks & Kerns, 1996). The likelihood of having symptoms severe enough to warrant a diagnosis of depression may be three to five times more likely in individuals who suffer from persistent pain conditions compared to non-patient groups (Magni, Marchetti, Moreschi, & Merskey, 1993; Von Korff, Dworkin, Le Resche, & Kruger 1988). Particularly alarming is the relation chronic pain has with suicide. One longitudinal study reported that in chronic back pain sufferers the risk of suicide was nine times greater compared to a control group (Pettinen, 1995). Others have given lower, but nonetheless disquieting, estimates that suicidal ideation and intent are on average three times and twice as high, respectively, when compared to the general population (Tang &Crane, 2006). Edwards and colleagues (2006) found that in a mixed chronic pain sample 32% reported either passive of active suicidal ideation.

Individuals with TMDs are not excluded from the negative impact pain may have on mood. In one sample of patients seeking treatment from TMD symptoms lasting longer than 6 months, nearly 40% of patients scored in the range of severe depression on a self-report measure (Manfredini, Borella, Fevero Ferronato, & Guarda-Nardini, 2010). Gender differences in depressive symptoms for TMD samples also exist, with females tending to be more depressed than males (Auerbach, Laskin, Frantsve, & Orr, 2001). In one intervention study of those with TMDs, individuals who saw the greatest improvement in their pain intensity, also saw the greatest improvement on measures of psychological symptoms, including lower depression scores (Ohrbach & Dworkin, 1998).

Cognitive explanations for the relation between pain and depression have been offered suggesting that pain acts as a unique stressor and that over time pain related cognitions can

become distorted when an individual's coping resources are expended (Banks & Kerns, 1996). The meaning that chronic pain holds for an individual can often alter one's sense of self and erode self worth. This may activate other negative and distorted thoughts by way of schema activation resulting in generalized feelings of hopelessness that goes beyond the domain of pain (i.e., depressive symptoms; Beck, 1976). These distortions may shape the description and expression of pain, and ultimately influence the treatment that a chronic pain patient is willing to pursue (DeGood & Tait, 2001).

Pain-related catastrophizing is one of the more prominent examples of a distorted pain related cognition. Indeed, the relation between pain-related catastrophizing and depressives symptoms is strong, with zero order correlations of $r = 0.57$ in mixed pain samples and $r = 0.60$ in TMD samples (Geisser, Robinson, Keefe, & Weiner, 1994; Turner et al., 2001). At one point it was suggested that the strength of the relation between pain-related catastrophizing and depressive symptoms was indication of construct redundancy (Jensen, Turner, Romano, & Karoly, 1991; Sullivan & D'Eon, 1990), making pain-related catastrophizing simply a superfluous variable in attempting to understand the psychosocial nature of pain. However, several studies have demonstrated that pain-related catastrophizing continues to make a unique contribution in the prediction of chronic pain outcomes over and above depression scores. Catastrophizing has been found to mediate the relation between depressive symptoms and the affective and evaluative experience of pain (Geisser, et al., 1994). Additionally, pain-related catastrophizing continue to significantly predict suicidal ideation after controlling for depression scores (Edwards et al., 2006). While it is clear that depression and catastrophizing are two separate, but highly related constructs, it is common for researchers in the pain literature to statistically control for depressive symptoms when investigating pain-related catastrophizing's

impact on pain outcomes (Quartana et al., 2009).

Sleep Disturbances. An important part of the clinical picture for both pain and depression is the presence of sleep disturbance. The DSM-IV lists insomnia and hypersomnia as possible symptoms of major depressive disorder (American Psychiatric Association, 2000). While this would seem to suggest sleep disruptions arise as a function of depressive mood, the relation between sleep disturbances and depression is complex, often producing discrepant findings about the causal direction of the relation (Taylor, 2008). In looking at instances of comorbidity, one study indicated that initial symptoms of insomnia occur at the onset of the first depressive mood episode in 29% of cases (Ohayon and Roth, 2003). Yet, epidemiological studies have found that insomnia is a risk factor for developing depression (Buysse, Angst, Gamma, Ajdacic, Eich, & Rössler, 2008; Johnson, Roth, & Breslau, 2006; Taylor, Lichstein, & Durrence, 2003). Still others have found that insomnia may perpetuate or exacerbate depressive symptomatology that is already present (Pigeon et al., 2008).

The relation between sleep and pain can be similarly difficult to isolate given it is generally conceived of as bidirectional (Lautenbacher, Kundermann, & Krief, 2006; Roehrs & Roth, 2005; Smith & Haythornthwaite, 2004). Several studies have demonstrated that sleep deprivation has a hyperalgesic effect, meaning a greater sensitivity to pain, (Brand, Gerber, Pühse, Holsboer-Trachsler, 2010), which is often demonstrated in a reduction in pain thresholds in laboratory pain procedures (Onen, Alloui, Gross, Eschallier, & Dubray, 2001). In one study, Raymond and colleagues (2001) found that subjective sleep quality predicted next-day pain ratings of inpatients with burn injuries, with poorer sleep quality predicting higher pain ratings. The opposite however was not found, as initial pain ratings of pain intensity were not predictive of sleep the following night. Poor sleep has also been found to reduce cognitive ability to

manage pain (Kundermann, Krieg, Schreiber, & Lautenbacher, 2004). Several studies have also demonstrated that chronic low back pain can delay sleep onset compared to non-pain controls (Kelly, Blake, Power, O'Keeffe & Fullen, 2011). Still another study found that pre-sleep cognitive arousal, not pain severity, was the primary predictor of sleep quality in a sample of chronic pain patients (Smith et al., 2000).

Disruptions in sleep have been found in samples of patients who suffer with chronic orofacial pain, sometimes resulting in pain related awakening, the frequency of which has been found to be positively correlated with pain intensity (Benoliel, Birman, Eliav, & Sharav, 2009). TMDs are often a source of chronic facial pain which can alter sleep patterns (Brousseau, Manzini, Thie, & Lavigne, 2003; Riley et al., 2001). Yatani and colleagues (2002) compared the sleep quality of 137 TMD patients seeking care at a facial pain center. Median cutoffs were used to divide the sample into poor and good sleepers. Poor sleepers scored significantly higher on over half of the subscales which make up the Multi-Dimensional Pain Inventory. Hierarchical regression analyses revealed that poorer sleep quality was significantly predicted by greater pain intensity, more psychological distress, and less perceived life control. Another study found that in a sample of patients diagnosed with obstructive sleep apnea who required oral appliance therapy, 52% also presented with symptoms of TMDs, the most common symptom being pain (Cunali et al., 2009). In one study, sleep problems were found to partially mediate the association between arousal caused by psychological distress and pain (Davis et al., 2010). One longitudinal study of orofacial pain patients found some support for the hypothesis that pain may result in negative affect which goes on to reduce sleep quality in pain patients (Riley et al., 2001).

Statement of the Problem

In summary, previous research has established the importance of pain-related catastrophizing in both laboratory induced pain sensations, as well as in naturally occurring chronic pain conditions. Medical interventions, including surgical procedures that are employed with the primary goal of alleviating pain or correcting a problem in functioning are also impacted by pain-related catastrophizing. Although a deleterious effect on post-intervention functioning and pain related outcomes has been established for numerous medical conditions, its impact on similar interventions for TMDs, a painful and often debilitating group of disorders, has not been established. However, numerous studies have established that greater levels of pain-related catastrophizing have a negative impact on the pain-experience of those suffering from TMDs. Moreover, pain-related catastrophizing has been implicated as an important process variable in psychological interventions in TMD samples. By establishing the relevance of pain-related catastrophizing to the outcomes associated with medical interventions for TMDs, oral surgeons and other dental and medical professionals may be better informed about the circumstances under which psychological interventions can lead to improved outcomes.

Additionally, this study seeks to understand the role of pain-related catastrophizing in operant or communal coping terms by examining the responses of significant others as perceived by the patient. Although some work has been done regarding these associations in the chronic pain populations, it has yet to be incorporated into studies also examining outcomes following medical interventions for TMDs. As has been described, the responses of significant others may alter the pain experience in various ways due to their reinforcing or punishing nature (Romano et al., 1992). Additionally, perceived responses are related to levels of pain-related catastrophizing in chronic pain samples. As such, in the current study one would expect perceived spousal

responses to be related to post-operative pain and functional outcomes and that this relationship will interact with levels of catastrophizing. It should be noted that in prior studies which have examined how perceived solicitous and punishing responses impact the catastrophizing-pain relationship only the hopelessness dimension of catastrophizing construct was measured. The current study will employ a multi-dimensional measure to better account for the magnification of threat that pain poses and for the rumination about the pain experience.

The current study thus had two primary aims. The first was to evaluate whether the relation between pain-related catastrophizing and patient outcomes for TMD patients was consistent with findings for other medical conditions. Interventions consisted of either a non-surgical intervention, including occlusal splint delivery or referral to physical therapy, or one of the aforementioned surgical procedures commonly utilized in the treatment of TMDs. Patient data was obtained at baseline and three post-intervention time-points. The second aim was to further elucidate the role of social responding, and more specifically the role of patients' perception of significant other responses, in pain-related catastrophizing for TMD patients. To this end the relevance of a communal coping interpretation of the catastrophizing-pain outcome link as it relates to TMD interventions was explored and the following specific hypotheses were tested:

Hypothesis 1. Based on the consistent finding that pain-related catastrophizing is positively correlated with pain in TMD samples (Turner, Edwards, Slick & Katz, 2009) and is predictive of intervention outcomes for several other medical conditions (Edwards et al., 2009) it was expected that baseline levels of pain-related catastrophizing will predict post-intervention levels of pain. This relation will be positive, such that as catastrophizing increases levels of pain will

also increase. This relation was expected to be observed for all follow-up time points and to be demonstrated for patients receiving both surgical and non-surgical interventions.

Hypothesis 2. There will be a negative relationship between baseline levels of pain-related catastrophizing and post-intervention measures of functioning, such that as catastrophizing increases scores on functional range of motion outcome measures will decrease. This expectation is based on the impact that pain-related catastrophizing has on functioning and disability following surgical intervention in other medical conditions, in particular knee replacement (Calderon, Paiva, & Ring, 2008; Riddle, Wade, & Jiranek, 2010). Again, this relation was expected for all follow-up time points and will be demonstrated for both surgical and non-surgical patients.

Hypothesis 3. Consistent with findings in mixed-chronic pain samples (Boothby et al., 2004) and spinal cord injury patients (Giardino et al., 2010), it was expected that there would be an interaction between pain-related catastrophizing and perceptions of partner responding such that:

a. TMD patients who perceive greater levels of solicitous responses among their significant others and who are high in pain-related catastrophizing at baseline will have higher pain scores at follow-up time points (See Figure 1).
b. Patients who perceive greater levels of solicitous responses among their significant others and who are high in pain-related catastrophizing at baseline will experience poorer (less) range of motion at follow-up time points.
c. TMD patients who perceive greater levels of punishing responses among their significant others and who are high in pain-related catastrophizing at baseline will have higher pain scores at follow-up time points.

d. Patients who perceive greater levels of punishing responses among their significant others and who are high in pain-related catastrophizing at baseline will experience poorer (less) range of motion at follow-up time points.

e. Patients who perceive lower levels of solicitous and punishing responses among their significant others and who are low in pain-related catastrophizing at baseline will experience improved (greater) range of motion at follow-up time points.

f. Patients who perceive lower levels of solicitous and punishing responses among their significant others, but who remain high in pain-related catastrophizing at baseline will continue to have moderately high pain scores at follow-up time points. Additionally, poorer (less) range of motion at follow-up time points will also characterize this component of the interaction.

g. Patients who perceive lower levels of solicitous and punishing responses among their significant others and who are low in pain-related catastrophizing at baseline will have lower pain scores and will experience improved (greater) range of motion at follow-up time points.

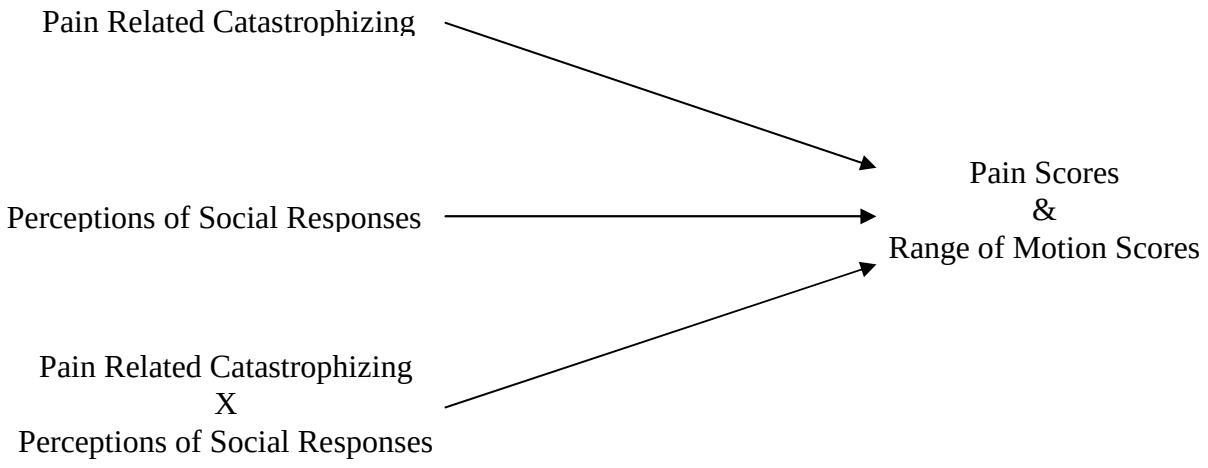

Figure 1. Hypothesis 3

Hypotehsis 4. Consistent with previous findings (Buenaver et al., 2007; Cano, 2004; Giardino et al., 2003) the relation between pain-related catastrophizing and perceived spousal responses was expected to be moderated by pain duration, such that high catastrophizing will be related to more perceived solicitous spouse responses in patients with shorter pain durations, but less perceived solicitousness at longer pain durations (See Figure 2).

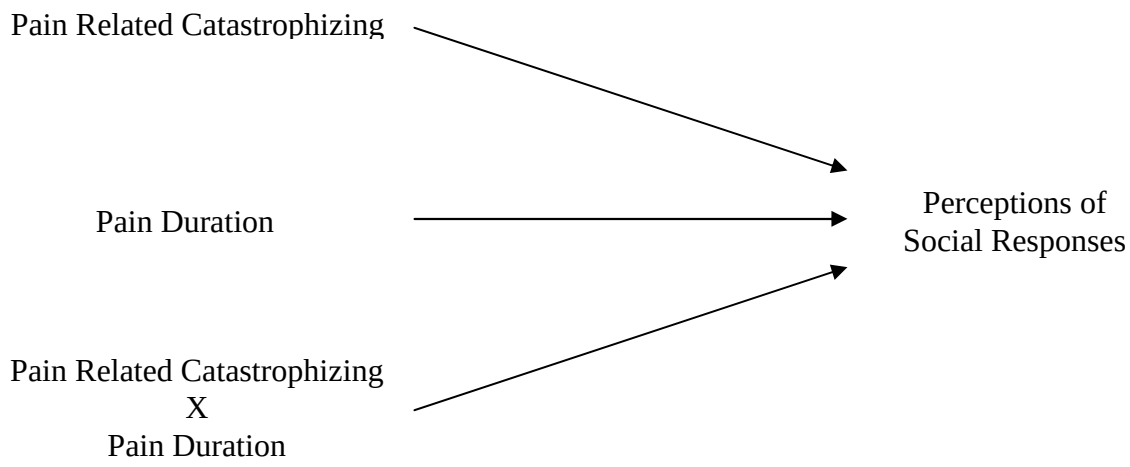

Figure 2. Hypothesis 4

Hypothesis 5. Given the longitudinal nature of the data, a mediational hypothesis regarding the relation between pain-related catastrophizing and outcomes will also be tested, with perceptions of social responses as the mediating variable. Based on a prior cross-sectional study with a mixed-chronic pain sample (Buenaver et al., 2007), it was anticipated that the perception of punishing responses will mediate this relation, but that solicitous responses will not (See Figure 3). Specifically:

a. The relation between pain-related catastrophizing at baseline and post-intervention levels of pain at the at second and third follow-up time points will be mediated by the patient's perception of significant other responding at the first follow-up time point. Both punishing and solicitous perceptions will be tested.

b. Similarly, the relation between pain-related catastrophizing at baseline and range of motion scores will also be mediated by the patient's perception of significant other responding at the first follow-up time point. Both punishing and solicitous perceptions will be tested.

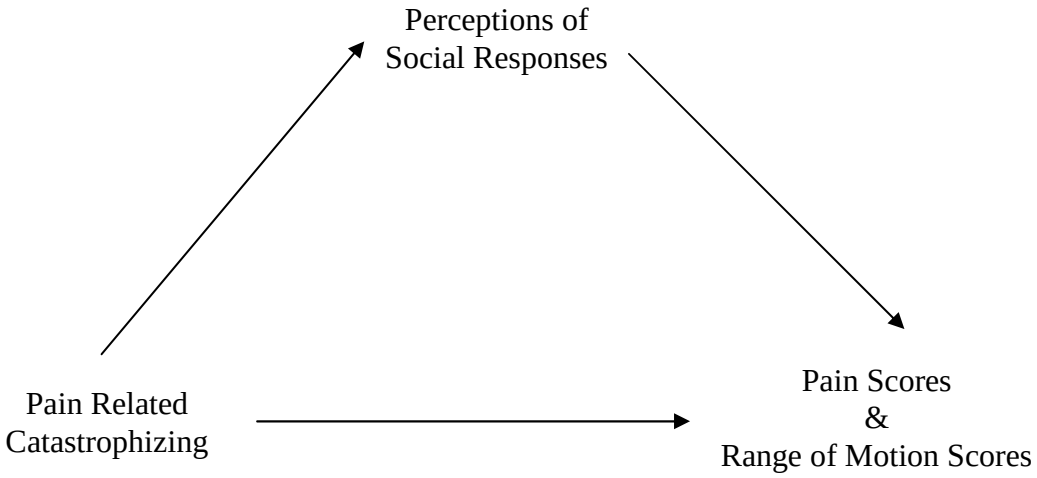

Figure 3. Hypothesis 5

Method

Participants

Data were collected on 94 adult patients suffering from pain and dysfunction due to temporomandibular disorders (TMDs). Patient data were derived primarily from two oral and maxillofacial surgery (OMFS) clinics located in the South-Atlantic region of the United States. Data from three of the study patients were collected from the East North Central region of the United States. The sample was primarily female (93%) with an average age of 41. All patients were formally diagnosed with TMDs by one of two clinic oral surgeons. Patients were included in the current study if they received one of five primary interventions including: physical therapy, occlusal splint, arthrocentesis, arthroscopy, or arthroplasty (open joint). Data sources included archived data found in electronic medical records, paper questionnaires and brief telephone interviews (See Procedure for a description of clinics). Consent was obtained from patients with ongoing treatment and for data obtained using brief phone interviews. Patients were not compensated for use of their data in the study and inclusion in the study was voluntary for patients with non-archived data. The dataset was de-identified in accordance with ethical standards for research established by the American Psychological Association and requirements set forth by Virginia Commonwealth University's Institutional Review Board.

Measures

Demographics. Demographic information was collected from patient charts for all participants for descriptive purposes. Specifically, information gathered included gender, race, age, education level, and marital status. Duration of pain, use of pain medications, and health histories were also collected from patient charts.

West Haven-Yale Multidimensional Pain Inventory (MPI). The MPI is a 52-item instrument designed to measure the relevant psychosocial, cognitive and behavioral aspects of responses to pain (Kerns, Turk, & Rudy, 1985). The MPI is divided into three sections consisting of 12 subscales in total. Section I (20 items) elicits information regarding (I.1) pain severity, (I.2) activity interference, (I.3) perceived life control, (I.4) perceived level of social support, and (I.5) affective distress. The second section, which is the most germane to the current study, contains 14 items and assesses perceived responses of significant others which are either (II.1) punishing, (II.2) solicitous, or (II.3) distracting. Section III (18 items) assesses the patients' levels of engagement in common daily activities such as (III.1) household chores, (III.2) outdoor work, (III.3) activities away from home, and (III.4) social activities. All items are rated on a 7 point Likert-type scale (see Appendix A). In sections II and III, items are anchored by descriptors indicating the frequency of an occurrence (0 = "Never," 6 = "Very often"). In section I these anchors vary by item (e.g., "Not at all irritable," 6 = "Extremely irritable"). In addition to providing quantitative measures of the aforementioned subscales, Turk and Rudy (1988) have introduced a classification system based on patterns of responding that can be used to predict how patients will respond to treatment. These include dysfunctional, interpersonally distressed, and adaptive coping subtypes. Support exists for the validity of these subtypes, with the dysfunctional subtype responding more poorly to interventions compared to the other two groups (Asmundson, Norton, & Alterdings, 1997).

Pain Catastrophizing Scale (PCS). The PCS was introduced to provide conceptual clarity to the construct of catastrophizing. At the time of its development, the primary components which characterized catastrophizing differed from one investigator to the next (Sullivan et al., 1995). The PCS is a 13 item self-report scale designed to measure various

dimensions of pain-related catastrophizing in both clinical and non-clinical populations. Each item is scored on a five point Likert-type scale (see Appendix B) with higher scores reflecting greater degrees of the construct being measured. This measure is composed of three subscales: rumination, helplessness, and magnification. In creating the PCS, Sullivan and colleagues (1995) sought to design a measure that would integrate the multiple cognitions previously associated with catastrophizing and suggested that catastrophizing was a unitary construct with multiple dimensions. Specifically, the helplessness subscale of the PCS, with the exception of one item, is composed of five of the six items that make up another commonly used catasrophizing measure, the catastrophizing subscale of the Coping Strategies Questionnaire (Rosentiel & Keefe, 1983; Turner & Aaron, 2001). In a sample of 438 undergraduate students, the rumination and helplessness subscales were found to be highly correlated (r = -.50), while rumination (r = .32) and helplessness (r = -.30) each showed moderate correlations with magnification. Each subscale demonstrated good internal consistencies with alpha coefficients of .87, .60 and .79 for rumination, magnification and helplessness, respectively (Sullivan et al., 1995). The authors of the PCS also examined the construct validity of the measure by comparing questionnaire responses to responses given during a structured interview. As expected those scoring high on the PCS reported a greater frequency of catastrophic thoughts. Additionally, Sullivan and colleagues (1995) reported a six-week test re-test correlation of r = .75, demonstrating the temporal stability of responses elicited by the measure. The factor structure of the PCS has also been replicated by others (Osman et al., 1997). Additionally, the PCS has been used with both TMDs (Campbell, Kronfli et al, 2010) and surgical samples (Kahn et al., 2011).

Short-Form of the McGill Pain Questionnaire (SF-MPQ). The SF-MPQ was developed for use in specific research and clinical settings when the time to obtain information is

limited (Melzack, 1987). It consists of 15 descriptors of pain, 11 of which assess sensory dimension of pain (e.g., throbbing, shooting) while 4 assess affective categories related to pain experience (e.g., sickening, fearful). Each descriptor is rated on a Likert-type scale in terms of intensity ranging from 0 (none) to 3 (severe) with reference to the past week (see Appendix C). The total of these scales form both 'Sensory Pain' (range = 0–33) and 'Affective Pain' scores (range = 0–12). The SF-MPQ also contains a 10-cm horitonzal visual analogue scale (VAS) of pain and Present Pain Intensity (PPI) index. The PPI asks patients to indicate their current level of pain intensity on a scale from 0 (no pain) to 5 (excruciating). This measure has been shown to have the sensitivity needed to detect change due to treatment at statistical levels.

Centers for Epidemiologic Studies Depression Scale (CES-D). The CES-D is a 20 item questionnaire designed to assess depressive symptoms including (Radloff, 1977). It consists of depressive symptoms (e.g., mood, somatic complaints, social interaction, and motor functioning) which are rated on a Likert-type scale ranging from 0 (“None or rarely [less than 1 day per week]”) to 3 (“Most or all the time [5–7 days per week]”), with four items worded in a positive direction (see Appendix D). Instuctions direct the patient to response to items with regard for the past 7 days (“During the past week.”). A total CES-D score can range from 0 to 60, with a higher score indicating the endorsement of greater depressive symptoms. Scores greater than 16 are considered in the “depressive” range. Among patients with chronic pain scores of 16 and 27 indicate minor and major depressive symptoms, respectively (Geisser, Roth, & Robinson, 1997). The internal consistency of the CES-D has been demonstrated, with Cronbach alphas ranging from .85 to .90 (Radloff, 1977). Additionally, other studies have demonstrated the validity of using the CES-D in patients with both physical disability (Berkman et al., 1986) and chronic pain (Turk & Okifuji, 1994).

Insomnia Severity Index (ISI). The ISI is a seven-item scale designed to measure the severity of sleep problems in patients over the prior two weeks with regard to the diagnostic criteria for primary insomnia found in the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV; American Psychiatric Association, 2000; Bastien, Vallieres, & Morin, 2001). Items are rated on a five point Likert-type scale (see Appendix E). The items evaluate (a) difficulty falling asleep, (b) difficulty staying asleep, (c) problems with waking up too early, (d) current sleep pattern satisfaction, (e) interference with daytime functioning, (f) if sleep problem is noticeable to significant others, and (g) level of distress caused by the sleep problem. Total scores range from 0 to 28, with higher scores indicating greater self-report of insomnia severity. Bastien and colleagues (2001) offer the following specific diagnostic cutoffs: 0 to 7 (no clinically significant insomnia); 8 to 14 (subclinical insomnia); 15 to 21 (moderate insomnia); and 22 or greater (severe insomnia). The reliability and validity of the ISI for the purpose of quantifying insomnia severity has been demonstrated and it has been cited as clinically useful as a screening device for insomnia, as well as an outcome measure in treatment research (Bastien et al., 2001).

Range of Motion (ROM). As part of routine clinical examination patients presenting to all clinics will obtain ROM data at baseline and both follow-up time points (Wright, 2005). This generally involves three measurements taken during the visit. The maximum vertical opening measurement is determined by the millimeter distance between the maxillary (top) central incisors and the mandibular (bottom) central incisors. Lateral movement is measured by placing a ruler over the maxillary (top) central incisors and having the patient move his or her mandible as far as possible in both directions. Finally, protrusive movements are measured by having the patient move their lower jaw as far forward as possible while the practitioner measures the

distance the mandibular (bottom) incisors are in front of the maxillary (top) incisors. For the purposes of this study, maximum vertical opening (MVO) was used as the primary ROM outcome.

Procedure

Data analysis for the current study was performed on data that were primarily collected during the ongoing treatment of patients being seen in two dental clinics in the Richmond, VA area and one clinic associated with the Ohio State University. Some data for third follow-up time points were obtained via telephone contact. Local clinic data included the patients referred to the private practice of Dr. Gregory Ness, D.D.S. and an associated residency training program, both of which are housed in the Oral and Maxillofacial Surgery Department at the VCU Medical Center. Patient data were also obtained from the private practice of Dr. Gregory Zoghby, D.D.S. at Commonwealth Oral & Facial Surgery (COFS). Finally, data were obtained from a separate private practice clinic conducted by Dr. Ness that is part of Ohio State University's Oral and Maxillofacial Surgery Department in the College of Dentistry. As part of the routine care process all patients referred to these clinics for treatment of temporomandibular disorders completed initial intake paper work. The aforementioned measures made up part of these packets. Each packet was accompanied by a letter (see Appendix F) introducing the patients to the practice and giving them notification of how the information they are supplying might be used. They were informed that this information is of clinical utility and that they may be asked for their permission to include it in a future study. They were also told that they are not obligated to have their information used in a study and could refuse this permission at any time.

For active patients, prior to having data included in the present study, dental treatment providers spoke with patients to obtain their permission. Patients received an IRB approved

consent form when first introduced to the clinic. They were told that the information they provide is useful to their care and should be completed, but that they were in no way obligated to give consent for its use in the current study. For patients who were inactive (had completed their questionnaire and treatment and were no longer seen in the clinic) a waiver of consent has been approved by the VCU IRB for the collection of retrospective medical record data at one site. For active patients from whom consent was obtained consent was documented in the study files of each clinical site.

Patients returned their intake forms from the mailed packets at their initial appointment. Patients who had not filled out these forms completed them in the waiting room prior to their initial appointment. At the initial consultation, in addition to the aforementioned forms, a routine dental examination was performed. Functional range of motion data was collected at this time and at each visit thereafter. Based on the initial consultation a decision was made as to what type of intervention would be in the best interest of the patient. Patients completed selected subscales of the MPI, SF-MPQ, PCS, CES-D and ISI (see Appendix A – D) and were assessed for functional limitations in ROM approximately one-three weeks post-intervention and then again at a two-three month follow-up appointment. At approximately six months post-intervention, follow-up data were also obtained for ROM and Pain Severity. A majority of patients provided Pain Severity ratings by phone, while ROM data was obtained during clinic visits. Patients received the same level of care regardless of whether or not their data was used in the study.

Table 1.
Timeline of Data Collection

Measure Name	Domain	Baseline	Follow-up 1	Follow-up 2	Follow-up 3
MPI	Pain Severity	X	X	X	X
	Interference	X			
	Support	X			
	Life-Control	X			
	Affective Distress	X			
	Punishing Responses	X	X	X	
	Solicitous Responses	X	X	X	
	Distracting Responses	X	X	X	
SF-MPQ		X	X	X	
CES-D	Depressive Symptoms	X	X	X	
PCS	PCS Total	X	X	X	
	PCS Magnification	X	X	X	
	PCS Rumination	X	X	X	
	PCS Helplessness	X	X	X	
ISI	Total ISI	X	X	X	
ROM	Range of Motion	X	X	X	X

Note. WHYMPI = West Haven-Yale Multidimensional Pain Inventory; MPQ-SF = McGill Pain Questionaire - Short Form; CES-D = Center for Epidemiologic Studies Depression Scale; PCS = Pain Catastrophizing Scale; ISI = Insomnia Severity Index; ROM MVO = Range of motion; X denotes data collected at indicated time point.

Design and Data Analysis

The current study employed a longitudinal, correlation design. Data were collected in four waves, including pre-intervention baseline data, and at three follow-up time points. Because data collection procedures involved multiple sites and various intervention procedures at those sites, there was a possibility for heterogeneity within the participant sample. Multilevel regression approaches were explored as a possible analytic approach to account for these differences and take advantage of the clustered nature of the data. Bickel (2007) recommends determining the need for multilevel regression by first calculating the intraclass correlation

coefficient (ICC). The ICC measured the degree of dependence among patients nested within intervention, and interventions nested within clinics. In other words, this provided an indication as to what proportion of variability in outcome data (i.e., pain and range of motion) was a function of intervention and site differences.

For descriptive purposes preliminary analyses were conducted for the various groups at the site and intervention level to determine differences among demographic variables. Significant differences among these variables were accounted for in multiple regression analyses by including those variables as covariates in the first step of the model. The baseline levels of primary outcome measures, ROM MVO scores and Pain Severity, were also included as covariates. Bivariate correlations among all predictors were examined for multicollinearity. Correlation analysis was used to identify potential covariates of dependent variables. Independent sample t-tests were conducted for those demographic variables which are dichotomous (e.g., gender) for this same purpose. Covariates were identified by a significant relation to the dependent variable at $p < .05$. Hierarchical multiple regression analyses were performed to test Hypothesis 1 and 2. For each hypothesis covariates were entered at the first step of the statistical model followed by main predictor variables. Regression was used to examine the relations between the various intervention procedures (e.g., splint delivery versus surgical procedure).

Hypothesis 3, that there would be an interaction effect between pain-related catastrophizing and perceptions of partner responding in predicting both Pain Severity and ROM MVO, was also tested using hierarchical regression with the cross-product interaction terms (e.g., pain-related catastrophizing x perception of solicitous responding) entered into the last step of the model. Any significant interactions were to be examined using the post-hoc analysis

procedures outlined by Holmbeck (2002). Hypothesis 4, that pain duration would moderate the relation between catastrophizing and perceptions of social responding was tested using this same analysis procedure.

Hypothesis 5, that perception of significant other responding would mediate the relation between pain-related catastrophizing and post-intervention outcomes of Pain Severity and ROM MVO, was tested using hierarchical multiple-regression and the criteria outlined by Baron and Kenny (1986). Accordingly, specific conditions were examined to determine if mediation was present. These are: (1) that the IV (pain-related catastrophizing) must be significantly related to the DV (pain and range of motion scores); (2) the IV (pain-related catastrophizing) must be significantly associated with the mediator (perceived punishing/solicitous responses); (3) the mediator (perceived punishing/solicitous responses) must be significantly related to the DV (pain and range of motion scores) after controlling for the IV (pain-related catastrophizing); and finally, there must be a significant reduction in the strength of the relationship between the IV (pain-related catastrophizing) and the DV (pain and range of motions scores) after controlling for the mediator (perceived punishing/solicitous). As is customary, covariates were entered in to the first step of the regression model prior to demonstrating the data meets the aforementioned conditions.

Results

Demographics and Descriptive Data

Data were obtained from 94 patients identified as having a TMD diagnosis and subsequently receiving surgical (e.g., arthroscopy) or non-surgical intervention (e.g, occlusal splint). As specified, patient data were obtained from three clinics. Patients were categorized based on primary TMD ICD-9 diagnostic codes into three groups: (1) those for whom the temporomandibular joint was the primary concern (ICD-9 code 524.xx), (2) those for whom myofascial pain was the primary diagnosis (ICD-9 code 729.xx), and (3) those who received a combination of these diagnoses. For the sake of parsimony, patients with data from OSU's maxillofacial and oral surgery clinic (N = 3) were combined with that of MCV's maxillofacial and oral surgery clinic because they had the same primary provider. Analyses where this is not the case are noted. All analyses were performed using SPSS version 21. Three patients were removed from the data set because they received treatment from both MCV and Commonwealth Oral and Facial Surgery (COFS). The three patients from OSU's clinic were all female aged 50, 40, and 21years and pain durations of 12, 360, and 18 months, respectively. Two of the three patients were married. Two patients received primary diagnoses of the temporomandibular joint and subsequently received arthrocentesis and arthroscopy, whereas one received a primary diagnosis of myofascial pain, the intervention for which was occlusal splint. Demographic data (Table 2) for MCV (without the aforementioned OSU patients) and COFS clinics are presented below, as well as descriptive data on pain duration, TMD diagnoses and primary interventions (Table 3).

Table 2

Demographic Characteristics of the Study Sample

	MCV Sample				COMS Sample				Total			
	M	*SD*	n	%	*M*	*SD*	n	%	*M*	*SD*	n	%
Age	41.4	16.2			41.7	16.7			41.2	16.3		
Gender												
Male			4	6.1			3	12.5			7	7.5
Female			62	93.9			21	87.5			86	92.5
Race												
Caucasian			54	81.8			18	78.3			72	85.7
African American			8	12.1			3	13			11	13.1
Multi/Bi-racial			0	0			1	4.3			1	1.2
Marital Status												
Single			20	32.3			6	30			27	31.8
Divorced			6	9.7			1	5			7	8.2
Married			34	54.8			13	65			49	57.6
Widowed			2	3.2							2	2.4
Education Level												
Some High School			2	3.3			9	40.9			2	2.4
High School Graduate			32	52.5			11	50			41	49.4
College Graduate			22	36.1			2	9.1			33	39.8
Graduate Studies			5	8.2			0	0			7	8.4

Note. Total study $N = 94$. *n*'s vary within table.

Table 3

Diagnoses and Interventions

	MCV Sample				COMS Sample				Total			
	M	*SD*	n	%	*M*	*SD*	n	%	*M*	*SD*	n	%
Pain Duration (months)	71.8	94.3			82.1	101.5			74.5	95.7		
Diagnoses												
Joint Diagnosis			32	49.2			9	36			43	46.2
Myofascial Pain Diagnosis			15	23.1			13	52			29	31.2
Combination Diagnosis			18	27.7			3	12			21	22.6
Primary Intervention												
Physical Therapy			4	6.1			9	36			13	13.8
Occlusal Splint			21	31.8			7	28			29	30.9
Arthrocentesis			21	31.8			2	8			24	25.5
Arthroscopy			8	12.1			2	8			11	11.7
Arthroplasty-Open Joint			12	18.2			5	20			17	18.1

Note. Total study $N = 94$. *n*'s vary within table.

Of the 94 patients who received some form of intervention, follow-up data were obtained on 65 for pain severity at a minimum of one time point. The average interval (days) between intervention and follow-up time point 1 (M = 16.28, SD = 9.69), time point 2 (M = 71.11, SD = 9.69) and time point 3 (M = 213.21, SD = 64.74) did not differ significantly by intervention. There was no difference across the various interventions for average days to follow-up at time point 1, F (4,59) = 1.889, follow-up time point 2, F (4,59) = .483, or follow-up time point 3, F (4,33) = 1.299.

Patients with follow-up data did not differ from those with only baseline data in terms of pain duration (t(84) = -1.46), baseline levels of pain related catastrophizing (PCS Total Score, (t(81) = -.868), depressive symptoms (CES-D Score, t(77) = -.880), pain severity (t(80) = -.032), or sleep disturbance (ISI, t(77) = -.519). However, patients did differ in terms of range of motion as measured by maximum vertical opening (ROM MVO), with returning patients characterized by greater impairment in ROM (M = 31.18, SD = 11.39) compared with non-returning patients (M = 36.48, SD = 8.72), t(82) = 2.140, p = .035.This suggests that patients with less functional impairment were less likely to return for follow-up visits. Inspection of frequency data for TMD diagnosis and intervention revealed that only 15.6% of patients with a primary diagnosis of myofascial pain provided follow-up data. A greater proportion of non-surgical intervention, namely occlusal splint delivery and referral to PT, also characterizes this subset of the data, as these interventions make up 75.9% of patients without pain severity data over the three follow-up time points. A chi square test was performed to examine the relation between surgical versus non-surgical intervention and presence of follow-up appointments. Results indicate that patients receiving non-surgical intervention were significantly less likely to have follow-up visits, X^2 (1) = 11.31, p<.01. This was unrelated to the source of patient data as patients without follow-up

appointments were not more likely to be treated by MCV than COFS, X^2 (1) = .108. MCV patients and OMFS patients also did not differ significantly in terms of pain duration (t(58) = .075), or baseline levels of ROM MVO (t(55) = .316), pain severity (t(58) = .341), pain catastrophizing (t(58) = 1.1185), depressive symptoms (t(57) = -.342), or sleep disturbance (t(56) = -.892).

Interventions were also coded for level of invasiveness with occlusal splint representing the least invasive intervention followed by PT, arthrocentesis, arthroscopy, and arthroplasty being the most invasive. Level of invasiveness was not correlated with baseline levels of pain catastrophizing, sleep disturbance, depressive symptoms, pain duration or pain severity at any time point. Consistent with the aforementioned findings regarding intervention, ROM MVO and likelihood of follow-up, invasiveness was correlated with ROM MVO at baseline (r(56) = -.59, p< 0.01) and at follow-up time point 1 (r(50) = -.61, p< 0.01). Surgical interventions are more invasive and likely to be used for patients with a joint diagnosis while less invasive interventions (e.g., occlusal splint) are used to treat myofascial pain diagnoses. A major characteristic of those with a primary joint diagnosis was greater limitations in ROM MVO compared to those with muscular pain. As such, this relationship between level of invasiveness and ROM MVO during a diagnostic period and initial follow-up are expected.

Of the 65 patients with follow-up pain severity data, 48 were treated at MCV, 14 at COFS, and 3 at OSU. For this subset of patients the frequency of TMD diagnoses and primary interventions are presented in Table 4 below. Additionally, pain medications prescribed for or being used by patients, as documented in the medical chart, are presented. Overall, 12 patients were prescribed opioid medication to control their pain beyond routine care. Of those, two patients were prescribed tricyclic anti-depressants and were using NSAIDs to manage their pain,

while one other patient was documented to also be using NSAIDS with gabapentin. Three additional patients were documented to be using just one other medication in addition to opioids; two were NSAIDs and one was gabapentin. Patients prescribed opioid medications did not significantly differ from patients using other medications or patients not using medications in terms of pain duration ($t(58) = 1.282$), baseline PCS scores ($t(58) = .292$), pain severity at baseline or any follow-up time points ($t(58) = .305$;$t(50) = .152$;$t(43) = 1.298$; $t(25) = 1.138$), or ROM for any study time point ($t(55) = -.086$; $t(49) = .388$; $t(48) = -.151$; $t(14) = -.934$).

Table 4
Diagnoses, Interventions, and Pain Medication Use in Patients with Follow-Up Data

	MCV Sample*		COMS Sample		Total	
	n	%	n	%	n	%
Diagnoses						
Joint Diagnosis	29	58	8	57.1	37	57.8
Myofascial Pain Diagnosis	6	12	4	28.6	10	15.6
Combination Diagnosis	15	30	2	14.3	17	26.6
Primary Intervention						
Physical Therapy	2	3.9	2	14.3	4	6.2
Occlusal Splint	13	25.5	3	21.4	16	24.6
Arthrocentesis	17	33.3	2	14.3	19	29.2
Arthroscopy	8	15.7	2	14.3	10	15.4
Arthroplasty-Open Joint	11	21.6	5	35.7	16	24.6
Concurrent Pain Medication Use						
NSAID/Tylenol	14	45.2	7	87.5	21	53.8
Vicodin	8	25.8	0	0	8	20.5
Gabapentin	4	12.9	0	0	4	10.3
Percocet	1	3.5	1	12.5	2	5.1
Tramadol	2	6.5	0	0	2	5.1
TCA	2	6.5	0	0	2	5.1

Note. N = 65. *MCV Sample includes n = 3 OSU patients.

Internal consistency estimates were computed for the measures of pain-related catastrophizing, depressive symptoms, sleep disturbance, pain severity, and perceptions of social responding. Alphas for pre-intervention scores on the PCS, CES-D and ISI were .91, .90, .93, respectively. Internal consistency estimates for the WHYMPI subscale for perceptions of Punishing Responses and Solicitous Responses ranged from .74 to .82 and .84 to .86 across pre-intervention and initial follow-up time points, respectively. Cronbach alphas for WHYMPI Pain Severity across four times points ranged from .81 to .91.

Pearson product moment correlations were conducted to partially test the first hypothesis that pre-intervention levels of pain related catastrophizing will predict follow-up measures of pain severity and range of motion scores. Prior to analyses, variables were examined for normality and were found to not be skewed. Scatter plots were examined visually and assumptions of linearity and homoscedasticity were met. Bivariate correlations between predictor and outcome variables across study time points, as well as means and standard deviations are presented in Tables 5 and 6 below. Compared to similar research using mixed chronic pain samples, the current sample reported lower baseline levels of pain severity (M = 3.57 versus 4.6) and pain related catastrophizing (M =19.13 versus 33.8; Buenaver et al., 2007). However, levels of pain severity (M = 3.57 versus 3.33; Turner et al., 2001), pain related catastrophizing (M =19.13 versus 15.1; Buenaver et al., 2012) and depressive symptoms (M = 15.76 versus 12.17; Litt, Shafer, Ibanez, Kreutzer, & Tawfik-Yonkers, 2009) were similar to those of other TMD study samples. Additionally, mean baseline levels of sleep disturbance were comparable to that of other TMD samples (10.26 versus 9.3; Quartana et al., 2010). As hypothesized there was a significant positive relation between Total PCS Score and WHYMPI Pain Severity at all follow-up time points, ranging from r = .39 to .51, as well as a significant

negative correlation between Total PCS Score and ROM MVO, but only for initial follow-up, r = -.34, $p < 0.05$.

Table 5

Means, Standard Deviations, and Correlations between Baseline Measures

	M	*SD*	1	2	3	4	5	6	7
1. PCS Total - BL	19.13	11.37	1						
2. Punishing - BL	1.02	1.02	.36 (57)**	1					
3. Solicitous - BL	3.19	1.65	.18 (58)	-.13 (57)	1				
4. CES-D - BL	15.76	11.12	.47 (59)**	.38 (56)**	.03 (57)	1			
5. ISI - BL	10.26	7.56	.42 (58)**	.51 (55)**	-.05 (56)	.59 (57)**	1		
6. Severity - BL	3.57	1.44	.51 (60)**	.21 (57)	.23 (58)	.26 (59)*	.092 (58)	1	
7. ROM - BL	31.18	11.39	-.21 (53)	-.07 (50)	-.10 (51)	-.20 (52)	-.18 (52)	-.29 (53)	1

Note. N's for each correlation presented in ( ).

PCS = Pain Catastrophizing Scale; BL = Baseline; Punishing = WHYMPI Punishing Responses Subscale; Solicitous = WHYMPI Solicitous Responses Subscale; CES-D = Centers for Epidemiologic Studies Depression Scale; ISI = Insomnia Severity Index; Severity = WHYMPI Pain Severity Subscale; ROM = Range of Motion Maximum Vertical Opening.
* $p < 0.05$, two tailed, ** $p < 0.01$, two tailed.

Table 6

Means, Standard Deviations, and Correlations between Baseline Measures and Follow-up Measures

	M	*SD*	6	7	8	9	10	11
1. PCS Total - BL	19.13	11.37	.50 (49)**	-.34 (47)*	.39 (41)*	-.15 (46)	.49 (26)*	.11 (15)
2. Punishing - BL	1.02	1.02	.27 (48)	-.25 (48)	.20 (38)	.16 (43)	.56 (25)**	.19 (15)
3. Solicitous - BL	3.19	1.65	.17 (48)	-.05 (46)	.23 (39)	-.09 (44)	.08 (26)	-.07 (15)
4. CES-D - BL	15.76	11.12	.32 (49)*	-.03 (47)	-.02 (40)	.02 (45)	.43 (26)*	-.06 (15)
5. ISI - BL	10.26	7.56	.25 (48)	-.03 (45)	-.06 (40)	.18 (44)	.43 (26)*	-.11 (14)
6. Severity - F1	2.19	1.22	1					
7. ROM - F1	33.35	9.43	-.29 (45)	1				
8. Severity- F2	1.84	1.49	.54 (37)**	-.34 (36)*	1			
9. ROM - F2	37.16	8.43	-.25 (42)	.55 (44)**	-.46 (41)**	1		
10. Severity - F3	1.77	8.44	.26 (20)	-.22 (20)	.53 (21)*	-.09 (41)*	1	
11. ROM - F3	38.25	11.70	-.13 (13)	.62 (15)*	-.10 (13)	.86 (15)**	.30 (11)	1

Note. Correlations among baseline measures presented in Table 5. N's for each correlation presented in ( ).

PCS = Pain Catastrophizing Scale; BL = Baseline; Punishing = WHYMPI Punishing Responses Subscale; Solicitous = WHYMPI Solicitous Responses Subscale; CES-D = Centers for Epidemiologic Studies Depression Scale; ISI = Insomnia Severity Index; Severity = WHYMPI Pain Severity Subscale; ROM = Range of Motion Maximum Vertical Opening; F1 - F3 = Follow-up timepoint 1 - 3.
* $p < 0.05$, two tailed, ** $p < 0.01$, two tailed.

Differences on baseline and outcome measures were examined for the various diagnostic groups using one-way analyses of variance. Diagnostic categories (see Table 4) did not significantly differ in pain duration, F (2,56) = .457, baseline measures of depressive symptoms, F (2,55) = .223, pain related catastrophizing, F (2,56) = .495, or sleep disturbance, F (2,54) = .768. Pain severity did not significantly differ by diagnostic category at any study time-points. However, RMO MVO significantly varied by diagnostic category at pre-intervention, F (2,53) = 4.705, p< .05.Post-hoc examination of these differences was conducted using several procedures including Bonferonni, Tukey, and Hochberg's GT2 and planned contrasts examining this revealed significantly greater ROM MVO for those with primary myofascial pain diagnoses (M = 40.78, SD = 8.18) compared to those with primary joint diagnoses (M = 28.41, SD = 11.33), t(53) = -3.057, p = < .01.

Differences on primary study measures were also examined for the various intervention groups using one-way analyses of variance. Patients receiving the various interventions (see Table 4) did not significantly differ in pain duration, F (4,55) = .290, baseline measures of depressive symptoms, F (4,54) = 1.020, pain related catastrophizing, F (4,55) = .023, or sleep disturbance, F (4,53) = .508. Intervention groups also did not differ in terms of pain severity at any study time-points, but differed in RMO MVO scores pre-intervention, F (4,52) = 7.694, p< .001, and at initial (time 1) follow-up, F = (4, 46) = 11.264, p< .001. Differences were not found in ROM MOV for the final two follow-up time points. Post-hoc examination of differences pre-intervention and at follow-up time point 1were conducted using Bonferonni, Tukey and Hochberg's GT2. This revealed significant differences between Occlusal Splint and Arthroscopy as well as Arthroplasty interventions for pre intervention ROM MVO and differences between Occlusal Splint and Arthroplasty initial follow-up time point 1. Contrasts examining differences

between Occlusal Splint RMO MVO and a Arthroscopy/Arthroplasty grouping indicated significant differences at pre-intervention $t(52) = 5.101, p < .001$, and initial follow-up time point 1, $t(46) = 4.1029$, $p < .001$. ROM MVO was also significantly different for Arthrocentesis compared to Arthroplasty at pre-intervention, $t(52) = 3.242$, $p < .01$, and follow-up time point 1, $t(46) =$, $p < .01$.

Exploring the Nested Nature of the Data

After cleaning the raw score data and computing scale scores for the predictors and dependent measures, the database was restructured to examine the appropriateness of performing multilevel analyses given the nested nature of the data. As suggested by many (Bickel, 2007; Cohen, Cohen, Aiken & West, 2003; Heck, Thomas, &Tabata, 2010) a null multilevel model was first specified to derive an intraclass correlation (ICC). The null model is a model that does not feature predictors. The goal of this is to partition the variance in the outcome into within and between group components. The ICC derived from this model is an indication of how correlated observations are within clusters or "nests." That is, it indicates how much of the variance in data at the individual level is non-unique and can be attributed to the group variance. The Wald *Z* also provides a test of statistical significance for differences between intercepts across different interventions and clinics. Convention suggests that if ICC is above 0.05 and the Wald *Z* is significant then multilevel analyses may be warranted. Initial multilevel analyses were conducted with individual patients (level1) nested within clinics (level 3) and interventions (level 2) nested within individual patients at each clinic. SPSS produced an ICC of < .0000, but did not produce a *Wald Z* test for variance at the clinic level for both Pain Severity and ROM MVO. Others have reported that a *Wald* Z will not be computed when the variance for a random component of a multilevel analysis is negligible (Biderman, 2012; Heck et al., 2010). They suggest removing the

term, in this case clinic assignment, and treating it as a fixed effect. For a second set of analyses, individual patients (Level 1) were nested within interventions (Level 2). In this case, the variance within individuals on measures of pain severity and ROM, were examined in the context of within group variations that might occur for the various intervention approaches (e.g., PT, splint, surgical intervention). Dependent variables of pain severity and ROM scores were specified in the null model. The proportion of variance that was between intervention groups for WHYMPI Pain Severity was low and non-significant (ICC = 0.019; *Wald Z* = 0.686). Similarly ROM MVO scores were non-significant (*Wald Z* = 1.309) with and an ICC of .0246.

Identifying Covariates

In order to examine whether or not pre-intervention levels of pain-related catastrophizing are predictive of Pain Severity and ROM MVO, covariates of outcomes were identified prior to regression analyses using independent samples t-tests and Person product-moment correlations. Pre-intervention levels of Pain Severity and ROM MVO were both included as covariates in models with respective outcomes. Level of education was recoded into a binomial variable which included individuals who had a high school education or less and those who achieved above a high school level of education. Demographic variables including gender, level of education, age, marital status, and race/ethnicity were tested as possible covariates. Only gender was significantly related to outcomes, with males (M = 44.40,SD = 3.78) having higher ROM MVO at follow-up time point 2 compared to females (M = 36.36, SD = 8.45),$t(48) = 2.091$, $p<0.05$. Continuous variables identified through bivariate correlations, as displayed in Tables 5 and 6, included depressive symptoms, which was significantly correlated with pain severity at follow-up time point 1, $r(47) = .32$, $p< 0.05$, and follow-up time-point 3, $r(24) = .43$, $p< 0.05$. Sleep disturbance was also identified as a possible covariate at of pain severity at follow-up time point

3, $r(24) = .43$, $p < 0.05$. Use of pain medications was also examined earlier and considered as a possible covariate in the prediction of pain severity and ROM MVO outcomes, but as indicated above use of these medications was not significantly associated with outcome measures. Pain duration was positively associated with Pain Severity at follow-up time point 1, $r(45) = .333$, $p < 0.05$.

Multiple Regression Analyses

Prior to performing multiple regression analyses, standardized residuals were plotted against standardized predicted values to check assumptions of linearity and homoscedasticity. Histograms and normal probability plots were visually inspected to determine normality. Assumptions of normality, linearity, and homoscedasticity were all met. Assumptions of multicollinearity were met after examining collinearity statistics and were determined to be satisfactory.

Hypothesis 1. Hierarchical regression analyses were conducted to test the predictive ability of pre-intervention levels of pain-related catastrophizing, while controlling for identified covariates. Total PCS score was correlated with Pain Severity at all 3 follow-up time points. Because of this 3 separate regression analyses were run. Each regression model included baseline Pain Severity, ISI and CES-D scores, and pain duration as covariates in the model. Results of these regression analyses are presented below (Table 7). Analyses reveal pre-intervention levels of pain catastrophizing significantly predict higher levels of pain severity across all three follow-up time points, over and above baseline pain severity, depressive symptoms, sleep disturbance, and pain duration.

Table 7

Results of regression analyses testing pain related catastrophzing as a predictor of pain severity at follow-up time-points.

Step	Variable	B	SE	β	R^2	R^2 Change
DV = Pain Severity at F1; *n* = 44						
1	Pain Duration	.002	.002	.147		
	Severity BL	.037	.026	.247*		
	CESD BL	.018	.021	.156		
	ISI BL	.014	.029	.088	.225	.225*
2	Pain Duration	.002	.002	.154		
	Severity BL	.005	.001	.002		
	CESD BL	.006	.019	.051		
	ISI BL	.008	.027	.154		
	PCS Total BL	.043	.016	.417*	.313	.088*
DV = Pain Severity at F2; *n* = 35						
1	Pain Duration	.001	.003	.044		
	Severity BL	.141	.114	.247		
	CESD BL	.014	.036	.094		
	ISI BL	.022	.043	.113	.083	.083
2	Pain Duration	.001	.002	-.043		
	Severity BL	.001	.113	.002		
	CESD BL	.030	.032	.206		
	ISI BL	038	.039	.195		
	PCS Total BL	.078	.027	.591**	.289	.206**
DV = Pain Severity F3; *n* = 23						
1	Pain Duration	.002	.002	.145		
	Severity BL	.291	.153*	.299		
	CESD BL	.013	.021	.115		
	ISI BL	.020	.028	.126	249	.249*
2	Pain Duration	.001	.002	.135		
	Severity BL	.133	.167	.137		
	CESD BL	.004	.021	.033		
	ISI BL	.009	.027	.057		
	PCS Total BL	.037	.018*	.367	.322	.073*

Pain Severity = WHYMPI Pain Severity Subscale; F1-3 = Follow-up time point 1-3; BL = Baseline; CES-D = Centers for Epidemiologic Studies Depression Scale; ISI = Insomnia Severity Index; PCS Total = Pain Catastrophizing Scale Total Score.

* $p < 0.05$, two tailed, ** $p < 0.01$, two tailed.

Hypothesis 2. Hierarchical regression analyses were conducted to test the hypothesis that pre-intervention levels of pain related catastrophizing would be predictive of reduced ROM MVO scores. That is, pain related catastrophizing would be negatively associated with this intervention outcome. PCS Total score was significantly correlated with ROM MVO only at follow-up time-point 1 (See Table 6) and only a single regression analysis was conducted for that time-point. As was described above, TMD diagnostic category and type of intervention were associated with differences in ROM MVO scores, although only intervention was associated with significant differences in ROM MVO score for follow-up time point 1. Also, because treatment is often a function of diagnosis, intervention was chosen as the covariate to enter into the model. Gender was also included, because it was related to ROM MVO at other time-points. Two options were examined for including the non-binomial categorical variable of intervention in the regression model. Given the similarities between non-surgical and surgical interventions in terms of RMO MVO, intervention was recoded to reflect surgical and non-surgical interventions. Additionally, intervention was recoded as dummy variables, which allows the various levels of intervention to be represented in the regression model using ones and zeros to convey group membership. Creating dummy variables requires identifying a "control" by which other categories are compared. Given prior analyses examining Occlusal Splint as a potential common intervention against which other interventions differed in terms of ROM MVO, Occlusal Splint was selected as the control category. Separate regression models were run examining both forms of inclusion of the covariate and outcomes were comparable. Analyses reveal that pre-intervention levels of pain catastrophizing significantly predict poorer ROM MVO scores at follow-up time point 1, over and above baseline ROM MVO, gender, and intervention. Regression featuring dummy variables is presented below (Table 8).

Table 8

Results of regression analyses testing pain related catastrophzing as a predictor of ROM MVO at follow-up time point 1.

Step	Variable	B	SE	β	R^2	R^2 Change
1	Gender	-4.684	4.085	-.123		
	ROM MVO BL	.301	.120	.334*		
	Splint vs PT	-2.823	4.583	-.074		
	Splint vs Arthrocentesis	-5.001	2.926	-.242		
	Splint vs Arthroscopy	-1.386	3.858	-.053		
	Splint vs Arthroplasty	-15.570	3.421	-.680**	.525	.525**
2	Gender	-4.766	4.026	-.126		
	ROM MVO BL	.218	.132	.242		
	Splint vs PT	-1.729	4.579	-.046		
	Splint vs Arthrocentesis	-5.530	2.906	-.268		
	Splint vs Arthroscopy	-3.269	4.020	-.125		
	Splint vs Arthroplasty	-15.987	3.383	-.699**		
	PCS Total BL	-.232	.081	-.281*	.603	.078**

Note. $n = 53$.

RMO MVO = Range of motion maximum vertical opening; BL = Baseline; PCS Total = Pain Catastrophizing Scale Total Score.

* $p < 0.05$, two tailed, ** $p < 0.01$, two tailed.

Hypothesis 3. To test whether there was an interaction between perceptions of social responding (punishing responses and solicitous responses) and pain-related catastrophizing in predicting pain severity, cross-product interaction terms were computed and subsequently entered into the final step of regression models examining outcomes at study follow-up time points. Consistent with procedures described by others (Aiken, Aiken, Cohen & West, 2003; Holmbeck, 2002; Keith, 2006) interaction terms were derived from centered main predictor variables (e.g., PCS Total x WHYMPI Punishing Response).

There was not a statistically significant interaction between pain related catastrophizing and either perceptions of punishing responses or solicitous responses across any of the three study follow-up time points. As indicated in Table 9 there was a trend for the interaction of

perceived solicitousness and pain related catastrophizing as a moderator in predicting pain severity at follow-up time point 1.

Table 9

Results of regression analyses testing the interaction between pain related catastrophizing and perceived solicitous responses in predicting pain severity at initial follow-up.

Step	Variable	B	SE	β	R^2	R^2 Change
1	Pain Duration	.002	.002	.145		
	CESD BL	.003	.021	.025		
	ISI BL	.011	.028	.067		
	Solicitous BL	.097	.114	.124		
	PCS Total BL	.041	.017	.407	.316	.316*
2	Pain Duration	.001	.001	.130		
	CESD BL	.000	.020	.002		
	ISI BL	.008	.027	.047		
	Solicitous BL	.415	.204	.528		
	PCS Total BL	.094	.033	.922		
	PCS Total BL x Solicitous BL	-.016	.009	-.756†	.375	.059†

Note.n = 43.

CES-D = Centers for Epidemiologic Studies Depression; BL = Baseline; ISI = Insomnia Severity Index; Solicitous = WHYMPI Solicitous Responses Subscale; PCS = Pain Catastrophizing Scale.
* $p < 0.05$, two tailed, † $p = 0.07$, two tailed.

In earlier regression models, pre-intervention pain related catastrophizing was not associated with ROM MVO scores at follow-up time point 1 over and above the type of intervention a patient received. Consequently, interactions between pain catastrophizing and perceptions of significant other responding were not tested in predicting ROM MVO scores at time point 1. To increase power, change scores from baseline to initial follow-up were computed for ROM MVO. Because change scores accounted for norm based gender differences in ROM MVO at baseline, gender was removed from the model as a covariate. An interaction was not found between pain related catastrophizing and perceived punishing, β= .186, $t(45) = .707$, *ns*, or solicitous responses, β= .106, $t(45) = .313$, *ns* in the prediction of ROM MVO at time point 1.

Hypothesis 4. Cross-products were derived to test the interaction between pain duration and pre-intervention levels of pain-related catastrophizing in predicting perceptions of social responding. Pain duration did not moderate the relation between catastrophizing and perceived solicitous, β= .143, *t*(52) = 1.041, *ns*, or punishing responses, β= .173, *t*(51) = .669, *ns*.

Hypothesis 5. Tests of the hypothesis that perceptions of social response, solicitous or punishing, would mediate the relation between Pain Severity and ROM MOV at follow-up time points were examined using the criteria presented by Baron and Kenny (1986) with SPSS macros procedures provided by Preacher & Hayes (2004). Specifically, mediation is present when there is a significant relation between the predictor and criterion construct, there is a significant relationship between the predictor and mediator, the mediator continues to predict the criterion after controlling for the predictor, and the relationship between the predictor and criterion is significantly reduced when the mediator is present I the equation. In the current study, perception of punishing responses was unrelated to pain severity at follow-up time point 1, β = .036, *t*(48) = .842, *ns*, time point 2, β = .124, *t*(35) = .785, *ns*, and time point 3,β = .919, *t*(23) = .741, *ns*. Similarly, the mediating variable of perceptions of solicitous responses was not significant for all three follow-up time points. ROM MVO scores were also not related to punishing or solicitous response across all three follow-up time points, meaning that the criteria for mediation could not be satisfied.

Discussion

The purpose of the present study was to examine the role of pain-related catastrophizing in patient outcomes following surgical and non-surgical interventions for TMDs. Non-surgical interventions included occlusal splint delivery or referral to physical therapy, while surgical procedures consisted of arthrocentesis, arthroscopy, or arthroplasty – open joint. In order to conduct the study, patient data were drawn from medical charts, paper questionnaires and telephone contact. Data were obtained prior to intervention (baseline) and at three post-intervention follow-up time points. Pain severity and range of motion (ROM) scores were utilized as outcome measures and their relations with baseline levels of pain-related catastrophizing were examined as bivariate relations and in multiple regression models controlling for covariates. The current study also sought to understand the interpersonal nature of pain-related catastrophizing in operant or communal coping terms. To do this significant others' responses as perceived by patients were examined to determine their impact on intervention outcomes. Given that pain-related catastrophizing significantly predicted outcomes, perceived solicitous and punishing responses of significant others were examined as both moderators and mediators of this relationship. The role of pain duration was also examined as it related to the perception of significant other responses, because the interpersonal nature of pain coping and reaction of significant others may change over time. As such, pain duration was examined as a potential mediator of the relationship between pain-related catastrophizing and perceptions of significant other responses.

The Relation Between Pain-Related Catastrophizing and Outcomes

Pain Severity. The current study is the first known attempt to investigate the effectiveness of pain-related catastrophizing as a predictor of post-intervention outcomes

following non-surgical and surgical intervention for TMDs. Prior studies have demonstrated that greater pain in TMDs is associated with higher levels of pain-related catastrophizing, and that catastrophizing is an important variable to consider when patients are learning to manage pain associated with TMD through psychological intervention (Castrillon et al., 2008; Turner et al., 2007). The current study found that pain-related catastrophizing predicted post-intervention pain severity at all three follow-up time points, with increased levels of pain catastrophizing being related to increased pain severity. This suggests that catastrophizing is an important variable to consider in surgical and less invasive non-surgical intervention approaches. Baseline levels of pain catastrophizing and pain severity were not significantly different across the three primary diagnostic categories in the current study, nor were significant differences found between the five interventions ultimately provided to patients. Previous literature has demonstrated a robust relationship between pain catastrophizing and pain outcomes in a variety of surgical populations and chronic pain diagnoses (Edwards et al., 2009; Pavlin et al., 2005; Riddle, Wade, Jiranek, & Kong, 2010; Sullivan, Tanzer, Reardon, Amirault, Dunbar, & Stanish, 2011). The predictive ability of pain catastrophizing in the current study is consistent with these findings.

Pain catastrophizing was predictive over and above pain duration, sleep disturbance and depressive symptoms. This result was obtained despite the finding of a high correlation between depressive symptoms and pain-related catastrophizing that is consistent with prior literature (Arnow et al., 2011; Jensen, Moore, Bockow, Ehde, & Engel, 2011). Due to the strong associations between pain-related catastrophizing and depressive symptoms, statistical control of negative affect (e.g., CES-D) is commonplace when examining associations of pain catastrophizing (Quartana et al., 2009). The literature has continued to confirm this relation in TMD samples. For example, catastrophizing and depression were both found to be associated

with the onset of clinically significant pain and the progression of TMD pain and disability in an 18 month prospective cohort study (Velly et al., 2011). Reduction in TMD pain following CBT interventions has also been shown to significantly reduce depressive symptoms and these gains have been maintained up to one year following intervention (Litt, Shafer, & Kreutzer, 2010). Although not the case in the current study, depressive symptoms have been found to be significantly greater in patients with exclusively myofascial pain diagnoses compared to an exclusively joint pain group (Giannakopoulos, Keller, Rammelsberg, Kronmuller, & Schmitter, 2010; Schmitter, Kellers, Giannakopoulos, & Rammelsberg, 2010).

The effectiveness of catastrophizing in continuing to predict pain severity, even with the inclusion of pain duration in the regression model is not surprising. Pain catastrophizing is often predictive of pain ratings during aversive stimulation procedures administered in standard experimental pain studies with otherwise healthy participants (Campbell, Quartana et al., 2010; Goodin, McGuire, Allshouse, et al., 2009). That is, patients who do not experience ongoing, chronic pain do not necessarily have "a pain duration" in any long-term sense, but pain catastrophizing nonetheless influences their ability to attend to and appraise acute pain levels. Moreover, the emergence of the relation between catastrophizing and pain early in life (Sullivan et al., 2001) and the tendency to conceptualize catastrophizing as a dispositional construct is consistent with findings that pain duration does not influence the ability of catastrophizing to predict pain severity (Quartana et al., 2009). Overall, the duration of a person's pain is not related to fluctuations in pain-related catastrophizing.

Sleep disturbance was also controlled for as a covariate in all of the models predicting pain severity, although bivariate relationships between sleep disturbance and pain severity were significant only for the most distant follow-up time point. Few studies have examined the

relationship between sleep and pain in TMD samples. One study of patients with TMDs sought to clarify the bidirectional relation between sleep and pain by examining the temporal associations between naturalistic fluctuations in pain and insomnia using a cross-lagged analysis technique (Quartana, Wickwire, Klick, Grace & Smith, 2010). This study found that initial month increases in insomnia were associated with daily pain ratings the next month. One difference between that study and the current study is the measure of sleep disturbance. The referenced study used the Pittsburgh Sleep Quality index which measures a broader construct of sleep quality, which is related to sleep disturbance. It is unlikely that measurement differences alone account for the lack of relationship between initial sleep disturbance and initial follow-up outcomes because the ISI (used in the current study), and the PSQI are highly correlated (r = .80; Morin, Belleville, Belanger, & Ivers, 2011). Additionally, participants in the study by Quartana and colleagues (2010) and the current study reported comparable average ISI ratings which fell in the lower end of the subthreshold insomnia range (M = 9.3 and M = 10.26, respectively). It is not possible to determine if sleep disturbance increased in the month preceding the final follow-up time point and was predictive of pain severity in the current study. However, one alternative explanation for why sleep disturbance was related only to distant measures of pain severity is that the ruminative nature of catastrophizing may have become more pronounced over time, as pain was reduced, but not completely eliminated in some patients. One study of patients with myofascial temporomandibular disorder (Buenaver et al., 2012) found that sleep disturbance was positively associated with pain catastrophizing, and that the relationship between sleep disturbance and pain severity was partially mediated by the ruminative subdomains of pain-related catastrophizing.

It is important to note that pain-related catastrophizing did not significantly vary across diagnostic categories or the intervention approach ultimately chosen. This is consistent with prior research which found that while patients within various TMD diagnostic categories may be similar in terms of physical examination and ultimate treatment approach, they vary significantly across and within groups in terms of the psychological characteristics which influence their ability to cope with and adjust to chronic pain (Rudy et al., 1989; Turk, Rudy, Kubinski, Zaki, & Greco,1996).

Range of Motion. Bivariate relationships between pain-related catastrophizing and functional ROM scores for maximum vertical opening were obtained only at follow-up time point 1. As a result, only the initial follow-up ROM scores were examined using multivariate analyses. Gender, diagnostic category and intervention approach were all related to ROM scores at follow-up, with intervention being the only covariate that was related to ROM at initial follow-up. The relation between diagnostic category and ROM within 1-2 weeks of intervention was expected given that ROM often provides diagnostic information in TMD evaluation (Laskin, 1995; Wright & North, 2009) and patients are still in early stages of treatment or recovery from surgical intervention at that time.

Results suggest that pre-intervention levels of catastrophizing are not an early indicator of poor response with objective measures such as range of motion. The lack of significant relationships at follow-up time points 2 and 3 are consistent with previous studies which have found no association between pain catastrophizing and measures of jaw impairment (Turner et al., 2001; Turner et al., 2005). There are no prior studies of the influence of pain-related catastrophizing on functional outcomes following medical intervention for TMD. But studies have examined this relation in other surgical samples. One study found that scores on the

Western Ontario and McMaster Universities Arthritis Index (WOMAC), a measure of physical functioning, were predicted by pain-related catastrophizing one year following total knee arthtroplasty (TKA; Sullivan et al., 2011). Given that the WOMAC is a self-administered questionnaire it is likely to be heavily influenced by pain-related catastrophizing, in contrast to the objective ROM scores obtained in the current study, which were based on objective measurements by a trained professional.

There are several studies that examine post-surgical intervention functioning using the WOMAC as it relates to pain catastrophizing, but many do not report objective outcomes such as range of motion of ratings based on observations by trained professionals (Peters et al., 2007). One study found that the number of days it took recovering patients to bend their leg at a 90 degree angle was positively associated with pain-related catastrophizing (Kendell, Saxby, Farrow, & Naisby, 2001). Another study found that lower knee functioning, as measured by an examiner-based measure known as the Knee Society Rating System, was associated with lower levels of problem-focused coping (Lopez-Olivo et al., 2011). Some studies have examined objective outcomes by proxy. Pain catastrophizing has been related to longer hospital stays following TKA (Witvrouw, Pattyn, & Almqvist, 2009) and return to sports activities in athletes following anterior cruciate ligament repair, with catastrophizing being related to lower rates of returning to sports (Tripp et al., 2011).

Another variable commonly cited in studies of return to function following TKA or other major joint surgeries is kinesiophobia, or fear of movement (Sullivan et al., 2009; 2011). The post-surgical movement necessary following TKA for rehabilitation is naturally different than that required for patients with TMDs. Nonetheless, movement of the jaw may be considerably fear inducing among this population contributing to deconditioning, poor functional outcomes

and increased pain. The relative equivalence of this variable across different conditions is unknown and a measure of kinesiophobia was developed only recently (Visscher, Ohrbach, van Wijk, Wiilkosz, Naeije, 2010). Future studies examining the role of pain catastrophizing in post-intervention outcomes, particularly those that involve movement related intervention, such as PT, should include measures of kinesiophobia to help determine its relative importance.

Mechanisms of Action

A variety of explanations have been provided in the pain literature regarding mechanisms of action related to deleterious effects of catastrophizing on pain severity, dysfunction, and poor adjustment to pain. Some have contended that pain-related catastrophizing arises as a function of an appraisal process related to the threat of painful stimuli and a tendency to over interpret pain as threatening and uncontrollable (Lazurus & Folkman, 1984; Severeijns et al., 2004). These appraisals are associated with some form of coping attempt. Generally in the pain literature, coping approaches are examined along the dimensions of active versus passive coping. The coping strategies employed are believed to alter both the perception of the intensity of the pain and one's perception of their ability to manage or tolerate the pain (Flor & Turk, 2006). Those high in catastrophizing may engage in more maladaptive and passive coping styles to manage their pain, which tend to increase attention to pain.

Indeed, those higher in pain-related catastrophizing also demonstrate greater attentional bias towards pain. One study found that in a sample of chronic low back pain patients the relation between catastrophic pain-related thoughts and response times on a distraction task was mediated by attention to pain during a standardized lifting activity (Goubert et al., 2004). Additionally, pain-related catastrophizing has been associated with several cognitive distortions related to pain including overgeneralizations (assuming the outcome of one experience applies to

all similar experiences), selective abstraction (attending to those negative aspects of experiences selectively), and lack of generalization of corrective experiences (reluctance to generalize an outcome where lesser pain is experienced to other similar experiences; Goubert, Crombez, & Danneels, 2005). As for the current study, patients with TMDs may have experienced greater helplessness if they were higher in pain-related catastrophizing and surgical or non-surgical interventions did not meet their expectations for pain reduction.

Pain-related catastrophizing may also be associated with increased pain by way of altered physiological responses to stress and pain. For instance, pain catastrophizing has been associated with increased muscle tension in patients with back pain (Quartana, Burns, & Lofland, 2007). This may present similarly in TMDs where some studies have found catastrophizing is associated with greater muscle pathology (Galdon et al., 2006; Turner et al., 2001). Catastrophizing has also been associated with altered hypothalamic-pituitary-adrenal (HPA) axis activity and less variability in diurnal cortisol activity in patients with low back pain (Johansson et al., 2008). Cortisol is a glucocorticoid which is released in response to stress. Cortisol has anti-inflammatory effects by reducing the synthesis of proinflammatory cytokines. Chronic stressors, such as pain, disrupt cortisol signaling resulting in an overproduction of proinflammatory cytokines (Robles et al., 2005). Interleukin-6 (IL-6) is a proinflammatory cytokine, meaning it is involved in the body's natural immune response to defend against invading organisms, a major process of which is inflammation. Increases in IL-6 are associated with increases in inflammation, and possible increases in pain. Although pain itself is a stress inducing stimulus, pain-related catastrophizing appears to play a role in how the body responds. In one study of healthy participants undergoing experimental pain procedures, catastrophizing was associated with elevations in cortisol as well as IL-6 (Edwards et al., 2008).

In patients with TMDs, pain catastrophizing has been associated with greater interruption of the morning cortisol decline in response to experimental pain testing (Quartana et al., 2010). One study of TMDs found that patients with lower levels of optimism also had significantly higher levels of IL-6 under experimentally manipulated stress (Costello et al., 2002). IL-6 has also been positively associated with arthroscopic findings related to level of internal derangements of the TMJ (Sandler, Buckley, Cillo, & Braun, 1998).

Naturalistic stress, which can result in the activation of the HPA system and increase the aforementioned biomarkers, is associated with slowed wound healing (Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995; Marucha, Kiecolt-Glaser, & Favagehi, 1998) which has been demonstrated following a variety of surgical interventions (Kiecolt-Glaser, Page, Marucha, MacCallum, & Glaser, 1998; Broadbent, Petrie, Alley, & Booth, 2003). Given research demonstrating greater catastrophizing is associated with increased HPA activation in TMDs and that this type of activation has been associated with slowed rates of wound healing following surgical intervention, it is plausible that stress-related physiological changes may mediate the relation between pain catastrophizing and pain severity following surgical intervention.

Pain-Related Catastrophizing and Communal Coping Variables

As mentioned, several mechanisms of action have been proposed by which pain-related catastrophizing may result in increased levels of pain severity. The communal coping aspect of pain-related catastrophizing has been demonstrated to play a role in the increase in pain severity in a variety of chronically painful conditions (Boothby et al., 2004; Buenaver et al., 2007; Cano, 2004; Giardino et al., 2003) . The communal coping model is based, in part, on Fordyce's (1979) operant conceptualization of how acute pain develops into chronic pain through the reinforcement of maladaptive "pain behaviors" by way of social responses. This has

been demonstrated in observational studies of patients with musculoskeletal pain and their spouses, wherein increases in pain behaviors and pain severity were related to negative and solicitous responses (Romano et al., 2000).

Several studies have examined the role of punishing and solicitous responses in the relationship between catastrophizing and pain outcomes, with some researchers finding significant positive relations between catastrophizing and punishing responses whereas others have found relations only between catastrophizing and solicitousness (Grant et al., 2002; Raichle et al., 2007). One study found that the relationship between catastrophizing and pain outcomes is moderated by social responses, with a stronger catastrophizing-pain relationship for those perceiving high levels of solicitous responding (Giardano et al., 2003). The current study did not find a significant interaction between catastrophizing and punishing responses or catastrophizing and solicitous responses in predicting either pain or ROM outcomes. However, a trend for moderation was observed that would be consistent with findings by Giardano and colleagues (2003). Punishing and solicitous responses also did not mediate the relationship between catastrophizing and outcomes. This is also consistent with previous findings (Buenaver et al., 2007). Some have found that pain duration is a possible explanatory factor for the discrepant findings mentioned earlier, with shorter pain durations related to stronger associations between pain catastrophizing and solicitous responses, but not punishing responses (Buenaver et al., 2007; Cano, 2004). In the current study pain duration did not moderate the relation for either type of response.

All prior work in this area has been conducted via cross-sectional designs. The current study supports many of the previous findings with longitudinal data in the context of ongoing intervention. It is also important to note that previous work in this area has been conducted with

samples of patients with mixed pain, none of which included TMDs. The items on the WHYMPI that make up the perceived Punishing and Solicitousness subscales may not be sufficiently specific to the forms of impairment associated with TMD. The WHYMPI was designed using a mixed chronic pain sample, comprised mostly of patients with chronic low back (Kerns, Turk, & Rudy, 1985). Hence, items that address perceptions of significant other responding (e.g., "Takes over my jobs or duties."), may not be relevant for TMD patients because impairment with mobility or broad physical strength may not be as salient. Because of this TMD patients may not engage in the same interpersonally salient behavioral coping repertoire that a patient with low back pain might. Ultimately, it is difficult to know at this time if the communal coping model is a relevant framework for explaining pain severity as a function of catastrophizing in TMD patients.

Limitations and Future Directions

There are several limitations to the current study. Although it was assumed that patients were adherent with regard to occlusal splint use and active engagement in PT (and corresponding home exercise plans) , it was not possible to obtain information about the nature of the PT provided or amount of follow-through by patients with regard to occlusal splint use. Although the current study was not intended to compare effectiveness of interventions, the inclusion of a control group may have aided the interpretation of results particularly given that the nature of the PT intervention was unclear.

Also with respect to interventions, the nature of expectations for outcome may influence the outcomes of medical interventions themselves, particularly for patients who are higher in catastrophizing and may have unrealistic expectations (Sullivan et al., 2011). The current study did not assess patient expectancies and it is possible expectations for pain reduction may play a

role in pain severity following non-surgical and surgical interventions for TMDs. Future studies should incorporate a measure of expectations for pain reduction to see if pain catastrophizing continues to predict pain severity over and above those expectations.

The current study examined solicitous and punishing responses in the context of the communal coping model, which assumes that catastrophizing is associated with pain behaviors that ultimately elicit responding from the social environment. Although the current study examined patient perceptions of social responses as they relate to assistance with pain, the nature of the actual responses themselves is unknown, because collateral information from significant others was not obtained and actual observations of patient-significant other interactions were not made. Also, the perceived significant other responses assessed in the current study represent only a small sampling of the potential responses that a spouse or partner might make, or be perceived to make, in response to pain behaviors. It is possible that aspects of significant other responses that influence pain-related catastrophizing were not considered because they were not addressed in the WHYMPI subscales. Future research examining social coping, and the possible responses of significant others is needed in TMDs. Currently an instrument that corresponds to the Punishing and Solicitous subscales of the WHYMPI does not exist and may need to be developed to fully understand the nature of communal coping with regard to this population.

Moreover, only 58% of the current study sample reported being married. Although the WHYMPI instructions are clear with regard to identifying a significant other, regardless of marital status (e.g., parent, child, sibling), it is not absolutely certain that non-married patients completed the WHYMPI with a significant other in mind. Future research should also consider the temporal changes that may occur in perceptions of others' responses using longitudinal designs. Extant studies have only made retrospective reference to pain duration to explain

concurrent findings, without consideration for how perceptions of significant others may change over time.

The current study utilized a convenience sample, obtained primarily from two OMFS clinics located within the same metropolitan area in the South-Atlantic region of the United States. This somewhat limits the generalizability of the current findings to the population of TMD patients as a whole. This study also did not include a measure of masticatory and non-masticatory jaw activity limitations, which may not correspond to objective ROM findings.

The current findings suggest that pain-related catastrophizing may play a role in the continued subjective pain experience of patients following medical interventions for TMDs, while the objective findings suggest that catastrophizing is only predictive of poorer range of motion in the immediate follow-up after intervention. Future research into determining how best to triage patients with psychosocial and behavioral components to their ongoing pain, possibly based on brief screenings of pain-related catastrophizing, is needed. This is an important endeavor, given CBT interventions for TMDs have been demonstrated to be effective in reducing associated depressive symptoms, pain severity, and pain related interference (Litt et al., 2010; Turner et al., 2006). Additionally, while the mechanism of action is still unclear, future studies that examine physiological correlates of pain-related catastrophizing in TMDs, particularly those involving the HPA and physiological stress response, will be important for determining if inflammatory processes mediate the relationship between psychological variables and pain outcomes. This inclusion may also shed additional light on the differences found in the current study between objectively measure outcomes (ROM) and subjective levels of pain.

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Appendix A

MULTIDIMENSIONAL PAIN INVENTORY

Kerns, Turk & Rudy (1985)

THE FOLLOWING SECTION ASKES YOU IMPORTANT QUESTIONS ABOUT YOUR PAIN. PLEASE RESPOND TO EACH QUESTION.
BEFORE YOU BEGIN, PLEASE ANSWER 2 PRE-EVALUATION QUESTIONS BELOW:

1. Some of the questions in this questionnaire refer to your "significant other". A significant other is *a person with whom you feel closest.* This includes anyone that you relate to on a regular or infrequent basis. It is very important that you identify someone as your "significant other". Please indicate below who your significant other is (check one):

□Spouse □Partner/Companion □Housemate/Roomate
□Friend □Neighbor □Parent/Child/Other relative
□Other (please describe):_____

2. Do you currently live with this person? □ YES □ NO

When you answer questions in the following pages about "your significant other", always respond in reference to the specific person you just indicated above.

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

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

0 1 2 3 4 5 6
No pain Very intense pain

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

0 1 2 3 4 5 6
No interference Extreme interference

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

0 1 2 3 4 5 6
No change Extreme change

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

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

0	1	2	3	4	5	6
No change						Extreme change

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

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

6. Rate your overall mood during the <u>past week</u>.

0	1	2	3	4	5	6
Extremely low mood						Extremely high mood

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

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

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

0	1	2	3	4	5	6
No change						Extreme change

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

0	1	2	3	4	5	6
No change						Extreme change

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

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

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

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

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

0 1 2 3 4 5 6
No suffering Extreme suffering

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

0 1 2 3 4 5 6
No change Extreme change

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

0 1 2 3 4 5 6
No change Extreme change

__ **Check here, if you are not presently working.**

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

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

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

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

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

0 1 2 3 4 5 6
No change Extreme change

18. During the past week, how irritable have you been?

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

19. How much has your pain changed your friendships with people other than your family?

0 1 2 3 4 5 6
No change Extreme change

20. During the past week, how tense or anxious have you been?

0 1 2 3 4 5 6
Not at all tense or anxious Extremely tense or anxious

B. In this section, we are interested in knowing how your significant other (this refers to the person you indicated above) responds to you when he or she knows that you are in pain. On the scale listed below each question, **circle a number** to indicate how often your significant other generally responds to you in that particular way when you are in pain.

1. Ignores me.

0	1	2	3	4	5	6
Never						Very often

2. Asks me what he/she can do to help.

0	1	2	3	4	5	6
Never						Very often

3. Reads to me.

0	1	2	3	4	5	6
Never						Very often

4. Expresses irritation at me.

0	1	2	3	4	5	6
Never						Very often

5. Takes over my jobs or duties.

0	1	2	3	4	5	6
Never						Very often

6. Talks to me about something else to take my mind off the pain.

0	1	2	3	4	5	6
Never						Very often

7. Expresses frustration at me.

0	1	2	3	4	5	6
Never						Very often

8. Tries to get me to rest.

0	1	2	3	4	5	6
Never						Very often

9. Tries to involve me in some activity

0 1 2 3 4 5 6
Never Very often

10. Expresses anger at me.

0 1 2 3 4 5 6
Never Very often

11. Gets me some pain medications.

0 1 2 3 4 5 6
Never Very often

12. Encourages me to work on a hobby.

0 1 2 3 4 5 6
Never Very often

13. Gets me something to eat or drink.

0 1 2 3 4 5 6
Never Very often

14. Turns on the T.V. to take my mind off my pain

0 1 2 3 4 5 6
Never Very often

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

1. Wash dishes.

0 1 2 3 4 5 6
Never Very often

2. Mow the lawn.

0 1 2 3 4 5 6
Never Very often

3. Go out to eat.

0 1 2 3 4 5 6
Never Very often

4. Play cards or other games.

0 1 2 3 4 5 6
Never Very often

5. Go grocery shopping.

0 1 2 3 4 5 6
Never Very often

6. Work in the garden.

0 1 2 3 4 5 6
Never Very often

7. Go to a movie.

0 1 2 3 4 5 6
Never Very often

8. Visit friends.

0 1 2 3 4 5 6
Never Very often

9. Help with the house cleaning.

0 1 2 3 4 5 6
Never Very often

10. Work on the car.

0 1 2 3 4 5 6
Never Very often

11. Take a ride in a car.

0 1 2 3 4 5 6
Never Very often

12. Visit relatives.

0 1 2 3 4 5 6
Never Very often

13. Prepare a meal.

0 1 2 3 4 5 6
Never Very often

14. Wash the car.

0 1 2 3 4 5 6
Never Very often

15. Take a trip.

0 1 2 3 4 5 6
Never Very often

16. Go to a park or beach.

0 1 2 3 4 5 6
Never Very often

17. Do a load of laundry.

0 1 2 3 4 5 6
Never Very often

18. Work on a needed house repair.

0 1 2 3 4 5 6
Never Very often

Appendix B

Pain Catastrophizing Scale (PCS)

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.
We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

0=Not at all
1=To a slight degree
2=To a moderate degree
3=To a great degree
4=All the time

When I'm in pain…

1. I worry all the time about whether the pain will end.	0	1	2	3	4
2. I feel I can't go on.	0	1	2	3	4
3. It's terrible and I think it's never going to get any better	0	1	2	3	4
4. It's awful and I feel that it overwhelms me.	0	1	2	3	4
5. I feel I can't stand it anymore.	0	1	2	3	4
6. I become afraid that the pain will get worse.	0	1	2	3	4
7. I keep thinking of other painful events.	0	1	2	3	4
8. I anxiously want the pain to go away.	0	1	2	3	4
9. I can't seem to keep it out of my mind.	0	1	2	3	4
10. I keep thinking about how much it hurts.	0	1	2	3	4
11. I keep thinking about how badly I want the pain to stop.	0	1	2	3	4
12. There's nothing I can do to reduce the intensity of the pain.	0	1	2	3	4
13. I wonder whether something serious may happen.	0	1	2	3	4

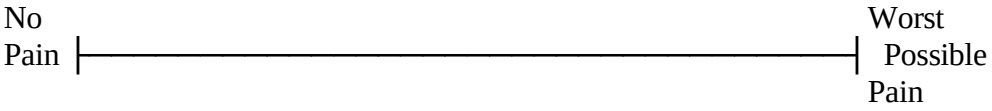
Appendix C

SHORT-FORM McGILL PAIN QUESTIONNAIRE

Please choose the words below that describe your pain *today*. If a word does not describe your pain, choose the 0 (*none*) for that word. For each word that does describe your pain, rate the intensity for that quality of your pain from 1 (*mild*) to 3 (*severe*).

	None	Mild	Moderate	Severe
Throbbing	0) _____	1) _____	2) _____	3) _____
Shooting	0) _____	1) _____	2) _____	3) _____
Stabbing	0) _____	1) _____	2) _____	3) _____
Sharp	0) _____	1) _____	2) _____	3) _____
Cramping	0) _____	1) _____	2) _____	3) _____
Gnawing	0) _____	1) _____	2) _____	3) _____
Hot-burning	0) _____	1) _____	2) _____	3) _____
Aching	0) _____	1) _____	2) _____	3) _____
Heavy	0) _____	1) _____	2) _____	3) _____
Tender	0) _____	1) _____	2) _____	3) _____
Splitting	0) _____	1) _____	2) _____	3) _____
Tiring-exhausting	0) _____	1) _____	2) _____	3) _____
Sickening	0) _____	1) _____	2) _____	3) _____
Fearful	0) _____	1) _____	2) _____	3) _____
Punishing-cruel	0) _____	1) _____	2) _____	3) _____

Rate the intensity of your pain on the two scales below. Make a mark on the line to indicate where your pain falls between *No Pain* and *Worst Possible Pain* and then circle the appropriate number on the second scale.



0 No pain
1 Mild
2 Discomforting
3 Distressing
4 Horrible
5 Excruciating

Appendix D

Below is a list of the ways you might have felt or behaved. Please check (X) how often you have felt this way during the past week.	DURING THE PAST WEEK			
	Rarely or none of the time (less than 1 day)	Some or a little of the time (1-2 days)	Occasionally or a moderate amount of time (3-4 days)	Most or all of the time (5-7 days)
1. I was bothered by things that usually don't bother me.				
2. I did not feel like eating; my appetite was poor.				
3. I felt that I could not shake off the blues even with help from my family or friends.				
4. I felt I was just as good as other people.				
5. I had trouble keeping my mind on what I was doing.				
6. I felt depressed.				
7. I felt that everything I did was an effort.				
8. I felt hopeful about the future.				
9. I thought my life had been a failure.				
10. I felt fearful.				
11. My sleep was restless.				
12. I was happy.				
13. I talked less than usual.				
14. I felt lonely.				
15. People were unfriendly.				
16. I enjoyed life.				
17. I had crying spells.				
18. I felt sad.				
19. I felt that people dislike me.				
20. I could not get "going."				

Appendix E

Please answer each of the questions below by circling the number that best describes your sleep patterns ***in the past week.*** Please answer all questions.

Please rate the current (past week's) **SEVERITY** of your insomnia problem(s):	None	Mild	Moderate	Severe	Very Severe
Difficulty falling asleep					
Difficulty staying asleep					
Problem waking up too early					

How **SATISFIED/DISSATISFIED** are you with your current sleep pattern?	Very Satisfied 0	Satisfied 1	Neutral 2	Dissatisfied 3	Very Dissatisfied 4

To what extent do you consider your sleep problem to **INTERFERE** with your daily functioning (eg, daytime fatigue, ability to function at work/daily chores, concentration, memory, mood, etc)?	Not at all Interfering 0	A Little 1	Somewhat 2	Much 3	Very Much Interfering 4

How **NOTICEABLE** to others do you think your sleeping problem is in terms of impairing the quality of your life?	Not at all Noticeable 0	A Little 1	Somewhat 2	Much 3	Very Much Noticeable 4

How **WORRIED/DISTRESSED** are you about your current sleep problem?	Not at all Worried 0	A Little 1	Somewhat 2	Much 3	Very Much Worried 4

Appendix F

Dear New Patient,

Welcome to the Faculty Practice of the Department of Oral and Maxillofacial Surgery at the Virginia Commonwealth University Medical Center! We are pleased that you have chosen to seek care for your temporomandibular joint (TMJ) disorder with us and we look forward to meeting you in person.

Because TMJ disorders are often complex, it is important that we gather detailed information from you in order to best diagnose and treat your problem. To help with this, we are enclosing a set of forms for you to fill out. Please take the time to complete them before you arrive for your appointment. We understand that the forms are lengthy, but they are designed to gather information that has been shown through extensive scientific research to be relevant to many TMJ problems. The medical information you provide will be reviewed at your appointment and will be very helpful to us as we work to help you.

We sometimes gather information about our patients for research purposes, and you may be asked for permission to include your information in one of these studies. However, unless you give us written permission, your health information will not be shared with anyone, as required by all laws and customs regarding protected health information. These forms collect the same information we need even if you do not participate, so please complete them even if you are sure you will not take part in any research. There is no obligation to participate, and whatever you decide will not change the care you receive from us.

As always, please remember to bring your medical and dental insurance cards and information with you so we can file benefit claims on your behalf. If you have TMJ x-rays, CT or MRI reports or (better still) CDs with images, please bring them as well. We are enclosing some additional maps and parking information that will help you find your way around the Health Sciences Campus. We look forward to welcoming you to our clinic in the Lyons Building of the School of Dentistry.

Sincerely,

Gregory M Ness DDS
Professor
Oral and Maxillofacial Surgery

Vita

Aaron Michael Martin was born on October 10th, 1983 in Burlington, North Carolina, but grew up on the Eastern Short of Maryland. He graduated James M. Bennett High School in 2001. He received his Bachelors of Arts degree from Salisbury University in December of 2004, graduating Summa Cum Laude. While there he majored in psychology with a minor in marketing. He received his Master of Science degree from Loyola College in September of 2007. He entered the doctoral program at Virginia Commonwealth University in the fall of 2007 and defended his doctoral dissertation in August of 2013. He completed his pre-doctoral internship in Clinical Health Psychology at VA Connecticut Healthcare System and will begin a postdoctoral fellowship at VA Connecticut in Fall of 2013.