THE EFFECTS OF AN EARLY INTERVENTION PROGRAM ON PHYSICAL SYMPTOMS IN A TMD POPULATION

by

KARA LORDUY

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In this journey together, we have each other for strength, inspiration, love, and to help each other remember when there are obstacles along the way… keep moving forward…one step at a time.

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ABSTRACT

THE EFFECTS OF AN EARLY INTERVENTION PROGRAM

ON PHYSICAL SYMPTOMS IN A TMD POPULATION

Kara Lorduy, PhD

The University of Texas at Arlington, 2012

Supervising Professor: Robert J. Gatchel

Aims: 1) Identify comorbid, non-specific symptoms of CSS, and TMD specific symptoms across three groups of Axis I RDC/TMD disorders, 2) investigate the influence of three interventions on TMD specific and comorbid symptoms of CSS and pain and pain-related disability, and 3) examine the influence of emotional distress on symptoms, pain, and pain-related disability. Methods: Participants were patients recruited from dental clinics within a major metropolitan area assessed for TMD non-specific symptoms of CSS using the Symptoms Checklist (Study 1) and TMD specific symptoms using the RDC/TMD (Study 2). In Study 2, participants at high-risk for chronicity were randomly assigned to a self-care (SC) or biobehavioral (BB) intervention and evaluated for their responsiveness immediately following treatment (T2). Results: In Study 1, we found that those with a TMD Muscle Disorder and those

with more than one TMD diagnosis had more symptoms of CSS. As predicted in Study 2, symptoms for Axis I Group I Muscle Disorders and Axis I Group III Bone Deficiencies and several of the target variables therewithin were significantly reduced immediately following Treatment. Moreover, emotional distress accounted for a substantial amount of the variance for physical symptoms and mediated comorbid symptoms of CSS. Conclusions: Comorbid symptoms are strongly related to myofacial TMD. Axis I Group I and Group III disorders are more responsive to the effects of intervention immediately following treatment compared to Axis I Group II Disc Displacements.

Key words: temporomandibular disorder (TMD), myofacial TMD, central sensitization syndrome (CSS), biobehavioral, emotional distress

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CHAPTER 1

OVERVIEW

Recently, the National Institute of Health (NIH) granted the Orofacial Pain: Prospective Risk-Assessment Intervention (OPPERA) program $19.1 million dollars to investigate demographic, genetic, autonomic, and psychosocial risk-factors associated with temporomandibular disorder (TMD) (Dworkin, 2011). There was a general expectation among the collaborators that the findings would reveal important mechanisms involved in the phenomenology of TMD that could potentially inform pain investigators about other musculoskeletal disorders (Dworkin, 2011). Indeed, there has been a growing awareness of the strong degree of overlap TMD shares with central sensitivity syndromes (CSS) including fibromyalgia (FM), chronic fatigue syndrome (CFS), and irritable bowel syndrome (IBS), which involve symptoms, such as muscle and joint aches, problems with sleep, feeling tired or unrested after sleep, cognitive problems, and gastrointestinal disturbances (Aaron, Burke, & Butchwald, 2000). In fact, 75% of those with FM meet the standards for TMD using the Research Diagnostic Criteria (RDC/TMD) (Plesh, Wolfe, & Lane, 1996). Moreover, it has been suggested that these disorders are a product of central sensitization (CS) (Yunus, 2007). Many refer to CS as a disease state constituted by a whole-organism stress response whereby dysregulation among many interdependent neuroendocrine and neurotransmitter systems promotes either the

maintenance or induction of chronic pain through augmentation of neural structures in the pain pathway (Chapman, Tuckett, & Song, 2008; Lyon, Cohen, & Quinter, 2011).

Alongside the amounting research informing the various mechanisms involved in CS and with the recognition that it dispels overly simplistic dualistic models that reduce pain to a series of stimulus-response processes (Greene, 2009), important strides have been made in identifying biopsychosocial factors of TMD (Dougall et al., 2012). As a result, many have conceptualized pain as a homeostatic emotion that is influenced by multiple bodily systems responsive to internal and external events (Craig, 2003). More comprehensive and realistic views of pain are invaluable to the health care community, which is readily adapting to a multidisciplinary approach through incorporation of systematic methods, such as computer-based-assessments and systems-based-tracking of patient reported outcomes (Swanholm, Lorduy, Noe, & Gatchel, 2012). Such patient-centered approaches are generally preferred by patients who experience greater treatment responsiveness and improved health status as a result (Swanholm, Noe, & Gatchel, 2012).

Likewise, the developing knowledge regarding the involvement of CS in the context of TMD, FM, CFS, and IBS has important implications for how these disorders are perceived within the field and in applied treatment settings (Yunus, 2008). There has been an indisputable agreement that these disorders are part of a larger continuum embodying amplified sensory and affective-motivational components of pain (Clauw, 2009). Yunus (2011) discusses the significance of introducing the term CSS for such disorders in place of terms like “somatoform disorders,” “medically unexplained symptoms,” “functional somatic syndrome,” and “chronic multisymptom illnesses,” which present certain connotations that needlessly imply that the pain

and comorbid symptoms experienced in these cases are purely psychological, and somehow, "not real." CSS conveys certain validity to the origin of these disorders in the wake of extensive research elucidating the physiological underpinnings associated with central sensitization (Yunus, 2012). Recent studies demonstrating the overlap among these disorders and the pathophysiology therewithin are also useful in clarifying the role of CS as a common etiological factor (Aaron, Burke, & Butchwald, 2000; Smart et al., 2012).

In contrast to the term TMJ, TMD is the preferred and more inclusive term for pain or impairment in the orofacial region, which is a diagnostic criterion that distinguishes it from other CSS (Aaron, Burke, & Butchwald, 2000). As such, it is a heterogeneous group of disorders that can be systematically classified using the RDC/TMD to diagnose Axis I: Group I Muscle Disorders, Group II Disc Displacements, Group III Bone Deficiencies, as well as, Axis II psychosocial disorders, such as depression and somatization (LeResche et al., 1992; Truelove et al., 2010). Preliminary data suggests that Group I Muscle Disorders, or myofacial TMD (m-TMD), have a more pronounced relationship with symptoms of CSS compared to the two other groups of Axis I disorders (Lorduy, Dougall, Haggard, & Gatchel, 2012). Where the prevalence of comorbidities within the TMD population is vastly becoming established (Cowley et al., 2011), less progress has been made in defining what accounts for this relationship (Aaron, Burke, & Butchwald, 2000). Therefore, pain investigators are intent on identifying important commonalities, which could potentially improve the efficacy of pain treatment.

Most health care professionals agree that early intervention is key for successful treatment (Gatchel, Polatin, & Mayer, 1995). The onset of pain can spur latent characteristics that interact progressively within a downward spiral without timely intervention (Gatchel, 2004).

Treatment success is also reliant on being able to identify those that are at risk for progressing through the pain stages in the transition from acute to chronic pain (Epker, Gatchel, & Ellis, 1999). Furthermore, it is important to identify resistant barriers to treatment so that they can be addressed. The biopsychosocial model of pain management and rehabilitation involves a systematic evaluation of overall symptomatology, including pain, impairment, and disability, as well as, psychosocial concerns, such as emotional distress, in order to be aware of the full magnitude and breadth of the patient's status (for review see Gatchel, Howard, & Haggard, 2010). This evaluation also allows for a risk assessment to be made and accurate classification of those at low-risk or high-risk for developing chronicity (Epker, Gatchel, & Ellis, 1999). A plethora of evidence suggests the collective benefits of biobehavioral and cognitive- behavioral skills training (CBST) are comparatively more cost- effective and efficacious at post- treatment and at the long- term follow- up within an acute TMD population (Bernstein & Gatchel, 2000; Gardea, 1998; Gardea, Gatchel, & Mirsha, 2001; Phillips et al., 2001).

The present research investigates the effects of an early intervention program on the prevalence of symptoms of TMD and symptoms of CSS. Additionally, we investigated the relationship between the prevalence of symptoms and emotional distress with respect to three different groups of Axis I (RDC/TMD) disorders, while examining the influence of emotional distress. This paper surveys many of the important topics regarding the dynamic interactions between pain-relevant biopsychosocial sequelae. Though the current review is not exhaustive, research demonstrating pain to be a complex multifaceted phenomenon that can manifest through a wide array of possible pathways is discussed. This paper emphasizes the powerful effects of psychosocial factors on the influence of central and peripheral processes involved in pain

processing. The appropriateness of this emphasis is two-fold. First, compared to the long-strides that have to be made before discovery of a "cure," psychosocial variables are good candidates for targeting in treatment as research has revealed well-validated means of identifying and modifying them. This not to say, that the biopsychosocial approach does not consider the physiological aspects involved in pain conditions or within the scope of treatment. Second, psychosocial variables account for a proportionately larger amount of the variance for patient responsiveness (Gatchel & Turk, 2008).

CHAPTER 2

THE EFFECTS OF AN EARLY INTERVENTION PROGRAM ON PHYSICAL SYMPTOMS IN A TMD POPULATION

Although 2.7% of the population meet the criteria for TMD as indicated by the RDC/TMD, roughly half of the population are estimated to have subclinical symptoms (Gersh et al., 2004). Among those with TMD, approximately 70% are women of reproductive age (Carlsson & LeResche, 1995). Musculoskeletal pain disorders pose a serious challenge to health care professionals and incremental economic burden, which equates to nearly $100 billion dollars each year in the United States alone (NIH, 1998). As pain is increasingly becoming conceptualized as a multi-dimensional phenomenon, the study of pain now spans many disciplines. Pain is agreed by most to be, “an unpleasant sensory and emotional experience associated with actual or potential tissue damage” (Merskey & Bogduk, 1994; pp. 210). However, within the context of treatment, pain can more appropriately be viewed as, “whatever the experiencing person says it is, existing whenever the experiencing person says it does” (Pasero & McCaffery, 1999). Self-reports of pain suggest there is a high degree of variability experienced by individuals within the pain population (Edwards & Fillingim, 2006). Where jaw pain is perhaps the most recognized symptom of TMD (Aaron, Burke, & Butchwald, 2000), it is commonly the case that those with this disorder are hypersensitive in areas remote from the orofacial region. Greenspan and colleagues (2011) demonstrated that those with TMD were

sensitive to different types of experimental pain beyond the orofacial region, suggesting the possibility of abnormal central processing of pain.

The dynamic interplay between pain and stress has been suggested as both an etiological consideration and exacerbating factor of pain conditions (Bennaroch, 2006). For instance, those with TMD were comparatively more sensitive to electrocutaneous stimulation and isometric contraction, which corresponded with mean arterial pressure (Mohn, Vassend, & Knardahl, 2008). Maxiner and colleagues (2011) observed those with TMD had altered autonomic function at rest and in response to physical and psychosocial stressors among indices of HPA axis function including heart rate, heart rate variability, blood pressure and baroflex sensitivity. This falls in line with an amounting body of research demonstrating dysregulation of autonomic function within this population (Robinson, Garofalo, & Gatchel, 2006). In extension, prolonged physical or psychological stress has demonstrated the ability to increase important pro-inflammatory cytokines, which contribute to the connectivity between peripheral and central processing of pain (Chapman, Tuckett, & Song, 2008). Meta analyses suggest that relaxation, biobehavioral, and cognitive-behavioral skills training (CBST), are effective methods of reducing self-reports of pain and range of oral opening, as well as, reducing the prevalence of TM symptoms (McNeely, Armijo, & Magee, 2006). Psychoneuroimmunological studies have contributed to our knowledge that these methods operate by reducing stress responding and restoring balance among neuroendocrine and neuroimmune systems (Antoni, 2003).

Understanding the pain-autonomic relationship and its role in TMD and the psychosocial (e.g., emotional distress, depression, anxiety) and physical comorbidities that are often associated with it is essential in structuring more effective pain rehabilitation and management programs

that help to circumvent the transition of acute to chronic pain and possibly the development of chronic widespread pain (Lyon, Cohen, & Quinter, 2011). First, stress is an inevitable part of the adaptive pain response (Craig, 2003). Second, some have suggested it plays a direct role in TMD pathogenesis (Ohrbach & McCall, 1996). Third, it plays a key role in regulating and modulating pain processing through neurochemical mechanisms, as well as, through cognitive-affective appraisals that shape the subjective experience. In the remainder of the paper, we will elaborate on each of these points (Edwards, Campbell, Jamison, & Wiech, 2009; Westman et al., 2011).

2.1 The Adaptive Pain Response

Pain is often considered with respect to its adaptive value (Craig, 2003). In response to a noxious stimulus, acute pain motivates an individual to evaluate the affected area, seek treatment if necessary, and limit activity in order to allow for healing (Bolles & Franslow, 1980; Wall, 1979;). The sympathetic nervous system (SNS) is innervated along the spinothalamic pathway and plays an integral role in processing pain and relaying somatosensory information (Craig, 2003; Donello et al., 2011). Thus, the activation of the autonomic nervous system is an inevitable consequence of nociception, or the neurochemical activity among nociceptors (i.e., C-fibers, Aδ-fibers, and large-area touch fibers) in response to substantive mechanical, thermal, or chemical stimulation in the periphery (Julius & Basbaum, 2001), and is constituted by the release of catecholamines, norepinephrine (NE) and epinephrine (E), from the adrenal medulla, which lead to the characteristic features of “fight-or-flight” (Cannon, 1914). Upon noxious stimulation, nociceptors release substance P (SP), a prominent neuropeptide that acts in the transmission of nociceptive activity. However, NE also initiates the release of SP (Lyon, Cohen, & Quinter,

2011). Both stress and nociception contribute to a profound and subsequent release of pro-inflammatory cytokines upon activation (Lyon, Cohen, & Quinter, 2011).

Where the SNS provides a more immediate response, the hypothalamic pituitary adrenal (HPA) axis serves as an additional "back-up" to homeostatic challenge (Selye, 1964). Both physical and psychological events can elicit the release of corticotrophin-releasing hormone (CRH) from the paraventricular nucleus (PVN) in the hypothalamus leading to a subsequent release of adrenocorticotropin hormone (ACTH), which acts on the adrenal cortex to release glucocorticoids in CNS and cortisol in the periphery. The HPA axis is a crucial intermediary involved in the regulation of the neuroendocrine, neuroimmune, and neurotransmitter systems (Blackburn-Munro & Blackburn-Munro, 2001). It is also important in down-regulating inflammation (Ordway, Klimek, & Mann, 2010) and modulates glutamate NMDA-stimulated activity from the hippocampus. The HPA axis also shares a reciprocal influence on 5-HT and NE (Blackburn-Munro & Blackburn-Munro, 2001), and is activated by cytokines, such as IL-1β, IL-6, TNF-α, and IL-1 (Hestad, Aukrust, Tonseth, & Reitman, 2009).

Generally, the effects of both acute and chronic stress, apart from traumatic contexts, are pain-facilitating via modulation of the descending pathway, which will be elaborated on later in the paper. Blackburn-Munro and Blackburn-Munro (2001) illustrate the dynamic interactions among neural structures and neurotransmitters involved in pain-autonomic processing, which are often altered as a result of prolonged pain or chronic stress exposure. Others have referred to this neural circuitry as the neuromatrix (Melzack, 2001). This conceptualization of pain is the culmination of many theories which have sought to define the physiological basis for pain that emerges irrespective of an instantiating stimulus, such as chronic pain that "persists beyond the

normal time of healing" (Mersky & Bogduk, 1994, pp. 210), and phantom pain, or the sensations experienced by amputees from their missing limb (Vase et al., 2011). Thus, the notion that the conscious perception of pain corresponded to collaborative activity among brain substrates that could be evoked with or without spinal input continues to receive growing support (Melzack, 2001; Becker & Schweinhardt, 2011). Moreover, augmented pain-autonomic function as a result of changes to features in the CNS is thought to be a common etiological factor among CSS (Yunus, 2007; Yunus, 2012).

2.2 Etiological Considerations of TMD

In the absence of nerve injury and with proper coping and pain management, acute TMD should dissipate over time. However, pain exposure can exploit genetic predispositions for developing psychosocial comorbidities (Bruns & Disorbio, 2005; Caspi et al., 2003). In other cases, premorbid psychosocial issues are regarded as etiologic (De Leeuw et al., 1994; Ohrbach & McCall, 1996). While many have attempted the "chicken-or-egg" debate (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997) the existence of comorbid psychosocial features, including psychiatric and personality disorders, are a very certain risk factor for the development of chronic TMD (Gatchel, 2004). Among those with a musculoskeletal disorder, psychosocial comorbidities have been estimated at 64.1%, while comorbid pain conditions are estimated at roughly 68.8% (Dersh et al., 2006; Dersh, Gatchel, Polatin, & Mayer, 2002). The OPPERA study identified genetic risk factors suggesting that those with TMD have a predisposition for maladaptive processing of DA, 5-HT, opioid, and cholinergic neurotransmitter systems (Smith et al., 2011). These systems are important in pain transmission, but are also implicated in HPA axis regulation (Blackburn-Munro & Blackburn-Munro, 2001). Moreover, catecholaminergic

function is important in depression and anxiety, which are the two primary psychosocial concerns implicated in TMD (Dersh et al., 2004; Dersh, Gatchel, Polatin, & Mayer, 2002). Additionally, dysregulation of the immune system has also determined to have a genetic basis in this population (Smith et al., 2011).

Elevations in inflammatory cytokines have been reliably observed within the TMD population (Campos, Campos, & Line, 2006). In response to acute, or time-limited stressors (physical or psychological), immune factors are redistributed into the periphery (Dougall & Baum, 2001). Once activated, systemic increases in proinflammatory cytokines, such as tumor-necrosis factor alpha (TNF-α) and interleukin- 6 (IL-6) among others, are observed. Chronic stress is defined as an excess in number of negative events within a six month time period (Dougall & Baum, 2001), and has been associated with an increase in IL-6 (Kiecolt-Glaser et al., 2003). Brief or mild episodes of depression are denoted by similar changes in immunological function (Hestad, Aukrust, Tonseth, & Reitan, 2009). Moreover, repeated social defeat induced hyperalgesia via neuroinflammation, which was reversed through administration of a cholecystokinin (CCK) antagonist directly into the rostro ventromedial medulla (RVM) (Rivat et al., 2010). The RVM is an important brain substrate comprised of “on cells” and “off cells” that are sensitive to neuropeptides and neurotransmitters including endogenous and pharmacological opioids (Bannister, Lee, & Dickenson, 2009). Moreover, in response to chronic stress CCK appears to be inversely related to the production of neuropeptide Y from the hypothalamus in response to mild chronic stress and induces depressive and anxiety-related behaviors (Kim et al., 2003). Additionally, sustained levels of cytokines, namely those that are commonly elevated among those with TMD and among chronically stressed individuals (IL-6, IL-1, TNFα), can act

on the hypothalamus and augment CRF inducing HPA axis dysregulation to bring about consequent dysregulation among neuroendocrine, neurotransmitter, and neuroimmune systems involved in pain regulation and depressive symptomatology (Anisman, 2008; Black, 1994; Campos, Campos, & Line, 2006). HPA axis hyperactivity has been implicated in certain types of depression, panic disorder, obsessive-compulsive disorder, and alcoholism, where HPA axis hypoactivity has been implicated in other types of depression, FM, and CFS (Gameiro, Andrade, Nouer, & Veiga, 2006). HPA axis dysfunction is associated with hypersensitivity (Lariviere & Melzack, 2000). Moreover, others have found cortisol to be protective during experimental pain (Michaux, Magerl, Anton, & Treede, 2012).

Euteneuer and colleagues (2010) demonstrated the interaction between emotional distress and immunological factors that bring on hypersensitivity to pain. Specifically, they found that depressed individuals had higher levels of TNF-α and this demonstrated a robust correlation with pressure pain thresholds suggesting that one of the ways in which depression contributes to pain pathology is through dysregulation of the autonomic and immunological systems. Furthermore, longitudinal studies have demonstrated inflammatory cytokines are associated with a number of maladaptive cognitive-behavioral adjustments and these effects have been validated with animal models (Dantzer et al., 2008). Such behaviors parallel with symptoms of depression including changes in food and water intake, social withdrawal, reduced pleasure in sexual activity, fatigue, and cognitive disturbances (Dantzer et al., 2008). Administration of TNF-α induced hypersensitivity (Sorkin & Doom, 2000), while administration of an antagonist for TNF-α has demonstrated to ameliorate depressive symptoms (Tyring et al., 2006; as seen in Euteneuer et al., 2010). Again, these studies suggest inflammatory cytokines may explain the connectivity among

peripheral and central features involved in hypersensitivity and hyperreactivity (Chapman, Tuckett, & Song, 2008; Lyon, Cohen, & Quinter, 2011).

Beyond disturbances in the regulation of neuroendocrine, neurotransmitter, and neuroimmune systems, there are other peripheral factors that contribute to the hypersensitivity and the development of orofacial pain (Chapman, Tuckett, & Song, 2008; Lyon, Cohen, & Quinter, 2011). Celik and Mutlu (2011) identified a positive relationship between the prevalence of latent trigger points and symptoms on the Beck Depression Inventory (BDI). Prior to the onset of pain, latent trigger points (LTrPs) cause abnormal muscle contraction, poor coordination, and total balance (Simons, Travel, & Simons, 1999; Simons 2004). Moreover, it may contribute to the high prevalence of subclinical signs and symptoms observed in the general population (Gersh et al., 2008). Shah and Gilliams (2008) determined the biochemical milieu of activated trigger points, or myofacial trigger points (MTrPs), which evoke pain and are associated with joint and disk abnormalities, tension-headaches, migraines, complex regional syndrome, and spinal and pelvic pain, etc. (Borg-Stein & Simmons, 2002). Specifically, they found elevations in SP, calcitonin gene reactive protein (CGRP), bradykinin (BK), 5-HT, NE, TNFα, Il-8, IL-6, and IL-β (Shah & Gilliams, 2008). MTrPs cause constant stimulation of nociceptors, perhaps mimicking what many laboratory studies have demonstrated using various methods of repetitive stimulation of afferent nerve fibers reliably evoking peripheral and central sensitization (Fuchs & Peng, 2003; Woolf, 2011). The biochemical properties of the MTrPs suggest that TMD represents the convening influence of multiple bodily systems that lead to changes in motor and sensory neurons that lead to the development of CS (Shah & Gilliams, 2008).

More central to the notion that CS should be considered as an etiological factor, studies have demonstrated the role of inflammatory factors, such as CGRP, are important in a number of events that lead to CS through increased membrane excitably, long-term-potentiation (LTP), morphological changes involved in the synapses of neurons, and disinhibition of neurons in the dorsal horn of the spine (Buldyrev et al., 2006; Latremoliere & Woolf, 2009; Woolf, 2011). CGRP regulates brain derived neurotropic growth factor (BDNF), an important neurotrophin involved in trigeminal neuroplasticity (Buldyrev et al., 2006). All of the afferents in craniomandibular region innervate the trigeminal ganglion in lamina I of the dorsal horn (Craig, 2004). It has also been found that CGRP is released from trigeminal nerves, where it stimulates mitogen activated protein kinases (MAPK), as well as, glial and astrocytes that activate sensory nerves and promote cellular changes that comprise CS (Cady, Glenn, Smith, & Purham, 2011). IL-1 has also demonstrated the ability to induce pain via modulation of NMDA activity in the trigeminal nucleus specifically involved in the processing of deep orofacial input (Guo, Wang, Watanabe, & Shimizu, 2007). Other studies have suggested that changes in cellular activity in the spine leads to subsequent changes among neural substrates in the brain (Craggs et al., 2012). For instance, Younger, Shen, Goddard, & Mackey (2010) identified altered gray matter volume (GMV) in several brain regions that overlap with those observed in other pain populations, such as the anterior cingulate cortex (ACC), thalamus, somatosensory (SM), putamen, hippocampus, midbrain, cerebellum, and limbic system. However, the finding that those with m-TMD had less GMV in the trigeminal sensory pathway most likely distinguishes this population from others and underpins the specificity, or diagnostic criterion, of TMD.

2.3 Biopsychosocial Modulatory Mechanisms

The difference between adaptive and maladaptive pain responding is thought to be a function of key biopsychosocial factors (Gatchel, 2004). Where the previous section discusses psychosocial comorbidities and genetic predispositions as etiological considerations through their influence on the regulation of multiple bodily systems and role in amplified pain processing, this section discusses the indirect influence on the processing of pain through altering brain activity and modulation of the descending pathway (Becker & Schweinhardt, 2012). As such, these factors can also be regarded for their importance in the maintenance of pain and risk for chronicity. In line with this notion, the OPPERA study identified four primary constructs among 21 various psychosocial factors that predicted chronic TMD. These constructs include overall psychological function, affective distress/stress, passive coping, and active coping (Fillingim et al., 2011).

In line with the widely accepted notion that emotional distress occurs when environmental pressure exceeds one's ability to cope (Lazarus, 1974), the West Haven-Yale Multidimensional Pain Inventory (MPI) is commonly used in order to assess an individual's level functioning in social, occupational, and physical domains (Etscheidt & Steiger, 1995). Pain investigators have used the MPI to categorize three groups of copers including, adaptive copers, dysfunctional copers, and interpersonally distressed copers, each of which have demonstrated varying levels of treatment responsiveness (Kerns, & Rudy, & Turk, 1985). Moreover, Gardea (1998) found elevations on the Minnesota Multiphassic Personality (MMPI) scale 2 to be predictive of treatment outcomes, which was further supported by Bernstein and Gatchel (2000). The MMPI-2 is a well-validated measure for assessing 10 different subscales that characterize

different dimensions of mental health and psychopathology including hypochondriasis, depression, hysteria, psychopathic deviate, masculinity/femininity, paranoia, psychoasthenia, schizophrenia, hypomania, and social introversion (Butcher, Dahlstrom, Graham, Tellegen, & Kaemer, 1989). Additionally, a particular pattern of elevation on certain scales within the MMPI-2 referred to as the "disability profile" has been strongly related to poor health outcomes (Haggard, Stowell, Bernstein, & Gatchel, 2008). It is common for this subgroup of individuals to be particularly resistant to medical explanation and reassurance (Bernstein & Gatchel, 2000). These qualities create a number of complexities that are better addressed within the biopsychosocial context. Such research argues for the need for comprehensive treatment of pain conditions.

As pain illness is increasingly becoming understood as a confluence of internal and external influences that affect neuroendocrine, neuroimmune, and cognitive-affective systems (Chapman, Tuckett, & Song, 2008; Lyon Cohen, & Quinter, 2011), evidence validating the biopsychosocial perspective compels a compatible and systematic approach to pain management and rehabilitation (Gatchel, Howard, & Haggard, 2010). Such an approach targets maladaptive pain cognitions and behaviors with the knowledge that these factors modulate important pain-autonomic interactions involved in pain processing (DeCharmes et al., 2005; Gatchel et al., 2007; Robinson, Garofalo, & Gatchel, 2006). For example, catastrophizing is an overly pessimistic appraisal that is highly associated with depression and poorer physical health (Sullivan & Neish, 1998). In fact, catastrophizing 10 weeks prior to surgery was predictive of post-operative self-reports of pain and pain-related disability (Sullivan, 2001). Catastrophizing is considered a negative orientation or mind- set that is characterized by an amplified perception of

pain (Westman et al., 2011). For example, it accounts for approximately 31% of the variance for pain ratings (Sullivan & D' Eon, 1990). The negative expectations that catastrophizers hold can be observed in statements that overestimate potential threat in relatively benign settings (e.g., dental clinics), such as "I wonder if something serious may happen" (Chaves & Brown, 1987). Moreover, it has been demonstrated in functional magnetic resonance imaging (fMRI) studies that this cognitive appraisal enhances pain perception much like anticipation or nocebo effects have been suggested to enhance pain perception (Gracely, 2004). Specifically, they found that apart from depression, catastrophizing augments the perception of pain through altering activation of regions in the brain responsible for anticipation to pain (medial frontal cortex, cerebellum), attention to pain (dorsal ACC, dorsolateral PFC), as well as, in the emotional aspects of pain through activation of the claustrum, which is interacts with the amygdale (Gracely, 2004). Additionally, the level of catastrophizing corresponded to pain-relevant brain activity (amygdale, ACC) to electrical shock (Seminowicz & Davis, 2006). Moreover, it appears that polymorphisms on the COMT gene, a gene involved in dopamine processing, are highly related to catastrophizing and degree of pain severity (George et al., 2008), as are high levels of stress and inflammation (Hestad et al., 2009).

Many pain enhancing cognitions and behaviors do not directly influence spinal reflexes (France 2002; as seen in Edwards et al., 2009), but instead, influence pain perception through modulation of supraspinal input via the descending pathway in what is referred to as "top-down" modulation of pain (Tracey, 2012). Top-down modulation is achieved by the activity of higher cortical brain areas, such as the PFC and OFC, on lower order brain substrates, such as the ACC, which is involved in pain processing and pain modulation, thalamus, amygdale, and midbrain

regions. The periageductal gray (PAG) and RVM have been strongly implicated in pain-inhibitory and pain- facilitory pain processing serving as an interface between ascending spinal impulses and descending supraspinal mediators (Tracey, 2012). The interactions among these brain regions explain the pain enhancing effects of catastrophizing, as well as, how prior experiences, mood and emotional states, as well as, environmental cues influence the perception of pain (Bingel & Tracey, 2008). Atlas, Bolger, Lindquist, and Wager, (2010) investigated the mediating effects of cues on self-reports of pain and brain activity. They found that cues mediated pathways involved in pain sensation (lateral pathway) and the affective-motivational aspects of pain (medial pain pathway). Specifically, cues influenced the perception of pain by modulating the ACC, insula, and thalamus. The cues were suggested to influence pain perceptions through evoking anticipatory activity in the OFC and striatum (Atlas et al., 2010).

The Gray-McNaughton (2000) theory of anxiety supports the notion that emotionally salient cues are processed in such a way so as to facilitate learning, or the pairing of those cues with consequential events. Moreover, CCK is elevated during stress and is thought to aid in memory consolidation (Gulpinar & Yegen, 2004). Evolutionarily, this is thought to be advantageous (Gray-McNaughton, 2000). Also, when anxiety provoking cues are available, pain processing from the hippocampus becomes amplified (Delgado & Jaffe, 2011). In line with this theory and supporting research, one study investigated modulation of self-reports of pain through inducing mood states prior to pain exposure (Roy et al., 2009). Displays of pleasant and unpleasant pictures modulated pain perception through differential levels of brain activity in the insula, paracentral lobule, parahippocampus, thalamus, and amygdale. Moreover, they demonstrated that the perception of pain was also influenced by modulation of the connectivity

of frontal and subcortical structures. Specifically, interactions between the PFC, parahippocampus, and brainstem played a large part of the modulation of pain perception by mood states (Roy et al., 2009). Moreover, mood and affective states can influence monoamine function to influence neurotransmission along the pain pathway (Ziegler & Herman, 2002; Becker & Schweinhardt, 2012). Suzuki and colleagues (2002) demonstrated that 5-HT supraspinal input from the descending pathway is important in determining membrane excitability in the dorsal horn of the spine. Donello and colleagues (2011) demonstrated the importance of NE in descending inhibition of pain. Blackburn-Munro and Blackburn-Munro (2001) present a model demonstrating the interrelatedness among neuroendocrine, neurotransmitter, and neuroimmune systems in the processing of pain and the deleterious effects of chronic stress and allostatic load on key intermediaries, such as the HPA axis.

2.4 Pain stages: The transition from acute to chronic pain

The above outlined biopsychosocial risk factors influence the likelihood that an individual will either follow along an adaptive path leading to recovery, or a maladaptive path leading to chronicity. The latter has been presented in a model by Gatchel (2004). As described, it is common for individuals to have mild levels of fear and state anxiety, which is distinct from trait anxiety in pathological conditions, at stage 1 upon pain onset. Emotional regulation and behavior modification through provision of an educational framework, CBST, and biobehavioral modalities are effective in promoting recovery if addressed early (Epker, Gatchel, & Ellis, 1999). However, without conscientious efforts to incorporate appropriate coping and pain management strategies with symptom onset, pain can persist over the course of several months leading to stage 2 (Gatchel, 2004). Among the myriad of psychosocial issues at this stage, learned

helplessness, depression, and anxiety are particularly concerning with depression being the most prevalent psychiatric disorder among pain populations (Dersh et al., 2006). After several months of increased or sustained pain and stress responding, the pain condition becomes progressively debilitating, and resources for coping are depleted leading to further dysregulation among multiple bodily systems and changes in central features in the CNS that characterize CS (Shah & Gilliams, 2008). At stage 3, the patient becomes chronic and adopts a "sick role," wherein problematic pain behaviors are incidentally reinforced through secondary gain of compensation and excuse from roles and responsibilities from those in the experiencing individual's social network (Gatchel, 2004).

2.5 Biopsychosocial Approach to Pain Management

Over the past couple of decades, there has been a growing trend for patients to seek additional treatment outside of the medical setting (McCarberg & Passik, 2005). Despite its growing popularity, pain patients are reluctant to disclose utilization of alternative services to physicians. Twenty-eight percent of patients indicated using additional, alternative treatment, although only 63% reported not informing their family health practitioner (McCarberg & Passik, 2005). Ethical concerns regarding the efficacy of non-traditional treatments are another point of contention within the health community. Issues of liability are a consideration by physicians, although, if the patient is receiving additional care without the physician's awareness or without their referral, this concern is relatively minimal. Part of the dislike for non-traditional approaches by physicians has to do with the fact that such approaches are assumed to operate without an awareness of the specific mechanisms involved in achieving its non-specific effects (McCarberg & Passik, 2005). For instance, biobehavioral, meditation, massage, among others, are observed

to have stress-reducing effects (McCarberg & Passik, 2005). The effects of stress reduction reverberate with respect to patient reporting of symptoms, mood, frequency of illness, immunological function, and self- reports of pain and pain-related interference (McCarberg & Passik, 2005). All of these are sufficient and characteristic indicators of treatment response and success.

In response to the need for more effective pain treatments for musculoskeletal disorders, such as temporomandibular disorder (TMD), there has been a growing appreciation for Interdisciplinary Pain Rehabilitation Programs (IPRPs) that incorporate the biopsychosocial model (BPS) (Turk, Loeser, & Monarch, 2002). The BPS model addresses the dynamic interactions between physical factors (e.g., pain) and psychosocial factors (e.g., emotional distress) (for review see Gatchel, Howard, & Haggard, 2010). Despite the mounting evidence to support its efficacy, there remains some reluctance in the assimilation of IPRPs since 1979 when the World Health Organization (WHO) reported on the limitations of the traditional, biomedical approach to treatment of pain illness saying, "The medical model provides an efficient approach to disorders that can be prevented or cured- the impact of illness is relieved secondarily as the underlying condition is brought under control- (but) it is incomplete because it stops short of the consequences of disease" (Broose, 2004 pp. 1). This statement highlights the inherent disregard for the distinction between disease and illness in the medical field. Where some do not agree with making such distinctions (Yunus, 2008), wholistic, patient-centered approaches have demonstrated important advantages for disorders, such as TMD (Bernstein & Gatchel, 2000; Gatchel et al., 2007).

The innocuous assumption of the biomedical model is that pain stems from a curable impetus (Brose, 2004). When patients become ill or experience pain, they will commonly seek treatment assuming their healthcare provider can "fix" the problem (Barsky & Klerman, 1983). Though the goals and assumptions of the healthcare provider and the treatment expectations of the patient are not always at the level of awareness, they can complicate the dynamics in the patient-healthcare provider relationship and potentially perpetuate feelings of hopelessness in the patient as their condition shows intermittent patterns of remission and recurrence (Crombez, Eccleston, Baeyens, & Eeleu, 1998). This is because many pain conditions like TMD and FM do not have known causes or cures. The BPS approach to pain and disability operates with the realization of this fact, while recognizing the current function of effective treatment is to serve as a means to facilitate rehabilitation and pain management (Gatchel, 2004; Gatchel et al., 2007). It is for this reason among others that the BPS perspective has been perhaps the most efficacious perspective and approach to treating pain illness (Gatchel & Okifuji, 2006).

One of the advantages of the BPS approach is that it provides an educational framework that helps the patient understand their condition and the scope of their treatment more realistically (Gatchel, 2004; Gatchel et al., 2007). For example, the BPS model explains how the dynamics of "real" physiological factors including increased stress hormones and inflammation interact with cognitive-affective distortions (e.g., catastrophizing) and maladaptive pain behaviors (e.g., fear-avoidance and withdrawal) that promote and exacerbate their pain condition (Edwards, Campbell, Jamison, & Weich, 2009; Westman et al., 2011). The BPS model is useful in validating the impact the condition has on nearly every aspect of the patient's life (Gatchel, 2004; Swanholm, Lorduy, Noe, & Gatchel, 2012). At the same time, the patient is informed on

healthy coping mechanisms and other stress management techniques, made more aware of their endorsement of maladaptive pain cognitions and behaviors, and encouraged to be more proactive. As a result, IPRPs extinguish the false hope for an elusive cure and restore a sense of personal control and utility in the patient (Gatchel, Howard, & Haggard, 2010).

Moreover, the BPS perspective considers the high prevalence of comorbid physical symptoms and psychosocial comorbidities among those in the non-cancer, pain population (Dersh et al., 2006; Dersh, Gatchel, Polatin, & Mayer, 2002;). It has been helpful in understanding how emotional distress acts a primary factor is crucial in the pathogenesis of TMD (Meeus & Nijs, 2007). Additionally, there is a breadth of research regarding the pathways through which pre-existing psychosocial features can increase emotional distress and exacerbate pain pathology (Becker & Schweinhardt, 2011). For instance, the serial-stress system-based view suggests that chronic stress causes neuroendocrine and subsequent neurotransmitter dysregualtion prior to the onset of pain, whereas the serial-transmitter- based view suggests that neurotransmitter dysregulation precedes pain onset and neuroendocrine dysregulation. However, disturbances among features of the pain-autonomic systems could potentially occur simultaneously leading to multiple systems dysregulation as suggested in the parallel view (Lyon, Cohen, & Quinter, 2008; Becker & Schweinhardt, 2011). However these events take place, they represent important commonalities among CSS and are thought to explain many of the overlapping symptoms (Banister, Bee, & Dickenson, 2009). Thus, it is presumed that the beneficial effects of intervening factors, such as proper coping and pain management, are achieved because they help restore balance among these factors and ameliorate comorbid symptoms (Robinson, Garofalo, & Gatchel, 2006).

Where the BPS model has been useful in delineating the pathogenesis of TMD among other pain conditions, implementation of effective intervention early in the course of pain onset is imperative for preventing chronicity and increasing the likelihood of long-term responsiveness (Gatchel, Stowell, Wildenstein, & Riggs, 2006). It is common knowledge that prevention and early intervention are fundamental in treatment success for any condition. The clinical- and cost-effectiveness of the BPS approach and of IPRPs has been, in large part, the result of its ability to identify those at high- risk for developing chronicity and to provide a multimodal intervention while they are in the acute stages of illness (Gatchel & Okifuji, 2006; Keller, Hayden, Bombardier, & van Tudler, 2007). Because of the capacity of the BPS model to address the added complexities of pain illness, it has comparable clinical- and cost- effectiveness at the immediate post- treatment evaluation, and better long-term outcomes over medical treatments alone (Gatchel & Okifuji, 2006; Keller, Hayden, Bombardier, van Tudler, 2007).

2.6 Hypotheses

The purpose of the present research was to investigate the biopsychosocial factors of TMD. We have conducted two studies using selected samples from an overarching protocol conducted by Gatchel and colleagues. The following is an outline of the specific aims and respective hypotheses for Study 1 and Study 2:

Study 1

1. Identified physical symptoms (TMD-specific and TMD-nonspecific) within a sample of individuals with acute TMD.

- H_1= Those with Axis I RDC/TMD Group I Muscle Disorders would have more comorbid physical symptoms than those with Axis I Group II and Group III disorders.

Study 2

2. Examined the influence of two different interventions on physical symptoms, pain, and pain-related disability.

    - H_2= Biobehavioral intervention (BB) would cause more improvement in physical symptoms (TMD- specific and TMD non-specific) compared to self-care (SC).

    - H_3= BB would cause more improvement in pain and pain-related disability compared to SC and LR.

3. Investigated the influence of emotional distress on physical symptoms, pain, and pain-related disability.

    - H_4= Emotional distress would mediate the effects of Intervention on physical symptoms immediately following the intervention (T2).

    - H_5= Emotional distress would mediate the effects of Intervention on pain and pain-related disability at T2.

CHAPTER 3

STUDY 1

3.1 Study 1 Methods

Participants

Participants were included on the basis of having symptoms of acute TMD as defined by the RDC/TMD. Potential participants were excluded if they had had previous occurrences of TMD, or if they had symptoms for longer than six months prior to enrollment in the study. Additionally, participants were excluded if they had a pain-exacerbating chronic illness that could have potentially interfered with accurately assessing or investigating TMD, or if they were younger than 18 years of age. Participants were recruited upon referral from participating health clinics and educational settings within a major metropolitan area. Alternative modes of recruitment included flyers, handouts, personal contact, and academic talks. Upon consenting and screening participants were compensated $20.00 for their time. In addition to any benefits garnished from the intervention, participants received $50.00 at each of the four assessments. If the participants acquired a considerable travel expense for their involvement in the study, they were given a gas card. (Please refer to Table 1 for a flow chart of the sample selections for Study 1).

Measures

Physical symptoms

As described by Garofalo et al. (1998), the Research Diagnostic Criteria (RDC)/TMD is a comprehensive diagnostic evaluation because of its multitaxial classification system. Similar to the Diagnostic Statistical Manual (DSM), the RDC/TMD identifies the physical components on Axis I. Axis I is comprised of three different groups of physical disorders (Derogatis, 1983). Muscle Disorders constitute Group 1, Disc Displacements are designated within Group 2, and other joint conditions (e.g., arthritis, arthralgia, and arthrosis) are included in Group 3 (LeResche, Fricton, Mohl, Sommers, & Truelove, 1992). The Symptoms Checklist is a 138 item inventory of demographic, medical history, and physical symptoms that have commonly been identified in those with 10 different conditions (Aaron, Burke, & Butchwald, 2000). Specifically, these symptoms pertain to CFS, FM, IBS, TMD, chronic headaches, low-back pain, chemical sensitivities, post-concussion syndrome, irritable bladder syndrome, and pelvic pain.

Psychosocial symptoms

Axis II of the RDC/TMD is allocated for psychosocial concerns, as well as, self- reports of pain and pain- related disability. Pain is measured using the Characteristic Pain Inventory (CPI), which has been extensively validated as a viable predictor of patient's outcomes (Derogatis, 1983). This scale measures the average pain, the most intense pain, and the current pain. The Graded Chronic Pain Scale (GCPS) is a seven-item questionnaire that measures pain-related interference in daily activities, recreational (social) activities, and ability to work (Dworkin & LeResche, 1992). Additionally, Axis II includes the Symptom Checklist-90, which provides a global index of psychosocial health (Derogatis, 1983). The SF-36 Health Status

Survey is another well-validated measure of overall health including both physical and psychosocial variables (Ware et al., 1995). There are eight subscales four of which pertain to a physical health (PH) component (Ware et al., 1995). These subscales include physical function, role physical, bodily pain, and general heath. Whereas, the other four subscales including, mental health, role emotional, social function, and vitality pertain to the mental health (MH) component (Ware et al., 1995). The Beck Depression Inventory (BDI) has been widely used and well-validated as a measure of symptoms of depression (Beck, Steer, & Garbin, 1988). Specifically, it assesses negative cognitions about the self, the world, and the future.

Study 1

The purpose of this study was to identify TMD non-specific symptoms of CSS among various groups of Axis I RDC/TMD disorders. This research interest was formed in light of recent evidence demonstrating an apparent overlap between TMD and disorders of CSS, such as CFS, FM, and IBS, to name a few (Aaron, Burke, & Butchwald, 2000; Yunus, 2008). This study serves the goal of identifying which one of the Axis I RDC/TMD disorders has the strongest overlap with symptoms of CSS. Additionally, emotional distress has been consistently observed among those with TMD and CSS and suggested as a primary etiological or exacerbating factor (Blackburn-Munro & Blackburn-Munro, 2001). Therefore, this study also investigates the influence of emotional distress in the prevalence of physical symptoms. It is expected that symptoms of CSS will be more prevalent among those with an Axis I RDC/TMD Group I Muscle Disorder compared to the other Axis I RDC/TMD disorders.

Design

As part of an ongoing protocol by Gatchel and colleagues, the present study conformed to a one-way MANOVA model. The independent variable (IV) for this analysis was Axis I with five levels including no diagnosis, Axis I RDC/TMD Group I, Axis I RDC/TMD Group II, Axis I RDC/TMD Group III, and more than one diagnosis. The prevalence of TMD non-specific symptoms of CSS as indicated by the Symptom Checklist served as the DVs for this analysis. Additionally, we investigated whether emotional distress mediated the relationship between Axis I and TMD non-specific symptoms of CSS once we had redefined the Axis I RDC/TMD groups in order to be more sensitive to which groups of Axis I disorders were more strongly related to these symptoms. For this analysis, we used a MANCOVA with Axis I as the IV with three levels including a no diagnosis group, Axis I RDC/TMD Group I, and Axis I RDC/TMD Group I and Group III combined. Comorbid symptoms of CSS were the DVs and emotional distress was a covariate in this analysis and was indicated by scores on the BDI, as well as, the MH component of the SF-36 Health Status Survey.

Procedure

Upon recruitment, Master's level psychologists specialized in pain management and rehabilitation scheduled the initial screening, informed consent, and the baseline assessment (i.e., T1). Participants were included on the basis of the inclusion/exclusion criteria as specified in the Participants section. Participants were informed of their rights and responsibilities as participants in the study, and provided with a brief description of the study during informed consent. For example, participants were made aware that they would be asked to complete an assessment of their physical and psychosocial symptoms along with personal information pertaining to their

demographic and medical background at four specified time points over the course of their two year involvement in the study. Upon obtaining informed consent and eligibility screening, a risk assessment was conducted using the data from the initial baseline assessment.

Statistical Analysis

The purpose of Study 1 was to identify comorbid symptoms of CSS and investigate the role of emotional distress. For the group comparisons, Holm-Bonferroni corrections were used to reduce Type I error. The appropriateness of this method of Type I correction for our multivariate analyses stems from its capacity to not overcorrect when there are several dependent variables included in the analyses. First, we investigated comorbid symptoms of CSS among various types of Axis I RDC/TMD disorders. In line with existing research demonstrating a high comorbidity of overlapping symptoms among disorders, such as TMD, CFS, FM, and chronic headaches, we investigated the prevalence of symptoms of CSS across several groups of TMD disorders including those with no Axis I diagnosis, those with a Group I Muscle Disorder, those with a Group II Disc Displacement, those with a Group III Bone deficiency, and those with more than one diagnosis. For this analysis, we conducted a multivariate analysis of variance (MANOVA) with Axis I as the independent variable (IV) and symptoms of CSS as the dependent variables (DV)s. The outcomes for these analyses were observed at baseline (T1) in order to observe the prevalence of comorbid symptoms and its relationship with emotional distress with respect to three different types of Axis I RDC/TMD disorders prior to the effects of Intervention.

Having established that the prevalence of comorbid symptoms of central sensitivity syndrome (CSS) were more prevalent among those with an Axis I Muscle Disorder and those with more than one diagnosis and having determined that those that had more than one diagnosis

were primarily those with an Axis I RDC/TMD Group I Muscle Disorder and a Group III Bone Deficiency, we refined our group comparisons in effort to more adequately investigate the observed relationships in symptomatology. In order to identify the prevalence of comorbid symptoms of central sensitivity syndrome (CSS) among those with no diagnosis, those with only an Axis I RDC/TMD Group I Muscle Disorder, and those with a Group I Muscle Disorder and a Group III Bone Deficiency, we conducted a MANOVA with Axis I as the IV with three levels including no diagnosis, Axis I Group I Muscle Disorder, and Axis I Group I and Group III disorder combined) and symptoms of CSS as the DVs. Having investigated these aims at T1 prior to intervention, we are able to interpret these findings with respect to our first aim of identifying which Axis I RDC/TMD disorder is most strongly related to symptoms of CSS.

In a continued effort to establish the nuances of comorbid symptoms with respect to Axis I RDC/TMD disorders and to clarify the role of emotional distress where this symptomatology is indicated, we conducted the following analyses at baseline in order to interpret our findings prior to examining the effects of Intervention. We will return to our investigation of these analyses across both time points in Study 2 designed for the purpose of our second aim of investigating the effects of Intervention on TMD specific and TMD non-specific symptoms. In effort to investigate whether emotional distress mediated comorbid symptoms of CSS, we conducted a MANOVA to determine whether the requisite relationship between our covariate (CV), emotional distress as indicated by the BDI and SF-36 MHC, and (IV), Axis I, was present in our sample. To do so, we compared emotional distress among those with no diagnosis, those with an Axis I Group I Muscle Disorder, and those with both an Axis I Group I Muscle Disorder and Group III Bone Deficiency. In order to fulfill another requirement prior to mediational analyses,

we conducted bivariate correlations in order to determine whether the requisite relationships between our CVs, emotional distress, and dependent variables DVs including total physical symptoms, symptoms of CFS, FM, chronic headaches, IBS, low back pain, chemical sensitivities, irritable bladder syndrome, post-concussion syndrome, and pelvic pain were present in our sample at both Time points (T1 and T2). For this analysis, we examined the relationship of emotional distress and comorbid symptoms of CSS across both Time points as this analysis serves as a prerequisite for our group comparisons examining the effects of Intervention and mediation of the effects of Intervention by emotional distress in the Study 2. Once we had observed the required relationships between our variables were present in our sample and that there was a multivariate effect for Axis I prior to mediation, we conducted a multivariate analysis of covariance (MANCOVA) with Axis I as the IV with three levels including, those with no diagnosis, those with an Axis I Group I Muscle Disorder, and those with both an Axis I Group I Muscle Disorder and Group III Bone Deficiency. Symptoms of CSS at T1 were the DVs for this analysis so as to allow for interpretation of our first aim prior the influence of Intervention.

3.2 Study 1 Results

TMD Non-Specific Symptoms: Comorbid Symptoms of CSS

All variables were conducted using the appropriate scoring protocol which accounted for missing data using mean imputation. As expected, there were no differences among the demographic variables between our intervention groups. The analyses revealed that the 272 participants included in our sample (see Figure 1 for a flow chart of our sample selection). The current study investigated comorbid symptoms of CSS among various types of Axis I RDC/TMD disorders. In line with existing research demonstrating a high comorbidity of

overlapping symptoms among disorders, such as TMD, CFS, FM, and chronic headaches, we investigated the prevalence of symptoms of CCS across several groups of TMD disorders including those with no Axis I diagnosis, those with a Group I Muscle Disorder, those with a Group II Disc Displacement, those with a Group III Bone deficiency, and those with more than one diagnosis. For this analysis, we conducted a multivariate analysis of variance (MANOVA). As expected, the analysis revealed a multivariate effect for Axis I, $F(44, 1040) = 1.61$, $p < .05$, $\eta^2 = .06$. Univariate analysis demonstrated that Axis I influenced total physical symptoms, $F(4, 267) = 2.40$, $p = .05$, $\eta^2 = .04$. Additionally, we determined that Axis I also influenced symptoms specific to CFS, $F(4, 267) = 3.50$, $p < .05$, $\eta^2 = .05$, FM, $F(4, 267) = 2.91$, $p < .05$, $\eta^2 = .04$, and female pelvic pain, $F(4, 267) = 3.78$, $p < .05$, $\eta^2 = .05$ (see Table 1). Although we had predicted that those with a Group I disorder would have more comorbid symptoms of CSS, the analysis revealed that those with more than one diagnosis had sgnificantly more total comorbid symptoms compared to those with no diagnosis and those with only an Axis I RDC/TMD Group III disorder. In partial support of our expectations, those with more that one diagnosis had significantly more symptoms of CFS than all other groups except those with only an Axis I RDC/TMD Group I disorder. Those with more that one diagnosis had significantly more symptoms of FM than all other groups except those with only an Axis I RDC/TMD Group I disorder. Those with more than one diagnosis and those with an Axis I RDC/TMD Group I disorder had significantly more symptoms of female pelvic disorder compared to those with no diagnosis. Also, those with an Axis I Group I disorder had more symptoms of chemical sensitivities compared to those with no diagnosis.

Having established that the prevalence of comorbid symptoms of central sensitivity syndrome (CSS) were more prevalent among those with an Axis I Muscle Disorder and those with more than one diagnosis and having determined that those that had more than one diagnosis were primarily those with an Axis I RDC/TMD Group I Muscle Disorder and a Group III Bone Deficiency, we refined our group comparisons in effort to more adequately investigate the observed relationships in symptomatology. In order to identify the prevalence of comorbid symptoms of central sensitivity syndrome (CSS) among those with no diagnosis, those with only an Axis I RDC/TMD Group I Muscle Disorder, and those with a Group I Muscle Disorder and a Group III Bone Deficiency, we conducted a MANOVA. As expected, the analysis revealed a multivariate effect for Axis I, $F(22, 332) = 2.45$, $p < .05$, $\eta^2 = .14$. Univariate analysis demonstrated that Axis I influenced total physical symptoms, $F(2, 175) = 4.67$, $p < .05$, $\eta^2 = .05$. Additionally, we determined that Axis I also influenced symptoms specific to CFS, $F(2, 175) = 5.79$, $p < .05$, $\eta^2 = .06$, FM, $F(2, 175) = 5.46$, $p < .05$, $\eta^2 = .06$, IBS, $F(2, 175) = 3.05$, $p = .05$, $\eta^2 = .03$, low back pain, $F(2, 175) = 3.06$, $p < .05$, $\eta^2 = .03$, post-concussion syndrome, $F(2, 175) = 3.55$, $p < .05$, $\eta^2 = .04$, and female pelvic pain, $F(2, 175) = 6.69$, $p < .05$, $\eta^2 = .07$ (see Table 2). We expected that those with an Axis I Group I and Group III would have more symptoms compared to those with only a Group I disorder and those with no diagnosis. In line with our expectations, those with both Axis I disorders had more total comorbid symptoms, as well as symptoms of CFS, FM, chronic headaches, and low back pain, compared to those with only an Axis I Group I disorder and those with no diagnosis. Additionally, those with both disorders had more symptoms of IBS compared to those with no diagnosis. Those with an Axis I Group I disorder had more symptoms of female pelvic disorder compared to those with no diagnosis. In

contrast to our expectations, those with no diagnosis had more symptoms of post-concussive syndrome compared to the other two groups.

Figure 1. Sampling and Flow of Participants through an Randomized Early Intervention Study

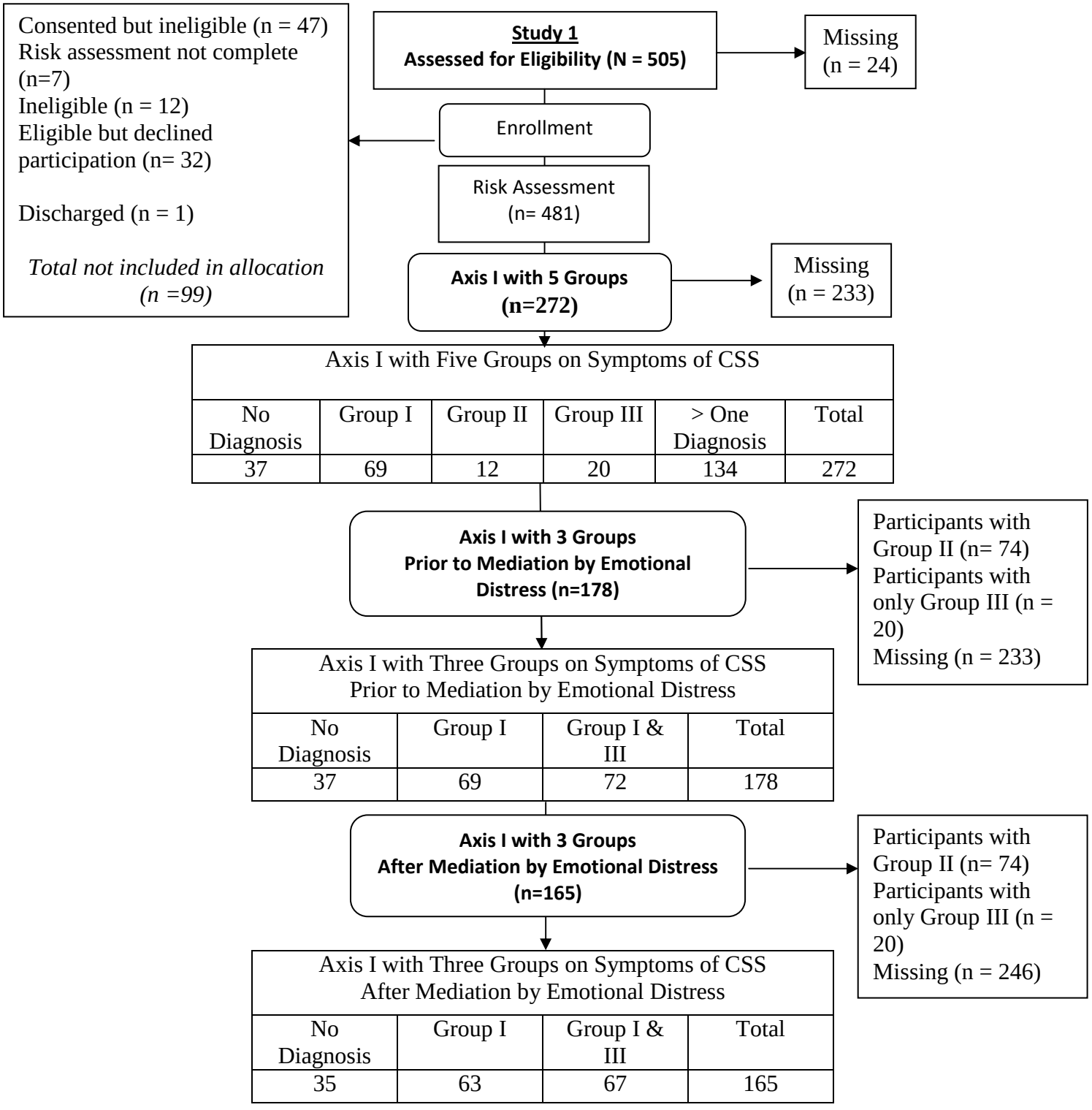


Table 1. Mean Proportion of Symptoms of CSS

as a Function of Axis I with Five Groups of Disorders

	Axis I RDC/TMD Disorders With Five Groups (n = 272)				
	No Diagnosis (n = 37) M (SE)	Group I (n= 69) M (SE)	Group II (n= 12) M (SE)	Group III (n= 20) M (SE)	> One Diagnosis (n= 134) M (SE)
Total Physical Symptoms	.19 (.02)	.22 (.02)	.17 (.04)	.17 (.03)	.24 (.01)*
CFS	.30 (.04)	.36 (.03)	.26 (.07)	.29 (.05)	.41 (.02)*
FM	.29 (.04)	.36 (.03)	.25 (.07)	.29 (.05)	.39 (.02)*
Chronic Headaches	.13 (.05)	.12 (.03)	.13 (.08)	.11 (.06)	.19 (.02)
Low Back Pain	.08 (.04)	.11 (.03)	.19 (.07)	.06 (.06)	.14 (.02)
IBS	.18 (.05)	.23 (.04)	.10 (.08)	.11 (.07)	.24 (.03)
Chemical Sensitivities	.02 (.02)	.08 (.02)*	.07 (.04)	.05 (.03)	.05 (.01)
Irritable Bladder	.08 (.03)	.11 (.02)	.08 (.04)	.09 (.03)	.11 (.01)
Post-Concussion	.23 (.05)	.10 (.03)	.11 (.08)	.09 (.06)	.11 (.02)
Female Pelvic Pain	.06 (.03)	.20 (.02)*	.18 (.05)	.11 (.04)	.17 (.02)
Male Pelvic Pain	.03 (.01)	.01 (.01)	.01 (.02)	.01 (.01)	.02 (.01)

Note. The table above displays a main effect for Axis I on comorbid symptoms of CSS. Although we had predicted that those with a Group I disorder would have more comorbid symptoms of CSS, the analysis revealed that those with more than one diagnosis had sgnificantly more total comorbid symptoms compared to those with no diagnosis and those with only an Axis I RDC/TMD Group III disorder. In partial support of our expectations, those with more that one diagnosis had significantly more symptoms of CFS than all other groups except those with only an Axis I RDC/TMD Group I disorder. Those with more that one diagnosis had significantly more symptoms of FM than all other groups except those with only an Axis I RDC/TMD Group I disorder. Those with more than one diagnosis and those with an Axis I RDC/TMD Group I

disorder had significantly more symptoms of female pelvic disorder compared to those with no diagnosis. Also, those with an Axis I Group I disorder had more symptoms of chemical sensitivities compared to those with no diagnosis.

Table 2. Mean Proportion of Symptoms of CSS as a Function

of Axis I with Three Groups of Disorders Prior to Mediation by Emotional Distress

	Axis I RDC/TMD Disorders With Three Groups (n = 178)		
	No Diagnosis (n = 37) M (SE)	Group I (n= 69) M (SE)	Group I & III (n= 72) M (SE)
Total Physical Symptoms	.19 (.02)	.22 (.02)	.28 (.02)*
CFS	.30 (.04)	.36 (.03)	.45 (.03)*
FM	.29 (.04)	.36 (.03)	.44 (.03)*
Chronic Headaches	.13 (.05)	.12 (.03)	.22 (.03)*
Low Back Pain	.08 (.04)	.11 (.03)	.20 (.03)*
IBS	.18 (.05)	.23 (.04)	.32 (.04)*
Chemical Sensitivities	.02 (.02)	.08 (.02)	.07 (.02)
Irritable Bladder	.08 (.03)	.11 (.02)	.13 (.02)
Post- Concussion	.23 (.05)*	.10 (.03)	.10 (.03)
Female Pelvic Pain	.06 (.03)	.20 (.02)*	.18 (.02)
Male Pelvic Pain	.03 (.01)	.01 (.01)	.02 (.01)

Note. The table above displays a main effect of Axis I with three groups of disorders on comorbid symptoms of CSS. We expected that those with an Axis I Group I and Group III would have more symptoms compared to those with only a Group I disorder and those with no diagnosis. In line with our expectations, those with both Axis I disorders had more total comorbid symptoms, as well as symptoms of CFS, FM, chronic headaches, and low back pain, compared to those with only an Axis I Group I disorder and those with no diagnosis. Additionally, those with both disorders had more symptoms of IBS compared to those with no diagnosis. Those with an Axis I Group I disorder had more symptoms of female pelvic disorder

compared to those with no diagnosis. In contrast to our expectations, those with no diagnosis had more symptoms of post-concussive syndrome compared to the other two groups.

Emotional distress

In order to investigate the theory that symptoms of TMD and symptoms of CSS are related and are thought to be promoted and maintained by emotional distress, we conducted the following analyses in order to better understand the influence of emotional distress. In effort to investigate whether emotional distress mediated comorbid symptoms of CSS, we conducted a MANOVA in order to determine whether the requisite relationship between our covariate (CV), emotional distress as indicated by the BDI and SF-36 MH at T1, and (IV), Axis I, was present in our sample. To do so, we compared emotional distress among those with no diagnosis, those with an Axis I Group I Muscle Disorder, and those with both an Axis I Group I Muscle Disorder and Group III Bone Deficiency. We found support for our expectation for group differences, $F(4, 344) = 2.65$, $p < .05$, $\eta^2 = .03$, such that those with a Muscle Disorder and a Bone Deficiency had more symptoms of depression on the BDI, $F(2, 172) = 5.06$, $p < .05$, $\eta^2 = .06$, and lower mental health on the SF-36 MH, $F(2, 172) = 3.27$, $p < .05$, $\eta^2 = .04$, compared to those with no diagnosis (see Figures 2 and 3).

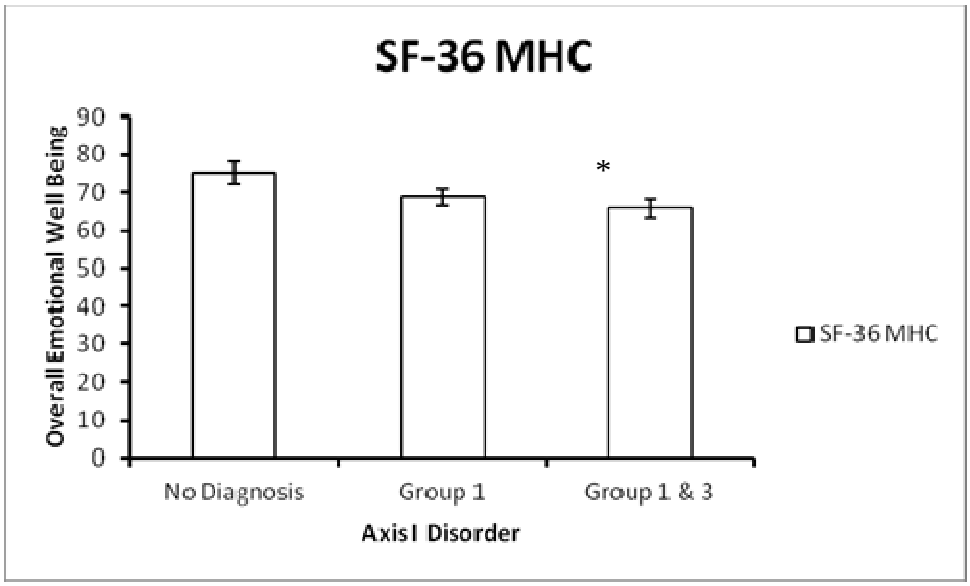


Figure 2. Overall Emotional Well-Being as a Function of Axis I wth Three Groups of Disorders

Note. The figure above displays a main effect for Axis I with three groups of disorders for overall emotional well-being. Higher scores indicate better overall mental well-being. Specifically, those with more than one diagnosis had significantly less emotional well-being compared to those with no diagnosis.

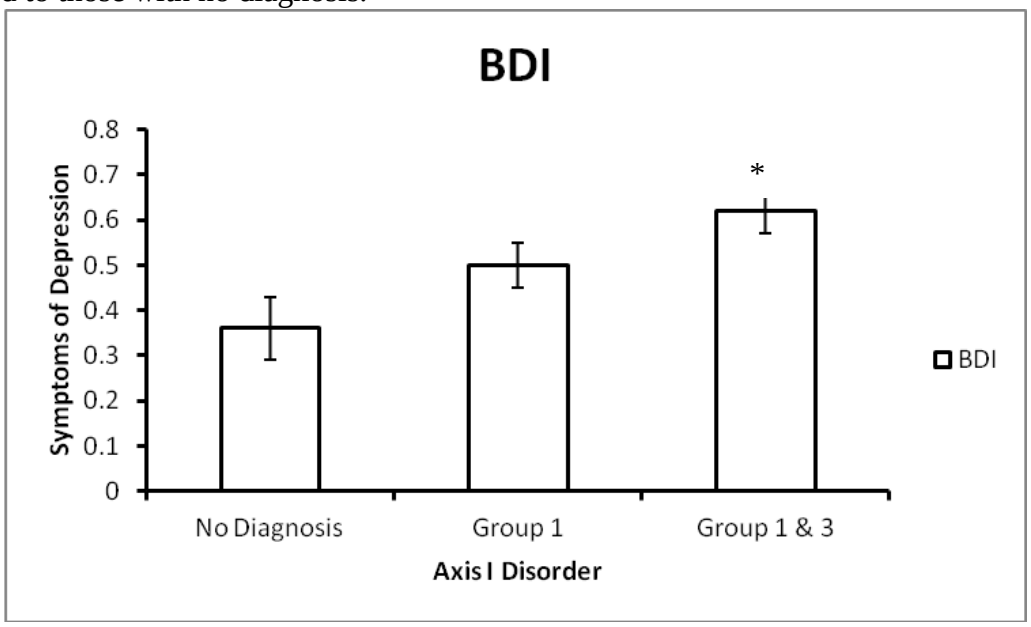


Figure 3. Symptoms of Depression as a

Function of Axis I with Three Groups of Disorders

Note. The above figure displays a main effect of Axis I with three groups of disorders for symptoms of depression. Higher scores indicate greater symptoms of depression. Those with more than one diagnosis had significantly more symptoms of depression compared to those with no diagnosis.

In order to fulfill another requirement prior to mediational analyses, we conducted bivariate correlations in order to determine whether the requisite relationships between our CVs, emotional distress, and dependent variables DVs including total physical symptoms, symptoms of CFS, FM, chronic headaches, IBS, low back pain, chemical sensitivities, irritable bladder syndrome, post-concussion syndrome, and pelvic pain were present in our sample at both Time points (T1 and T2). As expected, there was a strong positive relationship between symptoms of CSS and BDI ($r=.57$; $p< .05$) indicating a higher prevalence of symptoms corresponds to higher prevalence in symptoms of depression and a strong negative relationship with SF-36 MHC ($r=-.47$; $p< .05$) suggesting lower mental health corresponded to a higher prevalence of symptoms at Time 1. We observed similar trends at Time 2 for BDI ($r=.56$; $p< .05$) and SF-36 MHC ($r=-.34$; $p< .05$). For a display of these analyses, please refer to Table 3.

Table 3. Pearson Correlation Coefficients for

Symptoms of CSS and Emotional Distress Over Time

	BDI (T1)	BDI (T2)	MH (T1)	MH (T2)
Total Physical Symptoms (T1)	r= .57* p = .00	r= .44* p = .00	r = .47* p = .00	r = .24* p = .01
Total Physical Symptoms (T2)	r= .47* p = .00	r= .56* p = .00	r = .45* p = .00	r = .34* p = .00
CFS (T1)	r= .59* p = .00	r= .46* p = .00	r = .48* p = .00	r = .30* p = .00
CFS (T2)	r= .48* p = .00	r= .56* p = .00	r = .44* p = .00	r = .43* p = .00
FM (T1)	r= .63* p = .00	r= .45* p = .00	r = .50* p = .00	r = .34* p = .00

Table 3. – continued

FM (T2)	r= .52*	r= .61*	r = .48*	r = .44*
	p = .00	p = .00	p = .00	p = .00
IBS (T1)	r= .34*	r= .24*	r = .34*	r = .15
	p = .00	p = .01	p = .00	p = .08
IBS (T2)	r= .27*	r= .24*	r = .30*	r = .18*
	p = .00	p = .01	p = .00	p = .04
Low Back Pain (T1)	r= .27*	r= .17*	r = .19*	r = .11
	p = .00	p = .05	p = .03	p = .21
Low Back Pain (T2)	r= .35*	r= .43*	r = .31*	r = .14
	p = .00	p = .00	p = .00	p = .10
Irritable Bladder	r= .23*	r= .17*	r = .19*	r= .02
Syndrome (T1)	p = .01	p = .05	p = .03	p = .81
Irritable Bladder	r= .03	r= .18*	r = .13	r= .01
Syndrome (T2)	p = .71	p = .03	p = .14	p = .88
Chemical Sensitivities	r= .29*	r= .47*	r = .38*	r = .20*
(T1)	p = .00	p = .00	p = .00	p = .02
Chemical Sensitivities	r= .33*	r= .30*	r = .35*	r = .07
(T2)	p = .00	p = .00	p = .00	p = .40
Post-Concussive	r= .13	r= .11	r = .02	r= .07
Syndrome (T1)	p = .14	p = .22	p = .83	p = .44
Post-Concussive	r= .15	r= .15	r = .09	r= .02
Syndrome (T2)	p = .08	p = .10	p = .33	p = .84
Female Pelvic Syndrome	r= .15	r= .09	r = .08	r= .04
(T1)	p = .09	p = .33	p = .34	p = .68
Female Pelvic Syndrome	r= .08	r= .06	r = .02	r = .11
(T2)	p = .36	p = .46	p = .82	p = .21

Note. The above table displays the correlations for symptoms of CSS and the CVs BDI and MH at T1 and T2. The correlation coefficients are provided at both time points with the p values provided below each r value.

Once we had observed the required relationships between our variables were present in our sample and that there was a multivariate effect for Axis I prior to mediation, we conducted a multivariate analysis of covariance (MANCOVA). The analyses revealed an effect for Axis I, $F(22, 302)= 2.33$, $p< .05$, $\eta 2 = .15$, on total comorbid symptoms of CSS. Also, BDI, $F(11, 150)= 3.37$, $p< .05$, $\eta 2 = .20$, and SF-36 MHC, $F(11, 150)= 2.06$, $p< .05$, $\eta 2 = .13$ were significant mediators of Axis I on comorbid symptoms of CSS. Emotional distress partially mediated symptoms of CFS, $F(2, 160)= 3.85$, $p< .05$, $\eta 2 = .05$, symptoms of post concussive syndrome,

$F(2, 160)= 5.63$, $p< .05$, $\eta 2 = .07$, and female pelvic disorder, $F(2, 160)= 6.21$, $p< .05$, $\eta 2 = .07$. Specifically, those with an Axis I RDC/TMD Group I and Group III disorder had significantly more symptoms of CFS compared to those with no diagnosis. Those with only an Axis I RDC/TMD Group I disorder and those with an Axis I RDC/TMD Group I and Group III disorder had more symptoms of post concussive syndrome and female pelvic disorder. Also, total physical symptoms, $F(2, 160)= 1.77$, $p> .05$, $\eta 2 = .02$, symptoms of FM, $F(2, 160)= 2.47$, $p> .05$, $\eta 2 = .03$, and symptoms of low back pain, $F(2, 160)= 1.22$, $p< .05$, $\eta 2 = .02$, were fully mediated by emotional distress (see Table 4).

Table 4. Mean Proportion of Symptoms of CSS as a Function of Axis I with Three Groups of Disorders After Mediation by Emotional Distress

	Axis I RDC/TMD Disorders With Three Groups with Emotional Distress (n = 165)		
	No Diagnosis (n = 35) M (SE)	Group I (n= 63) M (SE)	Group I & III (n= 67) M (SE)
Total Physical Symptoms	.22 (.02)	.23 (.02)	.26 (.01)
CFS	.35 (.03)	.36 (.02)	.43 (.02)*
FM	.34 (.03)	.36 (.02)	.41 (.02)
Chronic Headaches	.15 (.04)	.13 (.03)	.19 (.03)
Low Back Pain	.10 (.04)	.12 (.03)	.17 (.03)
IBS	.20 (.05)	.22 (.04)	.31 (.04)
Chemical Sensitivities	.04 (.02)	.07 (.02)	.06 (.02)
Irritable Bladder	.10 (.03)	.10 (.02)	.12 (.02)
Post- Concussion	.27 (.05)*	.11 (.04)	.07 (.04)

Table 4. – continued

Female Pelvic Pain	.06 (.03)	.20 (.02)*	.17 (.02)*
Male Pelvic Pain	.03 (.01)	.01 (.01)	.02 (.01)

Note. The table above displays a main effect of Axis I with three groups of disorders partially mediated by emotional distress. As expected, total comorbid symptoms of CSS, as well as symptoms of FM, chronic headaches, low back pain, and IBS were fully mediated by emotional distress. Additionally, the analyses revealed that those with both disorders had significantly more symptoms of CFS compared to those with only a Group I disorder and those with no diagnosis. Those with only an Axis I RDC/TMD Group I disorder and those with an Axis I RDC/TMD Group I and Group III disorder had fewer symptoms of post- concussive syndrome and more symptoms female pelvic disorder.

CHAPTER 4

STUDY 2

4.1 Study 2 Methods

The purpose of Study 2 was to investigate the effects of Intervention on TMD specific symptoms of Axis I RDC/TMD disorders. It was expected that those in the BB Intervention group will have comparatively fewer TMD specific symptoms, comorbid symptoms of CSS, and pain and pain-related disability at the immediate post-evaluation (T1) compared to the baseline evaluation (T1). Also, we expected emotional distress to mediate the effects of Intervention.

Design

As part of an ongoing protocol by Gatchel and colleagues, the present study conformed to a 2(Intervention: SC-Self-care, BB-Biobehavioral) x 2(Time: T1-baseline and T2-post-intervention) mixed-factorial design. Time (T1 and T2) was the within- subjects independent variable (IV), and Intervention (SC and BB) was the between-subjects IV. The dependent variables (DV) included the prevalence of TMD specific symptoms as indicated by the RDC/TMD: 1), the number of muscle pain sites served as the DV for Axis I Group I Muscle Disorders; 2) the opening pattern, range, and joint sounds with opening served as the DV for Axis I Group II Disc Displacements; and 3) excursions and protrusions served as the DV for Axis I Group III Bone Deficiencies. Scores on the CPI and GCPS were the DV for pain and pain-related disability, respectively. Emotional distress was a covariate in many of the present

analyses and was indicated by scores on the BDI, as well as, the MH component of the SF-36 Health Status Survey.

Procedure

Once the participant was successfully enrolled in the study and had completed the baseline assessment (T1), the participant's pain ratings on the CPI and whether or not they had myofacial pain at two specified muscle sites as indicated by the RDC/TMD was entered into an algorithm developed by Epker, Gatchel, & Ellis (1999). This algorithm reliably classifies those at low- and high- risk for chronicity with a 91% accuracy. Those at low-risk comprised the LR intervention group. Those at high-risk were further randomized into the SC or BB intervention groups. Participants were told that if they decided to participate in the study, they would be asked to complete the assessment, which took approximately one and a half to two hours, at baseline (T1), post-intervention (T2), 12 months post-intervention, and again at 24 months post-intervention. Additionally, they were asked to provide information at three month intervals through telephone interviews with the research personnel. Moreover, they were told that they would be randomly selected to participate in one of three different intervention groups. One of the interventions (LR) was described as consisting of telephone interviews every three months for 24 months, while the other two interventions, SC and BB, were described as involving a series of six one and a half to two hour-long sessions within a six week to three month time period during which time they would be provided with an educational framework and pain management techniques for facilitating recovery.

The present study bases the intervention protocol from previous work conducted by Mirsha and colleagues, Gardea and colleagues, and Gatchel and colleagues as these studies have

been replicated and demonstrated reliability through the combined influence of CBST and biofeedback. As mentioned earlier, certain cognitive-affective factors can potentiate pain through modulation of pain-autonomic responding. The CBST aspect of the intervention targets these variables by challenging participants to become more aware of their endorsement of maladaptive automatic thoughts, pain behaviors, and poor coping. Patients were provided with an educational framework of how "physical," "psychological," and "social" factors interact dynamically in order to promote and maintain pain pathology. The goals and objectives of each of the CBST sessions were geared toward providing the patient with the skills to effectively manage their pain condition (Gatchel et al., 2006).

First, patients were challenged to identify cues and events that triggered arousal and pain (Gatchel et al., 2006). Patients were also asked to note the thoughts and behaviors they engaged in as a result of such triggers. In this way, the individual is made more aware of the specific triggers and pain cognitions that cause them stress and their pain symptomatology to worsen. Patients were also asked to set specific, attainable goals for the short-term and long-term, such as changing food or exercise habits, and rewarding themselves when those goals were met. Keeping track of improvements by assessing the frequency of maladaptive, cognitive-behavioral responses at the beginning of treatment and following improvements over the course of treatment puts the patient in the front seat of managing their condition (Gatchel et al., 2006). Second, patients were trained to distract themselves by focusing on music or things outside of pain. Pleasurable activities are important distracters and that can be used strategically to reward compliance or adaptive coping. Patient's were asked to prepare adaptive cognitive- behavioral response to negative events. Among some of maladaptive cognitions, are automatic thoughts or

appraisals of events including filtering, polarized thinking, overgeneralization, mind reading, catastrophizing, personalization, control fallacies, fallacy of fairness, blaming, should, emotional reasoning, fallacy of change, global labeling, being right, and heaven's reward fallacy. All of these cognitive appraisals involve unrealistic expectations or interpretations of the self, others, or the universe (Gatchel et al., 2006). In addition to enabling the patient to be more proactive in the management of their pain, the biofeedback aspect of the intervention helps restore a sense of personal control through enabling participant observe the influence they have over their physiological responding. This component has also demonstrated reliability and serves as a back-up for some individuals who may be resistant to professional or educational explanations (Bernstein & Gatchel, 2001).

Statistical Analysis

The purpose of Study 2 was to examine the effects of Intervention and investigate the role of emotional distress on TMD specific symptoms of Axis I RDC/TMD disorders. For the group comparisons, Holm-Bonferroni corrections were used to reduce Type I error. The appropriateness of this method of Type I correction for our multivariate analyses stems from its capacity to not overcorrect when there are several dependent variables included in the analyses. As a preliminary step in our analysis, we conducted a MANOVA with Intervention as the between subjects IV and Time as the within subjects IV and BDI and SF-36 MH as the DVs in order to determine whether the required relationship between Intervention and emotional distress for mediational analyses was present in our sample. In order to investigate the effects of Intervention on TMD specific symptoms, we investigated the influence of the two interventions on TMD-specific measures of an Axis I Group I Muscle Disorder, including the 20 different

muscle sites used to assess Axis I Disorders. A MANOVA was conducted with Intervention as the between subjects IV, Time as the within subjects IV, and the 20 muscles sites as the DVs. Although, we did not conduct mediational analyses as we had planned to do because of discovering that there was no relationship between Intervention and emotional distress, we conducted bivariate correlations the 20 muscle sites used to diagnose Axis I Group I Muscle Disorders and emotional distress at T1 and T2.

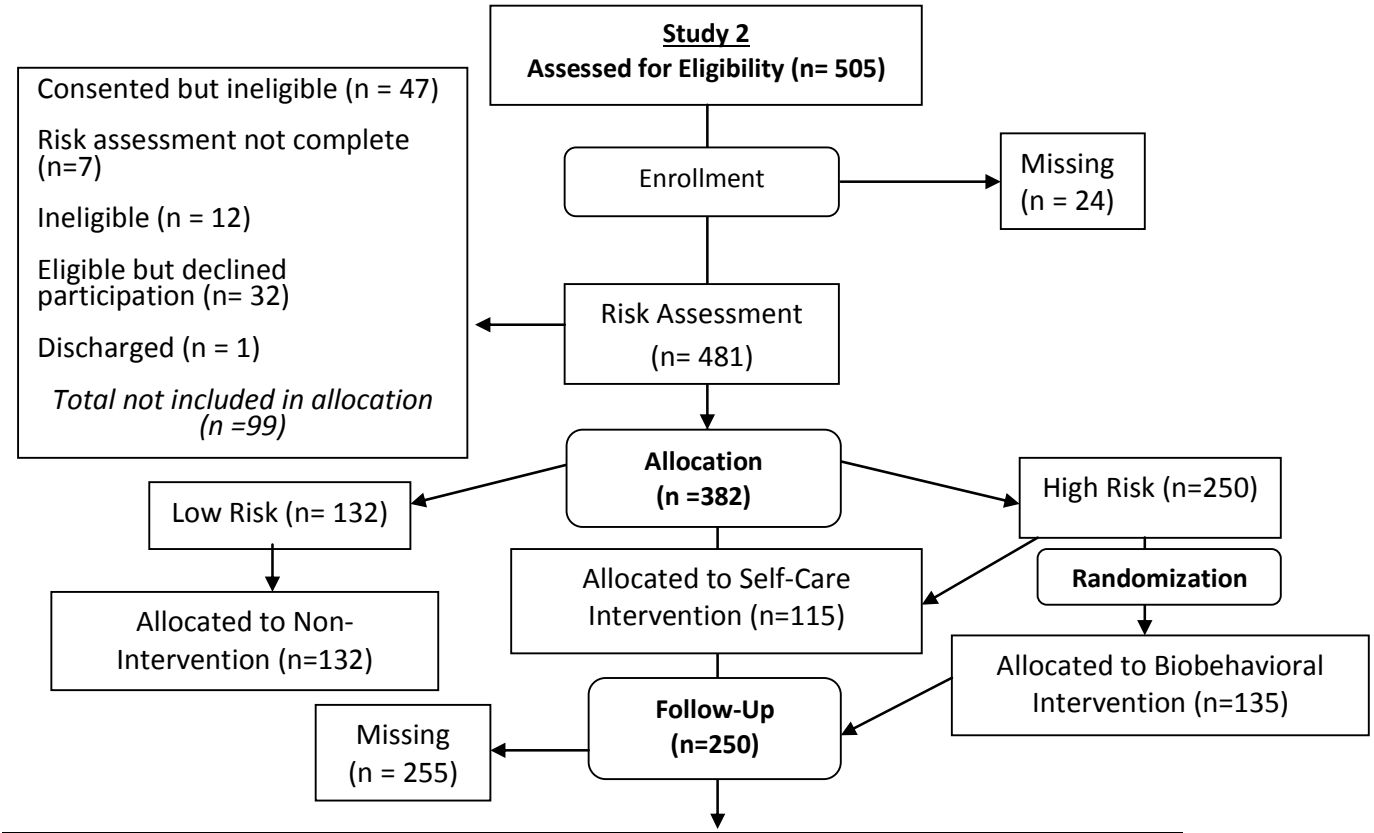
As a continued effort to examine the effects of Intervention on TMD specific symptoms, we investigated our expectation to observe the influence of the two Interventions on TMD-specific measures of Axis I Group II Disc Displacements, including TMD specific target variables used to diagnose Disc Displacements. A MANOVA was conducted with Intervention as the between subjects IV, Time as the within subjects IV, and the TMD specific target variables for Axis I Group II Disorders as the DVs. Although, we did not conduct mediational analyses with emotional distress as the mediator for this group of symptoms for reasons already specified, we conducted bivariate correlations for the target variables used to diagnose Axis I Group II Disc Displacements and emotional distress at T1 and T2. As a continued effort to determine the effects of Intervention on TMD specific symptoms, we conducted a MANOVA with Intervention as the between subjects IV, Time as the within subjects IV, and target variables used to diagnose Axis I Group III Bone Deficiencies as the DVs. Although, we did not conduct mediational analyses as we had planned for reasons already mentioned, we conducted bivariate correlations for the target variables used to diagnose Axis I Group III Bone Deficiencies and emotional distress at T1 and T2.

Having determined the effects of Intervention on TMD specific symptoms among the three groups of Axis I disorders, we conducted a MANOVA with Intervention as the between subjects IV, Time as the within subjects IV, and TMD non-specific symptoms of CSS as the DVs. Next, we conducted a MANOVA with Intervention as the between subjects IV, Time as the within subjects IV, and pain and pain related disability as the DVs to examine the effects of Intervention on pain and pain-related disability. Bivariate correlations were performed for pain and pain related disability and emotional distress for T1 and T2.

4.2 Study 2 Results

All variables were conducted using the appropriate scoring protocol which accounted for missing data using mean imputation. In order to ensure that our intervention groups were homogenous among the observed demographic variables including age, education, and number of children in the household, gender, ethnicity, marital status, and household income, initial analyses were conducted. Analyses of covariance (ANOVA)s were conducted to compare differences in age, education, and number of children among those in the low-risk, non-intervention group (LR), those in the high-risk, self-care group (SC), and those in the high-risk, biobehavioral group (BB). Chi-square analyses were conducted in order to detect unanticipated relationships between gender, ethnicity, marital status, and household income demographics and our three intervention groups, LR, SC, and BB. As expected, there were no differences among the demographic variables between our intervention groups (see Figure 4 for a flow chart of our sample and Table 5 for descriptives). The analyses revealed that the 250 participants included in our sample were predominately female (78.8%) with a mean age of 43.76 (SE = .94).

Figure 4. Sampling and Flow of Participant Selection for Study 2



Separate Analyses Conducted for Study 2	Intervention Group SC	BB	Total	Missing
The Effects of Intervention on Emotional Distress across Time	71	73	144	361
The Effects of Intervention on Group I symptoms across Time	71	76	147	358
The Effects of Intervention on Group II symptoms across Time	69	71	140	365
The Effects of Intervention on Group III symptoms across Time	51	56	107	398
The Effects of Intervention on Symptoms of CSS across Time	68	75	143	362
The Effects of Intervention on Pain & Disability across Time	74	77	151	354

Table 5. Demographics for the Selected Sample Including Age, Highest Grade Completed, Number of Children in Household, Gender, Ethnicity, Marital Status, and Household Income

Demographic	M (SE)	Demographic	n (%)
Age	43.76 (.94)	Gender	
		Male	50 (20.0%)
		Female	197 (78.8%)
		No information Provided	3 (1.2%)
Highest Grade Completed	15.07 (.14)	Ethnicity	
		White	166 (66.4%)
		Latino	30 (12.0%)
		Black	32 (12.8%)
Number of Children in Household	.77 (.07)	Asian	8 (3.2%)
		Other	8 (3.2%)
		Missing	1 (.4%)
		No information Provided	5 (2.0%)
		Marital Status	
		Married Together	129 (51.6%)
		Married Apart	2 (.8%)
		Widowed	7 (2.8%)
		Divorced	41 (16.4%)
		Separate	6 (2.4%)
		Never married	59 (23.6%)
		No information Provided	6 (2.4%)
		Household Income	
		$0- 14,999	29 (11.6%)
		$15,000- 24,999	19 (7.6%)
		$25,000- 34,999	22 (8.8%)
		$35,000- 50,000	22 (8.8%)
		> $50,000	148 (59.2%)
		No information Provided	10 (4.0%)

The Effects of Intervention on the Prevalence of TMD Specific Symptoms

As a preliminary step, a MANOVA was conducted with Intervention as the between subjects IV and Time as the within subjects IV and BDI and SF-36 MHC as the DVs in order to determine whether the required relationship between Intervention and emotional distress for mediational analyses was present in our sample (see Table 6). We did not observe differences between our Intervention groups for emotional distress, $F(2, 141) = .27$, $p > .05$, $\eta^2 = .004$, nor did we find an Intervention by Time interaction, $F(2, 141) = 1.05$, $p > .05$, $\eta^2 = .02$. Although emotional distress was reduced over Time, $F(2, 141) = 18.18$, $p < .05$, $\eta^2 = .21$ (see Table 8). Specifically, symptoms of depression decreased and emotional well-being improved over time. Because we did not observe the required relationship between Intervention and emotional distress, mediational analyses were not conducted for the following analyses including TMD specific symptoms for each of the three Axis I groups of disorders, or for the TMD non-specific symptoms of CSS, or pain or pain related disability.

Table 6. Main Effect of Time as a Function of Intervention Group Displayed at T1 and T2

	The Effects of Intervention Across Time (n = 144)			
	Self Care (SC) (n = 71)		Biobehavioral (BB) (n = 73)	
DVs	T1 M (SE)	T2 M (SE)	T1 M (SE)	T2 M (SE)
BDI*	.40 (.04)	.31 (.04)	.40 (.04)	.25 (.04)
MH*	72.04 (2.13)	78.07 (1.94)	72.62 (2.10)	78.10 (1.91)

Note. The table above displays a main effect of time as a function of Intervention group. Although we did not observe an interaction between Intervention and Time, we observed that symptoms of depression as indicated by BDI were significnatly reduced from T1 to T2. Similarly, we found that overall emotional well-being as indicated by the SF-36 MH was significantly imporved from T1 to T2.

Axis I RDC/TMD Group I Muscle Disorders

In order to investigate the effects of Intervention on TMD specific symptoms, we investigated the influence of the two interventions on TMD-specific measures of an Axis I Group I Muscle Disorder, including the 20 different muscle sites used to assess Axis I Disorders, a MANOVA was conducted with Intervention as the between subjects IV, Time as the within subjects IV, and the 20 muscles sites as the DVs. As expected, we observed a large multivariate effect for Time, $F(20, 126) = 3.20$, $p < .05$, $\eta^2 = .34$. However, we did not observe differences between our Intervention groups, $F(20, 126) = 1.01$, $p > .05$, $\eta^2 = .14$, nor an Intervention by Time interaction, $F(2, 126) = .99$, $p > .05$, $\eta^2 = .14$. Univariate analysis revealed that pain upon palpation was significantly reduced from T1 to T2. Specifically, Time reduced pain upon palpation in the Left mid temple $F(1, 145) = 6.22$, $p < .05$, $\eta^2 = .04$, right mid temple, $F(1, 145) = 4.04$, $p < .05$, $\eta^2 = .03$, left front temple, $F(1, 145) = 10.41$, $p < .05$, $\eta^2 = .07$, right side of face, $F(1, 145) = 4.36$, $p < .05$, $\eta^2 = .03$, left side of face, $F(1, 145) = 11.20$, $p < .05$, $\eta^2 = .07$, right jaw line, $F(1, 145) = 6.34$, $p < .05$, $\eta^2 = .04$, left jaw line, $F(1, 145) = 16.85$, $p < .05$, $\eta^2 = .10$, right throat, $F(1, 145) = 22.12$, $p < .05$, $\eta^2 = .13$, left throat, $F(1, 145) = 6.65$, $p < .05$, $\eta^2 = .04$, right under chin, $F(1, 145) = 15.76$, $p < .05$, $\eta^2 = .10$, left under chin, $F(1, 145) = 13.29$, $p < .05$, $\eta^2 = .08$, right tendon, $F(1, 145) = 5.40$, $p < .05$, $\eta^2 = .04$, and left tendon, $F(1, 145) = 15.43$, $p < .05$, $\eta^2 = .10$ (see Table 7).

Table 7.0 Main Effect of Time on Symptoms of an Axis I RDC/TMD Group I Disorder

	The Effects of Intervention Across Time (n = 147)			
	Self Care (SC) (n = 71)		**Biobehavioral (BB) (n = 76)**	
DVs	**T1**	**T2**	**T1**	**T2**
	M (SE)	*M (SE)*	*M (SE)*	*M (SE)*
Back temple: Temporal (posterior) Right	.14 (.05)	.09 (.04)	.18 (.05)	.12 (.04)
Back temple: Temporal (posterior) Left	.17 (.06)	.17 (.05)	.25 (.06)	.12 (.05)
Mid temple: Temporalis (middle) Right	.25 (.07)	.20 (.06)	.28 (.06)	.28 (.06)
Mid temple: Temporalis (middle) Left *	.31 (.07)	.16 (.05)	.29 (.07)	.21 (.05)
Mid temple: Temporalis (middle) Right*	.39 (.09)	.32 (.08)	.47 (.09)	.32 (.08)
Mid temple: Temporalis (middle) Left*	.56 (.09)	.38 (.08)	.49 (.09)	.28 (.07)
Under cheek: Masseter (origin) Right	.51 (.09)	.38 (.08)	.49 (.09)	.38 (.08)
Under cheek: Masseter (origin) Left	.49 (.10)	.42 (.08)	.51 (.09)	.36 (.08)
Side of face: Masseter (body) Right*	.52 (.10)	.58 (.09)	.93 (.10)	.54 (.09)
Side of face: Masseter (body) Left*	.78 (.11)	.58 (.09)	.88 (.11)	.41 (.08)
Jaw line: Masseter (insertion) Right*	.44 (.10)	.41 (.08)	.75 (.09)	.28 (.07)
Jaw line: Masseter (insertion) Left*	.54 (.09)	.31 (.07)	.66 (.09)	.36 (.08)
Throat: Posterior Mandibular region Right*	.59 (.12)	.32 (.08)	.93 (.11)	.36 (.08)
Throat: Posterior Mandibular region Left*	.61 (.11)	.51 (.09)	.70 (.11)	.11 (.04)
Under chin: Submandibular Region Right*	.32 (.08)	.10 (.04)	.34 (.08)	.17 (.05)
Under chin: Submandibular Region Left*	.32 (.08)	.17 (.05)	.40 (.08)	.13 (.05)
Outside joint: Lateral Pole Right	.45 (.10)	.47 (.10)	.55 (.09)	.54 (.09)
Outside joint: Lateral Pole Left	.54 (.10)	.65 (.23)	.59 (.09)	.34 (.22)
Tendon: Temporalis Tendon Right*	.51 (.11)	.41 (.10)	.83 (.11)	.57 (.09)
Tendon: Temporalis Tendon Left*	.73 (.12)	.42 (.09)	.76 (.12)	.41 (.08)

Note. The table above displays a main effect for symptoms of an Axis I RDC/TMD Group I disorder as a function of Intervention Group. Although, we did not observe the predicted interaction for Intervention and Time, pain upon palpation in the left mid temple, right front temple, left front temple, right side of face, left side of face, right jaw line, left jaw line, right throat, left throat, right under the chin, left under the chin, and right and left tendons was significantly reduced from T1 to T2.

Although, we did not conduct mediational analyses as we had planned to do because of discovering that there was no relationship between Intervention and emotional distress, we conducted bivariate correlations for the 20 muscle sites used to diagnose Axis I Group I Muscle Disorders and emotional distress at T1 and T2. As expected, there was a moderate relationship between TMD specific symptoms of Axis I Group I Muscle Disorder and BDI and SF-36 MH at T1 and T2. For a display of these analyses, please refer to Table 8.

Table 8. Pearson Correlation Coefficients for

Muscle Sites (right) and Emotional Distress Over Time

	BDI (T1)	BDI (T2)	MH (T1)	MH (T2)
Back temple: Temporal (posterior) – Right (T1)	r = .14 p = .14	r = .13 p = .15	r = - .04 p = .65	r = .01 p = .91
Back temple: Temporal (posterior) – Right (T2)	r = .20* p = .03	r = .36* p = .00	r = - .12 p = .22	r = - .20 p = .04
Mid temple: Temporalis (middle) – Right (T1)	r = .11 p = .25	r = .07 p = .44	r = .02 p = .86	r = - .11 p = .26
Mid temple: Temporalis (middle) – Right (T2)	r = .22* p = .02	r = .23* p = .01	r = - .12 p = .20	r = - .09 p = .32
Under cheek: Masseter (origin) – Right (T1)	r = .22* p = .02	r = .16 p = .09	r = - .09 p = .33	r = - .08 p = .42
Under cheek: Masseter (origin) – Right (T2)	r = .22* p = .02	r = .34* p = .00	r = - .22* p = .02	r = - .16 p = .08
Side of face: Masseter (body) – Right (T1)	r = .29* p = .00	r = .14 p = .15	r = - .23* p = .01	r = - .19* p = .04
Side of face: Masseter (body) – Right (T2)	r = .17 p = .07	r = .22* p = .02	r = - .08 p = .39	r = - .14 p = .13
Jaw line: Masseter (insertion) – Right(T1)	r = .28* p = .00	r = .18 p = .06	r = - .17 p = .07	r = - .20* p = .03
Jaw line: Masseter (insertion) – Right(T2)	r = .22* p = .02	r = .34* p = .00	r = - .21* p = .02	r = - .24* p = .01

Table 8. – continued

Throat: Posterior Mandibular Region – Right (T1)	r = .22* p = .02	r = .10 p = .29	r = - .06 p = .52	r = .00 p = .99
Throat: Posterior Mandibular Region – Right (T2)	r = .23* p = .01	r = .36* p = .00	r = - .20* p = .03	r = - .24* p = .01
Under chin: Submandibular Region-Right(T1)	r = .32* p = .01	r = .24 p = .24	r = - .18 p = .11	r = - .19 p = .34
Under chin: Submandibular Region-Right(T2)	r = .23* p = .02	r = .28* p = .00	r = - .22* p = .02	r = - .13 p = .18
Outside joint: Lateral Pole – Right (T1)	r = .24* p = .01	r = .10 p = .27	r = - .15 p = .11	r = - .09 p = .35
Outside joint: Lateral Pole – Right (T2)	r = .19* p = .05	r = .23* p = .01	r = - .06 p = .50	r = - .12 p = .21
Tendon: Temporalis Tendon – Right (T1)	r = .20* p = .03	r = .17 p = .06	r = - .08 p = .39	r = - .21* p = .02
Tendon: Temporalis Tendon – Right (T2)	r = 0.11 p = .23	r = .18* p = .05	r = - .11 p = .22	r = - .29* p = .00
Back temple: Temporal (posterior) – Left(T1)	r = .12 p = .20	r = .07 p = .44	r = - .03 p = .74	r = .06 p = .54
Back temple: Temporal (posterior) – Left(T2)	r = .18* p = .05	r = .31* p = .00	r = - .14 p = .14	r = - .1 p = .30
Mid temple: Temporalis (middle) – Left(T1)	r = .07 p = .46	r = .07 p = .49	r = - .01 p = .91	r = .01 p = .93
Mid temple: Temporalis (middle) – Left(T2)	r = .26* p = .00	r = .39* p = .00	r = - .14 p = .13	r = - .17 p = .07
Under cheek: Masseter (origin) – Left (T1)	r = .23* p = .01	r = .23* p = .02	r = - .17 p = .06	r = - .16 p = .09
Under cheek: Masseter (origin) – Left (T2)	r = .25* p = .01	r = .34* p = .00	r = - .28* p = .00	r = - .16 p = .09
Side of face: Masseter (body) – Left (T1)	r = .16 p = .09	r = .12 p = .18	r = - .15 p = .11	r = - .08 p = .40
Side of face: Masseter (body) – Left (T2)	r = .20* p = .03	r = .29* p = .00	r = - .16 p = .09	r = - .18 p = .06
Jaw line: Masseter (insertion) – Left (T1)	r = .22* p = .02	r = .12 p = .22	r = - .1 p = .29	r = - .12 p = .22
Jaw line: Masseter (insertion) – Left (T2)	r = .16 p = .10	r = .28* p = .00	r = - .18* p = .05	r = - .11 p = .23
Throat: Posterior Mandibular Region – Left (T1)	r = .30* p = .00	r = .23* p = .01	r = - .17 p = .07	r = - .12 p = .19
Throat: Posterior Mandibular Region – Left (T2)	r = .24* p = .01	r = .37* p = .00	r = - .19* p = .04	r = - .22* p = .02

Table 8. - continued

Under chin: Submandibular Region- Left (T1)	r = .32* p = .00	r = .24* p = .01	r = - .18* p = .05	r = - .19* P = .04
Under chin: Submandibular Region- Left (T2)	r = .27* p = .00	r = .33* p = .00	r = -.28* p = .00	r = - .19* p = .04
Outside joint: Lateral Pole – Left (T1)	r = .19* p =.04	r = .19* p = .04	r = - .09 p = .34	r = .01 p = .96
Outside joint: Lateral Pole – Left (T2)	r = .17 p = .07	r = .19* p = .04	r = - .11 p = .22	r = - .13 p = .16
Tendon: Temporalis Tendon – Left (T1)	r = .17 p = .07	r = .10 p = .31	r = - .09 p = .33	r = - .10 p = .30

Note. The above table displays the correlations for symptoms of Axis I RDC/TMD Group I disorder and the covariates BDI and MH at T1 and T2. The correlation coefficients are provided at both time points with the p values provided below each r value.

Axis I RDC/TMD Group II Disc Displacement

As a continued effort to observe the influence of the two interventions on TMD-specific measures of Axis I Group II Disc Displacements, including TMD specific target variables used to diagnose Disc Displacements, a MANOVA was conducted with Intervention as the between subjects IV, Time as the within subjects IV, and the TMD specific target variables for Axis I Group II Disorders as the DVs. We did not observe an Intervention by Time interaction, $F(12, 127) = .93$, $p > .05$, $\eta^2 = .08$. There was a marginal effect for Intervention, $F(12, 127) = 1.76$, $p = .06$, $\eta^2 = .14$ (see Table 9). The Univariate analysis revealed that those in the BB group had a greater maximum unassisted opening of the jaw, $F(1, 138) = 7.05$, $p < .05$, $\eta^2 = .05$, unassisted opening of the jaw, $F(1, 138) = 9.23$, $p < .05$, $\eta^2 = .06$, and maximum assisted opening of the jaw, $F(1, 138) = 6.94$, $p < .05$, $\eta^2 = .05$. Additionally, the analyses revealed a marginal effect for Time, $F(12, 127) = 1.78$, $p = .058$, $\eta^2 = .14$. Thus, the following results, should be interpreted with caution. The univariate analyses revealed that unassisted opening of the jaw, $F(1, 138) = 3.33$, $p <$

.05, η^2 = .02, muscle pain in the jaw, F(1, 138) = 7.40, p< .05, η^2 = .05, and click in the left jaw, F(1, 138) = 4.04, p< .05, η^2 = .03 was significantly reduced from T1 to T2.

Table 9. Means and Standard Errors for Symptoms of an Axis I RDC/TMD Group II Disorder

	The Effects of Intervention Across Time (n = 140)			
	Self Care (SC) (n = 69)		Biobehavioral (BB) (n = 71)	
DVs	T1 M (SE)	T2 M (SE)	T1 M (SE)	T2 M (SE)
Maximum unassisted opening	40.80 (.93)	41.33 (1.04)	44.42 (.91)	44.11 (1.03)
Unassisted opening	32.51 (1.16)	34.04 (1.10)	36.62 (1.15)	38.14 (1.08)
Muscle pain	1.44 (.17)	1.13(.15)	1.65 (.17)	1.21 (.14)
Maximum assisted opening	44.07 (.89)	44.00 (1.02)	47.55 (.88)	46.76(1.01)
Open of the right joint	.23 (.09)	.39 (.10)	.39 (.08)	.41 (.10)
Measure click on the right	3.36 (1.25)	3.97 (1.20)	4.10 (1.23)	3.96 (1.19)
Measure click on the left	5.50 (1.17)	4.20 (1.27)	1.85 (1.14)	4.90 (1.25)
Sound on the right	8.49 (.26)	8.26 (.31)	8.41 (.25)	8.03 (.31)
Excursion	10.74 (.59)	10.29 (.43)	11.09 (.59)	10.47 (.43)
Protrusion	7.03 (.34)	6.67 (.30)	7.06 (.34)	7.55 (.30)
Sound on the left	.29 (.07)	.28 (.09)	.13 (.07)	.25 (.09)
Joint sounds	.17 (.06)	.26 (.08)	.13 (.06)	.18 (.08)

Note. The table above displays the marginal means and standard errors for symptoms of an Axis I RDC/TMD Group II disorder as a function of Intervention Group across T1 and T2. We did not observe any significant results for this analysis in contrast to our expectations for an interaction between Intervention Group and Time.

Although, we did not conduct mediational analyses with emotional distress as the mediator for this group of symptoms for reasons already specified, we conducted bivariate correlations for the Axis I Group II diagnostic status and the target variables used to diagnose

Axis I Group II Disc Displacements and emotional distress at T1 and T2. There was no relationship between many of the Axis I Group II symptoms and BDI and SF-36 MHC at T1 or T2. For a display of these analyses, please see Table 10.

Table 10. Pearson Correlation Coefficients for Muscle Sites and Emotional Distress Over Time

	BDI (T1)	BDI (T2)	MH (T1)	MH (T2)
Maximum unassisted opening (T1)	r = .07	r = .05	r = -.12	r = -.09
	p = .54	p = .63	p = .27	p = .41
Maximum unassisted opening (T2)	r = -.07	r = -.03	r = .05	r = -.02
	p = .54	p = .76	p = .68	p = .88
Unassisted opening (T1)	r = -.15	r = -.10	r = .13	r = .02
	p = .16	p = .38	p = .24	p = .86
Unassisted opening (T2)	r = -.17	r = -.12	r = .12	r = -.02
	p = .11	p = .25	p = .26	p = .85
Muscle pain (T1)	r = .12	r = .14	r = -.22*	r = -.26*
	p = .28	p = .21	p = .04	p = .02
Muscle pain (T2)	r = .17	r = .25*	r = -.31*	r = -.15
	p = .11	p = .02	p = .00	p = .15
Maximum assisted opening (T1)	r = .16	r = .16	r = -.21*	r = -.17
	p = .13	p = .15	p = .05	p = .12
Maximum assisted opening (T2)	r = .05	r = .04	r = -.06	r = -.05
	p = .66	p = .69	p = .61	p = .62
Open of the right joint (T1)	r = -.04	r = -.02	r = .11	r = .13
	p = .75	p = .84	p = .31	p = .24
Open of the right joint (T2)	r = -.13	r = -.10	r = .32*	r = .11
	p = .23	p = .37	p = .00	p = .30
Click on the right (T1)	r = .12	r = .10	r = -.01	r = .04
	p = .29	p = .35	p = .90	p = .70
Click on the right (T2)	r = .08	r = .04	r = .05	r = -.05
	p = .47	p = .73	p = .66	p = .65
Click on the left (T1)	r = -.03	r = .03	r = .09	r = -.01
	p = .79	p = .78	p = .39	p = .91
Click on the left (T2)	r = .00	r = .00	r = .13	r = -.06
	p = .98	p = .99	p = .22	p = .59
Sound on the right (T1)	r = .09	r = .04	r = -.09	r = .03
	p = .42	p = .72	p = .44	p = .79
Sound on the right (T1)	r = .08	r = .00	r = -.13	r = .01
	p = .44	p = .99	p = .23	p = .93

Table 10. –continued

Excursion (T1)	r = .14	r = .16	r = -.16	r = -.09
	p = .18	p = .14	p = .15	p = .41
Excursion (T2)	r = .15	r = .15	r = -.13	r = -.24
	p = .17	p = .18	p = .25	p = .02
Protrusion (T1)	r = .03	r = .00	r = -.03	r = -.05
	p = .80	1.00	p = .80	p = .67
Protrusion (T2)	r = .04	r = .06	r = -.01	r = -.10
	p = .72	p = .56	p = .92	p = .37
Sound on the left (T1)	r = -.05	r = -.03	r = .14	r = .16
	p = .65	p = .77	p = .21	p = .15
Sound on the left (T2)	r = -.06	r = -.03	r = .07	r = .03
	p = .59	p = .80	p = .55	p = .78
Joint sounds (T1)	r = -.18	r = -.14	r = .27*	r = .26*
	p = .09	p = .19	p = .01	p = .02
Joint sounds (T2)	r = .21*	r = .17	r = -.06	r = -.13
	p = .05	p = .11	p = .59	p = .23

Note. The above table displays the correlations for symptoms of Axis I RDC/TMD Group II disorder and the covariates BDI and MH at T1 and T2. The correlation coefficients are provided at both time points with the p values provided below each r value.

Axis I RDC/TMD Group III Bone Deficiency

As a continued effort to observe the influence of the two interventions on TMD-specific measures of an Axis I Group III Bone Deficiency, including TMD specific target variables used to diagnose these disorders, we conducted a MANOVA was conducted with Intervention as the between subjects IV, Time as the within subjects IV, and the TMD specific target variables for Axis I Group III Disorders as the DVs. Analyses revealed a large multivariate effect for Time, $F(12, 94) = 2.58$, $p < .05$, $\eta^2 = .25$. However, we did not observe differences between our Intervention groups, $F(12, 94) = .76$, $p > .05$, $\eta^2 = .09$, nor an Intervention by Time interaction, $F(12, 94) = .49$, $p > .05$, $\eta^2 = .06$. The univariate analyses revealed that several of the TMD specific target variables for these disorders were effectively reduced from T1 to T2. Specifically, the prevalence of arthritis in left jaw was significantly reduced from T1 to T2, $F(1, 105) = 10.11$,

$p < .05$, $\eta^2 = .09$, as well as pain upon palpation in the left joint area $F(1, 105) = 10.11$, $p < .05$, $\eta^2 = .12$, and ongoing pain in the left jaw joint, $F(1, 105) = 12.94$, $p < .05$, $\eta^2 = .11$ (see Table 11).

Table 11. Main Effect of Time on Symptoms of an Axis I RDC/TMD Group III Disorder

	The Effects of Intervention Across Time (n = 107)			
	Self Care (SC) (n = 51)		Biobehavioral (BB) (n = 56)	
DVs	T1 M (SE)	T2 M (SE)	T1 M (SE)	T2 M (SE)
Right Arthralgia*	.37 (.07)	.22 (.06)	.38 (.07)	.16 (.05)
Right Osteoarthritis	.04 (.02)	.16 (.05)	.02 (.02)	.06 (.01)
Right Osteoarthrosis	.02 (.03)	.02 (.02)	.07 (.03)	.04 (.02)
Right Joint Pain On Palpation*	.49 (.07)	.31 (.06)	.54 (.07)	.27 (.06)
Ongoing Pain in Right Joint*	.69 (.07)	.47 (.07)	.55 (.07)	.34 (.07)
Pain in Right Joint on Opening	.51 (.07)	.47 (.07)	.41 (.07)	.34 (.07)
Left Arthralgia	.28 (.07)	.26 (.07)	.32 (.06)	.36 (.06)
Left Osteoarthritis	.00 (.01)	.02 (.02)	.02 (.01)	.04 (.02)
Left Osteoarthrosis	.02 (.02)	.00 (.02)	.02 (.02)	.04 (.02)
Left Joint Pain On Palpation	.33 (.07)	.33 (.07)	.45 (.07)	.43 (.07)
Ongoing Pain in Left Joint	.51 (.07)	.45 (.07)	.52 (.07)	.46 (.07)
Pain in Left Joint on Opening	.41 (.07)	.47 (.07)	.45 (.07)	.43 (.07)

Note. The table above displays the marginal means and standard errors for symptoms of an Axis I RDC/TMD Group III disorder as a function of Intervention group across T1 and T2. Although we did not confirm our expectations for an interaction between Intervention Group and Time, we found a main effect for Time. The analyses revealed that symptoms including right arthralgia, right joint pain on palpation, and ongoing pain in the right joint were significantly reduced from T1 to T2.

Although, we did not conduct mediational analyses as we had planned for reasons already mentioned, we conducted bivariate correlations for the target variables used to diagnose Axis I

Group III Bone Deficiencies at T1 and T2. As expected, there was a moderate relationship between several TMD specific symptoms of Axis I Group III Bone Deficiencies and BDI and SF-36 MHC at T1 and T2. For a display of these analyses, please refer to Table 12.

Table 12. Pearson Correlation Coefficients for

Axis I Group III Symptoms and Emotional Distress

	BDI (T1)	BDI (T2)	MH (T1)	MH (T2)
Right Arthralgia (T1)	r = .28* p = .01	r = .12 p = .23	r = -.10 p = .34	r = -.12 p = .26
Right Arthralgia (T2)	r = .15 p = .14	r = .20* p = .05	r = -.09 p = .41	r = -.14 p = .17
Right Osteoarthritis (T1)	r = .04 p = .68	r = .01 p = .92	r = -.14 p = .16	r = -.04 p = .74
Right Osteoarthritis (T2)	r = -.04 p = .73	r = .07 p = .50	r = .11 p = .30	r = -.03 p = .77
Right Osteoarthrosis (T1)	r = -.11 p = .27	r = -.13 p = .20	r = .14 p = .18	r = .14 p = .17
Right Osteoarthrosis (T2)	r = -.07 p = .53	r = -.10 p = .33	r = .08 p = .46	r = .10 p = .32
Right Joint Pain On Palpation(T1)	r = .29* p = .00	r = .19 p = .06	r = -.20* p = .05	r = -.18 p = .08
Right Joint Pain On Palpation (T2)	r = .19 p = .07	r = .27* p = .01	r = -.05 p = .60	r = -.18 p = .08
Ongoing Pain in Right Joint (T1)	r = .13 p = .21	r = .03 p = .81	r = .00 1.00	r = -.08 p = .43
Ongoing Pain in Right Joint (T2)	r = .07 p = .50	r = .05 p = .61	r = .10 p = .36	r = -.09 p = .38
Pain in Right Joint on Opening (T1)	r = .10 p = .34	r = .00 p = .99	r = .02 p = .84	r = -.10 p = .31
Pain in Right Joint on Opening (T2)	r = .14 p = .16	r = .16 p = .11	r = -.05 p = .62	r = -.13 p = .19

Table 12. Continued

	BDI (T1)	BDI (T2)	MH (T1)	MH (T2)
Left Arthralgia (T1)	r = .14 p = .18	r = .18 p = .08	-r = .07 p = .50	r = -.08 p = .45
Left Arthralgia (T2)	r = .22* p = .03	r = .29* p = .00	r = -.25* p = .01	r = -.27* p = .01
Left Osteoarthritis (T1)	r = .06 p = .57	r = -.02 p = .87	r = .09 p = .37	r = .08 p = .44
Left Osteoarthritis (T2)	r = -.08 p = .44	r = -.09 p = .37	r = .05 p = .61	r = .13 p = .22
Left Osteoarthrosis (T1)	r = -.18 p = .09	r = -.14 p = .18	r = .24 p = .02	r = .17 p = .09
Left Osteoarthrosis (T2)	r = .19 p = .07	r = .02 p = .86	r = -.01 p = .93	r = .05 p = .64
Left Joint Pain On Palpation (T1)	r = .22* p = .03	r = .28* p = .01	r = -.13 p = .21	r = -.13 p = .21
Left Joint Pain On Palpation (T2)	r = .27* p = .01	r = .32* p = .00	r = -.25* p = .01	r = -.27* p = .01
Ongoing Pain in Left Joint (T1)	r = -.05 p = .65	r = -.14 p = .16	r = .14 p = .17	r = .03 p = .80
Ongoing Pain in Left Joint (T2)	r = -.01 p = .92	r = .08 p = .44	r = .01 p = .92	r = -.12 p = .26
Pain in Left Joint on Opening (T1)	r = -.02 p = .85	r = -.03 p = .75	r = .13 p = .22	r = .00 p = .97
Pain in Left Joint on Opening (T2)	r = .01 p = .93	r = .02 p = .83	r = .03 p = .78	r = -.08 p = .42

Note. The above table displays the correlations for symptoms of Axis I RDC/TMD Group III disorder and the covariates BDI and MH at T1 and T2. The correlation coefficients are provided at both time points with the p values provided below each r value.

TMD Non-Specific Symptoms: Comorbid Symptoms of CSS

Having determined change in diagnostic status and TMD specific symptoms among the three groups of Axis I disorders, we conducted a MANOVA with Intervention as the between subjects IV, Time as the within subjects IV, and TMD non-specific symptoms of CSS as the DVs. We did not observe differences between our Intervention groups, $F(10, 126) = 1.08$, $p > .05$,

$\eta^2 = .08$, nor an Intervention by Time interaction, $F(10, 132) = 1.48$, $p > .05$, $\eta^2 = .10$. Although there was an effect for Time, $F(10, 132) = 2.11$, $p < .05$, $\eta^2 = .14$. As displayed in Table 13, total physical symptoms, $F(1, 141) = 4.15$, $p < .05$, $\eta^2 = .03$, and symptoms specific to CFS, $F(1, 141) = 5.60$, $p < .05$, $\eta^2 = .04$, were significantly reduced from T1 to T2. For a display of these analyses, please refer again to Table 3.

Table 13. Main Effect of Time on Symptoms of CSS as a Function of Intervention Group

	The Effects of Intervention Across Time (n = 143)			
	Self Care (SC) (n = 68)		Biobehavioral (BB) (n = 75)	
DVs	T1 M (SE)	T2 M (SE)	T1 M (SE)	T2 M (SE)
Total Physical Symptoms*	.23 (.02)	.23 (.02)	.19 (.02)	.15 (.02)
CFS*	.38 (.03)	.36 (.03)	.33 (.03)	.28 (.03)
FM	.35 (.03)	.36 (.03)	.32 (.03)	.26 (.03)
IBS	.21 (.04)	.22 (.03)	.18 (.04)	.14 (.03)
Low Back Pain	.09 (.03)	.15 (.03)	.07 (.02)	.06 (.03)
Irritable Bladder	.11 (.02)	.11 (.02)	.09 (.02)	.07 (.02)
Chemical Sensitivities	.05 (.01)	.04 (.01)	.04 (.01)	.02 (.01)
Post-concussive Syndrome	.12 (.03)	.18 (.04)	.10 (.03)	.08 (.03)
Female pelvic pain	.19 (.02)	.18 (.02)	.15 (.02)	.14 (.02)
Male Pelvic Pain	.01 (.01)	.00 (.00)	.01 (.01)	.01 (.00)

Note. The table above displays marginal means and standards errors for symptoms of CSS as a function of Intervention Group at both T1 and T2. Although there was no effect for an interaction between Intervention and Time like we had predicted, we observed a main effect for Time. The analyses revealed that total physical symptoms and symptoms of CFS were significantly reduced from T1 to T2.

The Effects of Intervention on Pain and Pain-related Disability

Having determined change in diagnostic status and TMD specific and TMD non- specific symptoms, we conducted a MANOVA with Intervention as the between subjects IV, Time as the within subjects IV, and pain and pain related disability as the DVs. We did not observe differences between our Intervention groups, $F(2, 148) = .54$, $p > .05$, $\eta^2 = .01$, nor an Intervention by Time interaction, $F(2, 148) = 2.47$, $p > .05$, $\eta^2 = .03$. However, there was an effect for Time, $F(2, 148) = 66.59$, $p < .05$, $\eta^2 = .47$. As displayed in Table 14, univariate analyses revealed that pain, as indicated by CPI, $F(1, 149) = 127.22$, $p < .05$, $\eta^2 = .46$, and pain related disability, as indicated by GCP, $F(1, 149) = 49.53$, $p < .05$, $\eta^2 = .25$, were significantly reduced from T1 to T2. Mediational analyses were not conducted for pain and pain-related disability because there was no relationship between Intervention and emotional distress, which is a requisite relationship prior to mediational analyses.

Table 14. Main Effect of Time for Pain and

Pain-related Disability as a Function of Intervention Group

	The Effects of Intervention Across Time (n = 151)			
	Self Care (SC) (n = 74)		Biobehavioral (BB) (n = 77)	
DVs	T1 M (SE)	T2 M (SE)	T1 M (SE)	T2 M (SE)
Pain*	64.23 (1.59)	49.37 (2.06)	64.11 (1.56)	46.02 (2.02)
Pain-Related Disability*	28.11 (2.72)	19.51 (2.29)	33.03 (2.67)	16.49 (2.25)

Note. The table above displays the marginal means and standard errors for pain and pain-related disability as a function of Intervention Group at T1 and T2. Although we did not observe the

predicted interaction, we observed a main effect for Time. Both pain and Pain-related disability were significantly reduced from T1 to T2.

Although, we did not conduct mediational analyses, we conducted bivariate correlations for pain and pain related disability. As expected, there was a moderate relationship between pain and BDI (r= .11: p> .05) and SF-36 MHC (r =-.13: p< .05) at Time 1. Moreover, we found similar results for pain and BDI (r= .20: p< .05) and SF-36 MHC (r =-.19: p< .05) at Time 2. As expected, there was a moderate relationship between pain-related disability and BDI (r= .28: p< .05) and SF-36 MHC (r =-.30: p< .05) at Time 1. Moreover, we found similar results for pain-related disability and BDI (r= .29: p< .05) and SF-36 MHC (r =-.25: p< .05) at Time 2.

CHAPTER 5

DISCUSSION

The specific aims of the current research were to: 1) identify comorbid, non-specific symptoms of CSS, across three groups of Axis I RDC/TMD disorders (i.e., Group I Muscle Disorders, Group II Disc Displacements, Group III Bone Deficiencies), 2) investigate the influence of two interventions (i.e., SC and BB) on TMD specific and TMD non-specific symptoms and pain and pain-related disability, and 3) examine the influence of emotional distress on the prevalence of symptoms (TMD [non] /specific) across two time points (i.e., T1 and T2). These aims were derived from a confluence of research demonstrating the clinical- and cost- effectiveness of biobehavioral interventions for musculoskeletal disorders (Gatchel & Okifuji, 2006), evidence demonstrating the interdependence of biopsychosocial factors (Dougall et al., 2012; Dworkin, 2011), and recent focus on the high degree of overlap of non-specific pain-inclusive symptoms among disorders, such as TMD, CFS, and FM, thought to be explained in part by multiple systems dysregulation and amplified pain-autonomic processing (Aaron, Burke, & Butchwald, 2000; Chapman, Tuckett, & Song, 2008; Lorduy, Dougall, Haggard, & Gatchel, 2012; Yunus, 2008).

In Study 1, we confirmed our hypothesis that comorbid TMD non-specific symptoms of CSS would be more prevalent among those with an Axis I Group I Muscle Disorder. Specifically, we found that those with an Axis I Group I Muscle Disorder and those with more than one Axis I diagnosis, a group constituted primarily with those that had both a Muscle

Disorder and an Axis I Group III Bone Deficiency, had more symptoms of CSS as indicated by the Symptom Checklist. This finding supports literature suggesting that the overlap between TMD and other disorders of CSS is more pronounced among those with myofacial TMD (Aaron, Burke, & Butchwald, 2000; Lorduy, Dougall, Haggard, & Gatchel, 2012). Where there is no existing literature directly explaining the shared pathology among myofacial TMD and symptoms of CSS, there is a segmented but developing body of research explaining the pathophysiological mechanisms involved which might contribute to CS (Celik & Mutlu, 2011; Chapman, Tuckett, & Song, 2008; Lyon, Cohen, & Quinter, 2011). For instance, it has been suggested that these disorders are unified by altered function along the pain pathway, which can be explained by psychosocial features, genetic predispositions, and altered neurobiological function (Becker & Schweinhardt, 2011; Smith et al., 2011; Younger et al., 2010). The stronger relationship between myofacial pain and symptoms of FM could arguably be the result of activated trigger points caused by dysregulation of NE, 5-HT, and several pro-inflammatory cytokines as a product of chronic stress responding (Shah & Gilliam, 2008), or otherwise exacerbated by chronic stress responding with compelling research to support that both are plausible (Celik & Mutlu, 2011; Becker & Schweinhardt, 2011; Lyon, Cohen, & Quinter, 2011). Activation of trigger points has demonstrated to bring on tonic contraction of motor fibers and subsequent neurobiological changes in the dorsal horn and brain substrates responsible for pain processing (McLean, Clauw, Abelson, & Liberzon, 2005; Shah & Gilliam, 2008). Moreover, these featured alterations along the pain pathway have demonstrated to be resultant from increases in stress, pain, and inflammatory factors caused by multiple systems dysregulation, which can exhaust the body's built-in regulatory defenses over time and operate in a "vicious

cycle" to bring about further tissue damage (Gatchel, 2004; Gatchel, Howard, & Haggard, 2010). For example, increases in CGRP correspond to areas in the dorsal horn responsible for deep orofacial pain and initiate powerful proinflammatory factors involved in the development of bone deficiencies (Cady, Glenn, Smith, & Durham, 2011; Guo et al., 2007). Both Axis I Muscle disorders and arthralgias have been correlated with indices of CS (Aaron, Burke, & Butchwald, 2000; Lorduy et al., 2012; Sullivan & D'Eon, 1990; Sullivan et al., 1991). Moreover, there is an extensive line of research demonstrating the reciprocal relationship between pain and stress and the contribution of altered pain-autonomic processing that leads to CS (Becker & Schweinhardt, 2011; Chapman, Tuckett, & Song, 2008; Lyon, Cohen, & Quinter, 2011). The synthesis of these areas of research offer a tentative explanation for our observation of myofacial TMD to be more strongly related to comorbid TMD non-specific symptoms of CSS (Lorduy et al., 2012).

In order to examine the relationship between TMD and comorbid TMD-nonspecific symptoms of CSS with more precision we refined our comparison of TMD groups in order to examine these relationships and the influence of emotional distress. Therefore, we compared the influence of emotional distress on symptoms of CSS among those with no diagnosis, those with only an Axis I Muscle Disorder, and those with both an Axis I Muscle Disorder and those with an Axis I Bone Deficiency. In line with the explanation provided in the previous paragraph, the results for this analysis favor the suggestion that the higher prevalence of comorbid symptoms of CSS coincides with progressive degeneration of muscle and bone tissue in the myofacial region. Specifically, we found the prevalence of comorbid symptoms of CSS was higher among those with both a Muscle Disorder and Bone Deficiency providing further support for the position that myofacial TMD possibly involves progressive dysregulation among multiple systems as

symptoms of depression increased in a similar fashion as described in the previous analysis of comorbid symptoms. Likewise, overall mental health declined from the no diagnosis group, to the Axis I Muscle Disorder group, and finally to the Axis I Muscle Disorder and Axis I Bone Deficiency combined group confirming our expectations. Furthermore, we confirmed our expectations that emotional distress would be positively correlated with TMD non-specific and TMD specific symptoms. Specifically, BDI ($r = .57$; $p < .05$) and SF-36 MHC ($r = -.49$; $p < .05$) were strongly correlated with TMD non-specific symptoms of CSS with symptoms of depression as indicated by the BDI as a stronger contributor compared to overall mental health as indicated by the SF-36 MHC. Among the psychosocial factors most emphasized as being involved in pain conditions and CS, depression has been the most consistently observed and extensively studied. For instance, Blackburn-Munro and Blackburn-Munro (2001) have presented a model depicting the intricacies of neurotransmitter and neuroendocrine systems in the regulation of pain-autonomic processing. They explain how imbalances in NE and 5-HT, both of which are implicated in depression, are relevant in the regulation of HPA axis function and transmission of pain in response to physical and psychological events (Becker & Schweinhardt, 2011).

In Study 2, we investigated the influence of two interventions (SC and BB) on TMD specific symptoms over Time (T1 and T2). Specifically, we expected that the TMD specific symptoms would be ameliorated overtime to the greatest degree by the BB intervention. We found partial support for this prediction for symptoms of an Axis I Group I Muscle Disorder. Where we did not observe an interaction between Intervention and Time, we did find that pain upon palpation at the majority of the 20 muscle cites used to determine diagnosis of a Muscle Disorder was significantly reduced from T1 to T2, or immediately following intervention.

However, there were no differences between our intervention groups. There was a marginal effect for unassisted opening of the jaw, reduction in muscle pain, and click in the jaw, which are target variables used for diagnosing Disc Displacements, from T1 to T2. Additionally, there was a marginal effect for differences between Intervention groups with those in the BB Intervention group reporting greater unassisted opening of jaw, unassisted opening of the jaw, and maximum assisted opening of the jaw joint, and click in the left jaw. Although there were no differences between the intervention groups as we had hoped, there was a reduction in arthritis in the left jaw, pain upon palpation of the jaw bone, and ongoing pain in the jaw joint. Similarly, we found that symptoms of CSS were reduced from T1 to T2, as well as pain and pain-related disability.

Additionally, we investigated the role of emotional distress on the prevalence of TMD specific and TMD nonspecific symptoms. Where we were not able to run the mediational analyses due to there not being the required relationship between our between subjects IV, Intervention, and our CV, emotional distress, we did observe that emotional distress fully mediated symptoms of CSS at T1. Moreover, we determined that the 20 muscle sites used to diagnose Axis I Group I Muscle Disorder, was strongly related to emotional distress. Similarly, the target variables used to diagnose these disorders were strongly related to emotional distress. However, many of the target variables for Axis I Group II Disc Displacements were not associated with emotional distress. Therefore, the disorders and symptomatology that has been more strongly emphasized with emotional distress as an etiological factor demonstrated the most improvement at T2 when emotional distress was reduced. With regard to the lack of difference between our intervention groups, it is possible that in the short-term, the benefits of attention and social support from clinicians in the SC intervention are comparable to the stress reducing effects

of the biobehavioral intervention. However, extant literature demonstrates that the effects of biobehavioral interventions generally do not show comparative improvements in the short-term (i.e., immediate post-evaluation), but are, instead, observed at the long-term follow-up (Epker, Gatchel, & Ellis, 1999; Gatchel et al., 2003). In other words, it is possible that the benefits to be gained by the BB are delayed as they are dependent on the establishment of new coping and thought patterns (Epker, Gatchel, & Ellis, 1999; Gatchel, 2004; Gatchel, Polatin, & Mayer, 1995; Edwards, Campbell, Jamison, & Wiech, 2009; Westman et al., 2011), which require additional effort and energy initially, but gradually appreciate over time to explain its comparable clinical- and cost- effectiveness (Bernstein & Gatchel, 2000; Gardea, 1998; Gardea, Gatchel, & Mirsha, 2001; Gatchel & Okifuji, 2006; Phillips et al., 2001).

Prior to interpreting these results presented in Study 2, it is important to note that the current results were derived from non-traditional means of analysis different from what is commonly used to investigate similar research aims. Thus, it is appropriate to consider these results, which were ascertained in a manner designed to be sensitive to minute differences among specific measures of Axis I RDC/TMD disorders, more conservatively, such that they may reflect clinical meaningfulness. In line with our predictions, the current analyses detected subtle changes in many of the key measures used to assess Axis I RDC/TMD diagnoses. Unfortunately, the current analyses are limited by the uneven sample sizes between each of the time points and consequent lack of power, and thus we were not able to investigate our prediction for an interaction between Intervention and Time fully.

In sum, both studies combined suggest that emotional distress influences the prevalence of symptoms and the severity of pain and pain-related disability. Moreover, symptoms of

depression are a stronger predictor of these facets of TMD, which falls in line with research suggesting that prolonged stress responding and activation of the autonomic nervous system leads to depression and comorbid physical symptoms through augmented pain-autonomic processing (Blackburn-Munro & Blackburn-Munro, 2001; Bruns & Disorbio, 2005; Caspi et al., 2003; Dersh et al., 2006; Dersh, Gatchel, Polatin, & Mayer, 2002; Lyon, Cohen, & Quinter, 2011). Where the current study was not intended to delineate the direct and indirect effects of depression on TMD symptomatology, there is evidence to suggest that restoration of the autonomic nervous system through early intervention is efficacious in decreasing TMD specific symptoms, pain and pain-related disability, and emotional distress through adaptive coping (Bernstein & Gatchel, 2000; Gardea, 1998; Gardea, Gatchel, & Mirsha, 2001; Gatchel & Okifuji, 2006; Phillips et al., 2001; Robinson, Garofalo, & Gatchel, 2006). The current analyses reveal substantive changes among diagnostic criteria prior to changes in actual diagnostic status. Although we did not observe differences between our Intervention groups, we must consider the point that the effects of the biobehavioral intervention are contingent upon replacing maladaptive responding with adaptive responding to pain. Therefore, it seems logical that the influence of stress reduction and restoration among interdependent neuroendocirne, neuroimmune, and neurotransmitter systems by biobehavioral interventions which are known to modulate these systems (Bernstein & Gatchel, 2000; Chapman, Tuckett, & Song, 2008; Gardea, 1998; Gardea, Gatchel, & Mirsha, 2001; Gatchel & Okifuji, 2006; Phillips et al., 2001; Robinson, Garofalo, & Gatchel, 2006), would be delayed initially and observed at the long-term follow-up. In any case our results suggest that the comorbid symptoms of CSS are highly related to myofacial TMD and that this symptomatology is influenced by emotional distress. For example, in Study 1,

symptoms specific to some of the disorders within the CSS spectrum and symptoms specific to myofacial TMD were fully mediated by emotional distress with BDI having a stronger influence on these symptoms compared to SF-36 MHC, or overall mental health. This falls in line with research suggesting chronic stress responding and consequent emotional distress (i.e., depression) are primary culprits of augmented pain-autonomic processing and CS. Moreover, the current research contributes to the growing knowledge regarding how TMD and symptoms of CSS are promoted by emotional distress, which is known to share a reciprocal relationship with multiple systems dysregulation and CS (Bruns & Disorbio, 2005; Dersh et al., 2006; Caspi et al., 2003; Dersh, Gatchel, Polatin, & Mayer, 2002).

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BIOGRAPHICAL INFORMATION

Kara Lorduy studied at the University of Texas at Arlington in the Health Psychology and Neuroscience doctoral program under the mentorship of Dr. Robert Gatchel. During her five and half years of graduate training she had the opportunity to work with Dr. Andrew Baum in the area of psychoneuroimmunology with an emphasis on the effects of acute stress and psychosocial influences on DNA damage and repair, as well as metabolic function. Kara developed her foundation in research studying with Dr. Odegard in the area of cognitive psychology. During which time, Kara investigated differences in brain activation among those with Gulf War Illness, as well as the use of metacognitive strategies at various ages across the lifespan. Kara earned her Master's degree investigating the biopsychosocial influences of temporomandibular disorders (TMD) including comorbid symptoms of central sensitivity syndrome (CSS). Drawing on this analysis, Kara extended her study of the pathophysiological mechanisms of pain and pain disorders, such as TMD, fibromyalgia, and chronic fatigue syndrome, etc. Extensive review of the dynamic nature and development of central sensitization (CS) among those with chronic pain disorders provided the rationale and support for the current investigation and dissertation project, which examined the influences of the effects of early intervention on TMD specific and TMD non-specific symptoms of CSS. While working with Dr. Gatchel, Kara had the opportunity to work with Dr. Fatma Gul at the Physical Medicine and Rehabilitation Department at the University of Texas Southwestern Medical School. Currently, Kara works in pediatric research at Children's Medical Center in Dallas, Texas.