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A COMPARISON BETWEEN MASTICATORY MUSCLE AND TEMPOROMANDIBULAR JOINT PAIN PATIENTS WITH REGARD TO THE PREVALENCE AND IMPACT OF POST-TRAUMATIC STRESS DISORDER SYMPTOMS

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ABSTRACT OF THESIS

A COMPARISON BETWEEN MASTICATORY MUSCLE AND TEMPOROMANDIBULAR JOINT PAIN PATIENTS WITH REGARD TO THE PREVALENCE AND IMPACT OF POST-TRAUMATIC STRESS DISORDER SYMPTOMS.

The purpose of this study was to evaluate masticatory muscle (MM) and temporomandibular joint (TMJ) pain patients regarding the prevalence of Post-traumatic Stress Disorder (PTSD) symptoms, and evaluate the level of psychological dysfunction and its relationship to PTSD symptoms in these patients. This study included 445 adult patients (male = 42, female = 403). Psychological questionnaires included the Symptom Check List-90-Revised (SCL-90-R), the Multidimensional Pain Inventory, the Pittsburgh Sleep Quality Index and the PTSD Check List Civilian. The total sample of patients was divided into two major groups: The MM group (n=242) and TMJ group (n=203). Each group was divided into three subgroups according to the presence of a stressor and severity of PTSD symptoms. Thirty six patients (14.9%) in the MM group and 20 patients (9.9%) in the TMJ group presented symptomatology of PTSD. MM and TMJ pain patients in the “positive PTSD” subgroups scored higher on all scales of the SCL-90-R ($p = .000$) than the other two subgroups and reached levels of distress that were indicative of psychological dysfunction. MM and TMJ pain patients in the “positive PTSD” subgroups were more often classified as dysfunctional than as adaptive copers and presented with more sleep disturbances than patients in the “no stressor” and “negative PTSD” subgroups. A somewhat elevated prevalence rate for PTSD symptomatology was found in the MM than in the TMJ group. Significant levels of psychological dysfunction appear limited to temporomandibular disorder patients with symptoms of PTSD.

KEYWORDS: Prevalence, TMD, PTSD, psychological dysfunction, sleep disturbances.

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July 19, 2005

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THESIS

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2005

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THESIS

A thesis submitted in partial fulfillment of the
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Chapter 1. Introduction

Post-traumatic Stress Disorder (PTSD) is defined as a type of anxiety disorder that can develop following an individual's exposure to an event perceived to be threatening or traumatic according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)[1], see Table 1. As a consequence of such exposure, a person may develop a recurring pattern of symptoms. These symptoms include persistent re-experiencing of the traumatic event, nightmares, recurrent and intrusive recollections, avoidance of the situations associated with the traumatic event, sleeplessness, and hypervigilance that must be present for more than one month [1]. Post-traumatic Stress Disorder can be classified as acute, when symptoms are present between one and three months or chronic when symptoms last for three months or more. Finally, PTSD can be classified as delayed onset when at least six months have passed between the traumatic event and the onset of the disorder. Post-traumatic Stress Disorder may coexist with others psychological disorders [2], [3] and also with chronic pain conditions such as fibromyalgia [4], [5], headache [6] and temporomandibular disorder (TMD) [7], [8].

Temporomandibular disorders (TMDs) comprise a number of clinical problems involving the masticatory muscles and/or the temporomandibular joint (TMJ) joint [9] that also have been associated with elevated levels of depression and anxiety [10], [11], [12], [13]. Studies comparing the two most common categories of TMD, masticatory muscle (MM) pain and TMJ/intracapsular pain, revealed that MM pain patients are more psychologically distressed than TMJ pain patients [14],

[15], [16], [17]. In general, psychological distress has been linked to increased pain level in a number of investigations [4], [12], [18], [19], [20].

Anxiety disorders, such as PTSD, may have the potential to magnify the subjective perception of pain [21]. Several studies have examined the relationship between PTSD and chronic pain [5],[18], [22], [23], [24], [25],. For instance, Sherman et al found in a sample of fibromyalgia patients that pain level, disability and affective distress was greater in those patients reporting PTSD symptoms than those who did not report such symptoms [4]. There are a small number of investigations reporting the comorbidity of PTSD and TMD [7], [8], [19]. A recent investigation in chronic orofacial pain patients revealed that the patients who reported symptoms of PTSD were more psychologically distressed and more prone to be classified with a dysfunctional profile than patients who did not report symptoms of PTSD [19]. An additional finding of this investigation was that clinically significant levels of psychological distress are strongly linked with PTSD. It also has been reported that traumatic experiences and more PTSD symptoms were observed in MM pain patients compared to TMJ pain patents [17], [26]. Apparently, the coexistence and interaction of chronic pain/ TMD and PTSD is related to an increased psychological distress, elevated levels of pain and greater disability. Such characteristics may influence a patient's adaptability to disease and treatment outcomes. Consequently, the successful management of patients with chronic pain/TMD requires assessment of comorbid psychological conditions. Thus, screening for PTSD should be included as part of a TMD patient's evaluation.

Table1. Diagnostic Criteria for PTSD, DSM-IV[1].

A	The person has been exposed to a traumatic event in which both of the following were present :
1	The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
2	The person's response to the event involves intense fear, helplessness or horror
B	The traumatic event is persistently re-experienced in one (or more) of the following ways:
1	Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions
2	Recurrent distressing dreams of the event
3	Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated)
4	Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
5	Psychological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
C	Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
1	Efforts to avoid thoughts, feeling, or conversations associated with the trauma
2	Efforts to avoid activities, places, or people that arose recollections of the trauma
3	Inability to recall an important aspect of the trauma
4	Markedly diminished interest or participation in significant activities
5	Feelings of detachment or estrangement from others
6	Restricted range of affect (e.g., unable to have loving feelings)
7	Sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or normal life span)
D	Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
1	Difficulty falling or staying asleep
2	Irritability or outbursts of anger
3	Difficulty concentrating
4	Hypervigilance
5	Exaggerated startle response
E	Duration of the disturbance (symptoms in criteria B,C, and D) is more than one month
F	The disturbance causes clinically significant distress or impairment in social, occupational or other important areas of functioning

PTSD: Post-traumatic Stress Disorder.

DSM-IV: Diagnostic and Statistical Manual of Mental Disorders-IV.

Chapter 2. Purpose of the Study

The aim of this investigation was to evaluate differences between MM pain patients and TMJ pain patients who sought treatment at the Orofacial Pain Center in the University of Kentucky with regard to the prevalence of PTSD symptoms. A second aim of this study was to analyze the level of psychological dysfunction and its relationship in regard to the presence and impact of PTSD symptoms in MM pain patients and TMJ pain patients. We hypothesized that the prevalence of PTSD symptoms would be higher in MM pain patients than in TMJ pain patients. In addition, given the complicated nature of PTSD symptoms we hypothesized that the presence of this symptomatology would influence the level of psychological dysfunction in MM and TMJ pain patients in several domains.

Chapter 3. Review of the Literature

3.1. Temporomandibular Disorders

3.1.2. Temporomandibular Disorders and Psychological Distress

According to the Research Diagnostic Criteria [27], TMDs are divided in two main categories: MM pain and TMJ (intracapsular) pain. Regarding etiology, TMD is often considered complex and multifactorial in nature. Factors such as trauma, constant deep pain input, orthopedic instability, parafunctional activities, systemic factors and emotional stressors have been implicated in the etiology of TMD [28]. Dworkin and LeResche [27] pointed out the importance of adding to the Axis I which, comprises the physical conditions responsible for the etiology of pain, a psychological aspect, the so called Axis II in the diagnosis of TMD. Axis II involves the psychological condition and its effects in producing and/or influencing the whole pain experience.

Several lines of evidence have linked TMD and psychological distress [11], [29], [30]. For instance, elevated levels of depression and anxiety are common findings in TMD patients compared with healthy controls [10], [16], [31], [32], [33], [34]. Psychological distress is more often associated with MM pain patients than TMJ pain patients [14], [15], [17]. In fact, MM pain patients report elevated levels of depression, pain disability and exposure to major life stressors when compared to intracapsular pain patients [20], [35]. Major life stressors in turn have been associated with high levels of pain, affective distress and disability in TMD

patients [20]. Lampe et al [36] noted that stressful life events such as childhood abuse and depression experienced by chronic pain patients had a significant impact on the occurrence of the chronic pain condition. In regard to TMD, Curran et al [12] reported that 68.9% of orofacial pain patients reported a history of physical and sexual abuse in an anonymous survey. The history of abuse was significantly correlated to depression, psychological distress and greater pain severity. There is also evidence suggesting that TMD patients suffer stressful life events prior to the onset of their symptomatology [20], [37], [38]. Overall, traumatic life experiences seem to interfere with the well-being of patients and may have a substantial link to the occurrence of TMDs.

The activity of the sympathetic portion of the autonomic nervous system is increased by emotional stress. This is the so called stress response or “fight or flight” response which is characterized by an increase in the arterial blood pressure, blood flow to muscles, muscle activity and mental activity [39]. Although, this increased autonomic activity is normal for acute stressors, major life events and/or persistent chronic stressors may also have long term consequences for an individual depending on how s/he perceives a situation and her/his general state of physical health [40]. McEwen in 1998 described these consequences as an increased activity of the “allostatic systems” (autonomic nervous, cardiovascular, metabolic and immune systems and hypothalamic-pituitary-adrenal (HPA) axis), the allostatic load theory, that may result in chronic overactivity of these systems [40]. It is known, for example, that chronic emotional stress may produce pain and increase its severity by precipitating and increasing activity in the central nervous

and musculoskeletal systems [41]. In addition, it has been demonstrated that there is increased cardiovascular activity and altered breathing rate in chronic TMD patients compared to normal controls [34].

An additional response to a stressor is the activation of the HPA-axis in response to stimulation of the sympathetic nervous system. This activation releases catecholamines that stimulate the secretion of corticotrophin-releasing factor (CRF) from the hypothalamus. Corticotrophin-releasing factor then induces the secretion of adrenocorticotropin hormone (ACTH), from the pituitary gland. It, in turn, mediates the release of cortisol from the adrenal cortex. This sequence of events that culminates in an increase of cortisol levels is a normal and ordinary response to any type of acute stress (physical or mental). The acute increase in cortisol is of significant benefit because glucocorticoids cause rapid mobilization of aminoacids and fats making them available for energy and for synthesis of other compounds [39]. However, chronic activation of the HPA-axis can cause pathophysiologic consequences such as down-regulation of the hippocampal glucocorticoid receptors and toxicity to hippocampal neurons leading to cognitive impairment that can alter memory [42], [43], [44].

Dysregulation of the HPA-axis has been related to several psychological disorders as well as to stress-related bodily disorders. In fact, hyperactivity of the HPA-axis that produces high levels of cortisol is an ordinary finding in affective disorders such as depression [13], [45], [46]. In contrast to affective disorders, stress-related bodily disorders like chronic fatigue syndrome and fibromyalgia are characterized by hypocortisolism [46], [47], [48], [49]. In addition, hypocortisolism has

been found in other somatoform disorders such as rheumatoid arthritis and asthma [50]. Indeed, hyporesponsiveness of the HPA-axis is associated with increased inflammatory cytokines [51] and its consequences have also been reported in animal studies [52]. The animals in these studies were very susceptible to autoimmune and inflammatory disturbances [52].

It also appears that dysregulation of the HPA-axis plays an important role in chronic pain and may actually predispose vulnerability for the development of chronic pain [50]. With TMDs, however, inconsistent findings have been reported in the neuroendocrinologic investigations. For instance, Jones et al in 1997 analyzing TMD patients and a control group noted that the TMD group was heterogeneous in regard to the levels of cortisol released in response to stress. The authors found TMD patients with increased cortisol levels and TMD patients whose cortisol levels were not different from healthy controls [53]. Other investigation revealed hypercortisolism in a group of 15 women with TMD patients compared to a control group [54]. Even though the patients were initially screened for psychological disorders, three subjects presented with symptoms of major depression. Interestingly, the methodology included the assessment of cortisol by plasma levels. It has been speculated that cortisol levels may fluctuate as a result of transient stressors in the environment including the actual stress of venipuncture or anticipatory anxiety associated with venipuncture [55]. Overall the findings suggest that chronic pain disorders including TMD potentially are related to dysregulation of the HPA-axis.

3.1.3. Temporomandibular Disorders and Sleep Disturbance

From the available literature, it is currently unknown whether chronic pain conditions produce a sleep disturbance or whether a sleep disturbance is significant in the initiation of the chronic pain condition itself. According to Okeson, sleep disturbances may be a systemic perpetuating factor that can cause the progression of an acute muscle pain to a chronic pain condition [28]. The available literature reveals a strong relationship between sleep problems and chronic pain. Poor sleep quality has been linked to chronic pain conditions [56], [57], [58], [59]. The deeper stages of sleep are important to restore function of the body systems such as the metabolic process. In fact, Moldofsky and Scarisbrick in 1976 noted that stage-four deprivation led to musculoskeletal symptoms such as muscle tenderness and stiffness in healthy subjects, but such symptoms were not observed following disruption of the rapid eye-movement (REM) sleep [60]. It appears that deprivation of the deeper stages of sleep may result in muscle pain because of the inability of an individual to repair damaged tissues. Patients with TMD frequently report sleep disturbance [61], [62], [63]. In regard to the two categories of TMD, patients in the MM pain category report more sleep disturbances compared to patients in the TMJ pain category [17], [64]. In addition, in TMD patients the sleep disturbance is related to elevated levels of pain severity and psychological distress [16], [30].

3.1.4. Summary

From the aforementioned review it is apparent that there is a positive relationship between TMD and emotional stressors. This relationship may disturb the well-being of the patient and may impact her/his ability to cope with the illness. Sleep disturbances often coexist with TMD and may be considered a contributing factor that may play an important role in the individual's recovery. Although, inconsistent findings of the neuroendocrinologic investigations have been reported in the literature, it seems that like in many other pain conditions, TMD is associated with dysregulation of the HPA-axis.

3.2. Post-traumatic Stress Disorder (PTSD)

Symptoms of PTSD have been reported in victims following rape [65], [66], motor vehicle accident [22], [67], [68], combat veterans experiences [69], medical conditions [70], [71] terrorist attacks [72], [73] and natural disasters [74]. Based on community studies, the lifetime prevalence of PTSD ranges from 1% to 14% [1]. Reports of at-risk individuals (e.g. combat veterans, survivors of natural disasters, terrorist attacks or criminal violence) revealed prevalence rates raging from 3% to 58% [1], [72], [75], [76].

Considering chronic pain patients, several lines of evidence suggest a high prevalence of PTSD symptoms in such patients [4], [5], [18], [24], [77]. In addition, PTSD co-occurs with other psychiatric diagnosis such as mood, anxiety and

substance abuse disorders. In fact, one study showed that approximately 80% of PTSD patients met criteria for at least one other psychiatric disorder [2]. Depression is the most widely investigated comorbid disorder [3]. For example, Hickling et al in 1992 analyzing headache patients who were victims of motor vehicle accident meeting diagnostic criteria for PTSD, noted that major depression was also present among those patients [22]. The literature documenting a positive relationship between PTSD and substance abuse is also extensive [78], [79], [80], [81], [82]. Indeed, recent studies noted a high prevalence of cigarette smoking in patients with current PTSD symptoms [83], [84]. The authors suggested a link among anxiety, PTSD, and substance abuse.

Dysregulation of the HPA-axis is a common finding in patients reporting symptoms of PTSD. Although inconsistent findings have been reported in the literature regarding cortisol levels in PTSD patients [55], there is a trend for PTSD to be linked to hypocortisolism [46], [55], [85], [86]. The inconsistent findings could be attributed to differences in the methodological assessment used in the studies. An additional factor that could contribute to these differences is the coexistence of others psychiatric disorders, such as depression, which may influence the findings since depression has been associated with hypercortisolism [87]. It seems that a variation of the psychiatric symptomatology over time in PTSD patients may also influence cortisol levels. In addition, the time frame between the trauma suffered by an individual and the investigation may also affect cortisol findings [55]. For example, there may be a difference in cortisol levels during or immediately after the traumatic event as compared to years after the traumatic event. It has

been reported that early life stressors may result in a persistent sensitization of the HPA-axis to stressors in adulthood [46]. It also has been suggested that chronic PTSD is associated with increased negative feedback inhibition of cortisol, due to altered glucocorticoid receptor activity [88]. Symptoms of PTSD such as increased response to stress, hypervigilance and arousal are consistent with dysregulation of the HPA-axis [88].

3.2.1. PTSD and Sleep Disturbances

Sleep disturbances are included in the symptomatology of PTSD according to the DSM-IV [1]. They are included in the category of re-experiencing symptoms, for example, nightmares, and in the category of arousal that contributes to difficulty falling or staying asleep. Sleep disturbances and nightmares are part of a normal and typical response following traumatic or threatening trauma [89]. However, for some individuals the sleep disturbance becomes a persistent problem. For instance, in a study of the survivors of the Oklahoma City bombing, 70% of the survivors reported sleep disturbance six months following the event [90]. In addition, sleep problems were the most common symptom reported by survivors of natural disaster [91], war prisoners [92] and holocaust victims [93].

The majority of the studies reporting association between sleep disturbances and PTSD used subjective measures such as self-reported symptoms, questionnaires and medical interviews [94]. Symptoms most frequently

reported by PTSD patients are difficulty falling or staying asleep, shorter sleep duration, restless sleep, daytime fatigue and nightmares [95], [96]. Such sleep disturbances may exacerbate symptoms of PTSD. In fact, several research groups have reported the clinical importance of the sleep disturbance's treatment in PTSD patients [97]. A causal relationship between sleep disturbance and PTSD has not yet been reported. Harvey et al in 2003 critically assessed the evidence on the prevalence and treatment of sleep disturbance of patients with PTSD. They concluded that there is a clear association between PTSD and sleep problems. However, the role sleep disturbances play in the mechanism of PTSD is unclear [89]. In other words, further studies are needed to determine whether a causal relationship between sleep disturbance and PTSD exists.

3.2.2. Summary

Following exposure to a traumatic or threatening incident an individual may develop symptoms of PTSD. According to the literature, PTSD has been linked to other psychiatric disorders such as depression and anxiety as well to chronic pain disorders. In addition, a positive relationship exists between PTSD and substance abuse and sleep disturbances. Similar to TMD, PTSD has been associated with dysregulation of the HPA-axis.

3.3. TMD and PTSD

Few studies have investigated the relationship between TMD and PTSD. Evidence reporting a high prevalence of PTSD symptoms in orofacial pain patients comes from clinical data [7], [8], [19]. Several lines of evidence suggest a higher prevalence of PTSD symptoms in the MM pain category compared to the TMJ pain category [17], [19], [26]. Aghabeigi in 1992 found that 15% of patients with chronic idiopathic pain had a history of PTSD which coincided with the pain onset [7]. Sherman et al in 1998 found a PTSD prevalence rate of 23% in chronic face pain patients [8]. In this study, patients with symptoms of PTSD reported higher levels of pain, greater affective distress and less control over their lives than patients without PTSD symptoms. Similarly, de Leeuw et al in 2005 also noted increased pain severity, affective distress and disability among orofacial pain patients with symptomatology of PTSD. A persistent finding in orofacial pain patients [19], headache patients [98] and neuropathic pain patients [99] is that the presence of PTSD symptoms may dictate elevated levels of psychological distress in several domains.

The findings with regard to pain level, affective distress and disability for TMD patients with PTSD symptoms are in accord with previous publications in chronic pain. For example, Geisser et al in 1996 demonstrated that chronic pain patients with elevated PTSD symptoms reported increased pain and affective distress [18]. In addition, Sherman and colleagues in 2000 evaluated a sample of fibromyalgia patients with and without symptomatology of PTSD noted higher level of pain, emotional distress, life interference and disability among patients reporting PTSD symptoms as compared to patients who did not report symptoms

of PTSD [4]. A common characteristic of patients with symptoms of PTSD and chronic pain is that these patients present with difficulty in coping and adapting to their pain [4], [19]. These patients are also frequently classified as dysfunctional or interpersonally distressed [4], [19], [100], on the MPI profile classification. The same tendency is observed in TMD, and seems to be more pronounced in muscle pain patients than in patients with primarily joint pathology [17], [64].

Based on this review, it seems that TMD, especially the MM pain category, and PTSD exhibit common symptoms such as depression, anxiety, sleep disturbances and dysregulation of the HPA-axis. In fact an overlap of symptoms between chronic pain in general and PTSD has been observed in the literature [23]. Asmundson et al in 2002 discussed two mechanisms that may explain the co-occurrence of these disorders, the shared vulnerability hypothesis and the mutual maintenance hypothesis. For the shared vulnerability hypothesis, an anxiety disorder may be a predisposing factor for both conditions, thereby increasing the susceptibility for development of both conditions [23]. Considering the mutual maintenance mechanism that was first introduced by Sharp and Harvey in 2001 [101], it is proposed that characteristics of either chronic pain or PTSD may reciprocally maintain or exacerbate the symptoms of the other disorder. It is postulated that for PTSD patients, chronic pain provides a persistent reminder of the trauma, thus in such patients an attentional bias towards the pain experience may exacerbate the pain condition. Furthermore, pain sensation may be exacerbated by high levels of anxiety. In addition, depression may maintain the symptomatology of PTSD and chronic pain. In

summary, the mutual maintenance hypothesis is based on the following mechanisms generated by distress: 1) attentional and reasoning biases; 2) anxiety sensitivity; 3) reminders of the trauma; 4) avoidance; 5) depression and reduced activity levels; 6) anxiety and pain perception and 7) cognitive demand from symptoms limiting use of adaptive strategies. The persistence of these factors may lead to disability.

A third mechanism that would confirm the coexistence of chronic pain and PTSD is the fear-avoidance model. Fear and avoidance may occur in response to chronic pain and may also be a symptom of PTSD [102]. In chronic pain, fear-avoidance occurs in response to the avoidance of movement that would increase pain sensation. In general, physiological symptoms such as increased blood flow, heart rate, or muscle tension may increase pain sensation and emphasize fear towards activities that will result in avoidance of such activities [103]. For PTSD, a characteristic symptom is the fear of thinking about or talking about the stressful experience, a symptom that would culminate in avoidance of activities or situations associated with the traumatic experience. In addition, Otis et al in 2003 discussed the triple vulnerability model as a fourth hypothesis linking the coexistence of chronic pain and PTSD [102]. The triple vulnerability model is characterized by a generalized biological vulnerability, a generalized psychological vulnerability and a more specific psychological vulnerability. Keane and Barlow in 2002 adapted this model to propose a hypothesis for the development of PTSD [104]. They suggested that in order to develop PTSD one must develop anxiety and an unpredictable and uncontrolled emotional reaction

to an event that resembles the traumatic event. Thus, when negative affect and the sense of uncontrollability are present, PTSD may develop. Otis et al in 2003 [102] proposed an extrapolation of this model to chronic pain whereby the pain experience itself could be perceived as an unpredictable and uncontrollable sensation leading to a lack of personal control over the pain. This in turn may lead to feelings of low self-efficacy, negative affect and avoidance of daily life situations. In summary, four models have been presented linking chronic pain and PTSD. The relationship between chronic pain and PTSD may have implications in treatment outcome for both conditions.

3.3.1. Summary

A high prevalence of PTSD in chronic pain patients including TMD patients is a common finding in the epidemiological studies. Chronic pain patients with PTSD symptoms frequently report higher levels of pain, affective distress and disability than chronic pain patients without PTSD symptoms. In addition, chronic pain patients with symptomatology of PTSD are more often classified with a dysfunctional profile than an adaptive coper profile on the MPI profile classification and present with clinically significant psychological distress on several domains. Due to the fact that both patients with chronic pain and patients with PTSD present with common symptomatology such as depression, anxiety, and/or dysregulation of the HPA-axis, an overlap of symptoms has been proposed. These mutual characteristics as proposed in four mechanisms

described above may influence prognostic and treatment outcomes of chronic pain patients with symptoms of PTSD.

3.4. PTSD, TMD and Treatment Outcomes

The effectiveness of biobehavioral strategies for management of TMD has been evaluated in a number of studies [105], [106]. In fact, biobehavioral approaches such as proprioceptive awareness training, relaxation, diaphragmatic breathing training and awareness and control of parafunctional activities have been indicated for short and long term management of TMDs, especially those with MM pain as the primary symptom [107]. Similarly, psychological approaches are currently identified as a first choice for treatment of PTSD [108]. Psychological treatments for PTSD include exposure therapy, which was first used by Black and Keane [109] to treat PTSD among combat veterans. Anxiety management that involves relaxation training, breathing retraining, trauma education, cognitive restructuring, or communication skill training can also have a favorable impact on the symptoms of PTSD [110]. In addition to psychological treatment, pharmacotherapy for PTSD has also been suggested [111]. In a recent report, Stein and coworkers systematically reviewed randomized controlled trials of pharmacotherapy for PTSD and concluded that medication should be considered as part of the treatment of PTSD. Although they could not demonstrate differences among classes of medication with regard to efficacy or better tolerance, the largest trials showing efficacy were those evaluating selective

serotonin re-uptake inhibitors. In addition, glucocorticoids (hydrocortisone) [112] and propranolol [113] have been suggested to prevent development of PTSD.

Regarding the coexistence of the two conditions, management of chronic pain patients with symptoms of PTSD should include treatment directed to the anxiety disorder and the pain disorder. In fact, simultaneous treatment of chronic pain and PTSD has been suggested in the literature[4], [23]. In view of the fact that chronic pain and PTSD may exacerbate each other's symptoms, management accomplishment of chronic pain patients with PTSD symptoms may be compromised. For example, depression in chronic pain patients has been associated with poor treatment response [114] and prematurely abandoning treatment [115]. In addition, a dysfunctional profile has been related to poor treatment outcome in TMD patients [15]. It seems logical that addressing all coexistent factors in both disorders may potentially lead to favorable treatment outcome, although further studies are necessary to determine whether it is necessary to address both conditions simultaneously in order to observe patient's improvement in general. Perhaps addressing one of the two conditions or even one aspect these conditions have in common such as depression and/or anxiety or sleep disturbances could be sufficient to have a positive treatment outcome in both chronic pain and PTSD.

Unfortunately, only a small number of studies addressing treatment outcome in chronic pain patients with PTSD symptoms have been reported in the literature. For instance, Hickling et al noted that patients with post-traumatic headache meeting criteria for PTSD required significantly longer cognitive

behavioral treatment than controls [116]. Recently, a case study evaluating the effects of PTSD treatment in chronic pain patients was reported [117]. The patient sample was composed of six females not responding to standard pain interventions such as surgery, physical therapy and medication including selective serotonin re-uptake inhibitors, non-steroids antiinflammatory and anticonvulsants. The PTSD treatment included a number of psychological approaches such as imaginal and in vivo exposure, relaxation techniques, social support, anger management and pleasant event scheduling. The authors found reduction in PTSD symptoms, improvement in dysfunction associated with pain, such as working status and time spent in bed following treatment. There was, however, no subjective reduction in pain reported by the subjects.

3.4.1. Summary

Addressing all coexisting factors may be the key to successful treatment in chronic pain patients with symptoms of PTSD. Behavioral treatment seems to be a promising approach for both chronic pain and PTSD. In fact, it appears that targeting PTSD symptoms may improve treatment outcomes overall in chronic pain patients. Additionally, educational strategies aimed at increasing patients' recognition of the potential association between PTSD and chronic pain may be helpful as well. However, the effect of such treatment has not been systematically evaluated. Further investigations are needed to evaluate long-term

treatment outcome and whether decreasing symptoms of PTSD has a positive effect in the treatment response of chronic pain patients.

Based on the aforementioned review of literature, the purpose of this study was to evaluate differences between MM pain patients and TMJ pain patients who sought treatment in an orofacial pain center in regard to the prevalence of PTSD symptoms. A second aim of this study was to analyze the level of psychological dysfunction and its relationship in regard to the presence and impact of PTSD symptoms in MM pain patients and TMJ pain patients. We hypothesized that the prevalence of PTSD symptoms and the level of psychological distress would be higher in MM pain patients than in TMJ pain patients. In addition, given the complicated nature of PTSD symptoms we hypothesized that the presence of this symptomatology would influence the level of psychological dysfunction both in MM and TMJ pain patients in several domains.

Chapter 4. Experimental Design and Methods

4.1. Participants

This study was a retrospective analysis of psychometric and sleep disorders data obtained from patients during the initial visit at an orofacial pain clinic as part of a standard evaluation protocol. The patient sample was selected from patients seen at the Orofacial Pain Center at the University of Kentucky, College of Dentistry from 1997 to 2005. Patients with both a primary and secondary diagnosis of MM pain or both a primary and secondary diagnosis of TMJ pain according to the Research Diagnostic Criteria [27] were eligible to participate in this study. Patients with a single diagnosis of either MM pain or TMJ pain were also eligible. Patients with a primary diagnosis of TMJ pain and a secondary diagnosis of MM pain or vice versa were not eligible. As part of the Orofacial Pain Center protocol all participants already have signed the standard "Patient Registration/Consent form" upon arriving for their initial evaluation.

All patients completed an orofacial pain questionnaire and a battery of psychological questionnaires as part of the initial evaluation/examination. The psychological questionnaires included the Symptom Check List-90-Revised (SCL-90-R) [118], the Multidimensional Pain Inventory (MPI) [119], the Pittsburgh Sleep Quality Index (PSQI) [120] and the PTSD Check list Civilian (PCL-C) [121]. These questionnaires embrace an extensive variety of symptoms and behaviors

that are important tools to develop a thorough treatment/management plan for chronic pain patients.

The Orofacial Pain Center initial evaluation/examination involves an extensive interview where a detailed patient's history is taking followed by conduction of clinical examination. The main objective of the history-taking is to acquire an accurate description of the patient's chief complaint; information about key elements of each complaint(s), including onset, location, intensity, duration and associated factors is obtained. In addition, a psychologic assessment is also obtained. The clinical examination is composed of physical measures such as blood pressure and pulse rate, cranial nerve examination, cervical evaluation, muscle palpation with special emphases on myofascial trigger points and pain referral, TMJ evaluation (palpation, loading, joints sounds, deviation and deflection), range of mandibular movement and an intraoral examination. For this study the examinations were performed by dentists with advanced training in the diagnosis of orofacial pain conditions. All examiners were trained in the Orofacial Pain Center of the University of Kentucky within the guidelines of the American Academy of Orofacial Pain [9].

The total sample of patients was divided into two major groups: Masticatory muscle (MM) and temporomandibular joint (TMJ) group. The MM group comprised patients with a primary and, when given, a secondary diagnosis of masticatory muscle pain [27]. The TMJ group comprised patients with a primary and, when given, a secondary diagnosis of TMJ/intracapsular pain [27]. Subsequently, each group was divided in three subgroups according to the

presence of a reported stressor (s) and severity of PTSD symptoms. The diagnoses of PTSD symptoms were based on the PCL-C, which corresponds to the DSM-IV criteria for PTSD (Table 2). Both groups were subcategorized as “no stressor”, “negative PTSD symptoms”, “positive PTSD symptoms” according to the presence of stressor and degree of PTSD symptomatology reported on the PCL-C. A score of 41 is considered the cut-off point for clinical significance of PTSD symptomatology [122]. The “no stressor” group comprised patients who did not report a stressor on the PCL-C. The “negative PTSD symptoms” group comprised patients who reported one or more stressor(s) on the PCL-C but did not meet criteria for PTSD symptoms (PCL-C score < 41). The “positive PTSD symptoms” group comprised patients who reported one or more stressor(s) on the PCL-C and met criteria for PTSD symptoms (PCL-C score ≥ 41).

4.2. Inclusion Criteria

Patients who presented with the following characteristics were included in the study:

1. At least18 years of age.
2. A single diagnosis of TMJ or MM pain or, when a secondary diagnosis was given, both primary and secondary diagnoses of either MM or TMJ pain according to the Research Diagnostic Criteria.
3. Pain duration of at least three months.

4. Pain intensity of at least three on visual analogue scale.
5. No stressor reported on the PCL-C for inclusion in the “no stressor” subgroup.
6. Stressor(s) reported and PCL score < 41 for inclusion in the “negative PTSD symptoms” subgroup.
7. Stressor(s) reported and PCL-C score ≥ 41 for the inclusion in the “positive PTSD symptoms” subgroup.

4.3. Psychometric Measures

For this study data from the SCL-90-R, MPI, PSQI and PCL-C were used. The SCL-90-R is a 90–item self–report inventory that is used to assess psychological symptoms and yield nine symptoms dimensions and three global indices of functioning. Patients were asked to rate each item on a 5-point scale (from 0 “not at all” to 4 “extremely”) for how much each item has distressed or bothered them during the last 7 days including the day of the examination. From the SCL-90-R the presence and extent of symptoms such as somatization, obsessive-compulsive, interpersonal sensitivity, anxiety, depression, hostility, phobic anxiety, paranoid ideation, and psychoticism were obtained. The reliability and validity of the SCL-90-R has been demonstrated in a great number of studies summarized by Derogatis in 1983 [123]. Test-retest reliabilities range from r = 0.78 to 0.90 for non-patient samples, and internal consistencies range from 0.77 to 0.90 [118].

The MPI includes three sections and contains 61 questions, which furnishes data regarding pain severity, perceptions of how pain interferes with life, appraisal of the amount of support received from spouse or significant other, perceived life control, affective distress including rates of depressed mood, irritability, tension, and social and general activities. In addition, it provides a patient profile classification, which includes dysfunctional, interpersonally distressed and adaptive coper profiles. These three profiles are considered the prototypic profiles. The category “Dysfunctional” comprises patients who report a high level of pain, distress, and disability and who feel pessimistic and helpless about their condition. The category “Interpersonally distressed” includes patients with the same characteristics as “Dysfunctional” but who also report poor social support. The category “Adaptive coper” includes patients who report low levels of pain, disability, and distress. In addition, three other profile classifications may be given. These include hybrid, anomalous and unanalyzable profiles. The “Hybrid” profile represents a combination of prototypic profiles. The “Anomalous” profile comprises MPI scale scores that make no sense to established theory; reasons for such profile could be random responding, reading or responding difficulties, or faking bad or good. Finally, the “Unanalyzable” profile is given when data are missing, and therefore, statistical analyses of the scores are not possible. Kerns et al [119] have demonstrated the validity of the MPI across chronic pain patients [119]. Test–retest reliabilities range from r = 0.68 to 0.86 and internal consistencies range from 0.73 to 0.90 [119].

The PSQI is an 18-item self-report measure used to appraise general sleep quality. It provides information regarding the number of hours spent in bed and asleep, sleep latency, frequency and reasons for awakening, difficulty returning to sleep after awakening, sleep efficiency and use of sleep medication. The PSQI has demonstrated test–retest stability (r = 0.85) and internal consistency (alpha= 0.83) and provides valid and reliable assessment to overall sleep quality and disturbance [120], [124].

The PCL-C is a 17-item self-report measure, used to assess the incidence of significant stressor(s) and prevalence of PTSD symptomatology. Before completing the 17-item measure, the patient is asked to identify any significant stressors s/he has experienced on a 15-item experience list. The list includes military combat, violent attack, being kidnapped, taken hostage, terrorist attack, torture, incarceration, natural or man-made disaster, severe auto accident, being diagnosed with a life-threatening illness, sudden injury/serious accident, observed someone hurt or killed, learned that your child has a life-threatening illness, and “others”. Subsequently, the patient is asked to identify the most significant stressor, indicate the date of occurrence and appraise how much the most significant stressor has bothered her/him in the past month on the 17-item measure. In this segment, 17 items are rated on a 5-point scale (1: “not at all”, 2: “a little bit”, 3: “moderately”, 4: “quite a bit”, and 5 “extremely”; see table 2). The PCL-C has exhibited test-retest stability (r = 0.96), good overall internal consistency (alpha = 0.92), and provides a valid and reliable assessment of the presence of PTSD symptoms [122].

4.4 Statistics Analyses

Initial analyses were conducted by comparing the two diagnostic (MM and TMJ) groups. Diagnostic, demographic, MPI profile, and prevalence of PTSD symptomatology data between the two groups were tested using chi-square analyses. Age, pain severity, and pain duration were tested using student's t-tests. After these initial comparisons, each diagnostic group was divided into three subgroups depending on prevalence and intensity of PTSD symptomatology (no stressor, negative PTSD symptoms and positive PTSD symptoms subgroup). Analysis of variance ANOVA was used to test differences between the two diagnostic groups and among the three subgroups with regard to data from the SCL-90-R, MPI, and PSQI. The potential for family-wise error due to multiple comparisons during ANOVA was controlled for by using the Bonferroni correction. Significance level for all other comparisons was set at p=.05. All statistical analyses were conducted using the Statistical Package for the Social Sciences, Release 11.0 (SPSS Inc; Chicago, III).

Table 2. The PTSD Checklist-Civilian Version (PCL-C) [125].

1	Repeated, disturbing memories, thoughts, or images of the stressful experience	1	2	3	4	5
2	Repeated, disturbing dreams of the stressful experience					
3	Suddenly acting or feeling as if the stressful experience were happening again (as if you were reliving it)					
4	Feeling very upset when something reminded you of the stressful experience					
5	Having physical reactions (e.g., heart pounding, trouble breathing, sweating) when something reminded you of the stressful event					
6	Avoiding thinking about or talking about the stressful experience or avoiding having feelings related to it					
7	Avoiding activities or situations because they reminded you of the stressful experience					
8	Trouble remembering important parts of the stressful experience					
9	Loss of the interest in activities that you used to enjoy					
10	Feeling distant or cut-off from other people					
11	Feeling emotionally numb or being unable to have loving feelings for those close to you					
12	Feeling as if your future somehow will be cut short					
13	Trouble falling or staying asleep					
14	Feeling irritable or having angry outbursts					
15	Having difficulty concentrating					
16	Being super alert, or watchful, or on-guard					
17	Feeling jumpy or easily startled					

PTSD: Post-traumatic Stress Disorder.

Chapter 5. Results

5.1 Sample size, demographics characteristics, pain variables and prevalence of PTSD symptoms

The total patient sample was comprised of 445 adult patients (male = 42; female = 403; mean age 37.25 ± 12.9 years). The MM pain group was composed of 242 patients (male = 23; female = 219) with a mean age of 38.27 ± 12.9 years. The TMJ pain group was composed of 203 patients (male = 19; female = 184) with a mean age of 36.0 ± 12.8 years. Pain severity measured by a visual analogue scale where “0” is no pain and “10” is the most extreme pain, was 6.9 ± 1.9 and 6.4 ± 2.0 respectively for the MM group and TMJ group. Pain duration reported by patients was 42.9 ± 55.7 months and 46.7 ± 74.5 months respectively for the MM group and TMJ group. There were no significant differences between the two groups in regard to gender (p=.55), age (p=.739), pain severity (p=.053), pain duration (p=.108) and demographic characteristics (see table 3).

Of the entire sample, 206 patients (46%; 48% of the MM group and 44% of the TMJ group) reported to have experienced one or more significant traumatic stressors. Fifty six patients (12.6%) of the total sample presented with symptomatology of PTSD. More patients in the MM group (14.9%) than in the TMJ group (9.9%), met criteria for PTSD symptoms, but the difference was not statistically significant (p=.280; see table 4).

In the MM group there were no significant differences among the three subgroups (no stressor, negative PTSD and positive PTSD symptoms subgroups) in regard to gender (p=.161), age (p=.384), pain severity (p=.986) and pain duration (p=.935). Significant differences, however, were found in smoking status (χ^2=6.657; p=.036) and marital status (χ^2 =18.961; p=.004) where patients in the "positive PTSD subgroup" were more likely to be smokers, divorced and less likely to be married than patients in the other two subgroups. Additionally, patients in the "positive PTSD symptoms" were more likely to be applying for or receiving disability than patients in the other two subgroups (χ^2=24.476; p=.000).

In the TMJ group there were no significant differences among the three subgroups in regard to gender (p=.425), age (p=.331), pain severity (p=.074), pain duration (p=.632) and smoking status (p=.125). Significant differences, however, were found on marital status where patients in the "positive PTSD subgroup" were more likely to be divorced and less likely to be married than patients in the other two subgroups (χ^2= 18.961; p=.004). Additionally, patients in the "positive PTSD symptoms" were more likely to be applying for or receiving disability than patients in the other two subgroups (χ^2=24.476; p=.000).

With respect to the presence of a stressor and / or PTSD symptomatology, there were no significant differences between the MM and TMJ group in the "positive PTSD symptoms" subgroups in regard to gender (p=.288), age (p=.634), pain severity (p=.631), pain duration (p=.513), disability (p=.566) and demographic characteristics in general (p>.05). There were no significant

differences between the MM and TMJ group in the “negative PTSD symptoms” subgroup in regard to gender (p=.533), age (p=.462), pain severity (p=.063), pain duration (p=.370), disability (p=.503) and demographic characteristics in general (p>.05). There were also no significant differences between the MM and TMJ group in the “no stressor” subgroup in regard to age (p=.124), gender, (p=.410), pain duration (p=.632), disability (p=.052) and demographic characteristics in general (p>.05). A significant difference however, was found between the MM and TMJ groups in the “no stressor” in regard to pain severity with patients in the MM group reporting more severe pain than patients in the TMJ group.(p=.007).

5.2 Psychometric Data

5.2.1 SCL-90-R

Analyses of SCL-90-R data revealed higher scores on all subscales for patients in the MM group as compared to patients in the TMJ group, although these differences were not statistically significant for most scales (see table 5 and figure 1). In the MM group, there were significant differences among the three subgroups (p=.000) for all nine subscales of the SCL-90-R. Post hoc tests revealed that these differences were due to significant higher scores on the subscales in patients who reported a stressor and met criteria for PTSD symptoms (“positive PTSD symptoms” subgroup) than in the other two subgroups (see figure 2). The same pattern was observed for the TMJ subgroups

(p=.000; see figure 3) for all subscales. Only patients in the “positive PTSD symptoms” subgroups of both the MM and TMJ group reached levels of distress that were indicative of psychological dysfunction on almost all subscales of the SCL-90-R (T-Score ≥ 63; see figures 1, 2 and 3).

Considering the presence of a stressor and / or PTSD symptomatology, there were no significant differences between the MM and TMJ group in the “no stressor” subgroups on the SCL-90-R scales. A significant difference, however, was found between the MM and TMJ groups in the “negative PTSD symptoms” subgroups on the “phobic anxiety” subscale of the SCL-90-R (p=.016). In addition, a significant difference was found between the MM and TMJ groups in the “positive PTSD symptoms” subgroups on the “somatization” subscale of the SCL-90-R (p=.010; see table 6).

5.2.2 MPI and MPI profile classification

Significant differences were found between the MM and TMJ groups on most MPI scales. The MM group had significant higher scores on “interference”, “affective distress” and “punishing responses” scales and presented with lower scores on “life control”, “support”, “distracting responses”, “activities away from home”, “social activities” and “general activities level” scales than the TMJ group (see table 7).

In the MM group, there were significant differences among the three subgroups for the following scales of the MPI: “interference”, “life control”,

“affective distress”, “support” and “punishing responses” (see table 8). Post hoc tests revealed that the “positive PTSD symptoms” subgroup had significantly lower scores on the “life control” scale and significantly higher scores on the “affective distress” and “punishing responses” scales than the other two subgroups (see table 8). For the MPI scales “interference” and “support” significant differences were found between the “no stressor” and “positive PTSD symptoms” subgroup with patients in the “positive PTSD symptoms” subgroup reporting more “interference” and less “support” than patients in the “no stressor” subgroup (see table 8).

In the TMJ group, there were significant differences among the three subgroups for the following scales of the MPI: “interference”, “life control”, “affective distress”, “punishing responses”, “distracting responses”, “activities away from home”, “social activities” and “general activity level” (see table 9). Post hoc tests revealed that the “positive PTSD symptoms” subgroup had significantly lower scores on the “life control”, “activities away from home”, “social activities” and “general activity level” scales and significantly higher scores on the “affective distress”, “punishing responses” and “distracting responses” scales than the other two subgroups (see table 9).

Considering the presence of a stressor and / or PTSD symptomatology, there were no differences between the MM and TMJ groups in the “positive PTSD symptoms” subgroups. Significant differences however, were found between the MM and TMJ groups in the “no stressor” as well as in the “negative PTSD symptoms” subgroups with regard to three scales of the MPI with patients

in MM group reporting more life interference and affective distress and less life control (see table 10).

Approximately 50% of all patients were classified in one of the three main MPI profiles. Significant differences were found between the MM group and the TMJ group with regard to the MPI main profile classification (see table 11; the three non-specific profiles were not analyzed). Patients in the MM group were more often classified as dysfunctional or interpersonally distressed than patients in the TMJ group.

In the MM group, the patients in the “positive PTSD symptoms” subgroup were significantly more often classified as dysfunctional and patients in the “no stressor” and in the “negative PTSD symptoms” subgroups were more often classified as adaptive copers (see table 12).

In the TMJ group, the patients in the “no stressor” and in the “negative PTSD symptoms” subgroups were more often classified as adaptive copers than patients in the “positive PTSD symptoms” (see table 12).

Considering the presence of a stressor and / or PTSD symptomatology, there were no significant differences between the MM and TMJ group in the “positive PTSD symptoms” subgroups for the three MPI profile classifications (see table 13). However, a significant difference was found between the “negative PTSD symptoms” subgroups where patients in the MM group were more often classified as interpersonally distressed than patients in the TMJ group (see table 13). A significant difference was also found between the “no stressor”

subgroups where patients in the MM group were more often classified as dysfunctional than patients in the TMJ group (see table 13).

5.2.3 PSQI

Subjectively reported sleep problems were significant higher for the MM group than for TMJ group (see table 14). In the MM group, the subgroup “positive PTSD symptoms” reported more sleep problems on most scales of the PSQI than the subgroups “no stressor” and “negative PTSD symptoms” (see table 15). The same trends were seen with TMJ patients in the subgroup “positive PTSD symptoms” who reported more sleep problems on all scales of the PSQI than TMJ pain patients in the subgroups “no stressor” and “negative PTSD symptoms” (see table 16).

There were no significant differences between the MM and TMJ groups in the subgroups “positive PTSD symptoms” for any of the scales of the PSQI. Significant differences, however, were seen between the MM and TMJ group in the “no stressor” as well as in the “negative PTSD symptoms” subgroups on most scales of the PSQI, with the MM group reporting more sleep-related problems than the TMJ group (see table 17).

Table 3. Demographic Characteristics.

Variable	MM Group N (%)	TMJ group N (%)	Chi-square	p
Female	219 (90.5)	184 (90.6)	0.003	0.546
Male	23(9.5)	19 (9.4)		
Married	142 (59.9)	117 (62.6)	3.49	0.322
Single	57 (24.1)	51 (27.3)		
Divorced	32 (13.5)	17 (9.1)		
Widowed	6 (2.5)	2 (1.1)		
Full time employment	117 (48.3)	98 (48.3)	6.78	0.341
Part time employment	28 (11.6)	21 (10.3)		
Unemployed	32(13.2)	36 (17.7)		
Disabled	27 (11.2)	15 (7.4)		
Retired	11 (4.5)	8 (3.9)		
Student	13 (5.4)	6 (3.0)		
Receiving or applying for disability	37(15.6)	22 (10.9)	2.08	0.095
Lawyer consult	18 (7.8)	9(4.6)	1.80	0.126
Smoker	64 (26.9)	51 (25.1)	0.18	0.378

MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
N: number of patients; %: percentage.

Table 4. Prevalence of Stressors and PTSD Symptoms.

	MM Group N (%)	TMJ Group N (%)	Total N (%)
No Stressor	126 (52.1)	113 (55.7)	239 (53.7)
Negative PTSD symptoms	80 (33.1)	70 (34.5)	150 (33.7)
Positive PTSD symptoms	36 (14.9)	20 (9.9)	56 (12.6)

PTSD: Post-traumatic Stress Disorder.
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
N: number of patients; %: percentage.
(χ^2 =2.547; p=.280)

Table 5. SCL-90-R Symptom Dimension Means and Standard Deviations between MM and TMJ Groups.

SCL-90-R Subscales	MM Group M (SD)	TMJ Group M (SD)	F	p
Somatization	61.8 (9.0)	58.0 (10.1	2.536	0.112
Obsessive-compulsive	57.7 (11.4)	54.9 (11.9)	0.517	0.472
Interpersonal sensitivity	54.9 (11.2)	52.9 (10.9)	0.140	0.780
Depression	57.9 (10.9)	54.1 (10.7)	.000	0.999
Anxiety	55.9 (10.9)	53.3 (11.3)	0.487	0.486
Hostility	55.3 (10.1)	53.1 (9.9)	0.126	0.723
Phobic anxiety	50.9 (9.4)	50.7 (9.3)	.023	0.880
Paranoid ideation	51.8 (10.8)	49.5 (10.1)	1.323	0.251
Psychoticism	55.9 (10.6)	52.1 (9.7)	3.850	0.050*

SCL-90-R: Symptom Check List- 90-Revised.
MM: masticatory muscle; TMJ: temporomandibular joint.
M: Mean; SD: Standard Deviation.

Table 6. Comparison of SCL-90-R Symptom Dimension Means and Standard Deviations between the MM and TMJ Groups on a Subgroup Level.

SCL-90-R subscales	Trauma Grouping	MM Group M(SD)	TMJ Group M(SD)	F	p
Somatization	No stressor	60.8 (8.3)	56.8 (9.5)	1.847	.175
	Negative PTSD	60.5 (8.0)	56.7 (9.6)	1.897	.171
	Positive PTSD	68.3 (10.9)	70.4 (7.1)	7.152	.010*
Obsessive-compulsive	No stressor	56.0 (11.0)	53.2 (11.6)	1.122	.291
	Negative PTSD	55.5 (10.8)	53.4 (10.2)	.815	.368
	Positive PTSD	68.7 (7.8)	69.5 (8.5)	.272	.604
Interpersonal sensitivity	No stressor	54.0 (10.4)	52.3 (10.7)	.130	.718
	Negative PTSD	51.5 (9.4)	50.7 (9.7)	.051	.821
	Positive PTSD	65.8 (11.7)	64.4 (9.7)	.670	.417
Depression	No stressor	56.5 (10.8)	53.2 (10.7)	.007	.934
	Negative PTSD	55.7 (9.5)	52.3 (9.5)	.126	.723
	Positive PTSD	68.0 (8.9)	66.1 (6.4)	.533	.469
Anxiety	No stressor	54.8 (10.2)	52.1 (10.3)	.015	.901
	Negative PTSD	52.6 (9.4)	51.3 (10.9)	3.845	.052
	Positive PTSD	67.5 (9.7)	67.2 (9.3)	.297	.588
Hostility	No stressor	53.5 (9.7)	52.6 (9.3)	.426	.514
	Negative PTSD	53.5 (8.4)	51.1 (9.2)	.688	.408
	Positive PTSD	65.8 (7.5)	63.7 (8.9)	.425	.517
Phobic anxiety	No stressor	49.0 (7.9)	49.6 (8.3)	.385	.536
	Negative PTSD	48.7 (6.6)	49.5 (8.9)	5.965	.016*
	Positive PTSD	62.1 (12.0)	60.9 (9.8)	2.762	.102
Paranoid ideation	No stressor	50.4 (9.5)	48.0 (8.8)	.358	.550
	Negative PTSD	49.4 (8.9)	48.4 (9.3)	.015	.903
	Positive PTSD	62.6 (12.6)	62.0 (11.5)	.683	.412
Psychoticism	No stressor	54.0 (10.0)	51.1 (9.0)	2.990	.085
	Negative PTSD	52.4 (8.5)	50.9 (8.2)	.246	.621
	Positive PTSD	64.9 (11.7)	62.8 (11.5)	.049	.825

SCL-90-R: Symptom Check List- 90-Revised.
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
M: Mean; SD: Standard Deviation.
PTSD: Post-traumatic Stress Disorder.
*: Statistically significant.

Table 7. Comparisons of MPI Scales Means and Standard Deviations between MM and TMJ Groups.

MPI Scale	MM Group M(SD)	TMJ Group M(SD)	F	p
Interference	33.6 (16.0)	25.0 (15.0)	30.142	.000*
Life control	49.4 (7.9)	53.0 (7.3)	26.405	.000*
Affective distress	47.4 (9.6)	43.5 (10.4)	27.082	.000*
Support	47.3 (11.1)	47.3 (10.0)	3.124	.045*
Punishing responses	46.5 (8.6)	45.0 (7.1)	10.998	.000*
Soliciting responses	49.9 (9.7)	48.1 (9.5)	.423	.655
Distracting responses	47.7 (9.7)	50.4 (40.2)	6.257	.002*
Household chores	55.7 (9.1)	55.6 (9.6)	1.394	.249
Outdoor work	54.6 (11.6)	54.1 (11.4)	.166	.847
Activities away from home	53.0 (10.0)	54. 2 (10.0)	5.114	.006*
Social activities	51.8 (10.0)	52.9 (9.4)	4.283	.014*
General activity level	55.2 (9.9)	55.9 (9.7)	2.743	.065

MPI: Multidimensional Pain Inventory.
MM: Masticatory Muscle; TMJ Temporomandibular Joint.
M: Means; SD: Standard Deviations.
*: Statistically significant.

Table 8. Comparisons of MPI Scales Means and Standard Deviations among the Three Subgroups in the MM Group.

MPI Scale	No stressor M(SD)	Negative PTSD M(SD)	Positive PTSD M(SD)	F	P
Interference	31.7[a] (16.2)	33.9[a,b] (15.5)	39.4[b] (14.2)	3.382	.036*
Life control	50.2[a] (8.0)	50.7 [a] (7.0)	43.8[b] (6.9)	11.842	.000*
Affective distress	46.0[a] (9.6)	46.7[a] (9.7)	53.9[b] (6.7)	10.464	.000*
Support	48.6 [a] (8.9)	47.3[a,b] (12.8)	42.5[b](13.3)	3.299	.039*
Punishing responses	45.6[a] (8.0)	45.7[a] (7.8)	51.5[b] (10.3)	5.690	.004*
Soliciting responses	49.5 (8.8)	48.0 (10.9)	49.1 (10.2)	.422	.656
Distracting responses	48.2 (9.5)	47.0 (9.9)	47.5 (10.6)	.308	.735
Household chores	55.9 (8.7)	55.2 (9.8)	55.7 (9.4)	.172	.842
Outdoor work	53.6 (11.4)	56.5 (11.6)	53.8 (12.0)	1.601	.204
Activities away from home	54.0 (10.0)	52.0 (10.3)	51.4 (10.0)	1.415	.245
Social activities	52.0 (10.0)	51.9 (10.2)	50.6 (9.7)	.301	.740
General activity level	55.5 (9.4)	55.4 (10.6)	54.2 (10.3)	.240	.786

MPI: Multidimensional Pain Inventory.
MM: Masticatory Muscle.
M: Means; SD: Standard Deviation.
PTSD: Post-traumatic Stress Disorder.
[ab] When superscripts are the same between 2 groups , post-hoc comparisons indicates no significant differences between group means. When superscripts are different, post-hoc comparisons indicate significant difference between group means at p≤.05
*: Statistically significant.

Table 9. Comparisons of MPI Scales Means and Standard Deviations among the Three Subgroups in the TMJ Group.

MPI Scale	No stressor M(SD)	Negative PTSD M(SD)	Positive PTSD M(SD)	F	P
Interference	23.3[a] (13.8)	23.7[a] (13.0)	39.6[b] (17.8)	11.962	.000*
Life control	53.3[a] (7.5)	53.9 [a] (5.6)	47.5[b] (9.4)	6.532	.002*
Affective distress	42.5[a] (9.7)	42.5[a] (10.5)	53.4[b] (9.7)	10.794	.000*
Support	48.3 (9.4)	45.2 (11.2)	49.1 (8.6)	1.959	.144
Punishing responses	43.9[a] (6.0)	45.3[a] (7.1)	50.3[b] (10.5)	5.628	.004*
Soliciting responses	48.8 (10.2)	46.5 (8.6)	50.8 (8.0)	1.644	.196
Distracting responses	47.4[a] (8.7)	46.4[a] (8.7)	50.5[b] (7.2)	5.825	.004*
Household chores	56.3 (9.3)	55.5 (9.4)	52.2 (11.6)	1.608	.203
Outdoor work	53.4 (11.4)	56.5 (10.0)	52.9 (15.0)	.862	.424
Activities away from home	54.9[a] (9.8)	55.0[a] (9.6)	47.4[b] (10.8)	5.296	.006*
Social activities	53.8[a] (9.4)	53.3[a] (8.5)	46.9[b] (10.3)	4.870	.009*
General activity level	56.4[a] (9.3)	56.7[a] (8.6)	50.0[b] (13.0)	4.147	.017*

MPI: Multidimensional Pain Inventory.
TMJ: Temporomandibular Joint.
M: Means; SD: Standard Deviation.
PTSD: Post-traumatic Stress Disorder.
[ab] When superscripts are the same between 2 groups , post-hoc comparisons indicates no significant differences between group means. When superscripts are different, post-hoc comparisons indicate significant difference between group means at p≤.05
*: Statistically significant.

Table 10. Comparison of MPI Scales Means and Standard Deviations between MM and TMJ group on the Subgroup Level.

MPI Scale	Trauma Grouping	MM Group M(SD)	TMJ Group M(SD)	F	p
Interference	No stressor	31.7 (16.2)	23.3 (13.8)	17.991	.000*
	Negative PTSD	33.9(15.5)	23.7 (13.0)	18.765	.000*
	Positive PTSD	39.4 (14.2)	39.6 (17.8)	.002	.961
Life control	No stressor	50.2 (8.0)	53.3 (7.5)	9.228	.003*
	Negative PTSD	50.7 (7.0)	53.9 (5.6)	9.374	.003*
	Positive PTSD	43.8 (6.9)	47.5 (9.4)	2.817	.099
Affective distress	No stressor	46.0 (9.6)	42.5 (9.7)	7.864	.005*
	Negative PTSD	46.7 (9.7)	42.5 (10.5)	6.720	.010*
	Positive PTSD	53.9 (6.7)	53.4 (9.7)	.054	.817
Support	No stressor	48.6 (8.9)	48.3 (9.4)	.059	.808
	Negative PTSD	47.3 (12.8)	45.2 (11.2)	.874	.352
	Positive PTSD	42.5 (13.3)	49.1 (8.6)	2.989	.092
Punishing responses	No stressor	45.6 (8.0)	43.9 (6.0)	2.728	.100
	Negative PTSD	45.7 ((7.8)	45.3 (7.1)	.089	.767
	Positive PTSD	51.5 (10.3)	50.3 (10.5)	.125	.725
Soliciting responses	No stressor	49.5 (8.8)	48.8 (10.2)	.282	.596
	Negative PTSD	48.0 (10.9)	46.5 (8.6)	.781	.379
	Positive PTSD	49.1 (10.2)	50.8 (8.0)	.293	.591
Distracting responses	No stressor	48.2 (9.5)	47.4 (8.7)	.351	.554
	Negative PTSD	47.0 (9.9)	46.4 (8.7)	.120	.729
	Positive PTSD	47.5 (10.6)	50.5 (7.2)	.922	.343
Household chores	No stressor	55.9 (8.7)	56.3 (9.3)	.106	.745
	Negative PTSD	55.2 (9.8)	55.5 (9.4)	.045	.833
	Positive PTSD	55.7 (9.4)	52.2 (11.6)	1.498	.226
Outdoor work	No stressor	53.6 (11.4)	53.4 (11.6)	.010	.921
	Negative PTSD	56.5 (11.6)	55.6 (10.0)	.244	.622
	Positive PTSD	53.8 (12.0)	52.9 (15.0)	.058	.811
Activities away from home	No stressor	54.0 (10.0)	54.9 (9.8)	.486	.486
	Negative PTSD	52.0 (10.3)	55.0 (9.6)	3.233	.074
	Positive PTSD	51.4 (10.0)	47.4 (10.8)	1.994	.164
Social activities	No stressor	52.0 (10.0)	53.8 (9.4)	1.797	.181
	Negative PTSD	51.9 (10.2)	53.3 (8.5)	.880	.350
	Positive PTSD	50.6 (9.7)	46.9 (10.3)	1.789	.187
General activity level	No stressor	55.5 (9.4)	56.4 (9.3)	.556	.457
	Negative PTSD	55.4 (10.6)	56.7 (8.6)	.655	.420
	Positive PTSD	54.2 (10.3)	50.0 (13.0)	1.684	.200

MPI: Multidimensional Pain Inventory.
M: Mean; SD: Standard Deviation.
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
PTSD: Post-traumatic Stress Disorder.*: Statistically significant.

Table 11. MPI Profile Classification between the MM and TMJ Groups.

MPI profile	MM Group N (%)	TMJ Group N (%)	Chi-square	p
Dysfunctional	46 (34.8)	21 (24.7)	9.328	.002*
Interpersonally distressed	27 (20.5)	10 (11.8)	7.811	.005*
Adaptive coper	59 (44.7)	54 (63.5)	.221	.638

MPI: Multidimensional Pain Inventory
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
N: Number of patients, %: percentage
*: Statistically significant.

Table 12. MPI Profile Classification among the Three Subgroups in the MM Group and in the TMJ Group.

MPI profile	Trauma Grouping	No stressor	Negative PTSD	Positive PTSD	Chi square	p
	MM Group					
Dysfunctional		25 (36.8)	12 (27.9)	9 (42.9)	9.440	.009*
Interpersonally distressed		11 (16.2)	10 (23.3)	6 (28.6)	1.556	.459
Adaptive coper		32 (47.1)	21 (48.8)	6 (28.6)	17.320	.000*
	TMJ group					
Dysfunctional		7 (15.2)	7 (25.9)	7 (58.3)	0.000	1.00
Interpersonally distressed		6 (13.0)	2 (7.4)	2 (16.7)	3.200	.202
Adaptive coper		33 (71.7)	18 (66.7)	3 (25.0)	25.00	.000*

MPI: .Multidimensional Pain Inventory:
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
N: Number of patients; %: percentage.
PTSD: Post-traumatic Stress Disorder.
*: Statistically significant

Table 13 Comparisons of MPI Profile Classification between MM and TMJ Group on the Subgroup Level.

MPI profile	Trauma Grouping	MM Group N (%)	TMJ Group N (%)	Chi-square	p
Dysfunctional	No stressor	25 (19.8)	7 (6.2)	10.125	.001*
	Negative PTSD	12 (15.0)	7 (10.0)	1.316	.251
	Positive PTSD	9 (25.0)	7 (35.0)	.250	.617
Interpersonally distressed	No stressor	11 (8.7)	6 (5.3)	1.417	.225
	Negative PTSD	10 (12.5)	2 (2.9)	5.330	.021*
	Positive PTSD	6 (16.7)	2 (10.0)	2.000	.157
Adaptive coper	No stressor	32 (25.4)	33 (29.2)	.015	.901
	Negative PTSD	21 (26.3)	18 (25.7)	.231	.631
	Positive PTSD	6 (16.7)	3 (15.0)	1.000	.317

MPI: .Multidimensional Pain Inventory:
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
N: Number of patients; %: percentage.
PTSD: Post-traumatic Stress Disorder.
*: Statistically significant.

Table 14. PSQI Means and Standard Deviation between the MM and TMJ Group.

PSQI Scales	MM group M (SD)	TMJ group M (SD)	F	p
Subjective sleep quality	1.6 (0.8)	1.3 (0.8)	13.228	.000*
Sleep latency	1.6 (1.0)	1.3 (1.0)	10.114	.002*
Sleep duration	1.3 (1.0)	1.1 (1.0)	5.370	.021*
Habitual sleep efficiency	1.0 (1.1)	0.7 (1.0)	6.774	.010*
Sleep disturbances	1.8 (0.6)	1.6 (0.6)	9.114	.003*
Use of sleep medication	1.4 (1.4)	0.9 (1.2)	15.359	.000*
Daytime dysfunction	1.3 (0.8)	1.1 (0.8)	6.046	.014*
PSQI total score	10.0 (4.4)	8.0 (4.2)	22.346	.000*

PSQI: Pittsburgh Sleep Quality Index.
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
M: Mean; SD: Standard Deviation.
*: Statistically significant.

Table 15. PSQI Means and Standard Deviations among the Three Subgroups in the MM group.

	No stressor M (SD)	Negative PTSD M (SD)	Positive PTSD M (SD)	F	p
Subjective sleep quality	1.6 (0.9)	1.5 (0.8)	1.9 (0.8)	2.315	.101
Sleep latency	1.6[a] (1.0)	1.5[a] (1.0)	2.2[b] (0.9)	6.339	.002*
Sleep duration	1.2[a] (0.9)	1.4[a, b] (1.0)	1.7[b] (1.1)	3.045	.050*
Habitual sleep efficiency	0.9 (1.1)	1.0 (1.1)	1.5 (1.9)	2.842	.060
Sleep disturbances	1.7[a] (0.6)	1.7[a] (0.6)	2.2[b] (0.6)	9.063	.000*
Use of sleep medication	1.3 (1.4)	1.3 (1.3)	1.7 (1.4)	.738	.479
Daytime dysfunction	1.3[a] (0.8)	1.2[a] (0.7)	1.8[b] 0.8)	6.326	.002*
PSQI total score	9.7[a] (4.4)	9.6[a] (4.4)	13.0[b] (4.0)	7.871	.000*

PSQI: Pittsburgh Sleep Quality Index.
MM: Masticatory Muscle; M: Mean; SD: Standard Deviation.
PTSD: Post-traumatic Stress Disorder.
[ab] When superscripts are the same between 2 groups , post-hoc comparisons indicates no significant differences between group means. When superscripts are different, post-hoc comparisons indicate significant difference between group means at p≤.05
*: Statistically significant

Table 16. PSQI Means and Standard Deviations among the Three Subgroups in the TMJ Group.

	No stressor M (SD)	Negative PTSD M (SD)	Positive PTSD M (SD)	F	p
Subjective sleep quality	1.2[a] (0.8)	1.4[a] (0.8)	1.9[b] (0.9)	6.529	.002*
Sleep latency	1.2[a] (1.0)	1.3[a] (1.0)	2.0[b] (1.1)	5.204	.006*
Sleep duration	1.1[a] (0.9)	0.9[a] (0.9)	1.8[b] (1.2)	5.772	.004*
Habitual sleep efficiency	0.7[a] (1.0)	0.5[a] (0.9)	1.5[b] (1.3)	5.619	.004*
Sleep disturbances	1.5[a] (0.6)	1.6[a] (0.6)	2.2[b] (0.6)	10.435	.000*
Use of sleep medication	0.7[a] (1.1)	1.0[a, b] (1.3)	1.4[b] (1.4)	3.437	.034*
Daytime dysfunction	1.0[a] (0.8)	1.0[a] (0.8)	1.9[b] (0.8)	10.200	.000*
PSQI total score	7.4[a] (4.0)	7.9[a] (3.8)	13.0[b] (4.4)	13.611	.000*

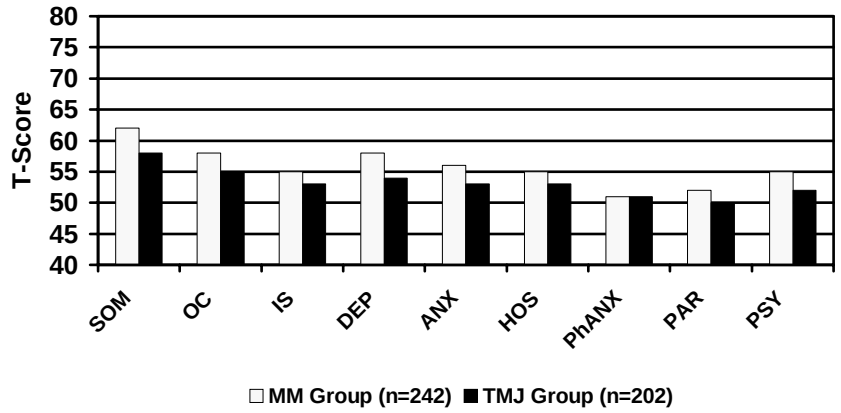
PSQI: Pittsburgh Sleep Quality Index.
TMJ: Temporomandibular Joint; M: Mean; SD: Standard Deviation.
PTSD: Post-traumatic Stress Disorder.
[ab] When superscripts are the same between 2 groups , post-hoc comparisons indicates no significant differences between group means. When superscripts are different, post-hoc comparisons indicate significant difference between group means at p≤.05
*: Statistically significant

Table 17. Comparison of PSQI scales between MM and TMJ Group on the Subgroup Level.

	Trauma grouping	MM group M (SD)	TMJ group M (SD)	F	p
Subjective sleep quality	No stressor	1.6 (0.9)	1.2 (0.8)	13.242	.000*
	Negative PTSD	1.5 (0.8)	1.4 (0.8)	1.601	.208
	Positive PTSD	1.9 (0.8)	1.9 (0.9)	.001	.976
Sleep latency	No stressor	1.6 (1.0)	1.2 (1.0)	8.687	.004*
	Negative PTSD	1.5 (1.0)	1.3 (1.0)	.987	.322
	Positive PTSD	2.2 (0.9)	2.0 (1.1)	.390	.535
Sleep duration	No stressor	1.2 (0.9)	1.1 (0.9)	.714	.399
	Negative PTSD	1.4 (0.9)	0.9 (0.9)	8.361	.004*
	Positive PTSD	1.7 (1.1)	1.8 (1.2)	.063	.803
Habitual sleep efficiency	No stressor	0.9 (1.1)	0.7 (1.0)	1.735	.189
	Negative PTSD	1.0 (1.1)	0.5 (0.9)	6.804	.010*
	Positive PTSD	1.4 (1.9)	1.4 (1.3)	.000	.992
Sleep disturbances	No stressor	1.7 (0.6)	1.5 (0.6)	9.011	.003*
	Negative PTSD	1.7 (0.6)	1.6 (0.6)	.544	.462
	Positive PTSD	2.2 (0.6)	2.7 (0.6)	.157	.694
Use of sleep medication	No stressor	1.3 (1.4)	0.7 (1.1)	15.097	.000*
	Negative PTSD	1.3 (1.3)	1.0 (1.3)	1.626	.204
	Positive PTSD	1.7 (1.4)	1.4 (1.4)	.418	.521
Daytime dysfunction	No stressor	1.3 (0.8)	1.0 (0.8)	5.569	.019*
	Negative PTSD	1.2 (0.7)	1.0 (0.8)	1.224	.270
	Positive PTSD	1.8 (0.8)	1.9 (0.8)	.413	.524
PSQI total score	No stressor	9.7 (4.4)	7.4 (4.0)	15.972	.000*
	Negative PTSD	9.6 (4.1)	7.9 (3.8)	6.559	.011*
	Positive PTSD	13.0 (4.0)	12.7 (4.0)	.058	.810

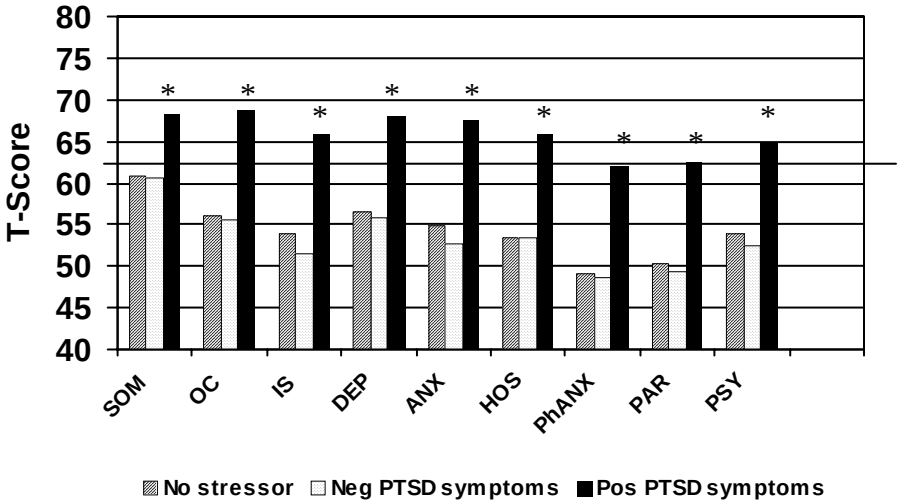
PSQI: Pittsburgh Sleep Quality Index.
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.
M: Mean; SD: Standard Deviation.
PTSD: Post-traumatic Stress Disorder.
*: Statistically significant.

Fig. 1. SCL-90-R Scores by Masticatory Muscle & Temporomandibular Joint Group.



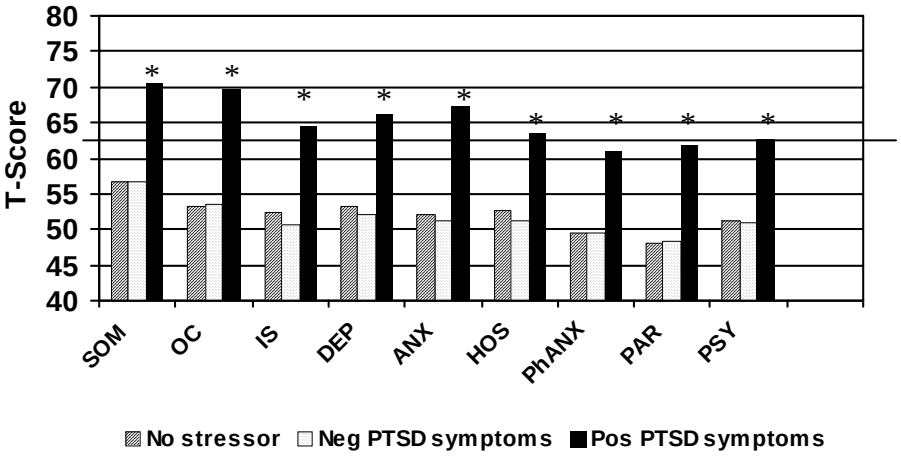
SCL-90-R: Symptom Check List- 90-Revised.
T-score ≥ 63: levels indicative of psychological dysfunction.
SOM: somatization; OC: obsessive-compulsive; IS: interpersonal sensitivity;
DEP: depression; ANX: anxiety; HOS: hostility; PhANX: phobic anxiety;
PAR: paranoid ideation, PSY: psychoticism.
MM: Masticatory Muscle; TMJ: Temporomandibular Joint.

Figure 2. SCL-90-R Score by PTSD Status in the Masticatory Muscle Group.



SCL-90-R: Symptom Check List- 90-Revised.
PTSD: Post-traumatic Stress Disorder.
T-score ≥ 63: levels of indicative psychological dysfunction.
Neg: negative; Pos: positive.
SOM: somatization; OC: obsessive-compulsive; IS: interpersonal sensitivity;
DEP: depression; ANX: anxiety; HOS: hostility; PhANX: phobic anxiety;
PAR: paranoid ideation, PSY: psychoticism.
*: Post-hoc comparisons indicate significant difference between the “positive PTSD symptoms” and the other two subgroups (p=.000).

Figure3. SCL-90-R Score by PTSD Status in the Temporomandibular Joint Group.



SCL-90-R: Symptom Check List- 90-Revised.
PTSD: Post-traumatic Stress Disorder.
T-Score ≥ 63: levels indicative psychological dysfunction.
Neg: negative; Pos: positive.
SOM: somatization; OC: obsessive-compulsive; IS: interpersonal sensitivity;
DEP: depression; ANX: anxiety; HOS: hostility; PhANX: phobic anxiety;
PAR: paranoid ideation, PSY: psychoticism.
*: Post-hoc comparisons indicate significant difference between the "positive PTSD symptoms" and the other two subgroups (p=.000)

Chapter 6. Discussion

Post-traumatic Stress Disorder symptoms were reported by 14.9% of patients with MM pain and by 9.9% of patients with TMJ pain. Analyses of the total sample revealed an overall prevalence of 12.6% for PTSD symptoms. The findings of this study are in agreement with previous studies in orofacial pain populations [7], [19]. In a recent investigation, de Leeuw et al [19] reported an overall prevalence rate of 14.7 % for PTSD symptoms in orofacial pain patients. Similar to previous studies, we observed a higher prevalence rate of PTSD symptoms in MM pain patients when compared to TMJ/intracapsular pain patients [19], [26]. The higher prevalence of PTSD symptoms in the MM group is not surprising given that several studies reported higher levels of psychological distress in MM pain patients than in TMJ pain patients [15], [14], [17]. Moreover, studies have shown that MM pain patients report more exposures to stressful life events than TMJ pain patients [20],[35].

Surprisingly, the overall prevalence rate of 12.6% of PTSD symptoms was similar to the lifetime prevalence of PTSD (1% to 14%) in the general population estimated by the DSM-IV [1]. Current PTSD prevalence rates, however, appear to be less than 10% [126], [127], [128]. Given these data, the prevalence of current PTSD symptomatology in the present study can be considered somewhat elevated. Nevertheless, our findings disagree with previous studies in chronic pain (fibromyalgia), where extremely elevated prevalence rates (approximately 55%) of PTSD symptoms have been reported [4], [5]. Such discrepancy could potentially be explained by individual differences among these study populations, such as

social support, family history, personality variables and preexisting mental disorders that may be involved in the development of PTSD [1], or methodological differences between the studies. On the other hand, the discrepancy between our studies and these other two studies could be a reflection of an increased vulnerability for PTSD symptoms in patients with chronic widespread pain conditions when compared to patients with a more localized pain condition such as TMD. Further studies are needed to clarify whether such relationship exists.

Previous studies have shown a relationship between the presence of PTSD symptoms and increased pain level [4], [18]. We were unable to confirm this relationship because in the present study there was no significant difference for both MM and TMJ among the PTSD subgroups in regard to pain severity. Such conflicting findings could potentially be explained due to methodological differences or different pain populations between this study and the previous studies. On the other hand, in accordance with these studies and two other studies in orofacial pain populations [8], [19] the present study indicates a positive relationship between PTSD symptoms and disability. Sharp et al in 2001 described a model to explain the overlap of symptoms between chronic pain and PTSD, the mutual maintenance hypothesis, whereby chronic pain and PTSD may reciprocally maintain or exacerbate the symptoms of both conditions that may lead to disability [101]. Nonetheless, a causal relationship between PTSD and disability can not be established with the present study due to its retrospective nature; further studies are need to determine whether such relationship exist.

The SCL-90-R data revealed somewhat higher scores for patients in the MM group than for patients in the TMJ group. An interesting finding of this study was that only TMD patients (both of the MM group and TMJ group) with PTSD symptomatology presented with elevated levels of psychological dysfunction (T-score ≥ 63) on the SCL-90-R. Evidence suggests that scores equal or greater than 63 are considered by most authors as the "cut-off" point for clinical significance [129]. Our findings are consistent with those presented by de Leeuw et al [19]. They observed that higher levels of psychological distress were limited to TMD patients who met criteria for PTSD symptomatology. Our findings are also similar to previous studies with headache [98] and neuropathic pain patients [99] where elevated levels of psychological distress were also linked to only patients meeting criteria for PTSD symptoms. These findings differ from those of previous studies, suggesting that MM pain patients report more psychological distress in general than TMJ pain patients [14], [15]. The discrepancy between our findings and the findings reported by these previous studies could be explained by the fact that these studies did not screen for PTSD symptomatology. Furthermore, this study contradicts the widely held concept that TMD patients are in general psychologically distressed. According to our findings, high levels of psychological dysfunction as measured on the SCL-90-R are likely to be associated with the presence of PTSD symptoms and not likely to be associated with TMD patients in general. Consequently, elevated SCL-90-R scores generally may indicate the presence of PTSD symptoms.

Significant differences were found between the MM and TMJ groups on most scales of the MPI. Our results are in agreement with previous studies of TMD patients that indicate higher levels of psychological distress and life interference in muscle pain patients than in TMJ pain patients [15], [17], [63], [130]. The fact that patients in the MM group presented with more life interference and higher levels of affective distress than patients in the TMJ group could potentially be a consequence of how high levels of psychological distress may interfere with a patient's coping skills. However, it is not possible to determine whether such an association exists with the present study; further studies are needed to elucidate this matter. It is noteworthy that no significant differences were found between the two diagnostic groups in the "positive PTSD symptoms" subgroups on the MPI scales. These findings are consistent with those findings reported by de Leeuw et al [19]. It is remarkable that TMJ pain patients potentially exhibited deficient coping skills and present with decreased level of social activities when the presence of PTSD is considered. Indeed, the severity of anxiety and life interference may be associated with the severity of PTSD symptoms in chronic pain patients generally [23]. Taken together, these findings further support the necessity for PTSD screening among TMD patients.

It appears that symptoms of PTSD may potentially interfere with patient's capacity to cope with her/his pain. This finding is also reflected in the fact that dysfunctional MPI profiles were more common than the adaptive coper profile amongst patients with PTSD symptomatology. Our results are in accordance with those reported in chronic pain patients with PTSD symptomatology [4], [19], [100] who

also presented more often with a dysfunctional profile than an adaptive coper profile. In addition, dysfunctional profiles may be associated with higher levels of anxiety [100] which, in turn, may potentially exacerbate the pain condition.

A potential explanation for these findings is the dysregulation of the HPA-axis that has been associated with inadequate coping strategies [50]. Alteration in the physiology of the HPA-axis may be related to somatic complaints such as myalgia, arthralgia and sleep disturbances in the absence of recognized pathological condition [131]. In addition, early life events, such as preterm birth, parental divorce, or childhood abuse may result in physiological vulnerability expressed as persistent sensitization of the HPA-axis [46]. In fact, dysregulation of the HPA-axis has also been linked to the development of both chronic pain [13, 50] and PTSD, that is characterized by maladaptive behavior [132]. In turn, this maladaptive behavior can be understood as a lack of inhibitory control [133].

Living systems are described as “self-organizing dynamic systems” that combine autonomic, attentional and affective systems into a functional and structural network [133,134]. These systems are likely to be controlled by inhibitory processes that allow them the necessary flexibility for efficient functioning through self-regulation and adaptability of the organism in the face of changing environmental demands [133,134]. Thayer and Lane in 2000 described how arousal associated with anxiety represents a dis-inhibition of circuits that are normally under inhibitory control [134]. Thus inhibitory failure may lead to maladaptive behavior at multiple levels of the organism which, in turn, may prevent recovery or a return to normal functioning.

Several studies have demonstrated a relationship between chronic pain and sleep disturbances [17],[30], [135]. In agreement with previous studies [17], [64], our findings revealed that the patients in the MM group reported more sleep problems than patients in the TMJ group. It is unlikely that these findings would be a consequence of increased pain severity [30] or increased pain duration [16] since in the current study no significant differences in regard to pain severity and duration were found between the two groups. A possible explanation for these findings could be the presence of psychological distress that has been associated with sleep disturbances pathogeneses in a number of studies [30], [64], [136].

Both MM and TMJ pain patients in the "positive PTSD symptoms" subgroup reported more sleep problems on most scales of the PSQI than MM and TMJ pain patients in the "no stressor" and in the "negative PTSD symptoms" subgroups. This finding is not extraordinary if the presence of PTSD symptoms is considered. Indeed, according to the DSM-IV [1] sleep disturbances are included in the symptomatology of PTSD. It is not possible to determine with the present study design whether the sleep disturbances were a response to the pain experience itself or whether they were associated to the symptomatology of PTSD. These findings may be associated to an overlap of symptoms between chronic pain and PTSD symptoms [23], [101] which in turn may exacerbate the symptomatology of both conditions. On the other hand, it also could be a response to alterations of HPA-axis, a common characteristic found in chronic pain patients as well as in PTSD patients [46]. Indeed, the HPA-axis plays

important roles in maintaining alertness and modulating sleep [137]. In addition, dysregulation of the HPA-axis has been associated with sleep disturbances in a number of studies [50], [138]. Nonetheless, sleep disturbances are remarkably common in chronic pain and in PTSD and should be addressed since it could be a major factor in chronic pain and PTSD symptomatology.

Given the coexistence of chronic pain and PTSD, appropriate management of chronic pain patients with symptoms of PTSD may possibly require treatment of both the anxiety disorder and the pain disorder. There are only a small number of studies addressing treatment outcomes in chronic pain patients with PTSD symptoms [116], [117]. Research suggests favorable treatment outcomes targeting symptoms of PTSD in chronic pain patients such as decreased PTSD symptomatology and improvement in dysfunction associated to pain [117]. The fact that in the present study, TMD patients with PTSD symptomatology were more often classified with a dysfunctional profile than an adaptive coper profile may further complicate interventions in such patients. Indeed, a dysfunctional profile has been related to poor treatment outcome overall in TMD patients and to treatment failure [15]. In addition, failure to recognize psychological distress has been associated with poor treatment response [114] and prematurely abandoning treatment [115]. It is likely that for successful treatment the multiple coexisting factors need to be addressed. Indeed, targeting PTSD symptoms may be a key factor in managing chronic pain patients with such symptomatology. Unfortunately, our study was not designed to evaluate treatment outcomes, although we acknowledge the need for well designed

longitudinal studies to answer questions such as whether management of PTSD would change treatment outcomes for chronic pain.

The present study has limitations due its retrospective design. It is not possible to determine a causal relationship between chronic pain and PTSD. It is also not possible to determine causal relationships among chronic pain, PTSD and psychological distress as well as among chronic pain, PTSD and sleep disturbances. An additional limitation is that this survey was conducted with patients who sought treatment for their TMD problem in a tertiary care center which could overestimate the prevalence rate of PTSD and the relationships among PTSD, psychological distress and sleep disturbances found in the present study as compared to what occurs in the natural environment. Given that the patients in this study could represent a more skewed pain population compared to the typical patients seem at a general practice. On the other hand, we implemented strict inclusion criteria in each diagnostic group (MM and TMJ group) whereby only patients with primary and when given a secondary diagnosis of MM pain (MM group) or TMJ pain (TMJ group) were included in order to have a more accurate sample. These inclusion criteria probably strengthen our findings given that our sample would be more representative of a more precise MM pain and TMJ pain population and thus decreasing the likelihood for potential errors associated with differential diagnosis.

Chapter 7. Conclusion

The present study replicates and extends previous investigations addressing the relationship between chronic pain and PTSD symptoms. Approximately 13% of patients reporting to an Orofacial Pain Clinic met criteria for PTSD symptomatology. A higher prevalence rate of PTSD symptoms was detected for patients in the MM group (14.9%) as compared to patients in the TMJ group (9.9%). This difference was not statistically significant; consequently, our primary hypothesis that the prevalence of PTSD symptoms would be higher for patients in the MM group than for patients in the TMJ group was not confirmed. There was, however, a trend suggesting a higher prevalence of PTSD symptomatology in the MM group when compared to the TMJ group. Analysis of our findings revealed that psychological distress measured on both SCL-90-R and MPI and sleep disturbances measured on the PSQI were linked to PTSD symptomatology in both MM and TMJ group. We also found that MM pain patients presented with more life interference, affective distress and sleep disturbances, and less life control than TMJ pain patients confirming and expanding previous studies addressing the differences between MM and TMJ pain patients. However, when the presence of PTSD was considered these differences were mostly maintained in the subgroups without PTSD symptomatology. Hence, the presence of PTSD appears to modulate not only the level of psychological distress in TMD patients and sleep disturbances, but also the differences between MM and TMJ groups. Further longitudinal research is

necessary to explore the relationship between chronic pain and PTSD patients and to devise effective multidimensional treatment.

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