ABSTRACT OF THESIS

COGNITIVE AND SOMATIC ITEM RESPONSE PATTERN OF OROFACIAL PAIN PATIENTS COMPARED TO FIBROMYALGIA PATIENTS AND A NON-PAIN CONTROL GROUP

Previous work has suggested that chronic pain patients report psychological distress through higher endorsement of somatic rather than cognitive signs of anxiety and depression. The present study compared female Non-Pain (n=52), Orofacial Pain (n=317) and Fibromyalgia (n=50) groups, on SCL-90-R Somatization, Anxiety and Depression raw scores and cognitive-somatic symptom patterning of the Anxiety and Depression scales. Comparisons were also made amongst orofacial pain diagnostic subgroups and subgroups based on Multidimensional Pain Inventory (MPI) classification groups. The Somatization, Anxiety and Depression scores were higher in the Orofacial Pain and Fibromyalgia than Non-Pain group and higher in the Dysfunctional than Adaptive Coper MPI Profile group. No differences in somatic-cognitive symptom patterning existed among the diagnostic or the MPI groups/subgroups. Orofacial pain patients endorsing cognitive items stronger than somatic items on the Anxiety and/or Depression scales showed a tendency towards more psychopathology (higher SCL-90-R scale scores) than the participants endorsing somatic items more so than cognitive items. In conclusion, study results indicate that differentiation of cognitive-somatic patterns does not contribute to increased understanding of chronic pain conditions.

KEYWORDS: Psychometrics (descriptive), Orofacial Pain, Fibromyalgia, Non-Pain, Anxiety, Depression, Cognitive aspects, Somatic aspects

Morten S. Hadsel
July 16, 2002

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THESIS

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The Graduate School
University of Kentucky
2002
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THESIS

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the College of Dentistry at the University of Kentucky

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Chapter One
Introduction

Pain as a multidimensional experience

After observation of patients with atypical facial pain, G. L. Engel concluded in 1951 that no pain exists without involvement of central nervous structures and in spite of having a physiological basis, pain is primarily a psychological phenomenon. These thoughts were further developed and proposed in the “Gate Control Theory” in 1965 by Melzack and Wall and expanded in 1968 by Melzack and Casey. Thereby a theory was presented that showed the parallel processing and integration of physiologic and “psychologic” elements in pain. In this way previous biomedical–mechanistic thought processes were incorporated into a bio-psychosocial model, and the importance of psychosocial factors in the field of pain was established.

The “Gate Control Theory” not only proposed a model for pain modulation at the dorsal horn level, but it also stated that ascending nociceptive information is transmitted via two main central pathways. One is the neospinothalamic tract to the ventrobasal thalamus and the somatosensory cortex, which is thought to represent the neurological basis for the sensory-discriminative pain dimension. The other is the “paramedian” pathway to the medial and intralaminar thalamus, the limbic system and the frontal lobe via and with connections to the reticular formation. This second pathway is proposed to be responsible for pain-related motivational drive, behavioral arousal and unpleasant affect (Melzack & Casey, 1968).

In addition to sensory/discriminative and emotional/motivational factors in the processing of nociception, other elements also play an important role in pain perception and need to be taken into consideration. These are cognitive/evaluative processes of higher brain centers, utilizing memorized information created by previous experiences involving painful events and their consequences, as well as genetically determined thinking and reaction patterns. The interaction between the sensory/discriminative, emotional/motivational and cognitive/evaluative systems leads to the resultant pain behavior. Pain behavior is the objectively observable, individual, non-verbal and verbal “external” expression of the subjective pain experience and accompanying suffering. Most of the activity in the effector organs involved in pain behavior is
mediated by physiological stress responses characterized by increased activity in the motor, endocrine, autonomic, immune and opioid systems.

Studies using functional MRI (Coghill et al, 1994) have suggested that the neuroanatomical substrates for the sensory-discriminative component of pain are the primary and secondary somatosensory cortices of the parietal lobe. The affective quality of pain involves primarily the anterior insular cortex of the frontal lobe. The affective-motivational aspects of pain are assigned to the anterior cingulate cortex, also of the frontal lobe. These findings in humans support the previous theories of the central pain pathways that were predominantly based on behavioral and neurophysiological observations in animals. They also confirm the necessity of considering both mind and body when dealing with pain.

Melzack integrated the original Gate Control concepts into his “Neuromatrix Theory” of pain (1989, 1990, 1992, & 1999) and proposed that besides aiming at restoring homeostasis, the previously discussed stress responses to nociception can produce tissue changes that subsequently act as additional inputs to the neuromatrix. Other inputs are considered to be sensory, emotional and cognitive stimuli from additional brain areas, as well as intrinsic neural inhibitory modulation. The neuromatrix is postulated to be a neuronal network consisting of somatosensory, thalamocortical and limbic structures producing “neurosignature” output patterns that activate perceptual, homeostatic and behavioral programs. A “neurosignature” output pattern is thought to be the individualized neuronal activation of these effector systems, based on genetic neuronal control and a multiplicity of other factors as mentioned above. It is important to acknowledge that according to the “Neuromatrix Theory”, these effector systems can be triggered independently of sensory input. Nevertheless, one can see that pain is proposed to be a multidimensional construct with both sensory and affective components, which also was stated by IASP in 1979.

For comprehension and didactic reasons it is tempting to maintain the separation between physiology and psychology in pain, although the reality may be even more intricate than one likes to believe. Sullivan (2001) for example, described the complexity involved in creating the pain experience through what he claims to be a product of many interpretational processes. He argues against the mind – body dualism of pain and states “There is no center of the brain where the pain observer sits; there is no point within the nervous system where interpretation of pain experience begins. There is no location within the organism where nociception becomes pain”
So, Sullivan actually goes a step further and questions if our knowledge really is good enough to neuroanatomically define pain or if individual pain experiences can be defined in terms of neuroanatomy at all.

**Emotions and pain**

General affective/emotional aspects of pain were postulated by Melzack to be potential inputs to the neuromatrix. Based on nociception and/or its causative/related events, these emotional aspects take part in the perceptual creation of the pain experience. Additionally, emotions are potentially found on the “output-side” as well. They can represent responses to the pain experience itself and – especially when pain persists – responses to possible consequences of the pain. Subsequently they can also serve as secondary inputs to the neuromatrix. Associated emotions experienced by chronic pain patients commonly include depression and anxiety (Gaskin et al., 1992).

Depression has been defined as “A temporary mental state or chronic mental disorder characterized by feelings of sadness, loneliness, despair, low self-esteem, and self-reproach; accompanying signs include psychomotor retardation or less frequently agitation, withdrawal from social contact, and vegetative states such as loss of appetite and insomnia” (Stedman’s Medical Dictionary, 27th edition). Banks & Kerns (1996) estimated a prevalence of 30-54% of depression in a clinic-based chronic pain population. This high prevalence underlies the major role of depression in chronic pain and suggests that depressive symptoms not only would dramatically change the behavior, especially with pain chronicity, but also quantitatively change the emotional input to higher centers in chronic pain patients.

The same influence seems also to hold true for other negative emotions, for example anxiety. Stedman’s Medical Dictionary (27th edition) defines Anxiety as “Fear or apprehension or dread of impending danger and accompanied by restlessness, tension, tachycardia, and dyspnea unattached to a clearly identifiable stimulus”. In 1996, McCracken et al. reported that anxiety accounted for 16-54% of the variance in pain report, disability, and pain-related behavior among persons with chronic pain. Again, these numbers confirm the quantitative importance of negative emotions among chronic pain patients, and support the value of the biopsychosocial model for pain.
Historically the chronic pain experience is known to be accompanied by strong negative emotions. It is therefore not surprising that depression, anxiety and anger correlate strongly with each other and also form a single affective distress factor when employed in structural equation modeling studies with chronic pain (Brown et al., 1996). There are other studies, however, that suggest each of these constructs contribute unique variance to the prediction of pain-related negative affect (Robinson & Riley in “Psychosocial Factors in Pain”, Gatchel & Turk ed, 1999). These findings suggest that the separate use of these constructs is justified.

The link between emotional distress and physical symptoms

Mechanisms linking emotional distress with physical symptoms include autonomic arousal, vigilance and misinterpretation (Sullivan & Katon, 1993) and somatic amplification (Barsky & Klerman, 1983). Somatic amplification is defined as an increased awareness of bodily sensations. Barsky et al. (1988) proposed that physiological events related to chronic pain could generate this sensitizing effect as a consequence of prolonged exposure to this stressor. The relationship can theoretically exist in the following ways: a) Negative emotion increases somatic sensitivity; b) Negative emotion causes pain/part of pain; c) Experience of chronic pain causes negative emotion and d) Negative emotion and pain are concomitant constructs, because of similar biological foundations (Brown, 1990; Banks and Kerns, 1996; Fishbain et al., 1997). These potential relationships are depicted in Figure 1.

Mediators between negative emotions and chronic pain

Theoretically, on an individual basis any one of the alternatives in Figure 1, as well as various combinations could be possible. Somatic amplification could of course also be preceding and leading to dysphoria. Cohen and Rodriguez (1995) underscored the bi-directional nature of biological, behavioral, cognitive and social pathways in the development and maintenance of affective and physical disorders. The complexity of these interactions implies that the relationship between pain and emotional factors is not of a direct nature, but seems to be mediated by other variables. Among several constructs suggested being responsible for the
Comorbidity of pain and negative emotions are Somatization and Dysfunctional Cognitive Processes (Robinson & Riley in “Psychosocial Factors in Pain” Gatchel & Turk ed, 1999).

Somatization has been defined in several ways. One definition is: “The process by which psychiatric or psychological factors present as physical symptoms” (Dickens & Creed in “Postgraduate Psychiatry”, Appleby et al. ed, 2001). This implies the experience of pain without actual nociception or the amplification of the pain-experience due to accompanying negative emotions. Cognition is defined by Stedman’s dictionary (27th edition) as “a generic term embracing the mental activities associated with thinking, learning and memory”. It can be seen that cognition is a very broad term encompassing different kinds of mental processes including logic and illogic ways of creating concepts, solving problems, making decisions and forming judgments. The role of cognition as a mediator between chronic pain and negative emotions is based on the assumption that thoughts intervene between events and emotional reactions. These thoughts could be based on previous experiences and/or related to the actual event. Catastrophizing represents one such intervening mental process, which seems to be of importance in chronic pain. It is defined as “a cognitive process characterized by negative expectations about future outcomes and lack of confidence” (Sullivan & D’Eon, 1990). Cognitive models of depression view negative cognition as distinct from, but related to symptoms of depression, underpinning the fact that they are two distinct constructs (Beck, 1976). The measurement of constructs like Somatization and Negative Cognition can be performed through the administration of psychometric tests. A Somatization or Cognition score achieved on a standardized psychometric test represents a numerical expression of the quantity and quality of the reported symptoms in question. The severity of the reported symptoms can be derived by comparing the score with known scores for a demographically matched group. The distance from the “norm-value” is usually expressed in standard deviations.

Role of somatic factors

Over time various definitions of somatization have been suggested, and Wilson et al. (1994) feel this has led to confusion about use of the term in any particular context. They underscore the necessity of distinguishing somatization as a psychiatric disorder from somatization as a dimension of personal functioning. For example Dworkin (in Gatchel and Turk
ed, 1999) defines somatization as “a dimension of personal functioning characterized by the tendency to report distress arising from multiple, non-specific physical symptoms accompanied by increased healthcare visits”. Somatof orm psychiatric disorder is defined (in DSM IV, 1994) as “the presence of a minimum of eight physical symptoms, distributed over multiple organ systems and not explained by a diagnosable medical condition”. So, although the basic definition of somatization is the same, the difference between somatization as a personal dimension and as a psychiatric disorder is dependent upon the quantitative and qualitative expressions of the somatic symptoms.

Somatization as part of a standardized, psychometric screening instrument reflects non-specific physical symptoms that are perceived as distressful (Escobar 1987, Simon 1993). Derogatis (1977) defines the Somatization subscale of the Symptom Checklist-90-Revised (SCL-90-R) as a dimension reflecting distress arising from perceptions of bodily dysfunction. He included symptoms from autonomically controlled systems (cardiovascular, respiratory and gastrointestinal), headaches, muscle pain and somatic equivalents of anxiety. He claims that these symptoms have a high prevalence in functional disorders, but may of course also reflect physical disease itself.

Grunau et al. (1994) searched for precursors of somatization by comparing extremely low birthweight premature infants (representing children exposed to early distress and pain) with full-term ones. They concluded that the etiology of somatization is a multidimensional problem and found non-optimal parenting as a contributing factor to development of inappropriate coping strategies with childhood pain. Noyes et al. (2001) reported that certain personality disorders, especially the obsessive-compulsive type and also self-defeating, depressive and negativistic personality traits contribute to somatization. Craig (1990) feels that social interactions and not intrinsic properties are decisive for the development of somatization. Dworkin (1994) also pointed out the contributing role of cultural factors and of medical institutions that focus exclusively on physical symptoms. After reviewing the literature relating to gender differences in somatization, Wool and Barsky (1994) stated that women report more functional symptoms than men do. As the literature contained many confounding variables and flaws, the authors did not consider this conclusion fully decisive. Bridges (1991) found a great similarity between primary care patients with somatic symptoms of distress and patients presenting with psychological symptoms. He stated that some evidence suggest “somatizers” to be less depressed and socially
distressed compared to “psychologizers”, but in a patient population this seems to be even less true.

Somatization scores seem to be elevated in chronic pain samples and positively correlated to the number of pain complaints (Walker et al., 1988; Dworkin, 1990). Perceived disability also increases linearly with increasing number of reported symptoms (Katon et al., 1991). The relationship between psychological distress and somatization has been reported in various ways. Some authors report that such distress, especially involving depression and anxiety, seems to be a primary determinant of somatic amplification i.e. non-painful experiences are interpreted in terms of pain (Simon & von Korff, 1991; Dworkin, 1994; Kosturek et al., 1998). Other authors can only confirm a co-existence between depression and somatic symptoms and between depressive disorders and pain complaints (Smith, 1992). Hiller et al. (2000) could not verify a general distinction between a “pure” pain disorder and a pain condition also involving multiple somatoform symptoms. They found equal amounts of general depression, dysfunctional attitudes and use of pain coping strategies in the two populations, but the multiple somatization group revealed higher degrees of affective and sensoric pain sensations and more pain related disabilities than did the “pure” pain group.

The Somatization construct has not only been used to predict properties concerning current patient conditions, but also prospective predictive properties of this construct have been reported. For example, it was shown in a prospective study of TMD-patients that the initial general somatic complaints were the best predictors of TMD pain at 2-year follow-up (Vassend et al., 1995), that somatization together with pain intensity and severity were the major predictors for the transition from acute to chronic TMD problems (Garofalo et al., 1998) and that somatization was a significant predictor of poorer treatment response in TMD patients (McCreary et al., 1992).

Role of cognitive factors

Several studies have examined the role of cognitive factors in the mediation between pain and psychological distress. A direct link between pain and depression could not be confirmed in a study by Rudy et al. (1988), but measures of perceived life interference and self-control were found to be significant intervening variables between pain and depression. Smith et al. (1994)
found that both depressed pain patients and depressed non-patients reported more cognitive distortion than their non-depressed counterparts. Sprock et al. (1983) compared depressed and non-depressed pain patients and normals and detected a significant positive correlation between degree of impairment in cognitive function and severity of depression. This finding was independent of pain report. In a study by Ingram et al. (1990), depressed chronic pain patients exhibited significantly more negative automatic thoughts than non-depressed pain patients and healthy controls. A recent study by Severeijns et al. (2001) found that catastrophizing was a potent predictor of pain intensity, disability and psychological distress. They reported no difference between chronic low back pain patients, patients with chronic musculoskeletal pain other than lower back and for pain patients excluding the previous groups. Jensen (1999) confirmed previous findings that patient beliefs about their chronic pain influence and is associated with their behavioral and psychological functioning. All these findings suggest that cognitive factors, particularly related to dysfunctional thought patterns, may lead to the development of dysphoria among chronic pain patients.

Ciccone (1984) concluded that behavioral interventions in chronic pain are effective because they facilitate the development of new thinking skills that explicitly challenge the cognitive causes of pain. Tota-Faucette et al. (1993) examined factors responsible for the outcome of multidisciplinary management of chronic pain inpatients. They found that increases in pain control and rational thinking were related to decreases in depression and anxiety, pain report and activity discomfort. They also studied the role of negative social cognitions, which include thoughts of being alone with ones pain, lack of social support and that no one else cares about ones suffering. It was found that when negative social cognitions decreased, so did depression at post treatment. It can be concluded that a change from irrational to rational thinking can lead to an improvement in general well-being, based on favorable changes in emotions, pain perception and/or activity level.

DeGood & Kiernan (1996) studied the perception of fault in patients with chronic pain. They compared a group that blamed someone else (e.g. employer or other driver) for their pain with a group not blaming anyone. The fault group reported greater concurrent mood distress and behavioral disturbance, as well as poorer response to past treatments and lower expectations of future benefits compared to the non-fault group. The authors claim that their data support the perception or attribution of blame as an under-recognized cognitive correlate of pain behavior,
mood disturbance and poor response to treatment. It seems reasonable to believe that strong emotions, feelings of anger and belief about a need for revenge and justice can give rise to the distress in patients holding someone else responsible for their pain, especially when the blamed party denies such responsibility.

Ethnic/cultural elements also seem to influence cognitive mediation in chronic pain. After studying chronic pain populations in New England and in Puerto Rico, Bates and Rankin-Hill (1994) found that ethnic/cultural background and locus of control style were the factors most often associated with statistically significant differences in pain intensity, pain responses and adaptation to the chronic pain experience. For the majority of the studied groups, ethnic/cultural identity was a predictor of locus of control style. The data also suggest that locus of control is not necessarily a permanent, unchanging cognitive characteristic, but may be altered by the chronic pain experience, behavioral interventions, and ethnic/cultural factors. Due to not only significant inter- but also intra-ethnic/cultural-group differences, the authors stress the need to assess each patient individually within the context of her/his total psychosocial and biocultural environment.

Some data exist on the effect of pain on cognitive processing. One example is Grigsby et al. (1995) who compared chronic pain patients with persons who had sustained moderate head trauma. The mean scores for both groups were lower than the normative mean on different information processing and motor tests. No inter-group differences were found on motor measures, but the pain patients performed more poorly than the head trauma group on the information processing tests. The results suggest that pain may disrupt cognitive performances, which depend on intact speed and capacity for information processing. One can clearly see that data exist in support of complex, bi-directional relationships concerning the mediators between pain and emotions.

**Depression and anxiety as compound constructs**

As discussed earlier, depression and anxiety can be viewed as separate constructs, but there have been questions as to if each of them represent a uniform construct. Considering depression, the debate originated due to the presence of cognitive and somatic items on the Depression scale and therefore a contribution of both cognitive and somatic symptoms to the Depression score (Rodin & Voshart, 1986; Buckelew et al. 1986; Geisser et al., 1997).
Concerning anxiety, the presence of physiological symptoms on most assessment scales used in pain patients have led to doubt of the uniformity of the construct (McCracken et al., 1992, 1996). Additionally, the complex nature of anxiety can also be seen from its common definition as consisting of components involving cognitive, physiological and behavioral/motor elements (Lang, 1968). Therefore, depression and anxiety can both be regarded as compound constructs with somatic and cognitive elements as important contributors.

Relative contributions of somatic and cognitive symptoms of anxiety and depression

Contrary to most studies, which have examined the role of either somatic or cognitive factors, some researchers have developed a more differentiated view. They have reported on the relative contributions of somatic and cognitive symptoms of anxiety and depression within the same study population, with and without physical symptoms and pain. In the following, a review of these studies will be presented.

Based on a model proposing a multidimensional nature of anxiety, Schwartz et al (1978) constructed the Cognitive-Somatic Anxiety Questionnaire (CSAQ), which is a trait anxiety inventory consisting of 14 items, seven cognitive and seven somatic items. The degree to which the participants typically experience the symptoms described by the different items when they are feeling anxious is to be rated between 1 and 5, where “1” represents “not at all” and “5” represents “very much so”. The CSAQ was administered to 44 subjects (predominantly females) participating in a physical exercise class, representing a somatically based activity. The CSAQ was also administered to 33 subjects (with equal gender ratio) practicing cognitively based, passive, daily meditation, representing a cognitively based activity. Although overall anxiety was not significantly different between the exercisers and the meditators, the exercisers reported significantly lower somatic than cognitive anxiety, while the meditators did not show any significant difference between the two anxiety modes. The authors concluded that anxiety is not an undifferentiated state. They proposed it is rather made up of patterns of specific psychobiological processes, which would be important to assess and take into consideration when dealing with affective disorders. They also question a unique generalized relaxation response and suggest, based on their findings, that the consequences of different relaxation techniques depend on the underlying biological system affected by the technique/procedure
applied. This specific “technique-sensitive” effect would then be superimposed on a generalized reduction in multiple physiological systems.

On the other hand, the authors were aware of a potential predispositional effect in that the two study groups could possibly differ in their initial cognitive-somatic patterning. This study was done in a cross-sectional manner on groups that had been performing either physical exercise or meditation for approximately 6 months. It could therefore be argued that the study is not providing any information about a possible “technique-sensitive” effect on or a change in the symptom patterning over time for groups performing the two different activities. Such differences would have to be studied in a longitudinal design.

Seven years after Schwartz’s study of cognitive-somatic symptom patterning in a non-pain population, DeGood et al. (1985) published a study involving chronic pain patients. They administered the same questionnaire as Schwartz et al. (Cognitive-Somatic Anxiety Questionnaire CSAQ, devised by Schwartz, Davidson and Goleman in 1978) to 100 chronic pain patients and 100 college students. They found that the overall, global anxiety was lower in the pain patients, but that the cognitive-somatic patterns of the responses differed between the two groups. The pain patients reported significantly more somatic anxiety while the students endorsed more cognitively oriented anxiety. The authors feel that the pain patients seem to fit the description of “alexithymia” (Difficulty in recognizing and describing one’s emotions, defining them in terms of somatic sensations or behavioral reactions). Unfortunately, the endorsements by the two groups could be confounded by their initial differences on either the cognitive or the somatic measures.

McCracken et al. (1998) studied a group of 210 adult, chronic pain patients and found that collateral, non-specific, physical symptom complaints were common in this group. The authors also discovered that physiological symptoms of pain-related anxiety and cognitive symptoms of depression were significant predictors of physical symptoms. The somatic anxiety component was the stronger predictor of the two. It is suggested that the results support a model in which non-specific physical complaints arise directly due to distressing circumstances of the pain experience and that emotional distress is more often a consequence than a cause of chronic pain. Based on these findings it seems that somatic distress (pain-related) correlates more strongly with physical complaints than general measures of distress do.
Wilson et al (1994) examined how clinical findings related to affective, cognitive and somatic symptom report. They combined information from the Somatization scale of the SCL-90-R (Derogatis 1977, 1983) and from their own Emotional/Cognitive distress scale-construct (based on removal of the somatic items from the SCL-90-R Anxiety and Depression scales) with clinical findings from a chronic TMD muscle disorder patient population. They reported that increased somatization and high pain intensity were strong predictors of widely dispersed pain on muscle palpation. The patients with high somatization were also more likely to present a painful placebo site on palpation. Affective and cognitive symptoms of psychological distress were less likely to be related to report of clinically widespread pain. The authors suggested these findings indicated that somatic and cognitive symptoms might relate differentially to characteristics of pain report. They also suggested that the inclusion of somatic items on psychometric scales of emotional/affective distress could confound this differentiated relationship.

Buckelew et al (1986) constructed a somatic and a cognitive subscale for depression and anxiety based on the SCL-90-R (Derogatis 1977,1983). They assigned the somatic and cognitive items on each of these scales to its own subscale and also assigned items to these subscales from the group of seven additional items on the SCL-90-R. This resulted in the following four subscales: Cognitive Anxiety, Somatic Anxiety, Cognitive Depression and Somatic Depression. Besides using the existing scales for Anxiety, Depression and Somatization on the SCL-90-R, they utilized the constructed subscales to compare the scores for chronic pain patients, psychiatric inpatients and new hospital employees. Each scale and subscale has a score range between zero and four, where four represents the highest endorsement. Each group contained 50 subjects, equally divided by gender, with an average age between 30 and 38 years old. The chronic pain group consisted of referred patients to the University of Virginia’s outpatient Pain Management Center with average pain duration of 78.1 months. This group was a mixture of patients with diverse primary pain locations: 68 % back, 14 % abdominal, 11 % headaches and 7 % other. The psychiatric patients were also representing several diagnostic groups, but 60 % were diagnosed with an affective disorder. The chronic pain patients reported the highest scores on somatization, as compared to the psychiatric inpatients and the hospital employees. The psychiatric inpatients reported the highest scores on anxiety and depression as compared to the chronic pain group and the hospital employees. Intra-group analysis showed equal levels of
somatic and cognitive endorsement of anxiety and depression for the psychiatric inpatients and the hospital employees. The chronic pain group showed higher somatic than cognitive subscores for both anxiety and depression than the two other groups did. The authors suggested that the cognitive-somatic symptom patterning for pain patients shown by DeGood et al in 1985 using the special CSAQ construct of Schwartz, was detectable when using a standardized questionnaire like the SCL-90-R. These findings led the authors to question the use of psychometric instruments based on norm values from different populations in the evaluation of medical patients. They suggested use of subscales to supplement the standard instruments, as equal test scores, independent on quantitative endorsement, could reflect extremely different item responses.

On analyzing an additional 150 pain patients, Buckelew et al. reported that they found a subgroup of 30 patients with higher scores on cognitive than on somatic symptoms on at least one of the anxiety and depression scales. They conveyed their impression that these patients were more “psychologically minded”, had often consulted mental health professionals for chronic pain or depression, took more personal responsibility and seemed more internally focused with regard to their health problem and health care than other patients. The authors advocated further research into the characteristics of this subset of pain patients endorsing inversed cognitive-somatic symptom patterning compared to the majority of pain patients.

The present study

Previous studies on the relationship between cognitive-somatic symptom patterning and pain, utilizing a standardized psychometric screening instrument (e.g. SCL-90-R) have only used global expressions of pain like “TMD” or “chronic pain” (e.g. Buckelew’s chronic pain group consisted of subjects with pain located to their back, abdomen, head or “other” areas) (Buckelew et al., 1986) or have only looked at one subset of diagnoses (e.g. Wilson’s group consisted of subjects with TMD-muscle pain) (Wilson et al., 1994). The studies have not used a specifically defined non-pain control group and have not compared different (regional and widespread) pain diagnoses and/or diagnostic subgroups. Further, somatic and cognitive patterning have not been related to MPI (Multidimensional Pain Inventory) profile groups, representing different expression of pain associated dysfunction.
The present study will make use of specific diagnoses, diagnostic subgroups, a non-pain control group and MPI profile groups. The objectives of the present study are to gain more information concerning the relationships between cognitive-somatic symptom patterning, anatomical distribution of pain and diagnostic subgroups of a regional pain. The first relationship will be evaluated by comparing psychometric data from patients with a widespread pain syndrome, patients with a regional, trigeminal pain and a non-pain control group. The second relationship will be analyzed using psychometric and clinical data from diagnostic subgroups.

A theoretical model (based on Okeson’s masticatory muscle model, 1993) was developed and is thought to represent a continuum starting with no pain, extending via “peripheral” regional pain, “centrally mediated” regional pain to a widespread, systemic pain. A demographically matched non-pain control group was expected to provide important data on existing, non-pain-related anxiety, depression and somatic/cognitive patterning. The regional pain syndrome was represented by orofacial pain, which was subdivided into diagnostic subgroups of “muscle disorder”, “intracapsular (TMJ) disorder” and “neuropathic pain disorder”. The orofacial pain muscle disorder subgroup was further subdivided into two groups. The first was a group consisting of the diagnostic entities Local Myalgia, Tendonitis and Muscular Co-contraction, representing diagnoses with less central/potentially more peripheral involvement. The second group comprised the diagnoses Myofascial Pain and Centrally Mediated Myalgia and was a diagnostic group with potentially more central nervous system involvement than the first group. The widespread/systemic pain was represented by fibromyalgia – a syndrome involving qualitatively altered nociception and thought to be a manifestation of an altered central nervous system processing of nociceptive stimuli (Bendtsen et al., 1997).

Aims and hypotheses

Female chronic Orofacial Pain patients and female Fibromyalgia patients from a tertiary treatment center and a demographically matched Non-Pain control group completed the SCL-90-R with the aims:

1) to compare among these different groups and among the diagnostic subgroups of the Orofacial Pain patients (Muscle group, Intracapsular group, Neuropathic group) the mean values for Somatization, Anxiety and Depression.
It was hypothesized that Somatization, Anxiety and Depression scores for the Orofacial Pain and Fibromyalgia groups would be higher than for the Non-Pain population. This was based on the assumption that Somatization correlate positively with pain intensity level, which was expected to be higher in the Neuropathic and Muscle subgroups and in the Fibromyalgia group compared to the Non-Pain group and the Intracapsular subgroup. The assumption was supported by the reports of elevated Somatization scores in chronic pain samples and a positive correlation between Somatization scores and the number of pain complaints (Walker et al., 1988 and Dworkin et al., 1990). As chronic pain patients often experience anxiety and depression (Gaskin et al., 1992; Banks and Kerns, 1996; McCracken, 1996), it was also hypothesized that Anxiety and Depression scores would be higher for the Orofacial Pain and Fibromyalgia groups than the Non-Pain group. It was predicted that the Neuropathic, Local Muscle, Central Muscle and Fibromyalgia groups would reveal higher Anxiety and Depression scores than the Intracapsular group, as the first four groups potentially would involve greater life interference from pain than the Intracapsular group.

2) to compare among the groups and the subgroups the type and degree of cognitive-somatic symptom patterning for the Anxiety and Depression subscales, after regrouping the somatic and cognitive items of these scales.

Based on published data on cognitive-somatic patterning (Buckelew et al., 1986), it was hypothesized that the Fibromyalgia and Orofacial Pain groups would reveal a higher somatic than cognitive component on the Anxiety and Depression scales. The Non-Pain control group was expected to reveal equal levels of cognitive and somatic scores on the Anxiety and Depression scales. All the Orofacial Pain subgroups were expected to show a greater expression of somatic than cognitive item responses on the Anxiety and Depression scales, but the Intracapsular subgroup was expected to show a smaller difference between the somatic and cognitive scores compared to the Neuropathic and Muscle subgroups. The Fibromyalgia group was expected to reveal the greatest difference between the somatic and the cognitive scores. These hypotheses were based on the assumptions from the previous paragraph combined with an expected strong positive correlation between the somatic subscale scores (on both the Anxiety and the Depression scales) and the Somatization score.

3) to compare among the different pain profile groups (“Dysfunctional”, “Interpersonally Distressed” and “Adaptive Copers” based on the Multidimensional Pain Inventory (MPI)) the
Somatization, Anxiety and Depression scores and to assess the relationship between cognitive and somatic subscale endorsements for these groups.

As a higher pain intensity level was expected for the Neuropathic and Muscle groups compared to the Intracapsular group and the pain level was thought to correlate with Somatization, Anxiety and Depression, it was hypothesized that the Dysfunctional and Interpersonally Distressed groups would reveal higher scores on Somatization, Anxiety and Depression compared to the group of Adaptive Copers. This was also supported by the finding that perceived disability increased linearly with increasing number of reported symptoms (Katon et al., 1991). Based on the assumptions in paragraph number 2, higher levels of somatic Anxiety and Depression among the Dysfunctional and Interpersonally Distressed groups compared to the Adaptive Coper group were expected. The Adaptive Copers were on the other hand thought to present either equal or higher levels of cognitive Anxiety and Depression compared to the two other groups.

4) to compare all SCL-90-R scale scores between the following two subsets extracted from the Orofacial Pain database:

a) Participants having a higher cognitive than somatic subscale score on the Anxiety and/or Depression scales (i.e. participants revealing a positive difference when the somatic subscale score is subtracted from the cognitive subscale score).

b) Participants having a higher somatic than cognitive subscale score on the Anxiety and/or Depression scales (i.e. participants revealing a negative difference when the somatic subscale score is subtracted from the cognitive subscale score).

The group endorsing cognitive items stronger than somatic items was, along with the thoughts of Buckelew et al (1986) expected to deal with their pain more on a cognitive level compared to the group endorsing somatic items stronger than cognitive ones. The latter group was expected to reveal a higher level of psychopathology and therefore also higher scores on the SCL-90-R scales compared to the other group.
Figure 1: Theoretical unidirectional relationships between negative emotions and pain
Chapter Two
Materials and Methods

Groups / Participants

The present study used data from three groups – two patient cohorts (groups B and C) derived from university based tertiary treatment centers and one population-based, Non-Pain control group (group A).

Group A / Non-Pain control group

Fifty-two female residents of the catchment area of the Kentucky Clinic in Lexington/Kentucky with the same age distribution as the total Orofacial Pain study group were recruited. The control population consisted of patients and accompanying persons recruited from the waiting areas in the College of Dentistry and the Clinic for Adult Dentistry at the University of Kentucky/Kentucky Clinic. When a person gave consent to participate after being informed about the study, she was asked to complete a brief questionnaire and to record gender, month and year of birth and place of residency. Additionally, psychometric data were collected. To be able to participate, the participants had to be pain free at the time of data collection and not have had any “chronic”, persistent or recurring pain during the preceding 6 months or for any period during their lives lasting more than 3 months. Pain referred to pain in any location of the body. The participants within each age cluster of the Non-Pain group were collected on a consecutive basis. Eleven questionnaires had to be discarded due to incompleteness and additional participants were recruited to achieve a total data set of 52 participants.

Group B / Orofacial Pain group

The participants that comprised this group had already been seen as part of a clinical consultation in the Orofacial Pain Center at the College of Dentistry at the University of Kentucky. For each of these participants clinical and psychometric data were available. Each participant had completed consent to have her data used for research purposes. Most participants
in the group had one or several diagnoses within the area of orofacial pain and these represented mainly a regional, trigeminal, chronic pain. All participants had been assessed and diagnosed according to the “Orofacial Pain - Guidelines for Assessment, Diagnosis, and Management”, published by The American Academy of Orofacial Pain in 1996 (Ed. J.P.Okeson). The complete Orofacial Pain database as of May 2002 consisted of 2,404 subjects and was used as the basis for selection and grouping of participants. The age range was 6 to 90 years with the majority of the subjects being 20 to 50 years old. 85% of the subjects were females and 15% males. The majority of the subjects were living in Kentucky at the time of examination. Participants were selected from the total database and divided into three groups for further analyses as follows: a) all female participants with a primary diagnosis of temporomandibular joint (TMJ) intracapsular disorder, b) all female participants with a primary diagnosis of neuropathic pain and c) all female participants with a primary diagnosis of muscle disorder. For all three groups, participants with secondary and/or tertiary diagnoses deviating from the primary one were excluded. The number of participants in the different subgroups can be found in Table 1. The Neuropathic Pain group was further subdivided in two groups: Episodic and continuous neuropathic pain. The Muscle Pain group was also subdivided into two categories:

I) Group with potentially less central nervous system involvement, represented by the diagnoses Local Myalgia, Tendonitis and Muscular Co-Contraction.

II) Group with potentially more extensive central nervous system involvement, represented by the diagnoses Myofascial Pain and Centrally Mediated Myalgia.

**Group C / Fibromyalgia group**

The data from this group had already been collected as part of an extensive study on fibromyalgia carried out in the Department for Physical Medicine and Rehabilitation at the Medical Center of the University of Kentucky. All participants had been diagnosed with fibromyalgia according to the 1990 American College of Rheumatology classification criteria. In the present study, this group was included to represent a global / widespread, chronic pain condition. The group consisted of 60 female participants, all residents of Kentucky. Ten participants had to be excluded due to incomplete data, resulting in a final study group of 50 female participants. Forty-five (91.8%) of these participants reported jaw pain as part of their
fibromyalgia complaint. The collected data consisted of demographic, clinical and psychometric information.

**Data for analyses**

**Demographic data**

For all the 3 main groups, age was recorded. The distribution of the number of participants for groups A, B and C as well as for the diagnostic subgroups of group B can be found in Table 1, which also includes the corresponding mean ages.

**Pain Ratings and Duration**

Duration of the pain complaint was recorded in months, from onset of the pain complaint until first evaluation / diagnosis in the Orofacial Pain Center for group B and from diagnosis until participation in the previously mentioned study by the University of Kentucky Physical Medicine Department for group C. Mean duration was in the range 66 months (Fibromyalgia) to 166 months (Local Muscle) and the median duration between 7 months (Central Muscle) and 36 months (Fibromyalgia) (Table 2).

Pain intensity was recorded through the patient’s pencil mark on a 100 mm VAS scale, where the left demarcation represented “no pain” and the right demarcation represented “the most imaginable pain”. The scale score was the distance from the “no pain” demarcation to the patient’s mark, measured in millimeters and recorded as average (VASAVE) during the week preceding the evaluation in the Orofacial Pain Center. VASAVE-scores were not available for the Fibromyalgia group. Jensen et al. (1986) have reported acceptable construct validity of the VAS scale, which also is in agreement with other pain intensity measurement methods. The mean score for VASAVE was between 32 for the Intracapsular group and 59 for the Neuropathic group (Table 2).
Psychometric data

For all groups

The multidimensional psychological symptom inventory “Symptom Check List-90–Revised” (SCL-90-R) (Derogatis et al 1976, 1983) was administered to all patients and also to the participants in the non-pain control group. This inventory consists of 90 psychological symptoms, each of which is rated on a scale 0 – 4, as to how much the specific symptom has been bothering the participant during the preceding week. A score zero represents “not at all”, one “a little bit”, two “moderately”, three “quite a bit” and four “extremely”. The 90 items are divided into 9 scales and 7 additional items. The scales are Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation and Psychotisism. Each scale score is obtained by dividing the sum of the item scores by the number of items for the specific scale, resulting in scale raw-scores between 0 and 4. In this study the raw-scores were used in the statistical computations and for group comparisons. Primarily the Somatization, Anxiety and Depression scales of the SCL-90-R were utilized in the present study. These scales are presented in Appendix B with their items listed. Satisfactory internal consistency, test-retest reliability and construct validity of the SCL-90-R have been reported (Derogatis, 1977). A copy of the SCL-90-R questionnaire is found in Appendix A.

To be able to quantify the somatic and cognitive aspects of the Anxiety and the Depression scales separately, their scale items were regrouped and the 7 additional items of the SCL-90-R were taken into account (according to Buckelew, 1986) as presented in Appendix C. The score for each subscale was calculated in the same manner as for the scale raw-scores. Reliability and validity data have not been published for these subscales. Chronbach’s Alpha was calculated for each subscale for each main group as a measure of internal consistency reliability in the present sample (Chronbach, 1951). An Alpha score of 0.7 or higher is considered acceptable. In this study all the Alpha values, except for Cognitive Anxiety in the Non-Pain group were higher than 0.7, suggesting acceptable internal consistency reliability for the four constructed subscales for all main groups except the Cognitive Anxiety subscale in the Non-Pain group (Table 3). Caution must be exerted when interpreting data involving the latter constellation.
The numeric values of the subscale scores themselves do not express anything per se and we are only interested in the relationship between the corresponding somatic and cognitive subscale scores for Anxiety and Depression within each group. To better express this relationship and for easier statistical computations, the mean cognitive value was subtracted from the mean somatic value. The resulting number being positive would mean that the somatic subscale score was larger than the cognitive subscale score. A negative number meant a larger cognitive than somatic subscale score.

For the non-pain control group

For clinical use, the SCL-90-R raw-scores are converted to t-scores, making the comparison of an individual patient’s scores with the general non-psychiatric patient population average (norm value) easier. A t-score of 50 represents the population average. In this study, the t-scores were calculated for all SCL-90-R scales in the non-pain control group to be able to compare this Kentucky based group to the norm values of the general population. The computed t-scores for the non-pain group are listed in Table 4. From these scores and their accompanying standard deviations it can easily be seen that there is no difference in SCL-90-R endorsements between the Kentucky control group and the general non-psychiatric patient reference population.

For the Orofacial pain group

The Multidimensional Pain Inventory (MPI) (Kerns, Turk & Rudy, 1985) was administered to all patients seen in the Orofacial Pain Center. The MPI scores take on values between 0 and 6, representing no and maximum positive endorsement respectively. Satisfactory internal consistency and test-retest reliability as well as construct validity for the MPI have been shown (Kerns et al., 1985). In this study, data from the MPI inventory were used to perform a profile patterning of the pain patients into the groups “Adaptive Copers”, “Dysfunctional” and “Interpersonally Distressed” according to Turk & Rudy, 1988. Adaptive Copers report high levels of social support and activity, and low levels of pain and pain-interference with their lives. Dysfunctional persons report high levels of pain-induced/-related psychological distress, pain-interference with their lives, high perception of pain and low activity levels. Interpersonally Distressed report poor social and domestic support in addition to a dysfunctional profile. The
profile grouping was performed using a “Profile centroid distance test”, which is a chi-square procedure testing the fit of a patient’s data with given profile-models.

**Protection of anonymity**

All data were released to and collected by the principal investigator in a way as not to disclose personal identifiers of the participants. IRB approval from the University of Kentucky was obtained and guidelines for the use of existing records were followed.

**Statistical analyses**

All analyses were conducted using the Statistical Package for the Social Sciences for Windows (SPSS V-10). Histogram analyses revealed that most outcome variables for the majority of the groups did not follow a normal distribution. Due to the fairly large number of subjects per group and the reduction in power when using non-parametric tests, it was decided to apply parametric statistics to the data analyses. A significance level of 0.05 was used for all statistical tests. Independent sample t-tests were used for comparison of two independent groups. Analyses of variance (ANOVA) were used for comparison of multiple groups. Analyses of co-variance (ANCOVA) were applied to correct for the influence of potential confounding factors like age, duration of pain (DURATION) and pain intensity level (VASAVE). For post hoc analyses, Levene’s test was applied to test for homogeneity of error variances across groups. Bonferroni’s test was utilized in the case of equal error variances. (This procedure accounts for the multiple tests and adjusts the significance level accordingly). Games Howell’s test was used in the case of unequal error variances. In this case, a Bonferroni-Holm procedure was then applied to adjust the significance levels according to the number of tests performed. Paired t-tests were used to test for equality between the somatic and the cognitive subscale scores of the Anxiety and Depression scales.
### Table 1: Number- and age-distribution of participants among the groups/subgroups.

<table>
<thead>
<tr>
<th></th>
<th>Groups</th>
<th>Non-Pain</th>
<th>Intracapsular</th>
<th>Neuropathic</th>
<th>Local Muscle</th>
<th>Central Muscle</th>
<th>Fibromyalgia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td></td>
<td>52</td>
<td>134</td>
<td>88</td>
<td>46</td>
<td>49</td>
<td>50</td>
</tr>
<tr>
<td>Mean age (Std. dev.)</td>
<td></td>
<td>39.62 (13.32)</td>
<td>41.48 (15.27)</td>
<td>45.44 (9.99)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 2: Pain duration and average pain intensity level for the Orofacial Pain subgroups and Fibromyalgia group.

<table>
<thead>
<tr>
<th></th>
<th>Groups / Subgroups</th>
<th>Groups / Subgroups</th>
<th>Non-Pain</th>
<th>Intracapsular</th>
<th>Neuropathic</th>
<th>Local Muscle</th>
<th>Central Muscle</th>
<th>Fibromyalgia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of pain (months)</td>
<td></td>
<td></td>
<td></td>
<td>Intracapsular</td>
<td>Neuropathic</td>
<td>Local Muscle</td>
<td>Central Muscle</td>
<td>Fibromyalgia</td>
</tr>
<tr>
<td></td>
<td>Mean (Std. error)</td>
<td>125.48 (25.49)</td>
<td>73.75 (19.70)</td>
<td>166.60 (48.95)</td>
<td>99.33 (35.01)</td>
<td>66.24 (10.16)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Median</td>
<td>25.49</td>
<td>18.00</td>
<td>12.00</td>
<td>7.50</td>
<td>36.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average pain intensity level (100 mm VAS scale)</td>
<td></td>
<td></td>
<td></td>
<td>Intracapsular</td>
<td>Neuropathic</td>
<td>Local Muscle</td>
<td>Central Muscle</td>
<td>Fibromyalgia</td>
</tr>
<tr>
<td></td>
<td>Mean (Std. error)</td>
<td>32.12 (2.37)</td>
<td>59.33 (2.79)</td>
<td>45.34 (3.78)</td>
<td>56.46 (3.61)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 3: Chronbach’s Alpha for the Anxiety and Depression subscales for the different diagnostic groups.

<table>
<thead>
<tr>
<th>Subscales</th>
<th>Cognitive Anxiety</th>
<th>Somatic Anxiety</th>
<th>Cognitive Depression</th>
<th>Somatic Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Pain</td>
<td>0.31</td>
<td>0.81</td>
<td>0.91</td>
<td>0.77</td>
</tr>
<tr>
<td>Orofacial Pain</td>
<td>0.86</td>
<td>0.80</td>
<td>0.92</td>
<td>0.79</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>0.75</td>
<td>0.81</td>
<td>0.93</td>
<td>0.71</td>
</tr>
</tbody>
</table>

Table 4: T-scores for the SCL-90-R scales for the Non-Pain group.

<table>
<thead>
<tr>
<th>Subscales</th>
<th>Mean score</th>
<th>Std. deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatization</td>
<td>49.02</td>
<td>8.90</td>
</tr>
<tr>
<td>Obsessive-Compulsive</td>
<td>50.04</td>
<td>9.55</td>
</tr>
<tr>
<td>Interpers. Sensitivity</td>
<td>50.90</td>
<td>9.43</td>
</tr>
<tr>
<td>Depression</td>
<td>49.27</td>
<td>9.35</td>
</tr>
<tr>
<td>Anxiety</td>
<td>47.67</td>
<td>8.51</td>
</tr>
<tr>
<td>Hostility</td>
<td>49.54</td>
<td>9.47</td>
</tr>
<tr>
<td>Phobic Anxiety</td>
<td>48.40</td>
<td>6.77</td>
</tr>
<tr>
<td>Paranoid Ideation</td>
<td>48.94</td>
<td>9.41</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>49.06</td>
<td>7.70</td>
</tr>
</tbody>
</table>
Chapter Three
Results

Corrections for co-variants

In the group comparisons, the mean values were corrected for potential confounding co-variants. Due to the different availability of these variants for the different groups, the following adjustments were made: In comparison of the Non-Pain, Orofacial Pain and Fibromyalgia groups, the mean values were corrected for age. In the comparison of the Orofacial Pain subgroups with the Fibromyalgia group, the mean values were corrected for age and duration of pain. Finally, in the comparison among the Orofacial Pain subgroups and the MPI profile groups, the mean values were corrected for age, duration of pain and the pain intensity level.

Cognitive-somatic patterning for the Anxiety and Depression scales

The mean somatic subscale score was higher than the mean cognitive subscale score for both the Anxiety and Depression scales in all groups and subgroups. Paired t-tests for intra-group comparisons of the mean cognitive and somatic subscale scores yielded significant non-equality of the mean subscores for both scales in all groups and subgroups (Table 5).

Analyses involving the Non-Pain group, Orofacial Pain group and Fibromyalgia group

Mean Somatization, Anxiety and Depression scores

The lowest mean values for the SCL-90-R Somatization, Anxiety and Depression scores were found in the Non-Pain group and the highest values in the Fibromyalgia group (Table 6).

Analyses of variance revealed a significant group effect for Somatization (F(2,367) = 38.95 / p < 0.001), Anxiety (F(2,367) = 8.61 / p < 0.001) and Depression (F(2,367) = 14.40 / p < 0.001). Post hoc tests revealed significant group differences between all the mean values for the Somatization, Anxiety and Depression scores.
Cognitive-somatic patterning for the Anxiety and Depression scales

The numeric difference between the mean subscale scores for the Anxiety and the Depression scales increased from the Non-Pain to the Orofacial Pain group and subsequently from the Orofacial Pain to the Fibromyalgia group (Table 6).

Analyses of variance revealed a significant group effect for the numeric differences in mean subscale scores for both the Anxiety (F(2,367) = 11.71 / p < 0.001) and the Depression (F(2,367) = 6.48 / p < 0.001) scales. Post hoc tests for the mean subscale differences for the Anxiety scale (CASA) revealed no significant difference between the Non-Pain and the Orofacial Pain groups. Post hoc analysis for the mean subscale differences for the Depression scale (CDSD) revealed significant difference between the Non-Pain and the Fibromyalgia groups. The mean value for the Orofacial Pain group did not differ from the other two groups.

Summary

The lowest mean Somatization, Anxiety and Depression scores were found in the Non-Pain group and the highest mean scores in the Fibromyalgia group, with the Orofacial Pain group taking on a middle value. The mean somatic subscale scores for Anxiety and Depression were higher than the corresponding mean cognitive subscale scores for the Non-Pain, Orofacial Pain and Fibromyalgia groups. For both Anxiety and Depression, there was a bigger difference between the mean somatic and cognitive sub-scale scores in the Fibromyalgia group than in the Non-Pain group. The somatic-cognitive subscale score differences for the Orofacial Pain group did not differ from the Non-Pain group regarding Anxiety and was not different from the Non-Pain and Fibromyalgia groups regarding Depression.

Subdivision of the Orofacial Pain group

For further analyses, the Orofacial Pain group was divided into three main subgroups: Intracapsular, Neuropathic and Muscle disorders. The Neuropathic group could potentially be further subdivided into “Episodic Neuropathic Pain” and “Continuous Neuropathic Pain”. The muscle pain group could additionally be subdivided into “Local Muscle Pain” and “Central
Muscle Pain” (see method section for description). A two-sample t-test was performed to compare the outcome variables between the “Episodic” and “Continuous” Neuropathic Pain groups and between the “Local” and “Central” Muscle Pain groups respectively, to find out if these additional subdivisions were appropriate.

Comparison of subjects with Episodic Neuropathic Pain vs. subjects with Continuous Neuropathic Pain

Of the 88 participants with Neuropathic Pain, 61 participants had Continuous and 27 participants had Episodic Neuropathic Pain. A two-sample t-test did not reveal any significant difference for any of the main outcome variables or potential co-variables between the two subdivisions of Neuropathic Pain (Table 7). Based on this result, the group “Neuropathic Pain” was kept as one group for further presentations and analyses.

Comparison of “Local” Muscle diagnoses vs. “Central” Muscle diagnoses

Of the 95 participants with Muscle Pain, 46 participants had “Local” and 49 participants had “Central” Muscle Pain. A two-sample t-test did not reveal any significant difference for any of the main outcome variables between the two subdivisions of Muscle Pain (Table 8). It did though, show a higher VASAVE pain intensity level for the “Central” group compared to the “Local” group (t(90) = -2.12 / p < 0.036). As the pain intensity level potentially could be a co-variant for the outcome variables, it was decided to keep the subdivision of the Muscle group for further analyses and presentations.

Analyses involving the Intracapsular Pain group, Neuropathic Pain group, Local Muscle Pain group, Central Muscle Pain group and Fibromyalgia group

Mean Somatization, Anxiety and Depression scores

The mean scores and accompanying standard deviations for the SCL-90-R Somatization, Anxiety and Depression scales are presented in Table 9.
Analyses of variance revealed a significant group effect for Somatization (F(4,310) = 22.94 / p < 0.001), Anxiety (F(4,310) = 10.50 / p < 0.001) and Depression (F(4,310) = 11.16 / p < 0.001). Post hoc tests for mean Somatization scores revealed no significant differences within the following clusters of groups: Intracapsular and Local Muscle; Neuropathic and Local Muscle; Neuropathic and Central Muscle; Central Muscle and Fibromyalgia. Post hoc tests for mean Anxiety and Depression scores revealed no significant differences within the following clusters of groups: Intracapsular; Neuropathic, Local Muscle, Central Muscle and Fibromyalgia.

Cognitive-somatic patterning for the Anxiety and Depression scales

Analyses of variance revealed a significant group effect for the numeric differences in subscale scores for the Anxiety scale (F(4,310) = 7.22 / p < 0.001) and for the Depression scale (F(4,310) = 3.45 / p < 0.009). Post hoc tests for the mean subscale differences for the Anxiety scale (CASA) revealed no significant differences within the following clusters of groups: Intracapsular and Local Muscle; Neuropathic, Local Muscle and Central Muscle; Neuropathic, Central Muscle and Fibromyalgia. Post hoc tests for the mean subscale differences for the Depression scale (CDSD) revealed no significant differences within the following clusters of groups: Intracapsular, Neuropathic, Local Muscle and Central Muscle; Intracapsular, Neuropathic, Central Muscle and Fibromyalgia.

Summary

The mean Somatization scores were higher for the Central Muscle and the Fibromyalgia groups than for the Local Muscle and Intracapsular groups. The mean Somatization score for the Neuropathic Pain group did not differ from the Local and the Central Muscle group scores. The mean Anxiety and Depression scores were higher for the Neuropathic, Local Muscle, Central Muscle and Fibromyalgia groups than for the Intracapsular group.

The mean somatic subscale scores for Anxiety and Depression were higher than the mean cognitive subscale scores for the Intracapsular, Neuropathic, Local Muscle, Central Muscle and Fibromyalgia groups. For Anxiety, there was a bigger difference between the mean somatic and the mean cognitive subscale scores in the Fibromyalgia group than in the Intracapsular and Local
Muscle group. The mean cognitive-somatic subscale score differences for the Neuropathic, Local Muscle and Central Muscle groups did not differ among each other. For Depression, there was a bigger difference between the mean somatic and the mean cognitive subscale scores in the Fibromyalgia group than in the Local Muscle group. The mean cognitive-somatic subscale score differences for the Intracapsular, Neuropathic and Central Muscle groups did not differ among each other or from any of the other groups.

**Analyses involving the Intracapsular Pain group, Neuropathic Pain group, Local Muscle Pain group and Central Muscle Pain group**

Due to the introduction of average pain level as an additional co-factor in the following analyses, the number of data responses vary from the analyses in the previous chapter. The descriptive statistics are therefore presented again as they also differ slightly from the previous chapter.

**Mean Somatization, Anxiety and Depression scores**

The mean scores and accompanying standard deviations for the SCL-90-R Somatization, Anxiety and Depression scales are presented in Table 10.

Analyses of variance revealed a significant group effect for Somatization (F(3,230) = 4.50 / p < 0.004), Anxiety (F(3,230) = 6.75 / p < 0.001) and Depression (F(3,230) = 5.37 / p < 0.001). Post hoc tests for mean Somatization scores revealed no significant differences within the following clusters of groups: Intracapsular, Neuropathic and Local Muscle; Neuropathic, Local Muscle and Central Muscle. Post hoc tests for mean Anxiety and Depression scores revealed no significant differences within the following clusters of groups: Intracapsular and Neuropathic; Neuropathic, Local Muscle and Central Muscle.
Cognitive-somatic patterning for the Anxiety and Depression scales

Analyses of variance revealed non-significant group effects for the numeric differences in subscale scores for the Anxiety scale (F(3,230) = 1.91 / p < 0.128) and for the Depression scale (F(3,230) = 2.02 / p < 0.111).

Summary

The mean Somatization scores were higher for the Central Muscle group than for the Intracapsular group. The mean Somatization scores for the Neuropathic and Local Muscle groups were not different from any of the groups. The mean Anxiety and Depression scores were higher for the Local and Central Muscle groups than for the Intracapsular group. The mean Anxiety and Depression scores for the Neuropathic group were not different from any of the groups.

The somatic-cognitive subscale score differences for Anxiety and Depression did not differ among any of the groups.

Analyses involving the MPI Profile groups: Adaptive Coper, Dysfunctional and Interpersonally Distressed (Based on the Orofacial Pain subgroups)

Mean ages for the MPI profile groups are presented in Table 11. Analyses of variance revealed no significant group effect for these mean ages (F(2,131) = 0.295 / p < 0.745).

Mean Somatization, Anxiety and Depression scores

The lowest mean values for the SCL-90-R Somatization, Anxiety and Depression scores were found in the Adaptive Coper group and the highest values in the Dysfunctional group (Table 11).

Analyses of variance revealed a significant group effect for Somatization (F(2,129) = 13.66 / p < 0.001), Anxiety (F(2,129) = 8.34 / p < 0.001) and Depression (F(2,129) = 13.40 / p < 0.001). Post hoc tests for mean Somatization scores revealed no significant differences within the
following clusters of groups: Adaptive Copers; Interpersonally Distressed and Dysfunctional. Post hoc tests for mean Anxiety and Depression scores revealed no significant differences within the following clusters of groups: Adaptive Copers and Interpersonally Distressed; Interpersonally Distressed and Dysfunctional.

**Cognitive-somatic patterning for the Anxiety and Depression scales**

The mean somatic subscore was higher than the mean cognitive subscore for both the Anxiety and Depression scales in all MPI profile-groups (Table 12). A paired t-test for intra-group comparisons of the somatic and the cognitive subscale scores yielded significant differences of all the corresponding subscores for both scales in all groups (Table 12).

Analyses of variance revealed non-significant group effects for the numeric differences in subscale scores for the Anxiety scale ($F(2,129) = 1.31 / p < 0.275$) and for the Depression scale ($F(2,129) = 0.73 / p < 0.486$).

**Summary**

There were no differences in mean age between the MPI profile groups. The mean Somatization scores were higher for the Interpersonally Distressed and Dysfunctional groups than for the Adaptive Copers. The mean Anxiety and Depression scores were higher for the Dysfunctional group than for the Adaptive Copers. The Interpersonally Distressed group did not differ from any of the other groups. The Anxiety and Depression subscale score differences were not different among the MPI profile groups.

**Comparison of the subsets COG and SOM**

Based on the Orofacial Pain subgroups, the following subsets were compared:

COG: Participants having a higher cognitive than somatic subscale score on the Anxiety and/or the Depression subscales.

SOM: Participants having a higher somatic than cognitive subscale score on the Anxiety and/or the Depression subscales.
MPI Profile distribution

The two subsets did not reveal any difference in MPI Profile distribution (Table 13). Chi Square value for Table 13 was 6.48 (df 5) / p < 0.263.

Mean SCL-90-R scale scores

Independent sample t-tests revealed significantly higher scores for COG than SOM for the following variables: SCL Anxiety, SCL Depression, SCL Obsessive Compulsiveness, SCL Interpersonal Sensitivity, SCL Hostility, SCL Phobic Anxiety, SCL Paranoid Ideation and SCL Psychotism. There were no evidence of differences between COG and SOM for Somatization, VASAVE, DURATION and age. See Table 14 for mean values and statistics.

Summary

The COG group revealed higher endorsement on all SCL-90-R scales except for Somatization. There were no differences between COG and SOM for Somatization, average pain intensity level, duration of pain, age and MPI profile distribution.
Summary of results

The mean Somatization, Anxiety and Depression scores were significantly different across the different groups with the Non-Pain group having the lowest scores and the Fibromyalgia group having the highest scores. The Orofacial Pain group scores were between the Non-pain and the Fibromyalgia groups. The mean Somatization, Anxiety and Depression scores were higher for the Central Muscle and Fibromyalgia groups than for the Intracapsular group.

In general, the mean somatic subscale scores for Anxiety and Depression were higher than the corresponding mean cognitive subscale scores. The mean Anxiety and Depression cognitive-somatic subscale score differences however, were greater in the Fibromyalgia group than in the Non-Pain group. When comparing the different Orofacial Pain subgroups and the Fibromyalgia group, the mean Anxiety and Depression cognitive-somatic subscale score differences were greater in the Fibromyalgia group than in the Local Muscle Pain group. No differences in the mean Anxiety and Depression subscale score differences could be shown among the Orofacial Pain groups.

For the Orofacial Pain participants, the mean Somatization, Anxiety and Depression scores were higher in the MPI Dysfunctional Profile group than in the MPI Adaptive Coper Profile group. Somatization scores for the Interpersonally Distressed Profile group were not different from scores obtained from the Dysfunctional Profile group. Anxiety and Depression scores for the Interpersonally Distressed Profile group were not different from scores obtained from the Adaptive Coper group and from those with a Dysfunctional profile. The mean ages as well as the mean Anxiety and Depression cognitive-somatic subscale score differences were not different among the MPI Profile groups.

For the Orofacial Pain group, the subset of participants having a higher cognitive than somatic subscale score on the Anxiety and/or Depression scales, compared to those participants who endorsed somatic items more than cognitive ones on the same scales, demonstrated higher mean scores for the following SCL-90-R scales: Obsessive-Compulsiveness, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation and Psychotisism. Mean Somatization score, pain intensity level, duration of pain, age and MPI profile distribution were not different between these two subsets of participants.
Table 5: Mean cognitive and somatic subscale score differences for SCL-90-R Anxiety (CASA) and Depression (CDSD) and paired t-test comparing the cognitive and somatic subscale scores for the Non-Pain, Intracapsular, Neuropathic, Local Muscle, Central Muscle and Fibromyalgia groups.

<table>
<thead>
<tr>
<th></th>
<th>cog-som</th>
<th>Std. dev.</th>
<th>t-value</th>
<th>df</th>
<th>p</th>
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<tr>
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<td>0.71</td>
<td>-7.92</td>
<td>77</td>
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<td>0.72</td>
<td>-4.56</td>
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</tr>
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<td>-0.69</td>
<td>0.72</td>
<td>-6.74</td>
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<td>0.76</td>
<td>-4.91</td>
<td>48</td>
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<tr>
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<td>-0.50</td>
<td>0.72</td>
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</tr>
<tr>
<td>Central Muscle</td>
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<td>0.60</td>
<td>-9.18</td>
<td>49</td>
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<td>-9.72</td>
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<td>0.71</td>
<td>-7.92</td>
<td>77</td>
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</table>

Table 6: Mean SCL-90-R Somatization, Anxiety and Depression raw-scores for the Non-Pain, Orofacial Pain and the Fibromyalgia groups.

<table>
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<tr>
<th></th>
<th>Somatization</th>
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<th>Depression</th>
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<tbody>
<tr>
<td></td>
<td>Mean score</td>
<td>Std. deviation</td>
<td>Mean score</td>
</tr>
<tr>
<td>Non-Pain</td>
<td>0.37</td>
<td>0.38</td>
<td>0.25</td>
</tr>
<tr>
<td>Orofacial Pain</td>
<td>0.73</td>
<td>0.66</td>
<td>0.51</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>1.46</td>
<td>0.76</td>
<td>0.72</td>
</tr>
<tr>
<td></td>
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<td>Std. dev.</td>
<td>t-value</td>
</tr>
<tr>
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</tr>
<tr>
<td><strong>Age (years)</strong></td>
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<td></td>
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<tr>
<td>Continuous NP Pain</td>
<td>48.16</td>
<td>11.40</td>
<td>-1.74</td>
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<tr>
<td>Episodic NP Pain</td>
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<tr>
<td><strong>Average pain intensity level (100 mm VAS scale)</strong></td>
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<tr>
<td>Continuous NP Pain</td>
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<td>Episodic NP Pain</td>
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<tr>
<td><strong>Duration of pain (months)</strong></td>
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<tr>
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<tr>
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<td>0.77</td>
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<td><strong>SCL-90-R Anxiety raw-score</strong></td>
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<td>0.57</td>
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<td><strong>SCL-90-R Depression raw-score</strong></td>
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Table 8: Mean values and statistics for the comparison between participants with Central Muscle and Local Muscle Pain.

<table>
<thead>
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<th></th>
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<th>Mean</th>
<th>Std. dev.</th>
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<th>df</th>
<th>p  &lt;</th>
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<td>Age (years)</td>
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<td></td>
<td>Central Muscle Pain</td>
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<tr>
<td>CDSD</td>
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Table 9: Mean SCL-90-R Somatization, Anxiety and Depression raw-scores for the Orofacial Pain subgroups and the Fibromyalgia group.

<table>
<thead>
<tr>
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<td>Mean score</td>
<td>Std. deviation</td>
<td>Mean Score</td>
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</tr>
<tr>
<td>Neuropathic</td>
<td>0.87</td>
<td>0.67</td>
<td>0.60</td>
<td>0.61</td>
<td>0.89</td>
<td>0.69</td>
</tr>
<tr>
<td>Local Muscle</td>
<td>0.75</td>
<td>0.66</td>
<td>0.66</td>
<td>0.70</td>
<td>0.97</td>
<td>0.99</td>
</tr>
<tr>
<td>Central Muscle</td>
<td>1.15</td>
<td>0.87</td>
<td>0.83</td>
<td>0.90</td>
<td>1.04</td>
<td>0.84</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>1.46</td>
<td>0.76</td>
<td>0.72</td>
<td>0.56</td>
<td>1.18</td>
<td>0.75</td>
</tr>
</tbody>
</table>

Table 10: Mean SCL-90-R Somatization, Anxiety and Depression raw-scores for the Orofacial Pain subgroups.

<table>
<thead>
<tr>
<th></th>
<th>Somatization</th>
<th></th>
<th>Anxiety</th>
<th></th>
<th>Depression</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean score</td>
<td>Std. deviation</td>
<td>Mean score</td>
<td>Std. deviation</td>
<td>Mean Score</td>
<td>Std. deviation</td>
</tr>
<tr>
<td>Intracapsular</td>
<td>0.47</td>
<td>0.44</td>
<td>0.26</td>
<td>0.37</td>
<td>0.46</td>
<td>0.56</td>
</tr>
<tr>
<td>Neuropathic</td>
<td>0.85</td>
<td>0.65</td>
<td>0.58</td>
<td>0.59</td>
<td>0.89</td>
<td>0.70</td>
</tr>
<tr>
<td>Local Muscle</td>
<td>0.74</td>
<td>0.68</td>
<td>0.68</td>
<td>0.73</td>
<td>0.99</td>
<td>1.04</td>
</tr>
<tr>
<td>Central Muscle</td>
<td>1.10</td>
<td>0.86</td>
<td>0.79</td>
<td>0.82</td>
<td>0.99</td>
<td>0.82</td>
</tr>
</tbody>
</table>
Table 11: Mean ages and mean SCL-90-R Somatization, Anxiety and Depression raw-scores for the MPI Profile groups: Adaptive Copers (ADAPT), Interpersonally Distressed (IPD) and Dysfunctional (DYSF).

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Somatization</th>
<th>Anxiety</th>
<th>Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (years)</td>
<td>Std. deviation</td>
<td>Mean score</td>
<td>Std. deviation</td>
</tr>
<tr>
<td>ADAPT</td>
<td>42.06</td>
<td>14.22</td>
<td>0.71</td>
<td>0.50</td>
</tr>
<tr>
<td>IPD</td>
<td>44.48</td>
<td>14.32</td>
<td>1.19</td>
<td>0.80</td>
</tr>
<tr>
<td>DYSF</td>
<td>43.16</td>
<td>14.53</td>
<td>1.39</td>
<td>0.87</td>
</tr>
</tbody>
</table>

Table 12: Mean cognitive and somatic subscale score differences for the SCL-90-R Anxiety (CASA) and Depression (CDSD) and paired t-test comparing the cognitive and somatic subscale scores for the MPI Adaptive Coper (ADAPT), Interpersonally Distressed (IPD) and Dysfunctional (DYSF) groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Subscale</th>
<th>cog-som</th>
<th>Std. dev.</th>
<th>t-value</th>
<th>df</th>
<th>p &lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADAPT</td>
<td>Anxiety (CASA)</td>
<td>-0.47</td>
<td>0.53</td>
<td>-7.33</td>
<td>67.00</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Depression (CDSD)</td>
<td>-0.60</td>
<td>0.63</td>
<td>-7.78</td>
<td>67.00</td>
<td>0.01</td>
</tr>
<tr>
<td>IPD</td>
<td>Anxiety (CASA)</td>
<td>-0.70</td>
<td>0.66</td>
<td>-5.01</td>
<td>21.00</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Depression (CDSD)</td>
<td>-0.72</td>
<td>0.59</td>
<td>-5.64</td>
<td>20.00</td>
<td>0.01</td>
</tr>
<tr>
<td>DYSF</td>
<td>Anxiety (CASA)</td>
<td>-0.71</td>
<td>0.68</td>
<td>-6.13</td>
<td>33.00</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Depression (CDSD)</td>
<td>-0.59</td>
<td>0.93</td>
<td>-3.79</td>
<td>35.00</td>
<td>0.01</td>
</tr>
</tbody>
</table>
Table 13: MPI Profile distribution for the COG and SOM subsets.

<table>
<thead>
<tr>
<th>Group</th>
<th>Count</th>
<th>Dysfunctional</th>
<th>Interpersonally Distressed</th>
<th>Adaptive Coping</th>
<th>Hybrid</th>
<th>Anomalous</th>
<th>Uninterpretable</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>COG</td>
<td>13</td>
<td>2</td>
<td>14</td>
<td>4</td>
<td>19</td>
<td>13</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>% within group</td>
<td>20.0%</td>
<td>3.1%</td>
<td>21.5%</td>
<td>6.2%</td>
<td>29.2%</td>
<td>20.0%</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>SOM</td>
<td>39</td>
<td>25</td>
<td>76</td>
<td>8</td>
<td>100</td>
<td>61</td>
<td>309</td>
<td></td>
</tr>
<tr>
<td>% within group</td>
<td>12.6%</td>
<td>8.1%</td>
<td>24.6%</td>
<td>2.6%</td>
<td>32.4%</td>
<td>19.7%</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>27</td>
<td>90</td>
<td>12</td>
<td>119</td>
<td>74</td>
<td>374</td>
<td></td>
</tr>
<tr>
<td>% within group</td>
<td>13.9%</td>
<td>7.2%</td>
<td>24.1%</td>
<td>3.2%</td>
<td>31.8%</td>
<td>19.8%</td>
<td>100%</td>
<td></td>
</tr>
</tbody>
</table>
### Table 14: Mean values and statistics for the comparison between the COG and SOM subsets.

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Std. dev.</th>
<th>t-value</th>
<th>df</th>
<th>p  &lt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>COG</td>
<td>38.91</td>
<td>15.70</td>
<td>-1.51</td>
<td>386</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>42.07</td>
<td>15.54</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average pain intensity level (100 mm VAS scale)</td>
<td>COG</td>
<td>42.06</td>
<td>29.41</td>
<td>-0.47</td>
<td>357</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>46.94</td>
<td>28.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of pain (months)</td>
<td>COG</td>
<td>35.81</td>
<td>66.58</td>
<td>-0.29</td>
<td>353</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>38.65</td>
<td>71.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Somatization raw-score</td>
<td>COG</td>
<td>0.80</td>
<td>0.72</td>
<td>0.71</td>
<td>373</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.74</td>
<td>0.70</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Obsessive-Compulsive raw-score</td>
<td>COG</td>
<td>1.10</td>
<td>0.88</td>
<td>4.09</td>
<td>373</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.68</td>
<td>0.73</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Interpersonal Sensitivity raw-score</td>
<td>COG</td>
<td>0.84</td>
<td>0.87</td>
<td>4.32</td>
<td>75.05</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.37</td>
<td>0.48</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Depression raw-score</td>
<td>COG</td>
<td>1.27</td>
<td>1.05</td>
<td>4.89</td>
<td>77.11</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.62</td>
<td>0.64</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Anxiety raw-score</td>
<td>COG</td>
<td>0.88</td>
<td>0.90</td>
<td>3.90</td>
<td>76.36</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.44</td>
<td>0.53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Hostility raw-score</td>
<td>COG</td>
<td>0.73</td>
<td>0.80</td>
<td>3.31</td>
<td>77.75</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.39</td>
<td>0.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Phobic Anxiety raw-score</td>
<td>COG</td>
<td>0.41</td>
<td>0.76</td>
<td>2.74</td>
<td>74.14</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.15</td>
<td>0.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Paranoid Ideation raw-score</td>
<td>COG</td>
<td>0.59</td>
<td>0.76</td>
<td>3.23</td>
<td>79.91</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.28</td>
<td>0.52</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCL-90-R Psychoticism raw-score</td>
<td>COG</td>
<td>0.50</td>
<td>0.69</td>
<td>3.75</td>
<td>73.32</td>
</tr>
<tr>
<td></td>
<td>SOM</td>
<td>0.18</td>
<td>0.34</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Chapter Four
Discussion

In agreement with the hypotheses, the Somatization and Anxiety scores were higher for the Orofacial Pain and Fibromyalgia groups than for the Non-Pain group. As predicted, this was true for the Depression scores also. When contrasting the Orofacial Pain subgroups, the Central Muscle group revealed higher Somatization, Anxiety and Depression scores than the Intracapsular group. This partly confirmed the hypotheses, as the expected higher scores for the Neuropathic and Local muscle groups than the Intracapsular group could not be confirmed. The Fibromyalgia group revealed higher Somatization, Anxiety and Depression scores than the Intracapsular group.

The mean somatic subscale scores for Anxiety and Depression were higher than the corresponding cognitive scores for all groups and subgroups. This was partly in agreement with the hypothesis, as the Non-Pain group was originally expected to reveal equal somatic and cognitive subscale scores. The hypothesized differential values for the Anxiety and Depression subscale score differences among the Orofacial Pain subgroups could not be confirmed. When introducing the Fibromyalgia and Non-Pain groups in the comparisons, the Fibromyalgia group demonstrated higher Anxiety and Depression subscale score differences than the Local Muscle and Non-Pain groups respectively, which only partly confirmed the original hypotheses.

The MPI Dysfunctional group revealed higher mean Somatization, Anxiety and Depression scores than the MPI Adaptive Coper group. This partly confirmed the hypotheses, which additionally had predicted dissimilarity between the MPI Adaptive Coper and Interpersonally Distressed groups. As the Dysfunctional group per definition reveal more psychological distress and a higher pain intensity level than the Adaptive Coper group, these findings were not surprising. As an adjustment was made for the average pain intensity level, the higher Somatization score for the Dysfunctional group than the Adaptive Coper group must be related to other physical complaints than the pain intensity. The hypothesized differential values for Anxiety and Depression cognitive-somatic subscale score differences could not be confirmed for the MPI Profile groups. This shows that no differentiated relationship between physical complaints and cognitive aspects of psychological distress exists among the different dysfunctional groups.
The subset of Orofacial Pain participants revealing a higher cognitive than somatic score on the Anxiety and/or Depression scales revealed a higher level of psychopathology expressed through higher scores on all SCL-90-R scales, except Somatization. These findings were the opposite of what was hypothesized. The findings could suggest that somatic subscale endorsement might be less related to development of psychopathology than cognitive subscale endorsement is. The fact that the mean somatic Anxiety and Depression subscale scores were higher than the mean cognitive subscale scores in all groups may suggest that somatic complaints are common also in a non-pain population, but that these complaints do not necessarily involve painful symptoms.

Consistent with the findings of Buckelew et al. (1986), the pain participants revealed higher Somatization scores than the Non-Pain participants. This would be expected due to more/stronger bodily symptoms in the Pain groups than in the Non-Pain group. These findings are also in agreement with McCracken’s finding of frequent somatic symptoms in a chronic pain population (1998). The higher endorsement of somatic than cognitive items on the Anxiety and Depression scales, which was reported for the pain group in the studies of Buckelew et al. (1986) and DeGood et al. (1985) was not found in the present study. Buckelew’s and DeGood’s mean scores were not adjusted for pain intensity or pain duration, and the somatic-cognitive patterning in that material was therefore probably dependent on one or both of these factors and not on group characteristics.

The finding that the subset of patients revealing higher Anxiety and/or Depression cognitive subscale scores than somatic subscale scores revealed higher scores on all SCL-90-R scales (except Somatization) while their pain intensity levels were the same, was directly opposite of the results reported by Buckelew et al.. Buckelew et al. felt that the analog subset from their own study represented more psychologically minded patients that already had sought treatment for mental health problems. Lower SCL-90-R scores for these subjects would therefore be expected. The findings from the present study may suggest that a high cognitive endorsement on the Anxiety and/or Depression scales does not express cognitive coping, but rather dysfunctional cognitive processes (e.g. catastrophizing).

In the present study, the creation of the COG and SOM subsets was based on both the Anxiety and the Depression scales for all Orofacial Pain participants from all Orofacial Pain diagnostic subgroups, but not containing clinical data. It was therefore not possible to evaluate if
either of the subsets (COG or SOM) would be derived from a specific scale (Anxiety or Depression). Further, it could not be evaluated if either of the subsets would be associated with specific total SCL-90-R Anxiety and/or Depression score levels or associated with specific diagnostic subgroups and/or clinical parameters.

Weinberger et al. stated (1979) that patients with repressive coping styles create a special clinical problem as they seem to be resistant to psychological interventions and seem to be highly prone to develop somatic complaints. Buckelew et al. drew similarities between repressors as described by Weinberger and chronic pain patients from their own study. Based on the present data, an analysis of cognitive-somatic item response patterning does not seem useful for “diagnosing” pain patients as about 80% of all pain patients in this study and also in Buckelew’s material revealed higher somatic than cognitive subscale scores.

Wilson et al. (1994) reported that high Somatization scores and high pain intensity scores were predictors of widespread muscle pain on palpation. A detailed comparison between Wilson’s study and the present study must be done with caution, as different methodologies were applied. Wilson et al. contrasted the non-somatic items on the SCL-90-R Anxiety and Depression scales with the SCL-90-R Somatization scale score. The present study also used the non-somatic items on the SCL-90-R Anxiety and Depression scales, but instead of the SCL-90-R Somatization score, the somatic items from the SCL-90-R Anxiety and Depression scales were used. Wilson et al. recorded pain on palpation while the present study used pain report by history. In the present study, the Central Muscle Group revealed higher Somatization scores than the Intracapsular group and the Pain groups demonstrated higher Somatization scores than the Non-Pain group. These findings could be interpreted as similar to the results of Wilson et al. If the higher Somatization scores for the Pain participants than the Non-Pain participants were based on group specific characteristics, on the extent of potential central involvement or on pain intensity level could not be decided, as the mean values in this comparison were not adjusted for the pain intensity level. This adjustment could however be performed for the mean values in the comparison of the Orofacial Pain subgroups.

Although muscle pain is classified as somatic pain in contrast to neuropathic pain and these two entities often are thought to underlie different pathophysiological mechanisms, it is still difficult to explain why only the Central Muscle Pain group and not the Neuropathic Pain group would reveal higher Somatization scores than the Intracapsular group, as both chronic
Central Muscle and Neuropathic pain would be considered conditions of central nervous system involvement. Until further and repeated data can support such a difference between the Central Muscle Pain and Neuropathic Pain groups, the present difference between these two groups must be regarded as to have occurred by chance and not to represent neurobiological differences between the groups. Wilson et al. also demonstrated that cognitive symptoms of psychological distress were less likely to be related to report of clinically widespread pain, but this could not be confirmed by the present study, as no group differences were detected for the Anxiety and Depression cognitive-somatic patterning.

Bridges (1991) compared primary care patients with somatic symptoms of distress and patients presenting with psychological symptoms. His findings suggested that “somatizers” were less depressed and less socially distressed than “psychologizers” were. While it is not known what pain-symptoms were in Bridges’ material, one could consider the SOM group from the present study as “somatizers” and the COG group as “psychologizers”. The finding that the COG group revealed more psychopathology than the SOM group would then resemble the findings of Bridges and suggest that most pain patients develop mainly somatic symptoms, but a subset of pain patients also reveal predominant psychopathology. It is not possible to evaluate if this psychopathology is secondary to pain and suffering or preceding / partially contributing to the pain condition.

There were several limitations of this study. As the average pain intensity scores were not available for the Fibromyalgia group, the mean scores could not be adjusted accordingly and limited conclusions could be drawn concerning potential differences between the Orofacial Pain and the Fibromyalgia group. In comparison of the Non-pain, Orofacial Pain and Fibromyalgia groups, as the mean values were only corrected for age, the higher Somatization, Anxiety and Depression scores, which were found for the Fibromyalgia group compared to the Orofacial Pain group must be interpreted with care, as the results could be confounded by other factors, e.g. pain intensity level and/or duration of pain. Further, the higher cognitive than somatic Anxiety and Depression subscale scores for the Fibromyalgia group than the Local Muscle and Non-Pain groups must also be interpreted with caution. The higher subscale score differences for the Fibromyalgia group could have resulted from differences in average pain intensity between the groups as in this comparison no correction was made for this factor.
The orofacial pain data were extracted from a database of over 2000 patients from a tertiary treatment center. Different patients saw different health professionals who may use different approaches in their diagnostic work up and also may have different priorities for their diagnostic listing of complaints. The data have been collected over many years with the possibility of change in diagnostic concepts over time. Although these factors are potential error sources, due to the size of the database and a fairly constant basic diagnostic strategy within the Orofacial Pain Center over time, it is felt that no segregation of errors within specific subgroups was likely to have significantly influenced patient classification.

The different subscales that were constructed to capture the differentiated cognitive-somatic item endorsements were based on the SCL-90-R Anxiety and Depression scales. These subscales were first introduced for this purpose and published by Buckelew et al. in 1986. It is important to remember that although these subscales also in the present study revealed satisfactory internal consistency reliability, the subscales have not been widely used and validity data have not been established. It is also important to keep in mind that psychometric evaluation of patients involves more than only the selected scales used in this study. On an individual basis it would be necessary to take data from the complete SCL-90-R into consideration.

Future research should include data containing information of pain intensity for fibromyalgia patients. In this way a comparison of SCL-90-R Somatization, Anxiety and Depression scores between the Orofacial Pain and the Fibromyalgia groups could be performed with mean values also adjusted for pain intensity. Then the role of pain intensity as a potential confounding factor could be established. Further, a regression analysis would reveal the relative contribution of the different co-factors for the SCL-90-R scale and subscale scores.

The present findings for the COG and SOM subsets suggest more detailed studies, especially concerning the relationship between the characteristics of the COG subset and type of psychological distress, e.g. if the expressed psychopathology is pain related or not. Further, longitudinal studies on the chronological development of dysphoria in the COG subset would be necessary to find out if the dysphoria precedes or follows the pain condition. It would also be interesting to find out how the COG and SOM subsets relate to the different diagnostic subgroups. Schwartz (1978) suggested that anxiety would be made up of patterns of specific psychobiological processes. Future research into the COG and SOM subsets may reveal interesting information about such patterns or about further subdivisions of the pain groups. Such
information could also give insight into potential predictive properties of the cognitive-somatic patterning.
Conclusion

Based on the results of this study, adding an analysis of somatic-cognitive item response patterning in the diagnostic work-up of chronic pain patients would not provide clinically useful information. Until data from prospective and retrospective longitudinal studies can substantiate the value for such an analysis for diagnostic purposes and its predictive utility for the development of pain chronicity, the differentiation of cognitive-somatic patterns remains an interesting heuristic exercise but does not contribute to developing a broader understanding of those factors that are associated with positive or negative clinical presentations.
Appendix A: Patient information letter and questionnaires
Collection of data for a non-pain control group to be used in the following study:

A comparison of response patterns between pain patients and persons with no pain.

Dear Madam / Sir:

In order to participate in this study you must presently be pain free and not have had any "chronic", persistent or recurring pain complaints during the last 6 months or for any period in your life lasting more than 3 months. Pain refers to any location in the body.

If you meet the above criteria, the Orofacial Pain Center at the University of Kentucky wants to invite you to take part in this project.

Please observe that your answers are totally anonymous, as you do not need to write down your name or other information, which could identify you.

Participation is absolutely on a volunteer basis. If you don't want to participate, this will have no negative effect (in general or as a patient at the University of Kentucky Medical Center) for you or the person(s) you are with today.

You will be asked to answer some questions about physical complaints as well as questions about your feelings and thoughts. After you have finished reading this page it will take you approximately 10 minutes to answer the questions.

The information we want to collect from pain free persons will be compared to information from people suffering from chronic pain. In this way it will contribute to our knowledge of pain in general, help us predict treatment outcome and to develop better treatments for people in pain.

You will not receive any reward for participating in this project, but your willingness to answer the questions may help doctors better understand and/or treat pain patients. We thank you in advance for the consideration and care for suffering people that you show by participating.

If you meet the inclusion criteria and have read this page and agree to participate, by filling out the questionnaires you give your consent for participation. The reason we do not ask you to give a written consent (sign this paper) is that your answers would then not be anonymous. In this way your privacy is guaranteed.
2. Demographic questionnaire (Non-Pain group)

If you have read the first page and agree to participate, please answer the following questions. Then proceed with the white/blue questionnaire (Skip the front page of the white/blue questionnaire!) Thanks again.

1. Gender
   Male  Female

2. a) Year of birth        19___
   b) Month of birth

3. Place of residency
   Kentucky  Outside Kentucky

If you previously have had any continuous pain lasting for more than 2 weeks but less than 3 months, please answer questions 4 and 5 before you continue with the white/blue questionnaire (Mark off all things that apply to you). If not, skip questions 4 and 5 and go directly to the white/blue questionnaire.

4. Was this previous pain lasting continuously for more than 2 weeks:
   a) Pain from illness treated with medicines
   b) Pain from illness treated with surgery
   c) Pain from illness that was treated in ways other than with medicines and/or surgery
   d) Pain not treated at all
   e) Pain after surgery
   f) Pain from trauma (car accident, sports accident, violence…)
   g) Pain resulting from habits (posture, work position…)
   h) Pain resulting from stress, depression, anxiety..
   i) Other / Unknown cause

5. Where was the pain located?
   a) Face / mouth / jaws
   b) Head other than in a)
   c) Upper back / neck / shoulders
   d) Lower back
   e) Lower part of body
   f) Any of the limbs
   g) Other
### INSTRUCTIONS:
Below is a list of problems people sometimes have. Please read each one carefully, and blacken the circle that best describes HOW MUCH THAT PROBLEM HAS DISTRESSED OR BOTHERED YOU DURING THE PAST 7 DAYS INCLUDING TODAY. Blacken the circle for only one number for each problem and do not skip any items. If you change your mind, erase your first mark carefully. Read the example before beginning, and if you have any questions please ask them now.

**EXAMPLE:**

<table>
<thead>
<tr>
<th>NOT AT ALL</th>
<th>A LITTLE BIT</th>
<th>MILDLY</th>
<th>QUITE A BIT</th>
<th>EXTREMELY</th>
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<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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</table>

**HOW MUCH WERE YOU DISTRESSED BY:**

1. **Headaches**
2. **Nervousness or shakiness inside**
3. **Repeated unpleasant thoughts that won't leave your mind**
4. **Faintness or dizziness**
5. **Loss of sexual interest or pleasure**
6. **Feeling critical of others**
7. **The idea that someone else can control your thoughts**
8. **Feeling others are to blame for most of your troubles**
9. **Trouble remembering things**
10. **Worried about sickness or carelessness**
11. **Feeling easily annoyed or irritated**
12. **Pains in heart or chest**
13. **Feeling afraid in open spaces or on the streets**
14. **Feeling low in energy or slowed down**
15. **Thoughts of ending your life**
16. **Hearing voices that other people do not hear**
17. **Trembling**
18. **Feeling that most people cannot be trusted**
19. **Poor appetite**
20. **Crying easily**
21. **Feeling shy or uneasy with the opposite sex**
22. **Feelings of being trapped or caught**
23. **Suddenly scared for no reason**
24. **Temper outbursts that you could not control**
25. **Feeling afraid to go out of your house alone**
26. **Blaming yourself for things**
27. **Pains in lower back**
28. **Feeling blocked in getting things done**
29. **Feeling lonely**
30. **Feeling blue**
31. **Worrying too much about things**
32. **Feeling no interest in things**
33. **Feeling fearful**
34. **Your feelings being easily hurt**
35. **Other people being aware of your private thoughts**
36. **Feeling others do not understand you or are unsympathetic**
37. **Feeling that people are unfriendly or dislike you**
<table>
<thead>
<tr>
<th></th>
<th>NOT AT ALL</th>
<th>A LITTLE BIT</th>
<th>MODERATELY</th>
<th>QUITE A BIT</th>
<th>EXTREMELY</th>
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<td>38</td>
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<td>Having to do things very slowly to insure correctness</td>
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<td>39</td>
<td>1 1 1 1 4</td>
<td>Heart pounding or racing</td>
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<td>40</td>
<td>1 1 1 1 4</td>
<td>Nausea or upset stomach</td>
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<td>41</td>
<td>1 1 1 1 4</td>
<td>Feeling inferior to others</td>
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<td>42</td>
<td>1 1 1 1 4</td>
<td>Soreness of your muscles</td>
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<td>43</td>
<td>1 1 1 1 4</td>
<td>Feeling that you are watched or talked about by others</td>
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<td>44</td>
<td>1 1 1 1 4</td>
<td>Trouble falling asleep</td>
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<td>45</td>
<td>1 1 1 1 4</td>
<td>Having to check and double-check what you do</td>
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<td>46</td>
<td>1 1 1 1 4</td>
<td>Difficulty making decisions</td>
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<td>47</td>
<td>1 1 1 1 4</td>
<td>Feeling afraid to travel on buses, subways, or trains</td>
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<td>48</td>
<td>1 1 1 1 4</td>
<td>Trouble getting your breath</td>
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<td>49</td>
<td>1 1 1 1 4</td>
<td>Hot or cold spells</td>
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<td>50</td>
<td>1 1 1 1 4</td>
<td>Having to avoid certain things, places, or activities because they frighten you</td>
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<td>51</td>
<td>1 1 1 1 4</td>
<td>Your mind going blank</td>
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<td>52</td>
<td>1 1 1 1 4</td>
<td>Numbness or tingling in parts of your body</td>
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<tr>
<td>53</td>
<td>1 1 1 1 4</td>
<td>A lump in your throat</td>
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<td>54</td>
<td>1 1 1 1 4</td>
<td>Feeling hopeless about the future</td>
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<td>55</td>
<td>1 1 1 1 4</td>
<td>Trouble concentrating</td>
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<td>56</td>
<td>1 1 1 1 4</td>
<td>Feeling weak in parts of your body</td>
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<td>57</td>
<td>1 1 1 1 4</td>
<td>Feeling tense or keyed up</td>
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<td>58</td>
<td>1 1 1 1 4</td>
<td>Heavy feelings in your arms or legs</td>
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<td>59</td>
<td>1 1 1 1 4</td>
<td>Thoughts of death or dying</td>
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<td>60</td>
<td>1 1 1 1 4</td>
<td>Overeating</td>
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<td>61</td>
<td>1 1 1 1 4</td>
<td>Feeling uneasy when people are watching or talking about you</td>
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<td>62</td>
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<td>Having thoughts that are not your own</td>
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<td>63</td>
<td>1 1 1 1 4</td>
<td>Having urges to beat, injure, or harm someone</td>
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<td>64</td>
<td>1 1 1 1 4</td>
<td>Awakening in the early morning</td>
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<td>65</td>
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<td>Having to repeat the same actions such as touching, counting, or washing</td>
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<td>66</td>
<td>1 1 1 1 4</td>
<td>Sleep that is restless or disturbed</td>
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<tr>
<td>67</td>
<td>1 1 1 1 4</td>
<td>Having urges to break or smash things</td>
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<td>68</td>
<td>1 1 1 1 4</td>
<td>Having ideas or beliefs that others do not share</td>
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<tr>
<td>69</td>
<td>1 1 1 1 4</td>
<td>Feeling very self-conscious with others</td>
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<tr>
<td>70</td>
<td>1 1 1 1 4</td>
<td>Feeling uneasy in crowds, such as shopping or at a movie</td>
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<td>71</td>
<td>1 1 1 1 4</td>
<td>Feeling everything is an effort</td>
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<td>72</td>
<td>1 1 1 1 4</td>
<td>Spells of terror or panic</td>
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<td>73</td>
<td>1 1 1 1 4</td>
<td>Feeling uncomfortable about eating or drinking in public</td>
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<td>74</td>
<td>1 1 1 1 4</td>
<td>Getting into frequent arguments</td>
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<td>75</td>
<td>1 1 1 1 4</td>
<td>Feeling nervous when you are left alone</td>
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<td>76</td>
<td>1 1 1 1 4</td>
<td>Others not giving you proper credit for your achievements</td>
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<td>77</td>
<td>1 1 1 1 4</td>
<td>Feeling lonely even when you are with people</td>
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<td>78</td>
<td>1 1 1 1 4</td>
<td>Feeling so restless you couldn’t sit still</td>
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<td>79</td>
<td>1 1 1 1 4</td>
<td>Feelings of worthlessness</td>
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<td>80</td>
<td>1 1 1 1 4</td>
<td>The feeling that something bad is going to happen to you</td>
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<td>81</td>
<td>1 1 1 1 4</td>
<td>Shouting or throwing things</td>
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<td>82</td>
<td>1 1 1 1 4</td>
<td>Feeling afraid you will faint in public</td>
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<td>83</td>
<td>1 1 1 1 4</td>
<td>Feeling that people will take advantage of you if you let them</td>
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<td>84</td>
<td>1 1 1 1 4</td>
<td>Having thoughts about sex that bother you a lot</td>
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<td>85</td>
<td>1 1 1 1 4</td>
<td>The idea that you should be punished for your sins</td>
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<tr>
<td>86</td>
<td>1 1 1 1 4</td>
<td>Thoughts and images of a frightening nature</td>
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<td>87</td>
<td>1 1 1 1 4</td>
<td>The idea that something serious is wrong with your body</td>
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<td>88</td>
<td>1 1 1 1 4</td>
<td>Never feeling close to another person</td>
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<td>89</td>
<td>1 1 1 1 4</td>
<td>Feelings of guilt</td>
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<td>90</td>
<td>1 1 1 1 4</td>
<td>The idea that something is wrong with your mind</td>
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</table>
Appendix B: Scales / Subscales
SCL-90-R scales utilized in this study

**Somatization (12 items)**

- Headaches
- Faintness or dizziness
- Pains in heart or chest
- Pains in lower back
- Nausea or upset stomach
- Soreness of your muscles
- Trouble getting your breath
- Hot or cold spells
- Numbness or tingling in parts of your body
- A lump in your throat
- Feeling weak in parts of your body
- Heavy feelings in your arms or legs

**Depression (13 items)**

- Loss of sexual interest or pleasure
- Feeling low in energy or slowed down
- Thoughts of ending your life
- Crying easily
- Feeling of being caught or trapped
- Blaming yourself for things
- Feeling lonely
- Feeling blue
- Worrying too much about things
- Feeling no interest in things
- Feeling hopeless about the future
- Feeling everything is an effort
- Feelings of worthlessness

**Anxiety (10 items)**

- Nervousness or shakiness inside
- Trembling
- Suddenly scared for no reason
- Feeling fearful
- Heart pounding or racing
- Feeling tense or keyed up
- Spells of terror or panic
- Feeling so restless you couldn’t sit still
- The feeling that something bad is going to happen to you
- Thoughts and images of a frightening nature
Subscales of Anxiety and Depression.

**Cognitive Anxiety (5 items)**

Suddenly scared for no reason
Feeling fearful
Spells of terror or panic
The feeling that something bad is going to happen to you
Thoughts and images of a frightening nature

**Somatic Anxiety (5 items)**

Nervousness or shakiness inside
Trembling
Heart pounding or racing
Feeling tense or keyed up
Feeling so restless you could not sit still

**Cognitive Depression (13 items)**

Thoughts of ending your life
Crying spells
Feelings of being caught or trapped
Blaming yourself for things
Feeling lonely
Feeling blue
Worrying too much about things
Feeling no interest in things
Feeling hopeless about the future
Feeling everything is an effort
Feeling of worthlessness
Thoughts of death or dying
Feelings of guilt

**Somatic Depression (7 items)**

Loss of sexual interest or pleasure
Feeling low in energy or slowed down
Poor appetite
Overeating
Trouble falling asleep
Awakening in the early morning
Sleep that is restless or disturbed
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- 1999 – 2002 Resident in the Orofacial Pain Center, University of Kentucky, USA