Somatosensory Amplification and Affective Inhibition Are Elevated in Myofascial Face Pain

Karen G. Raphael, PhD,* Joseph J. Marbach, DDS,* and Rollin M. Gallagher, MD, MPH†

*Department of Psychiatry, University of Medicine and Dentistry of New Jersey, New Jersey Medical School, and New Jersey Dental School of Oral Biology, Pathology, and Diagnostic Services; †Pain Medicine and Rehabilitation Center, Graduate Hospital, and Department of Psychiatry and School of Public Health, MCP Hahnemann University, Philadelphia, Pennsylvania

ABSTRACT

Objective. This study was designed to determine whether affective inhibition and somatosensory amplification are elevated in patients with a history of myofascial face pain (MFP). These processes may underlie a tendency to express distress in somatic rather than affective terms, leading to somatized or masked depression.

Design. Women (n = 162) with a history of MFP were compared with demographically equivalent women (n = 173) without MFP histories on self-report scales of affective inhibition and somatosensory amplification. Structured psychiatric interviews and health histories were conducted. In addition, a first-degree relative of 106 myofascial face pain subjects and 118 control subjects completed these same self-report scales.

Results. MFP cases and controls differed significantly on measures of affective inhibition and somatosensory amplification. History of depression or current psychological distress did not account for group differences. Elevated levels of somatosensory amplification were confined to MFP women with active symptoms. Finally, although both somatosensory amplification and affective inhibition showed a tendency to run in families, familial transmission did not account for case/control differences.

Conclusions. Affective inhibition and somatosensory amplification are likely to be elevated in patients with MFP. Although not accounted for by psychiatric symptomatology, the possibility that these response styles are reactive to coping with chronic face pain cannot be ruled out.

Key Words: Myofascial Face Pain; Affective Inhibition; Somatosensory Amplification; Masked Depression; TMD

One of the common disorders treated by pain clinicians is myofascial face pain (MFP) [1]. Characterized by pain in the muscles of mastication, it is considered to be the most prevalent type of temporomandibular disorder (TMD) [2].

The cause of MFP continues to be a subject of theory and research. As summarized recently by Molin [3], beginning in the 1950s [4,5], a paradigm shift occurred in which earlier mechanistic theories of etiology [6] were replaced by psychosocial theories. Today, integrationist, neurobiological and psychophysiological perspectives, based upon empirical animal and human studies, dominate theoretical discussion on the causes of chronic pain disorders (eg, [7–10]) and, specifically, myofascial facial pain (eg, [11]). However, more purely psychological theories, suggesting that pain is psychogenic, persist in the literature on MFP [12–15] and appear to unduly influence clinicians [16]. Many cases of MFP have been labeled “atypical facial pain of psychogenic origin” and viewed as part of a “masked” or “somatized” depression syndrome [12, 17–19]. This psychiatric model finds support in research documenting high rates of comorbid major depressive disorder [20–24]. In addition, reports suggesting the efficacy of antidepressants in its treatment [25] may be interpreted by some as evidence of the psychiatric origins of MFP.

The masked depression hypothesis has not been uniquely applied to MFP. This hypothesis, as well as a somewhat modified hypothesis that depressive syndromes and chronic pain syndromes of unknown origin share a pathogenic mechanism [26–30] has...
been applied to many painful disorders [31–38]. One recent study casts doubt on the hypothesis that MFP is a somatic manifestation of depression [39] because family patterns of depression in MFP indicate that depression is likely reactive to pain.

The link between depression and unexplained pain is difficult to disentangle. It has been noted [30] that research investigating the pain/depression relationship “needs to be closely linked to testing hypotheses derived from theoretical models . . . so that models can be either substantiated and refined or rejected” [30 (p. 31)].

Models [36] to explain the process underlying masked depression include a cognitive model, “affective inhibition,” in which an individual cannot or will not verbalize his or her feelings of sadness, or affect. This is akin to the psychoanalytically based concept of alexithymia [40,41], in which alexithymic individuals have difficulty in identifying and verbalizing feelings. The construct of somatosensory amplification [42] describes another pathway toward masked depression. Somatic amplifiers monitor and amplify normal body sensations. The combined effect of affective inhibition and somatosensory amplification may result in the expression of psychological distress in somatic rather than affective terms. If affective inhibition and the somatic focus of somatosensory amplification do occur more often in individuals with MFP, it is possible that these response styles are familially transmitted [36], either biologically or through social learning.

To date, studies examining these response styles specifically among MFP patients have been limited. One exception is a study of dental patients with pain of unknown origin [43] demonstrating that chronic facial pain patients tend to show more affective inhibition than either acute or pain-free dental patients, despite evidence of more affective disturbance in the chronic facial pain group.

The aim of the current investigation was to compare affective inhibition and somatosensory amplification in a group of MFP cases and demographically equivalent controls. In addition, a preliminary investigation of familial transmission of affective inhibition and somatosensory amplification was conducted by examining correspondence in response style between index cases or controls and their first-degree relatives.

Methods

MFP Cases

Records of patients with a history of MFP were drawn from the practice of a clinician (JJM) who specializes in the treatment of chronic facial pain. Only women were selected, because the overwhelming majority of those who seek treatment for MFP are women [44–46]. Only women who self-identified their race as white were selected, because there were few African-American or Hispanic women in the practice. Participants had to be current United States residents, between ages 18 and 65, and fluent English speakers. A sample of 443 was randomly selected for potential recruitment. They were selected from among 1,013 women who had sought treatment with the clinician at least once since 1979, and who, records indicated, had met diagnostic criteria for MFP at the time of consultation.

The diagnosis of MFP was made using International Association for the Study of Pain diagnostic criteria for temporomandibular pain-dysfunction syndrome (TMPDS) [47]: tenderness in one or more muscles of mastication together with a clicking or popping noise in the TMJ, and/or limitation of mandibular range.

For other purposes [39] not relevant to the investigation detailed below, we conducted a brief screening interview designed to oversample subjects with a history of major depressive disorder. After accounting for factors such as lack of current address, death, and other sources of demographic or diagnostic ineligibility, 198 patients were identified who met all study and screening criteria. Of these 198, 162 (82%) completed all study phases relevant to the current investigation. Further details of subject selection procedures are available in earlier reports [39].

Non-MFP Controls

MFP participants were asked to nominate one or more female acquaintances who were demographically similar to them but who did not have facial pain. Of 614 nominated acquaintances, 513 were randomly selected to participate in the study. Addresses were confirmed and initial contact was made with 498 acquaintances. Of the 498, 381 (77%) agreed to complete an initial screening interview. Of these women, 206 met all demographic and screening criteria; 173 (84%) eligible acquaintances completed the full study.

First-degree Relatives

Participating cases and controls were asked to provide consent for study staff to contact one or more of their first-degree relatives (parent, sibling, adult child). One first-degree relative was selected randomly from the roster of eligible family members (ie, ages 18–65) whom we had permission to contact. Forty-four MFP cases and 39 controls did not
provide permission to contact any first-degree relative. After accounting for direct refusal by family members, one first-degree relative of 106 (65% of 162) MFP cases and 118 (68% of 173) controls were enrolled.

Materials
All questionnaire materials were administered over the telephone. Other than the structured psychiatric assessment (see below), interviews were conducted by individuals possessing at least a masters degree in a health-related field. Mental health clinicians (PhD level or equivalent in experience) conducted psychiatric assessments.

Somatosensory Amplification (modified)
Although Barky’s [42] 10-item somatosensory amplification scale (SSA) has been shown in previous research to have adequate internal reliability, one of these 10 items (“I cannot stand pain as well as most people can”) is problematic for use in a sample of pain patients. To maintain adequate internal consistency, the 9-item scale was augmented with additional items (eg, “I sense foul odors that others might not notice,” “When my bladder is full, I’m often able to block out the sensation and wait a while before going to the bathroom” [reverse scored]), intended to similarly tap a respondent’s degree of sensitivity to normal body sensations. Responses to the 15-item scale were rated on a 5-point Likert scale. Internal consistency was adequate in both cases and controls (alpha = .76 and alpha = .71 respectively).

Affective Inhibition
The K-scale was used as a measure of psychological defensiveness or affective inhibition. Originally developed [48] specifically to identify psychological defensiveness or tendency to deny psychological symptoms, it has been used most often as part of the MMPI as a corrective factor for some of the other psychological scales in the inventory. Each of the 30 items (eg, “At times I feel like swearing,” “Criticism or scolding hurts me terribly”) is answered either true or false. In this sample, internal consistency was adequate among both cases (alpha = .73) and controls (alpha = .76).

Psychological Distress
Nonspecific psychological distress was assessed by the Demoralization Scale of the Psychiatric Epidemiology Research Interview [49]. The Demoralization scale has been shown to correlate highly with other measures of nonspecific distress [50]. As with the SSA scale, in order to ensure that symptoms of distress were not confounded with specific pain symptoms experienced more frequently by cases than controls, 2 of the original 27 items that were pain-related were removed. The remaining 25-item demoralization scale had high internal consistency in both cases (alpha = .89) and controls (alpha = .88).

History of Major Depression
Mental health clinicians were rigorously trained to directly interview study subjects by telephone to diagnose lifetime psychiatric disorders. The Structured Clinical Interview for DSM (SCID) [51], an instrument designed to enable trained clinicians to make reliable psychiatric research diagnoses according to the then-current DSM-III-R criteria, was used. Forty-six interview audiotapes, representing approximately every 12th completed SCID, were reviewed by both the clinical supervisor and a consultation/liaison psychiatrist to ensure reliability of the diagnosis of lifetime major depressive disorder, with a resulting Kappa of .86. The few discrepancies were resolved by consensus conference.

Health History
Health interviewers conducted a structured interview designed to record comprehensive physical health histories. As part of the interview, MFP symptoms and course, as well as current pain status, were assessed.

Analyses
Logistic regression was used to estimate the odds of case/control status as a function of SSA and K-scales, after controls for the effects of other factors. The odds ratio (OR) was used as an approximation of the relative risk of case status (being a patient with MFP), to show the increase in likelihood of case- ness for each point increase in SSA and K-scale score.

Results
Consistent with a masked depression hypothesis, cases and controls differed significantly on the somatosensory amplification scale (SSA) t = 2.42, P < .05; cases x = 2.74, controls x = 2.62, with the higher score of cases indicating a greater tendency to amplify normal body sensations. Cases and controls were also significantly different on affective inhibition as assessed by the K-scale (t = 2.87, P < .01; cases x = 14.25, controls x = 12.79), where scores represent the total number of items not en-
The scores on the two response style scales tended to negatively correlate to one another (r = -0.32 among cases and r = 0.35 among controls, both P < .0001).

In multivariate logistic regression analysis predicting case/control status from both SSA and K-scales, each measure was found to bear an independent relationship to case/control status (SSA: estimate = 0.925, SE = 0.27, P < .001, Odds Ratio = 2.2; K-scale: estimate: 0.105, SE = 0.028, P < .001, Odds Ratio = 1.11).

Because both measures were found to be associated with the demoralization scale (r = 0.38, P < .001 for SSA; r = -0.55, P < .001 for K-scale) we tested whether demoralization accounted for the relationship between case-control status and these characteristics, by adding demoralization to the logistic regression model. The resulting model found that the relationship between case-control status and SSA and K-scales was not accounted for by demoralization (SSA: estimate = 0.790, SE = 0.29, P < .01, Odds Ratio = 2.20; K-scale: estimate: 0.132, SE = 0.033, P < .0001, Odds Ratio = 1.14; demoralization: estimate = 0.380, SE = 0.22, P < .10, Odds Ratio = 1.46).

Similarly, we tested whether a lifetime history of major depressive disorder accounted for case-control differences on these response styles, by adding lifetime history of major depression to the logistic regression model. Again, the resulting model found that the relationship between case-control status and SSA and K-scale scores was not accounted for by a history of major depression (SSA: estimate = 0.906, SE = 0.275, P < .001, OR = 2.48; K-scale: estimate: 0.114, SE = 0.030, P < .0001, OR = 1.12; depression history: estimate = 0.271, SE = 0.247, P < .10, Odds Ratio = 1.31).

To test the possibility that somatosensory amplification was secondary to having a painful disorder, we compared the SSA score of those who said that their facial pain was “active” versus those who said it was “gone” or “in remission”. Of the subset who were able to categorize their current facial pain status, those with active or remitted facial pain (n = 130) had significantly higher tendencies to amplify normal body sensations than those who said their pain was gone (n = 29) (t = 2.21, P < .05). Active versus “pain gone” MFP cases did not differ in affective inhibition assessed by the K-scale (t = -0.34, P > .10).

Because we had the same self-report measures completed by first-degree relatives of cases and controls, we tested for familial transmission of these response styles. Among both cases and controls, there was a positive correlation between the index case or control’s score on the SSA scale and her first-degree relative’s SSA score (r = 0.18, P < .01; r = 0.16 in cases and r = .18 in controls P < .10). A trend toward a familial pattern of K-scale affective inhibition was also found in the combined sample (r = .12, P < .10). This pattern was actually confined to the cases (r = .30, P < .01) but was not found among controls (r = .01, P > .10). However, family members of cases versus controls were not significantly different from one another on these response styles (family SSA: cases x = 2.63, controls x = 2.56, t = 1.10, P > .10; family K-scale: cases x = 14.02, controls x = 13.80, t = 0.36, P > .10). Thus, although these response styles show some tendency to run in families, the hypothesis that elevation of these response styles is due to familial transmission was not supported in this sample.

Discussion

To our knowledge, this is the first study to examine levels of both affective inhibition and somatosensory amplification in MFP patients with and without major depression. These response styles could underlie a tendency toward masked or somatized depression in MFP patients and, therefore, are of clinical relevance [16].

Those women with a history of MFP showed elevated levels of affective inhibition and somatosensory amplification compared to a demographically equivalent sample of women without MFP. Multivariate analyses showed that differences between groups on these response styles were not due to differences in psychological distress or history of major depression.

Those who said that their MFP was gone did not differ from those who said their MFP was active or just in remission on levels of affective inhibition. These groups did differ on levels of somatosensory amplification. Those with active or remitted MFP showed greater somatosensory amplification than did those who said their MFP was gone. It may be tempting to conclude, then, that somatosensory amplification is reactive to facial pain. However, it is also possible that somatosensory amplification is a risk factor for a more recalcitrant form of MFP [52]. The causal relationship between somatosensory amplification and pain symptom variation is best untangled in the context of a longitudinal investigation.

Findings of elevated somatosensory amplification in MFP are consistent with other work, indicating that such individuals have enhanced ex-
perimental pain sensitivity [53] and a hypervigilant response style [54]. Neither laboratory nor questionnaire-based assessments can rule out the possibility that hypervigilance and somatosensory amplification are reactive to myofascial face pain. The monitoring of one’s bodily sensations may exert a tenuous sense of control over symptoms that are often highly variable within short periods of time [52].

Affective inhibition may well be a risk factor for the development of myofascial face pain. It appears to have trait-like characteristics, in that levels of affective inhibition did not vary with symptom remission among MFP patients. However, an alternative explanation exists. We have demonstrated [55] that patients with myofascial face pain are highly stigmatized by the notion that their facial pain is of psychological origin. It is conceivable that, in reaction to such treatment by health care providers and significant others, patients with facial pain tend to underreport normal affective experiences. Thus, a psychologically defensive response style could be reactive to the social experience of coping with chronic myofascial face pain.

This study found that levels of both affective inhibition and somatosensory amplification tend to run in families. The magnitude of the familial correlation was modest, and case versus control families did not significantly differ from one another. If our data had fully supported the hypothesis that familial factors account for case-control differences in affective inhibition or somatosensory amplification, the likelihood that these response styles are risk factors for MFP would be increased. This is because familial factors are likely to be present prior to the development of MFP. In fact, recent findings that MFP does not run in families [56] suggest that familial factors are unlikely to account for these response style differences in this particular pain disorder. However, familial transmission of affective inhibition and somatosensory amplification may play a clearer role in the pathogenesis of other painful disorders of unknown origin, such as fibromyalgia, a disorder that aggregates within families [57–58].

The concept of MFP as masked or somatized depression has, to a large extent, been replaced by more integrated biopsychosocial theories of etiology. Yet, the theory of masked depression in MFP persists. The current study shows that MFP patients express elevated levels of response styles involving affective inhibition and somatosensory amplification. The clinician treating MFP patients may observe these same elevations of response styles. Although tempting, it is premature to interpret these characteristics as indicators of an underlying masked depression. Cross-sectional observations such as these, in the context of both research and clinical practice, cannot disentangle cause from effect. As we await more definitive longitudinal research on risk factors for MFP, interpretive caution is advised.

References
18 Lehmann HJ, Buchholz G. Atypical facial neuralgia...


33 Magni G. On the relationship between chronic pain and depression when there is no organic lesion. Pain 1987;31:1–21.


