Use of a Structural Equation Model for Prediction of Pain Symptoms in Patients with Orofacial Pain and Temporomandibular Disorders

C. Ervin Davis, PhD
Assistant Professor
Department of Psychology
East Carolina University
Greenville, North Carolina

Charles R. Carlson, PhD
Professor and Chair
Department of Psychology

Jamie L. Studts, PhD
Associate Professor
Department of Behavioral Science

University of Kentucky
Lexington, Kentucky

Shelly L. Curran, PhD
Clinical Health Psychologist
Behavioral Medicine Associates
Milwaukee, Wisconsin

Rick H. Hoyle, PhD
Professor
Department of Psychology
Duke University
Durham, North Carolina

Jeffrey J. Sherman, PhD
Clinical Associate Professor
Department of Oral Medicine
University of Washington
Seattle, Washington

Jeffrey P. Okeson, DMD
Professor and Chair
Department of Oral Health Science
Director, Orofacial Pain Program
College of Dentistry
University of Kentucky
Lexington, Kentucky

Correspondence to:
Dr C. Ervin Davis, PhD
Department of Psychology
Rawl Building
East Carolina University
Greenville, NC 27858
Fax: 252-328-6283
Email: daviscl@ecu.edu

Aims: *To develop and test a biopsychosocial model using structural equation modeling for predicting orofacial pain symptoms in a sample of patients with masticatory muscle pain (MMP).* ***Methods:*** *Data were collected from clinic records of 251 adult patients who presented for initial evaluation to the Orofacial Pain Center at the University of Kentucky College of Dentistry and were subsequently diagnosed with MMP. Data were used to fit a model relating stressors, psychological distress, arousal, sleep problems, oral parafunction, and pain symptoms. Items from the Multidimensional Pain Inventory (MPI) and the IMPATH:TMJ, a comprehensive biopsychosocial assessment of patients with temporomandibular disorders (TMD), were used to construct a measurement model of five latent variables.* ***Results:*** *Estimation of the model indicated a good fit to the data and significant associations between stressors, psychological distress, arousal, sleep problems, and pain symptoms. Sleep problems partially mediated the relation between arousal and pain symptoms. Contrary to hypotheses, no association occurred between oral parafunction and pain symptoms, possibly indicating that any relationship between oral parafunction and pain symptoms may not exist.* ***Conclusion:*** *Results from the model tested in the present study are an additional step toward developing a more comprehensive biopsychosocial model explaining the nature and etiology of MMP in orofacial pain and TMD. With additional development and testing, it may also serve as an aid to planning interventions, especially psychosocial interventions targeting stress management, psychophysiological regulation, psychological distress, and sleep problems.* J OROFAC PAIN 2010;24:89–100.

Key words: biopsychosocial factors, chronic orofacial pain, masticatory muscle pain, structural equation model, temporomandibular disorders

Temporomandibular disorders (TMD) and orofacial pains are often complex multifactorial problems with prevalence rates of 5 to 7%[1] and predominantly affect women.[2] These disorders often are associated with many comorbidities, neuroendocrine-autonomic imbalances, significant pain, psychological stress, and disrupted mood systems.[3] Masticatory muscle pain (MMP) involves pain in the muscles of chewing[4] and is the predominant symptom in about 50% of orofacial pain cases.[1] Comorbid pain, such as cervical pain, headache, chronic fatigue, and fibromyalgia, are also common among persons with MMP.[5–10]

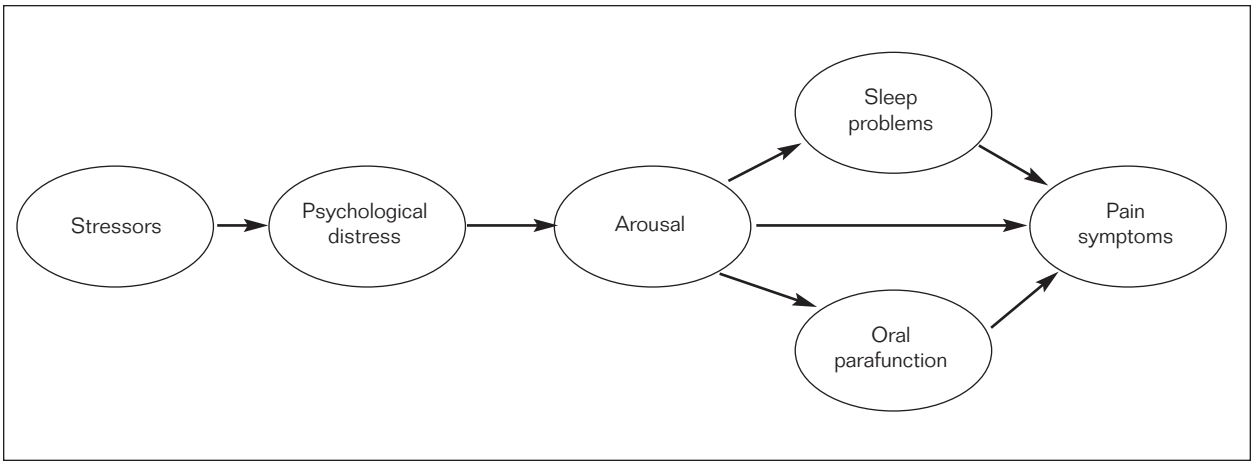

Fig 1 Hypothesized structural model predicting pain symptoms.

A review of TMD etiology emphasized the importance of integrating multiple physical and psychosocial factors in models of pain and disability.[11] One example of such integration of multilevel systems for chronic pain conditions was presented by Lackner et al[12] who tested a structural equation model (SEM) in chronic irritable bowel syndrome, relating pain sensation and affect to stages of pain processing. In patients with chronic low back pain, other researchers used a SEM to model coping and somatization,[13] and also pain catastrophizing, neuroticism, and pain-related vigilance and fear.[14] Of interest may be mediational models examining linkages between chronic stress and pathophysiology.[15]

The complexity of factors suggests that a SEM relating important biopsychosocial constructs to pain could improve theoretical understanding. The literature on associations between various biopsychosocial factors and orofacial pain has revealed many potentially important parameters such as oral parafunction, stress, arousal, autonomic and neuroendocrine changes, emotions, sleep disturbance, social, cognitive, and behavioral factors.[3,11,16]

Patients with MMP have demonstrated symptoms of psychological distress, higher arousal, heart rate and blood pressure under stress, along with more anxiety, depression, muscle tension, greater fatigue, and disturbed sleep than matched controls.[17,18] Patients with MMP also reported poorer sleep quality on the Pittsburgh Sleep Quality Index (PSQI) than headache patients.[19] In TMD patients, sleep problems have also been associated with increased pain, psychological distress, poorer coping, and lower perceived control.[20–22] In addition, external stressors, stressful life events, and posttraumatic stress disorder may lead to the development, exacerbation, or maintenance of chronic orofacial pain.[23–26] Patients also have shown higher masticatory muscle tension, oral parafunction, negative emotional states, financial strain, depression, stress, work strain, overload, lower social support, and lower work and life enjoyment and satisfaction.[6,10,24]

In summary, patients diagnosed with MMP evince numerous psychosocial symptoms that could predict or explain pain symptoms and suffering. In the present study, an initial explanatory biopsychosocial model was proposed that linked stressors, psychological distress, arousal, sleep problems, and oral parafunction to the development and maintenance of pain symptoms. This proposed model is consistent with the theory that arousal and autonomic and neuroendocrine activations to environmental challenges or stressors can produce exhaustion and damage to biological systems.[27,28] This model is also consistent with recent theories about the origins of orofacial pain and pain-stress response systems that involve the sympathetic nervous system, hypothalamic-pituitary-adrenal axis, and antinociceptive networks.[3] The specific aim of the present study was to develop and test the biopsychosocial model using structural equation modeling for predicting orofacial pain symptoms in a sample of patients with MMP. The study used SEM to test these hypothesized relationships in a large sample of clinic patients with a primary MMP diagnosis by using archival data from clinic records (Fig 1).

Materials and Methods

Sample

Archival data were collected and analyzed on 251 adult patients with a primary diagnosis of MMP who presented for initial evaluation at the Orofacial Pain Center at the University of Kentucky College of Dentistry over a 2-year period. The patients had previously consented to clinical and questionnaire data being used in research under an approved protocol of the University of Kentucky Medical Institutional Review Board. This clinical sample consisted of 225 (90%) female patients and 26 (10%) male patients. The average age of these patients was 35.3 years (range 12.9 to 81.3 years) and the average age was not different by gender $t = 0.84$, $P = .78$. Marital status of the patients was as follows: 47% married; 32% single; 12% divorced; 3% separated; 3% widow/widower; and 3% did not report. The ethnicity of the patients was 91% white, 1% black, and the remaining 8% included Asian, Native Americans, Hispanics, and other. Thirty-five percent of the patients reported working full-time, 10% part-time, 8% disabled, 5% unemployed, 4% retired, 10% homemaker, 16% student, and the remaining 12% gave no answer. The average patient reported having one to two children. The mean (± SD) pain duration was 1.6 ± 1.6 years. In addition to a primary diagnoses of muscle pain, 158 (62.9%) had a disc or temporomandibular joint (TMJ) problem, 64 (25.5%) had tension-type headache, 57 (22.7%) had cervical pain, 22 (8.8%) had migraine headache, 15 (6.0%) had neuropathic pain, and 11 (4.4%) had fibromyalgia (these numbers add to more than 100% because some patients received more than one secondary diagnosis).

Assessment

Clinical and standardized assessment information was collected from patient records, including information from a history and background questionnaire, the clinical chart, based on interview and physical exam from which diagnoses were obtained, and questionnaires completed by the patients, including the Multidimensional Pain Inventory (MPI)[29] and the Integrated Multi - dimensional Patient Assessment Tool for Health: Temporomandibular Joint (IMPATH:TMJ).[30]

Instruments

The data collected included scores from scales on the MPI, a 52-item questionnaire designed to examine the impact of chronic pain on patients' lives, response of others to pain, and participation in activities.[29] The MPI yields 12 subscales, including pain and affective distress scales used in the present model, and has demonstrated reliability and validity. The MPI scales have stability coefficients ranging from 0.62 to 0.91 and internal consistency ranging from 0.70 to 0.90. Also used, were scales from the IMPATH:TMJ.[30] The IMPATH:TMJ is designed to be used for general, integrated biopsychosocial assessment of TMD. It has stability of indices ranging from 0.69 to 0.93 and internal consistency from 0.74 to 0.87. The individual scales range from 1 to 28 with five anchor points, at the endpoints and at three intermediate points, with frequency or severity labels, such as "Never" = 1, "Sometimes," "Half the Time," "Usually," and "Always" = 28.

Creation of Model Variables

Six model variables (ie, five latent constructs and one directly measured variable) were created to test the proposed model and the specific study hypotheses. The five latent variables included: stressors, psychological distress, arousal, sleep problems, and pain symptoms. Each of the latent variables consisted of multiple indicators (or directly measured variables) which were selected from the assessments on the basis of content validity related to the latent variable of interest (Fig 2). The internal consistency of the five latent variables constructed was evaluated and each was greater than 0.90. Specific subscales and items used as latent variable indicators are described below. The construction of the model variables is described in detail in the following sections.

Stressors. This latent variable has three indicators and was formed from three item scales from the IMPATH:TMJ related to number of financial problems, amount of emotional support, and enjoyment of work or usual activities, in the past month. Thus, the stressor variable is a combination of indicators that potentially produce stress directly (eg, financial problems) and those that may buffer or moderate stress (support, enjoyment).

Psychological distress. This latent variable consisted of four indicators: the affective distress scale of the MPI, and five item scales from the IMPATH: TMJ, three of which were combined because they were highly correlated (average $r = 0.54$), had high

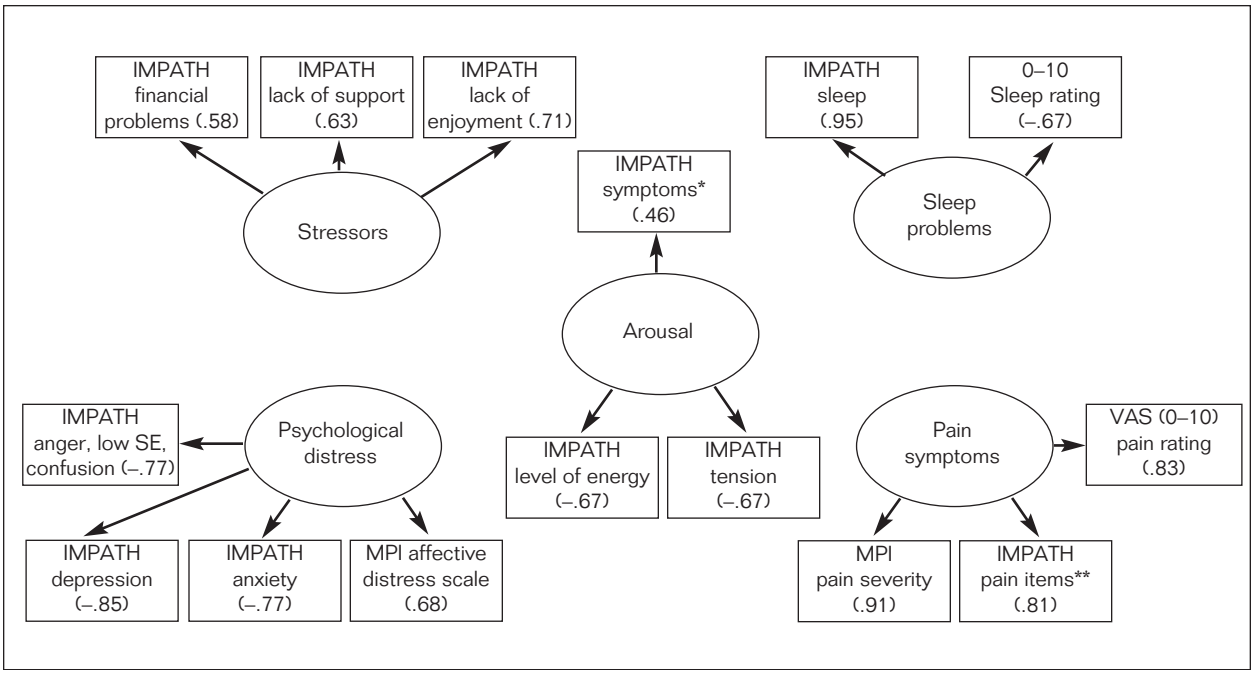

Fig 2 Measurement model with standardized factor loadings (in parentheses) and fit indices. All loadings are significant $P < .001$. Measurement error was included in the estimation, but is not shown in this figure. Fit indices: $\chi^2 = 183.3$, $df = 78$, n = 251, $P < .001$; CFI = 0.94; TLI = 0.92; RMSEA = 0.073 (90% CI: 0.06–0.09); SRMR = 0.068; ECVI = 1.07. *IMPATH symptoms are unusual sweating, weakness, shaking, short breath, racing heart rate, fatigue, cold hands, dizziness, fainting. **IMPATH pain items are pain frequency, duration, intensity, unpleasantness, difficulty to endure. The oral parafunction variable (not shown) was comprised of 16 IMPATH checklist items: grinding, clenching teeth, chewing gum, biting nails, holding or pressing tongue against teeth, biting lips, biting tongue, biting objects, touching or holding teeth together, holding jaw forward, rigid, or tense, waking with sore jaws, and being prevented from chewing—by pain or joint problem. SE = self esteem.

internal consistency (Cronbach's $\alpha = 0.8$), and improved measurement model fit and estimation. The IMPATH:TMJ depression and anxiety scales were used as separate indicators. The three scales combined by summing scores into a single indicator were ratings of anger frequency, feeling bad about yourself or low self esteem, and confusion about life (all rated in the past month).

Arousal. This latent variable has three indicators. One was formed by summing nine dichotomous (1 or 0), checklist items on the IMPATH:TMJ concerning physical problems related to high levels of arousal (ie, unusual sweating, general weakness, shaking or trembling, shortness of breath, racing heart, fatigue, cold hands, dizziness, and fainting). The other two indicators were two scales from the IMPATH:TMJ related to level of tension (How often are you tense in a typical day?) and level of energy (How is your energy level?).

Sleep Problems. This latent variable has two indicators and was formed from one interview question about overall sleep quality in the past month, and the other from the IMPATH:TMJ sleep scale rating sleep in the past month (What has your usual sleep been like in the past month?).

Pain symptoms. The pain symptoms latent variable included three indicators: *(1)* the pain severity scale of the MPI, *(2)* a visual analog scale (VAS) rating of current pain, and *(3)* the sum of five other ratings scales on the IMPATH:TMJ. The VAS ranged from 0 to 10, where 0 = "no pain" and 10 = "worst possible pain." The five scales from the IMPATH (which were combined as a third indicator variable by summing their scores) measured problem frequency (How often does the main problem occur?), duration (How long does it last?), intensity (How intense are symptoms?), unpleasantness (How unpleasant are symptoms?), and the difficulty of enduring pain. The pain symptom latent variable serves as the dependent, or criterion variable, in the model.

Oral parafunction. This variable was formed from a composite of 16 different dichotomous (ie, coded 1 or 0), self-reported checklist items on the

IMPATH:TMJ that are related to the presence of oral parafunctional behaviors and bruxism, oral habits or side effects of bruxism (ie, grinding, clenching teeth, chewing gum, biting nails, holding or pressing tongue against teeth, biting lips, biting tongue, biting objects, touching or holding teeth together, holding jaw forward, rigid, or tense, waking with sore jaws, and being prevented from chewing—by pain or jaw joint problem that you or others have noticed yourself doing). Scores on these items were combined into a single composite measured variable in the model by summing their values.

Statistical Analysis

The bivariate correlations between measured variables and the means and standard deviations (SDs) for these variables were calculated. The hypotheses were evaluated in the context of a SEM with maximum likelihood estimation using Amos 7.0.[31] The measurement and structural models were estimated and are described separately. The measurement model (see Fig 2) consisted of the evaluation of the 15 measured variables (not including oral parafunction) on each of the five latent variables which in turn were allowed to co-vary freely (covariance between all the latent variables). Measurement error for each of the measured variables was included in the model, although this is not shown in Figure 2 due to space limitations. This estimation procedure constitutes a confirmatory factor analysis (CFA) of the measurement model and has been recommended as the first step before evaluation of the structural model.[32] Goodness-of-fit indices were calculated for the measurement model and the factor loadings evaluated. These factor loadings are precisely the same as would be obtained in a traditional factor analysis.

Next, the hypothesized structural model was tested as shown in Figure 1. This model consists of hypothesized relationships among latent constructs. In the model, psychological distress and stressors are proposed to be precursors to the development of pain symptoms, mediated by the effects of arousal, sleep problems, and oral parafunction. Sleep problems and oral parafunction are hypothesized to partially mediate the effect of arousal on pain and symptoms. The path coefficients between the structural model elements, or latent variables, were evaluated along with model fit by using the selected fit indices described in the following paragraph. These path coefficients are the same as regression coefficients that would be obtained in a traditional multiple regression analysis.

The extent to which a proposed model corresponds to the observed relations is evaluated by various goodness-of-fit indices. Because of interpretational problems associated with exclusive use of the χ^2 approximation, various adjunctive indices of fit typically are consulted as well. Among the most common are the comparative fit index (CFI),[33] the Tucker-Lewis non-normed fit index (TLI),[34] and the root mean square error of approximation (RMSEA),[35] which were computed for the models estimated in the present study. The standardized root mean squared residual (SRMR), which is based on the difference in observed and implied covariance matrices,[36] and the expected cross-validation index (ECVI), which may be used to predict the appropriateness of a model for cross-validation[37] were also calculated. The CFI and TLI index the proportionate improvement in fit of a hypothesized model over a baseline model in which the covariances among the measured variables are constrained to zero. A value of 0.90 or greater for the CFI and TLI is considered sufficient justification for concluding that a model is consistent with the observed data.[38] The RMSEA is an index of the amount of misspecification of the model per degree of freedom, where values less than 0.05 indicate a good fit, values of 0.05 to 0.08 indicate a marginal fit, and values greater than 0.1 indicate an unacceptable fit.[39] The RMSEA is typically given as a 90% confidence interval (CI).

Similar to the strategy that underlies multiple regression, the goal of SEM is to account for variability in one or more outcomes—in the present case, muscle pain symptoms. The virtue of SEM is the ability to evaluate direct and indirect relations between predictors and outcomes (latent variables) and to separate measurement error from these latent variables. For instance, it was hypothesized that any relation between psychological distress and pain symptoms can be partially mediated by arousal, sleep problems, and oral parafunction, a relation between latent variables that cannot be tested using multiple regression. Additionally, biases due to measurement error in mediators may be reduced in the estimation of both indirect and direct effects through the use of SEM.[40] Other post-hoc tests can also be performed with the same SEM, for instance the direct effect of psychological distress on pain symptoms can be tested in the same model.

Another goal of SEM is to test a hypothesized pattern of relations believed to underlie the observed relations in a sample. The effect sizes of path coefficients between latent variables in the SEM in addition to their statistical significance can be examined.

Table 1 Correlation Matrix of Measured Variables

	Psychological distress				Stressors			Arousal			Sleep problems		Pain symptoms		
Latent/ measured variable	MPI affective distress	IMPATH anger, feeling bad, confusion	IMPATH anxiety	IMPATH depression	IMPATH financial problems	IMPATH lack of support	IMPATH lack of enjoyment	IMPATH symptoms	IMPATH level of energy	IMPATH tension	Interview sleep rating	IMPATH sleep	MPI pain	Pain (VAS)	IMPATH pain severity
Psychological distress															
MPI affective distress	1.000														
IMPATH anger, feeling bad, confusion	–0.542**	1.000													
IMPATH anxiety	–.517**	.564**	1.000												
IMPATH depression	–.525**	.703**	.650**	1.000											
Stressors															
IMPATH financial problems	–.292**	.388**	.480**	.477**	1.000										
IMPATH lack of support	–.417**	.541**	.395**	.470**	.359**	1.000									
IMPATH lack of enjoyment	–.402**	.425**	.480**	.576**	.576**	.448**	1.000								
Arousal															
IMPATH symptoms	.282**	.224**	–.296**	–.375**	–.156*	–.274**	–.280**	1.000							
IMPATH level of energy	–.505**	.393**	.511**	.496**	.368**	.366**	.536**	–.429**	1.000						
IMPATH tension	–.508**	.476**	.573**	.549**	.386**	.403**	.393**	–.281**	.397**	1.000					
Sleep problems															
Interview sleep rating	.385**	–.190**	–.357**	–.398**	–.257**	–.218**	–.361**	.328**	–.443**	–.387**	1.000				
IMPATH sleep	–.367**	.179**	.351**	.312**	.291**	.225**	.354**	–.276**	.346**	.290**	–.621**	1.000			
Pain symptoms															
MPI pain	.463**	–.254**	–.245**	–.390**	–.194**	–.257**	–.380**	.224**	–.380**	–.322**	.592**	–.396**	1.000		
Pain (VAS)	.383**	.769**	–.344**	–.380**	–.211**	–.220**	–.346**	.172**	–.373**	–.338**	.573**	–.369**	.769**	1.000	
IMPATH pain severity	.309**	–.231**	–.239**	–.289**	–.210**	–.226**	–.259**	.236**	–.373**	–.288**	.569**	–.372**	.744**	.648**	1.000
Oral parafunction	.316**	–.283**	–.291**	–.243**	–.160*	–.143*	–.114	.242**	–.157*	–.360**	.239**	–.094	.000	.090	.003

Two-tailed significance tests, * significant at .05. level; ** significant at .01 level.

Results

The Pearson product-moment bivariate correlation matrix of the 16 measured variables, including oral parafunction, is given in Table 1 and descriptive statistics for these variables are given in Table 2.

From the correlation matrix, it can be observed that nearly all the bivariate correlations between measured variables were significant. The magnitude of the significant correlations generally fell in the moderate to large effect size range,[41] especially for the measured variables making up psychological distress and pain symptoms, between the psychological distress and arousal variables, and also between the sleep problems and pain symptoms variables. Other correlations were in the small to moderate effect size range, including the correlations between measured variables on sleep problems, with the exception of correlations between pain symptom variables. Associations between other measured variables and oral parafunction were mostly small. Within each latent variable construct, the individual measured variables all correlated significantly (at the $P < .01$ level) for each latent construct which can be observed in the triangular submatrix portions of the correlation matrix. Between constructs, the correlations were also significant at the $P < .05$ or $P < .01$ levels, which can be observed in the rectangular submatrix portions of the overall correlation matrix. The only variable with nonsignificant correlations with other variables was the composite oral parafunction variable. These non-significant correlations were especially apparent between oral parafunction and the other measured variables indicating the pain symptom latent variable.

In checking the distributional characteristics of the data, the skewness and kurtosis of each of the measured variables were computed along with the multivariate kurtosis. Critical ratios for skewness ranged from 0.292 to –8.58. These skewness values were not considered to be a problem for model estimation.[42] For kurtosis, the critical ratios ranged from 0.34 to 6.65. The multivariate kurtosis (Mardia's coefficient) critical ratio was 10.46. It is generally recognized that normalized kurtosis coefficients, even in the 20s, cause no particular difficulty when using maximum likelihood estimation, so these values were considered to be well-within acceptable limits for valid model estimation.[43]

A pictorial diagram of the measurement model that was constructed and analyzed is shown in Figure 2. The standardized factor loadings are shown labeled in Figure 2. All of the factor loadings in the measurement model were significant.

Table 2 Descriptive Statistics for Measured Variables

Latent/ measured variables	Minimum	Maximum	Mean	SD
Psychological distress				
MPI affective distress	3.0	93.0	46.9	11.1
IMPATH anger, feeling bad, confusion	3.0	84.0	61.7	17.3
IMPATH anxiety	1.0	28.0	15.6	8.2
IMPATH depression	1.0	28.0	18.9	7.2
Stressors				
IMPATH financial problems	1.0	28.0	17.2	9.4
IMPATH lack of support	28.0	84.0	66.6	13.3
IMPATH lack of enjoyment	1.0	28.0	16.6	8.0
Arousal				
IMPATH symptoms	0.0	9.0	1.9	2.0
IMPATH level of energy	1.0	28.0	14.8	8.4
IMPATH tension	1.0	28.0	13.2	8.0
Sleep problems				
Interview sleep rating	0.0	7.3	3.3	2.1
IMPATH sleep	1.0	28.0	15.7	7.1
Pain symptoms				
MPI pain	6.0	70.2	13.5	14.0
Pain (VAS)	0.0	6.0	3.2	1.7
IMPATH pain severity	30.0	140.0	103.4	24.0
Oral parafunction	0.0	12.0	4.5	3.1

The estimated fit indices for the measurement model are also shown in Figure 2. The calculated adjunctive fit indices were a CFI of 0.94 and a TLI of 0.92 indicating a good fit, and a RMSEA of 0.074 (90% CI 0.06 to 0.09), indicating a marginal fit, as the upper bound of the CI exceeds 0.08.

The structural model with standardized statistical path estimates and fit indices is shown in Fig 3. The number of measured variables in the model was 16 resulting in a total of 136 elements in the observed matrix. The number of parameters to be estimated was 40, resulting in 136-40, or 96 degrees of freedom for the model estimated. The estimated model allowed measurement errors for MPI affective distress and MPI pain to covary (r = 0.39), and also the composite IMPATH:TMJ (anger, feeling bad, confusion) and IMPATH:TMJ depression variables were allowed to covary (r = 0.31). The post-hoc addition of these covariances improved model fit and is justified on the basis of these variables both being scales on the same instruments (the MPI and IMPATH) that contain common method variance that can be accounted for in the SEM.[44]

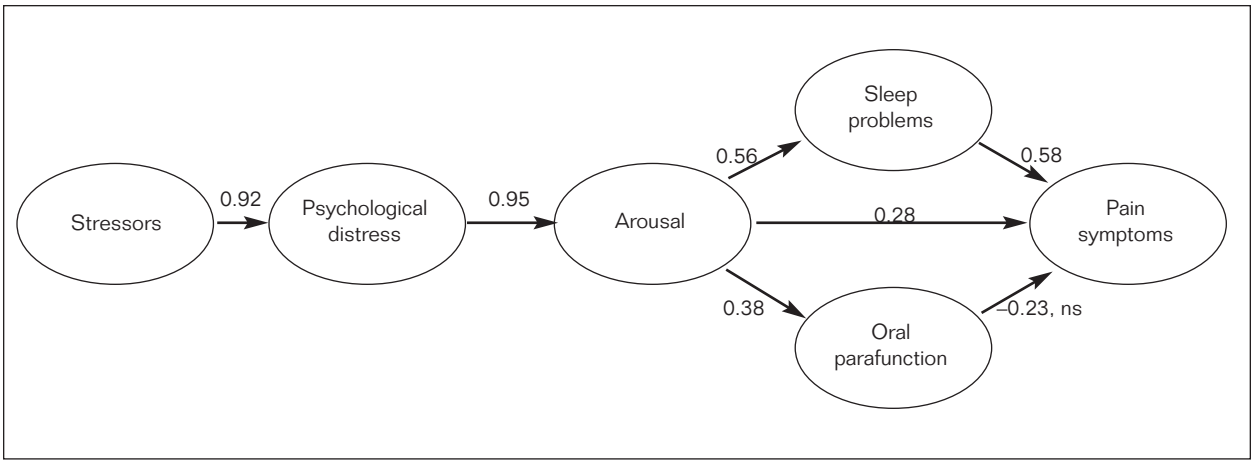

Fig 3 Structural model standardized statistical estimates and fit indices. All paths are significant ($P < .001$) except Oral parafunction to pain symptoms (ns = not significant). Fit indices: $\chi^2 = 228.2$, $df = 96$, n = 251, $P < .001$; CFI = 0.93; TLI = 0.92; RMSEA = 0.074 (90% CI: 0.06–0.09); SRMR = 0.052; ECVI = 1.09.

For the structural model shown in Fig 3 the estimated fit indices are also shown. The adjunctive fit indices were a CFI of 0.93 and a TLI of 0.92 indicating a good fit, and a RMSEA of 0.074 (with 90% CI of .06 to .09) indicating a marginal fit, as the upper bound of the CI exceeds 0.08. The hypothesized paths were significant in the model, with the exception of the path from oral parafunction to pain symptoms. Large effects were observed between stressors, psychological distress, and arousal (0.92, 0.95). A small effect was observed between arousal and pain symptoms (0.28), and a medium effect between arousal and sleep problems (0.56), and between sleep problems and pain symptoms (0.58). Also a small effect was observed between arousal and oral parafunction (0.38).

Self-perceived arousal was found to be a mediator of the effects of stressors and psychological distress on sleep problems and pain symptoms. The total effect of arousal on pain symptoms was 0.53 SD units (moderate effect size), which includes a direct effect of 0.25 and the indirect effect (mediated by sleep problems) of 0.28.

As an exploratory post-hoc analysis, an alternative structural path direct from psychological distress to pain symptoms was tested to evaluate the hypothesis that arousal, sleep problems, and oral parafunction could only partially mediate the relationship between psychological distress and pain symptoms; however, the path between psychological distress and pain symptoms was found to be nonsignificant (0.08, $P = .83$). In addition, the post-hoc hypothesis that a direct link exists between psychological distress and sleep problems was evaluated, but also found to be nonsignificant (–4.6, $P = .62$), as was the path between psychological distress and oral parafunction (–0.51, $P = .45$). Also note from the structural path coefficients, that while a positive and significant association existed between arousal and oral parafunction, no association was found between oral parafunction and pain symptoms.

Discussion

There has been considerable research exploring variables associated with pain symptoms in the facial region.[17,18,20,22,24,25,45,46] To consolidate this research, integrated multifactorial biopsychosocial models are an important step leading to better theory and treatment.[11] The plausible model tested in the present study was based on previous research on patients with MMP and TMD, which demonstrated relationships among stressors, psychological distress, arousal, sleep problems, oral parafunction, and pain symptoms. A case was made for the development of pain symptoms arising from the other variables, although the model could also apply to the exacerbation or maintenance of chronic MMP. In fact, for the clinical sample studied, patients had experienced pain for an average of 1.6 years and patients typically had more than one chronic pain condition. Also, in Table 2 the mean levels of variables comprising psychological distress, stressors, arousal, sleep problems, and pain symptoms are comparable to those of other patients with chronic pain in general and TMD specifically.[29,30]

In the present study, self-perceived arousal was found to be a mediator of the effects of stressors and psychological distress on sleep problems and pain symptoms. This particular finding may have several clinical and theoretical implications. The present model supports the importance of arousal as playing a central role in relation to pain symptoms. Treatments designed to reduce arousal, such as those reducing symptoms assessed in the present study (ie, unusual sweating, weakness, shaking, shortness of breath, racing heart rate, fatigue, cold hands, dizziness, fainting, tension, and level of energy), might lead to reduced pain symptoms. In fact, a recent randomized treatment study showed reductions in pain symptoms in TMD patients with the use of a combination of behavioral and cognitive techniques that deal directly with reduction of arousal and physical self-regulation, including training in diaphragmatic breathing and relaxation.[47]

As a further exploration of the role of arousal in mediating the relationship between psychological distress and pain symptoms, the study evaluated (post hoc) separately each direct path between psychological distress and pain symptoms, sleep problems, and oral parafunction. In each case, these path coefficients were nonsignificant with arousal in the model. In other words, arousal fully mediated the relationship between psychological distress and pain symptoms, extending support for the conclusion that arousal mediates the effect of psychological distress on pain symptoms.

While based solely on self-report data in the present study, arousal may be better assessed using physiological recording and may be associated with the development and maintenance of facial and other chronic pain disorders.[48] In previous work, MMP patients showed higher heart rate and blood pressure responses to laboratory stress than matched controls[17] and MMP patients had lower end-tidal CO_2 levels at rest.[18] Patients with myofascial pain also showed higher plasma cortisol, adrenaline, and noradrenaline responses in response to psychological stress and slower recovery of baseline levels than controls.[49] A SEM that included evoked potentials, pupil dilation, and skin conductance in pain-free volunteers in response to increasing levels of painful stimulation, showed that a combination of physiological variables related to pain threshold and the gradient of the pain response was the best-fitting and most parsimonious model.[50] Also self-reported physiological reactivity to pain predicted later increases in pain in patients with rheumatoid arthritis.[51]

Additionally, since a large and significant association was found between psychological distress and arousal, concurrent psychological therapies such as cognitive-behavioral therapy[52] or treatments designed to improve coping and develop stress management skills may also reduce arousal. Also as expected, stressors were strongly associated with psychological distress, supporting the hypothesis that the presence of stressors may be associated with psychological distress in these patients. The impact of stressors could be affected by interventions specifically designed to enhance coping skills, promote lifestyle changes, and increase social supports. Ultimately, such changes would be expected to lead to symptom relief. Psychological-based interventions to address the somatic symptoms of anxiety related to arousal are also a reasonable clinical course to pursue with facial pain patients. Progressive relaxation training[53–55] and indeed, cognitive-behavioral and electromyographic biofeedback interventions have been shown to be effective for a variety of muscle pain disorders.[52,56,57] Combined modality, individual patient-tailored treatments may be optimum.[58,59]

Stress, anger, and lack of enjoyment or satisfaction of life and work have been associated with chronic pain in previous studies.[6,10,60,61] However, one limitation of the present study was the lack of conventional indicators of stress, such as major life event or daily hassles scales. Further work could explore whether the current model would be supported with the inclusion of such information.

The present results clarify the roles played by sleep problems in association with pain symptoms and are consistent with studies linking poor sleep with chronic pain.[62,63] The total (direct) effect of sleep problems on pain symptoms was 0.58 (moderate effect size). In the model, sleep problems partially mediated the relationship between arousal and pain symptoms. One interpretation is that sleep problems appear as a result of arousal, but once they occur, they themselves lead to pain symptoms. It can be asserted that part of any intervention should focus on assessing and treating sleep problems. This conclusion is consistent with clinical reports and recommendations in the literature on TMD and orofacial pain.[64,65] Recent studies have shown that sleep problems are important factors influencing the onset and course of pain and TMD symptoms.[22,66,67] However, in a previous longitudinal study of orofacial pain patients, pain led to negative affect, which then led to problems with sleep.[22] Moreover, previous research has postulated a reciprocal relationship between sleep problems and pain, with sleep problems leading to

pain and pain problems further exacerbating problems with sleep.[68] Further research is warranted to understand more fully the role of sleep.

The positive association between arousal and self-reported oral parafunction was expected as part of the hypothesized mediational role that oral parafunction may play between arousal and pain symptoms. However, the association between oral parafunction and pain symptoms was nonsignificant in the tested model. Perhaps oral parafunction does not relate directly to pain symptoms or the patients did not accurately report their level of oral parafunction. A recent study demonstrated that masticatory muscle tension was significantly related to pain in patients with TMD[24]; however, muscle tension and oral parafunction may be distinct. Further research should explore where the oral parafunction variable best fits in the model.

Overall, the SEM provided necessary information on the likely and plausible relationships between stressors, psychological distress, arousal, sleep problems, and oral parafunction, as these variables are associated with pain symptoms in a large clinical sample of patients with MMP. There are limitations to the present study, especially related to the correlational nature of the data and the reliance on self-report, so the present results are not sufficient to confirm this particular model. Equally well-fitting, alternative models could be proposed which would hypothesize, for example, that pain symptoms originate due to factors not included in the current model, and then lead to sleep problems, arousal, and psychological distress. A SEM of this sort could be mathematically equivalent in terms of the estimation of path and fit coefficients; however, it would have different theoretical implications.[69] Further studies using longitudinal or experimental designs are needed to resolve these questions as this limitation is based upon the correlational research design, rather than limitations of a statistical approach, such as SEM, and would also apply had the present study used a different analysis such as multiple regression. The present model may demonstrate how the associations between psychosocial variables and pain tend to maintain one another in these MMP patients. An understanding of fundamental neurobiological mechanisms causing pain and leading to psychological distress could also be used to demonstrate causality. As yet unidentified factors could be hypothesized that would lead to increases in psychological distress, arousal, sleep problems, and pain symptoms. Ongoing research into the mechanisms associated with chronic orofacial pain may add other elaborations to the present model.

The current results also emphasize the value of a SEM for clarifying potentially fruitful areas of clinical investigation. Given the difficulty and expense of conducting epidemiologic studies and randomized clinical trials, the present SEM enables use of existing clinical data to establish directions for experimental clinical research. It provides an economical and parsimonious means to develop plans for formal clinical trials and longitudinal studies that have a reasonable probability for success.

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