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Potential Psychosocial Risk Factors for Chronic TMD: Descriptive Data and Empirically Identified Domains from the OPPERA Case-Control Study

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Abstract

Case-control studies have consistently associated psychosocial factors with chronic pain in general, and with temporomandibular disorders (TMD) specifically. Moreover, a handful of prospective studies suggest that pre-existing psychosocial characteristics represent risk factors for new onset TMD. The current study presents psychosocial findings from the baseline case-control study of the Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA) cooperative agreement. For this study, 1,633 TMD-free controls and 185 TMD cases completed a battery of psychosocial instruments assessing general psychosocial adjustment and personality, affective distress, psychosocial stress, somatic awareness, and pain coping and catastrophizing. In bivariate and demographically-adjusted analyses, odds of TMD were associated with higher levels of psychosocial symptoms, affective distress, somatic awareness, and pain catastrophizing. Among controls, significant gender and ethnic group differences in psychosocial measures were observed,

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consistent with previous findings. Principal component analysis was undertaken to identify latent constructs revealing four components: stress and negative affectivity, global psychosocial symptoms, passive pain coping, and active pain coping. These findings provide further evidence of associations between psychosocial factors and TMD. Future prospective analyses in the OPPERA cohort will determine if the premorbid presence of these psychosocial factors predicts increased risk for developing new-onset TMD.

Keywords

temporomandibular disorders; psychosocial risk factors; chronic pain; somatic awareness; catastrophizing

Introduction

Abundant evidence demonstrates that psychosocial factors contribute importantly to the experience of pain. In numerous case-control studies, investigators have reported that compared with pain-free controls, patients with chronic pain conditions show elevations on measures of psychosocial distress, environmental stress, catastrophizing, and somatic awareness, and these psychosocial variables are associated with poorer pain-related adjustment among patients with chronic pain [20,31,35]. In addition to these findings related to chronic pain in general, similar results have been reported in patients with temporomandibular disorders (TMD)[25,70]. For example, relative to pain-free populations, patients with TMD have reported higher levels of affective distress,[9,10,50] somatic awareness,[47,51] psychosocial stress, [13,48] and pain catastrophizing.[8,67] Also, personality characteristics, such as neuroticism, have been found to differ for TMD cases versus controls.[29,61] Recently, neuroticism was linked to increased gray matter thickness in the prefrontal cortex in TMD patients,[55] perhaps implicating neuroticism in cortical changes associated with altered pain processing in TMD. Further evidence of the importance of psychosocial factors in TMD derives from studies demonstrating an association between psychosocial measures and both the severity and persistence of TMD-related clinical symptoms. Specifically, cross-sectional studies show that higher levels of psychological distress are related to increased TMD pain and disability,[6,51,86] and longitudinal designs have demonstrated that psychosocial factors, such as somatic awareness and depression, represent risk factors for long-term persistence of TMD pain.[26,30,58]

While the above findings clearly demonstrate the clinical relevance of psychosocial factors in TMD, whether TMD pain drives psychological symptoms, or vice versa, is difficult to determine from studies of established TMD cases. Several prospective studies have demonstrated that pre-existing psychosocial factors predict the development of new onset chronic pain conditions, including chronic widespread pain,[53] regional musculoskeletal pain,[57] and low back pain.[43,44,57] However, few investigators have examined the role of psychosocial factors in predicting new onset TMD using prospective cohort designs. Slade and colleagues [75] assessed psychosocial factors in healthy females and followed them for a three-year period, during which time 8.8% of the sample developed new onset TMD pain. Regression models revealed that measures of depression, perceived stress, and mood state obtained at baseline were significant predictors of new onset TMD. A subsequent prospective study by Aggarwal et al.[1] showed that health anxiety assessed at baseline predicted risk for development of chronic orofacial pain over the ensuing two-year period. Thus, a paucity of prospective studies has examined whether premorbid psychosocial factors are associated with increased risk of TMD onset.

In addition to psychosocial variables, demographic factors have been associated with TMD risk. Specifically, sex differences in TMD are well-documented, as women are significantly more likely to be diagnosed with TMD than men. [18,42] Moreover, TMD prevalence varies with age, with peak prevalence occurring between the ages of 20 and 40, and lower prevalence observed in childhood and in older adults.[18,42] Ethnic and racial differences in TMD have also been identified in a handful of studies, for example, with African Americans showing lower prevalence rates than whites, especially in young adults.[34,66] It is notable that many of the psychosocial factors that have previously been related to TMD are also associated with demographic factors. For instance, compared to males, females report higher levels of psychological distress, somatic awareness, psychosocial stress, and catastrophizing.[3,4,23,76] This raises the possibility that the association of demographic factors with TMD could be mediated by psychosocial variables. Therefore, we report analyses examining gender, age, and ethnic/racial differences in psychosocial variables.

Given the findings described above, the assessment of psychosocial factors would be an integral component of any study designed to identify risk factors for onset and persistence of TMD. The Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA) study is a prospective cohort study that endeavors to discover causal determinants of TMD pain. The design of OPPERA is based on a conceptual model that hypothesizes that psychosocial factors represent a critically important intermediate phenotypic risk factor for development of TMD.[17] The findings presented below are restricted to the baseline assessment of psychosocial variables in a large group of people with examiner-classified TMD arthralgia, myalgia, or both ("TMD cases") and people who were found not to have TMD when examined ("controls"). The primary aim of this study was to characterize differences in psychosocial functioning across multiple psychosocial domains between TMD cases and controls. In order to accomplish this, a battery of psychosocial instruments was administered, with the goal of assessing psychosocial functioning across several domains that have been associated with chronic pain in previous research. Specifically, we included measures of personality and global psychological adjustment, mood and affect, psychosocial stress, somatic awareness, and pain coping and catastrophizing. While previous research has identified a variety of psychosocial variables that may be associated with TMD, few studies have assessed this broad array of psychosocial factors and determined their relationship specifically with TMD. A secondary aim of the study was to identify latent constructs across the multiple psychosocial measures that were administered. To that end, we conducted exploratory principal component analysis to investigate these putative latent psychosocial constructs.

Materials and Methods

Subjects

As described elsewhere,[74] the OPPERA baseline case-control study used advertisements, emails, flyers and word-of-mouth to recruit people who had chronic TMD (TMD cases) and people who did not (controls). They were recruited between May 2006 and November 2008 from communities in and around academic health centers at four US study sites: Baltimore, MD; Buffalo, NY; Chapel Hill, NC; and Gainesville, FL. At each study site, the target was to recruit 800 controls and variable numbers of cases based on local operational requirements, for a total of 3,200 controls and 200 cases. The actual number enrolled was 3,263 controls and 185 cases.

The classification of TMD was based on the Research Diagnostic Criteria for Temporomandibular Disorder.[21] In summary, cases met all three of the following criteria: during the telephone interview, (i) pain reported with sufficient frequency in the cheeks, jaw muscles, temples, or jaw joints during the preceding six months (at least 15 days in the

preceding month and at least five days per month in each of the five months preceding that); during the examination, (ii) pain reported in the examiner-defined orofacial region for at least 5 days out of the prior 30 days; and (iii) pain reported in at least three masticatory muscles or at least one temporomandibular joint in response to palpation of the orofacial muscles or maneuver of the jaw. Examiners defined the orofacial region by touching the following anatomical areas bilaterally: temporalis, preauricular, masseter, posterior mandibular, and submandibular. Controls met all six of the following criteria: during the telephone interview, (i) pain reported infrequently in the cheeks, jaw muscles, temples, or jaw joints (no orofacial pain in the preceding month and no more than four days per month in any of the five months preceding that); (ii) no more than four headaches per month within the preceding three months; (iii) never diagnosed with TMD; (iv) no use of night guard occlusal splint; and during the examination, (v) pain reported in the examiner-defined orofacial region for no more than 4 days in the prior 30 days; (vi) classified as having neither myalgia nor arthralgia. However, controls could be positive or negative with respect to pain in response to palpation or jaw maneuver. Additional study-wide criteria for all study participants were aged 18–44 years, fluent in English, not receiving orthodontic treatment, and not pregnant or nursing, and had negative responses to each of 10 questions about significant medical conditions and no history of facial injury or surgery. Cases and controls were not matched in this study. As reported by Slade, et al[74], cases were more likely to be female, non-Hispanic white, and were older than controls. All statistical case-control comparisons of psychosocial variables controlled for gender, ethnic group, and age (described below). Cases were also more highly educated than controls, but cases and controls were similar in socioeconomic variables.

This analysis uses data from all 185 recruited TMD cases and one half of the 3,263 recruited controls (1,633 people). The controls for this analysis were randomly selected so that data from participants in the reserved sample could be used for validation studies that will be reported elsewhere. The accompanying paper[74] gives a more detailed account of study recruitment, case-classification methods, and inclusion and exclusion criteria.

The OPPERA Study was reviewed and approved by institutional review boards at each of the four study sites and at the data coordinating center, Battelle Memorial Institute. All study participants verbally agreed to a screening interview done by telephone and they provided informed, written consent for all other study procedures. This study was conducted under the auspices of a Certificate of Confidentiality.

Psychosocial Instruments

For all instruments, participants had the option to complete the questionnaire via paper form or electronic PDF version. To distribute participant burden, some questionnaires were completed before the clinic visit (pre-clinic), while others were completed in-clinic immediately before the clinical examination. Missing questionnaire items were imputed using the expectation-maximization (EM) algorithm as described below. In general, if a subject skipped at least one but less than half the items in a questionnaire, the missing items were imputed. If they failed to complete at least half of the items in the questionnaire, we treated their summary score as missing. Information on missing values and imputation is provided in Supplementary e-Table 1.

Global Measures of Psychological Function

Eysenck Personality Questionnaire-Revised (Short Form, EPQ-R)—This 57-item true-false instrument assesses three personality dimensions: Extraversion, Neuroticism, and Psychoticism. The EPQ-R also includes a Lie scale, which reflects an effort to present oneself in the most positive light. Factor analysis of the EPQ-R supports the four scales, and

internal consistency of each of the scales is acceptable, with Cronbach's alpha ranging from 0.73 to 0.90.[28] Scales derived from the 57-item EPQ-R Short Form were found to correlate highly with scale scores from the original EPQ-R, with correlation coefficients ranging from 0.89 to 0.96 [27]. The EPQ has been widely used in previous research in chronic pain populations,[5,14,33,80] including TMD.[38,39]

Symptom Checklist 90-Revised (SCL-90R)—This 90-item instrument evaluates a broad range of psychological symptoms by having participants report the extent to which they have been bothered by each symptom on a five-category scale (not at all, a little bit, moderately, quite a bit, extremely). The SCL-90R provides measures of psychological distress across the following nine subscales: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism.[16] It also yields three Global Indices: the Global Severity Index (GSI), the Positive Symptom Distress Index (PSDI), and Positive Symptom Total (PST). This instrument has demonstrated good internal consistency (Cronbach's alpha for subscales ranging from 0.77 to 0.90) and test-retest reliability, which ranged from 0.78 to 0.90.[16] The SCL-90R has been widely used in research with numerous pain conditions,[2,12,62,64,73] including TMD.[37,41,59,82]

Measures of Affective Distress

State-Trait Anxiety Inventory (STAI)—The STAI includes two 20 item questionnaires, the State Anxiety Inventory and the Trait Anxiety Inventory.[77] For each item, participants are asked to indicate either how they "generally feel" (trait anxiety) or how they "feel right now" (state anxiety) using a four-category scale (not at all, somewhat, moderately so, extremely so). Test-retest reliability for the Trait Anxiety Scale has been adequate, ranging from 0.73 to 0.86 over intervals of 20 to 104 days.[77] As expected, State Anxiety is significantly less stable over time, given the transitory nature of anxiety states. Internal consistencies for both scales were high, with Cronbach's alphas of greater than 0.90.[77]

The Profile of Mood States-Bipolar (POMS-Bi)—This instrument consists of 72 mood-related items, and participants indicate the extent to which each item describes their mood state over the past week, including today using a four-category scale (much unlike this, slightly unlike this, slightly like this, much like this). This questionnaire assesses both positive and negative affective dimensions. It yields six subscale scores (Agreeable-Hostile, Elated-Depressed, Confident-Unsure, Energetic-Tired, Clearheaded-Confused, Composed-Anxious), as well as global indices of positive affect and negative affect. Given the state-like nature of the measure, test-retest reliabilities are understandably modest in magnitude, ranging from 0.33 to 0.72. The POMS has been well validated with other mood measures and is sensitive to subtle differences in affective state.[46]

Measures of Psychosocial Stress

The Perceived Stress Scale (PSS)—This 10-item scale assesses the perception of stress, that is, the degree to which an individual appraises situations as stressful [11]. For each item, participants indicate how often they felt or thought that way in the past month using a five-category scale (never, almost never, sometimes, fairly often, very often). The PSS yields a single overall perceived stress score by summing the numerical weights of each item, after reverse scoring four of the items. Internal consistency is good with Cronbach's alpha of 0.84 or greater, and construct validity has been demonstrated, as the PSS correlates significantly with other measures of stress appraisal.[11]

The Life Experiences Survey (LES)—This 57-item instrument assesses the frequency of life events that have occurred over the past year, as well as the impact of these events [72].

Impact ratings range from −3 (extremely negative) to +3 (extremely positive), with 0 indicating "no impact." There are multiple approaches to scoring the LES, which generally yield measures of frequency of positive, negative, and total events as well as positive, negative, and total impact of events. The test-retest reliability of positive impact was reported as low (0.10 and 0.53 across two samples), while reliabilities for negative and total impact were adequate, ranging from 0.56 to 0.88. Previous research indicated that scoring of impact based on individualized weights (i.e., each respondent's rating of impact) was the best predictor of psychological distress.[87] therefore, we report impact scores based on individualized weights in the included tables. For the LES, subjects who endorsed 40 or more items were excluded, on the grounds that they likely misunderstood the instructions for the questionnaire. Others were excluded from LES scoring if their answers were inappropriate based on their gender. (For example, male respondents who reported a pregnancy in the previous year were discarded.) This resulted in missing data for 240 participants (18 cases, 222 controls) on the LES. Frequency data and impact scores can be found in Supplementary e-Table 3.

The Lifetime Stressor List/PTSD Checklist-Civilian Version (LSL/PCL-C)—The LSL presents a checklist of 15 different traumatic events, and participants indicate which (if any) of these events they have experienced. For participants who endorse at least one item, they are then asked to identify the most significant stressor, and they complete the remaining 17-items regarding the extent to which they experience PTSD symptoms (e.g., repeated, disturbing memories of the experience) related to the selected traumatic event. Each item is endorsed on a five-category scale (not at all, a little bit, moderately, quite a bit, extremely). A total score is derived by summing the scores from each of the 17 symptoms. Test-retest reliability is high (0.96), and internal consistency ranged from 0.89 to 0.92.[84] In our sample, 912 controls and 83 cases did not endorse any traumatic events; therefore, PCL-C scores for these participants were set to missing.

Measures of Somatic Awareness

The Pennebaker Inventory of Limbic Languidness—(PILL) assesses the frequency with which individuals are bothered by each of 54 common physical symptoms and sensations on a five-category scale (never or almost never, less than 3 or 4 times a year, every month or so, every week or so, more than once every week). A single summary score is derived by summing each of the individual item responses, and PILL scores are related to the construct of somatic awareness or the general tendency to endorse physical symptoms. High internal consistency (alpha = 0.88) and adequate test-retest reliability (0.70 over two months)[63] have been reported. The PILL has been used as a measure of hypervigilance in fibromyalgia patients, who demonstrated higher scores on the PILL compared to arthritis patients and pain-free controls.[54]

The Kohn Reactivity Scale.[40]—This instrument consists of 24 items that assess an individual's level of reactivity or central nervous system arousability. Individuals respond to each item on a five-category scale ranging from "disagree strongly" to "agree strongly." The Kohn yields a single summary score created by summing all of the items, after reverse scoring half of the items. This measure has been reported to have adequate internal consistency, ranging from alpha of 0.73 to 0.83.[40] The Kohn has been shown to correlate negatively with pain tolerance [19] and it has been used as a measure of the construct of hypervigilance.[54]

Measures of Coping/Catastrophizing

The Coping Strategies Questionnaire-Revised (CSQ-R)—This CSQ-R is a revised version of the original CSQ,[71] which consists of 27 items relating to how individuals cope

with pain. Participants indicate the frequency with which they engage in specific coping activities when experiencing pain using a seven-category numerical scale, ranging from 0 (never do that) to 6 (always do that). It yields six subscales reflecting the pain coping strategies that individuals use: diverting attention, catastrophizing, praying and hoping, ignoring pain sensations, reinterpreting pain sensations, and coping self-statements. The subscales have shown adequate internal consistency, with Cronbach's alpha ranging from 0.72 – 0.91 in a sample of healthy young adults.[32] The CSQ-R has been shown to have stable factor structure in patients with chronic pain [69] and in healthy populations.[32] Because the catastrophizing scale from the CSQ-R is identical to the Helplessness scale from the PCS (see below), the CSQ-R's catastrophizing scale was excluded from all analyses.

The Pain Catastrophizing Scale (PCS)—This instrument consists of 13 items rated on a 5-point scale ranging from 0 (not at all) to 4 (all the time). Participants indicate the degree to which they have specified thoughts and feelings when experiencing pain. The measure assesses three dimensions of catastrophizing: Rumination, Magnification, and Helplessness, and a total score is calculated by summing the three subscales. Internal consistency has been shown to be adequate to very good, with coefficient alphas ranging from 0.66 to 0.87 across subscales.[78] The PCS has been validated for both clinical and nonclinical samples.[60,78]

Additional Measures

The Pittsburgh Sleep Quality Index (PSQI)—The PSQI is the most commonly administered self-report sleep measure. This 19-item instrument assesses sleep quality during the previous month across the following domains: Subjective Sleep Quality, Sleep Latency, Sleep Duration, Habitual Sleep Efficiency, Sleep Disturbances, Use of Sleep Medications, and Daytime Dysfunction.[7] Internal consistency was found to be high, with Cronbach's alpha of 0.83, and test-retest reliability for Global PSQI scores was 0.85 [7]. The PSQI has been used in previous research to identify sleep disturbance associated with TMD.[15,86] For the PSQI, errors in subjects' reporting of time values were corrected by algorithm (e.g. when 12:00 AM was confused with 12:00 PM or reporting 40:00 PM instead of 4:00 PM). For subjects who reported hours of sleep but not minutes, the value for minutes was set to 0.

The Medical Outcomes Study Short Form 12 Item Version (SF-12)—The SF-12 is a briefer measure of health-related quality of life derived by using 12 items from the original SF-36 instrument.[83] The SF-12 yields two scale scores, A Mental Health Composite Score (MCS) and a Physical Health Composite Score (PCS), which correlate highly with the MCS and PCS scores from the original SF-36, and test-retest reliability for the PCS and MCS measures ranged from 0.76 to 0.89. [83] The SF-12 has been found to distinguish patients with chronic pain conditions from healthy controls.[45,52]

Data Analysis

For the instruments with reversed scored items (KOHN, PSS, POMS-Bi, STAI), responses for the subjects who endorsed all minimum or all maximum values were considered invalid and set to missing. Imputation for missing values used the EM method in SAS proc MI, which finds maximum likelihood estimates for incomplete data under the assumption that the data are multivariate normal. Descriptive statistics for each summary score were generated using non-imputed data. Statistical significance of differences in mean scores was evaluated using the t-test derived from a least squares linear model in which study site was a covariate. Study site was used as a covariate because operational requirements during recruitment created different proportions of cases among sites (see Slade, et al.[74] for further detail). The relationship between each summary score and occurrence of TMD was expressed as the standardized odds ratio, calculated from an unconditional, binary logistic

regression model with study site as a covariate. To achieve this, the summary score was transformed to a unit-normal deviate. The transformation meant that odds ratios could be interpreted as the relative change in odds of TMD for each standard deviation of change in the summary score. A second logistic regression model generated a fully-adjusted estimate of the relationship, using additional covariates of age (in years) gender and race/ethnicity (dichotomized as white or non-white). A third logistic regression model using this imputed dataset calculated standardized odds ratios for each summary score, with adjustment for the same covariates as in the prior model.

All P-values were computed without adjustment for multiple tests, and we therefore refrain from nominating P=0.05 as a threshold for statistical significance. In this paper's case-control analysis, 21 psychosocial characteristics were investigated and, therefore, Bonferroni correction for the probability of type I error would yield a critical P value of 0.05/21 = 0.002. Using the same rationale, rejection of the null hypothesis concerning odds ratios would occur only if the 99.8% confidence interval excluded the null value of one. In general, though, we avoid drawing conclusions about statistical significance of associations, even with correction for multiple tests, because these papers report only univariate- or demographically-adjusted results. Furthermore, the Bonferroni adjustment is probably overly-conservative in this setting, where several measures are moderately correlated. Instead, we will reserve judgments about statistical significance to subsequent papers that will use multivariable modeling to consider multiple characteristics simultaneously, as proposed in the OPPERA heuristic model.[49]

Principal component analysis (PCA) was applied to this data set to identify putative latent variables, which potentially could serve in the future as a reduced set of variables to be exploited in multivariate analyses. The approach began with four steps, widely used in exploratory principal component analysis:[65] (1) variable selection; (2) evaluation of the correlation matrix; (3) extraction of initial components; and (4) rotation and interpretation of component loadings. This model is being used for purely exploratory purposes with regards to the structure of the latent variables and not for computation of summary scores for the various components.

Our primary interest focused on PCA loadings in controls for three reasons: (a) there are many more controls than cases, thereby improving statistical power to identify factors and estimate loadings; (b) our underlying conceptual model of TMD[49] proposes that relationships between putative risk factors might alter following onset of chronic pain, and we wanted to evaluate that possibility; and (c) the long-term goal of OPPERA is to identify risk factors for TMD among controls, so it is desirable to identify a reduced set of variables for that group. Thus, we fit separate PCA models for the TMD cases and controls.

Variables were selected for this analysis based primarily on conceptual grounds. Specifically, those measures that have been previously associated with TMD in case-control studies and were specified *a priori* in the OPPERA conceptual model[49] were included in the PCA. On this basis, the PSQI and SF-12 were excluded. Due to high levels of missingness (as described above), the LES and LSL/PCL-C were excluded. The variables included in the model are shown in Table 4. We elected to keep all 21 variables in the model even though some of the variables were highly correlated with one another. We wanted to see which measures could be combined into latent variables, and this required that all the variables be retained in the exploratory model. Although this has the potential to increase the variance of our estimates of the PCA loadings, this was not a major concern given our large sample size. As shown below, we estimated the variance of our PCA loadings using bootstrapping, and these variances were uniformly low despite the correlations among the input variables.

After imputing missing values as described above, 50 subjects still had at least one missing value among the variables included in the model. To retain these subjects in the model, we performed a second round of imputation. Specifically, we imputed any missing scale scores based on the observed scale scores from the remaining instruments for each participant. (We used the EM algorithm to perform this imputation under the assumption that the data are multivariate normal, as described previously.) After performing this second round of imputation, there were no remaining subjects with missing data.

PCA models were fit using the R statistical computing platform. All variables were normalized to have mean 0 and standard deviation 1 prior to fitting the models. Eigenvalues from the initial, unrotated solution were inspected using a scree plot. To help determine the optimal number of eigenvectors, a parallel analysis was performed to determine the number of components that exceeded what would be expected due to chance alone. Parallel analysis estimates the number of components to include in a PCA model by generating random data sets with the same numbers of observations and predictor variables as the original data.[81] The eigenvalues are computed for each random data set and averaged over all the data sets. When the average eigenvalue from these randomly generated data sets is larger than the corresponding eigenvalue of the original data, then the principal component associated with that eigenvalue is likely to be random noise.

After calculating the PCA eigenvectors, a promax rotation was applied to increase the interpretability of the resulting PCA loadings. The promax rotation produced loadings that were easier to interpret than the loadings resulting from orthogonal rotations, and other nonorthogonal rotations produced similar results. The rotated loadings are presented unless otherwise noted. The variance in the PCA loadings of each model was estimated by drawing 1,000 bootstrap samples for each data set and fitting a PCA model for each replicate. The 95% confidence bounds for the PCA loadings were estimated to be the 2.5% and 97.5% quantiles of the corresponding loading over the 1,000 bootstrap replicates.[24]

Results

The overall goal of this manuscript (and the others in this compendium) is to present the OPPERA methodology and to limit results in this initial report to general descriptive findings from the baseline data collection phase. We believe the univariate data analytic approach presented below accomplishes this most efficiently.

Case-Control Differences

Global Measures of Psychological Function (see Table 1 and Supplementary e-Table 2)—As shown in Table 1, TMD cases had higher mean scores than controls across all SCL-90R subscales of primary interest in this analysis: depression, somatization, hostility, and anxiety. Mean scale scores in cases were almost two times the corresponding mean for controls, and standardized odds ratios ranged from 1.4 for the Anxiety and Hostility scales to 2.1 for the Somatization scale. Odds ratios and their 95% confidence intervals were similar with and without adjustment for demographic characteristics, and using imputed versus non-imputed data.

Regarding personality as measured by the EPQ-R, TMD cases showed higher levels of neuroticism, on average, than controls, but no group differences in extraversion emerged. The standardized odds ratio was 1.6 for Neuroticism, this value decreasing slightly to 1.5 after adjustment for demographic characteristics and to 1.4 using imputed data. The 95% confidence intervals did not differ appreciably with or without adjustment for demographic characteristics or using imputed data compared to the non-imputed data.

Measures of Affective Distress (see Table 2 and Supplementary e-Table 3)— Compared to controls, TMD cases reported higher mean levels of both state and trait anxiety on the STAI. For both state and trait anxiety, standardized odds ratios were similar after adjustment and when using imputed data rather than non-imputed data. On the POMS-Bi, TMD cases reported higher Negative Affect and lower Positive Affect than controls. Standardized odds ratios were 0.8 for Overall Positive Affect Score and 1.5 for Overall Negative Affect Score. Odds ratios and their 95% confidence intervals were similar with and without adjustment for demographic characteristics and when using imputed data.

Measures of Psychosocial Stress (see Table 2 and Supplementary e-Table 3) —Relative to controls, TMD cases reported higher mean levels of perceived stress on the PSS. Standardized odds ratios were similar with or without adjustment for demographic characteristics, and when using imputed data versus non-imputed data. Data for the LES and the LSL/PCL-C can be found in Supplementary e-Table 3.

Measures of Somatic Awareness (see Table 3 and Supplementary e-Table 4)— PILL and Kohn scores, reflecting components of somatic awareness, were significantly higher, on average, for TMD cases than controls. For PILL scores, standardized odds ratios indicated a large difference between the groups (SOR=2.5), and the standardized odds ratios were generally unaffected by adjustment for demographic characteristics or by use of imputed versus non-imputed data. For Kohn scores, standardized odds ratios indicated moderate group differences, and odds ratios were slightly lower after adjustment for demographic characteristics and when using imputed rather than non-imputed data.

Measures of Coping/Catastrophizing (see Table 3 and Supplementary e-Table 4)—On the PCS, TMD cases had higher mean levels of catastrophizing than controls. This was true for all three subscales (Rumination, Magnification, Helplessness) and standardized odds ratios were similar across subscales, 1.5 for Magnification and Rumination and 1.6 for Helplessness. The odds ratios and their 95% confidence intervals were similar with or without adjustment for demographic characteristics and using non-imputed data or imputed data.

For other measures of pain coping, assessed using the CSQ-R, small but significant case-control differences emerged for Distraction and Praying & Hoping, with controls providing higher values for both measures. Standardized odds ratios and their 95% confidence intervals were unaffected by adjustment for demographic characteristics or by using imputed data compared to the non-imputed data. No significant case-control differences emerged for other CSQ-R scales.

Additional Measures (see Supplementary e-Table 5)—TMD patients on average reported poorer overall sleep quality on the PSQI compared to controls. Also, on the SF-12, Physical Component scores and Mental Component Scores were both lower (indicating poorer function) for cases versus controls.

Associations with Demographic Variables (Controls only)

Global Measures of Psychological Function (see Supplementary e-Tables 6, 7 & 8)—As shown in Supplementary e-Tables 6, 7, and 8, female controls reported higher mean scores on the SCL90R Depression and Somatization subscales; however, the effect sizes for these gender differences were small. Mean Somatization scores were slightly and significantly higher for the oldest participants. Significant ethnic group differences emerged for Depression, Anxiety and Hostility, with higher mean values among the non-white group. Regarding the EPQ-R, females reported greater mean levels of neuroticism than males, a

difference of moderate effect size. Age groups differed in extraversion, with lower extraversion associated with increasing age. Also, whites reported higher levels of extraversion than non-whites.

Measures of Affective Distress (see Supplementary e-Tables 9, 10 & 11)—No demographic group differences emerged for State Anxiety. For State and Trait Anxiety, non-whites reported higher mean values than whites, and age was associated with Trait Anxiety, such that the older group reported higher average scores. On the POMS-Bi, ethnic group was associated with Positive Affect, such that whites higher scores than non-whites, though the magnitude of the difference was small. A small but significant gender difference in Negative Affect emerged, with women reporting greater values, on average.

Measures of Psychosocial Stress (see Supplementary e-Tables 9, 10 & 11)—On the PSS, women reported higher mean levels of perceived stress than men, and non-whites reported greater scores, on average, than whites. Age differences in PSS scores were not significant.

Measures of Somatic Awareness (see Supplementary e-Tables 12, 13 & 14)—Age, gender, and ethnic group differences in PILL scores were found, such that younger participants, women, and non-Hispanic whites endorsed greater mean levels of somatic symptomatology. These group differences were small in magnitude. Regarding Kohn scores, significant age group, gender, and ethnic group differences emerged. Kohn scores were greater with increasing age. Females reported higher scores than males, and non-whites had greater values than whites.

Measures of Coping/Catastrophizing (see Supplementary e-Tables 12, A13 & A14)—On the PCS, women reported higher mean scores than men for two of the subscales, Rumination and Helplessness. Non-whites also reported higher mean levels on all three PCS subscales than whites, though the magnitude of these mean differences was small. Age differences in Helplessness scores emerged, with the oldest group reporting the highest mean values. For other measures of pain coping, assessed using the CSQ-R, gender differences were significant across most subscales. On average, women reported greater use of Praying & Hoping, while men reported higher scores on Coping Statements, Ignoring Pain Sensations and Reinterpreting Pain Sensations. Regarding ethnic group differences, non-whites reported significantly greater use of Praying & Hoping, Distraction, and Reinterpreting Pain Sensations, but lower mean values for Ignoring Pain. Age group differences emerged for Praying & Hoping and Distraction, mean scores increasing with greater age, and for Coping Statements, with lower mean scores in the oldest age group.

Principal Component Analysis—Figure 1 shows a scree plot of the PCA model for controls. The variance explained by each component decreases to less than 10% after the first four components. The parallel analysis showed strong evidence that components one through four were above the chance line (See Figure 1). The confidence intervals for the loadings of the first four components were generally very narrow, indicating a stable model. While the five-component model also showed relatively narrow confidence intervals, the four-component model appeared optimal based on the scree plot, the parallel analysis, and on conceptual grounds. Thus, we report a model based on four components. The loadings for the PCA model for controls are shown in Table 4, and the 95% confidence intervals for the loadings are shown in Supplementary e-Table 15. The first component, accounting for 19% of the variance, labeled Stress and Negative Affectivity, includes high loadings from both State and Trait Anxiety, Perceived Stress, and POMS Negative Affect and negative loadings for POMS Positive Affect and the EPQ-R extraversion Scale. Also the EPQ-R Neuroticism

scale loads positively on this component. The second component, labeled Global Psychological Symptoms, based on high loadings from all SCL-90-R scales, as well as the PILL, accounted for an additional 16% of the variance. The third component, Passive Pain Coping, accounted for an additional 14% of the variance. All three PCS subscales loaded highly on this factor, as did the Praying and Hoping subscale of the CSQ-R. The Kohn score loaded modestly on this component. The fourth component is labeled Active Pain Coping and includes positive loadings from the remaining CSQ-R subscales. This final component accounted for an additional 12% of the variance.

The loadings and 95% confidence intervals for the PCA for TMD cases are shown in Supplementary e-Tables 16 and 17. These confidence intervals are much wider than observed for controls. Indeed, with the exception of the loadings for the first component, essentially all of the loadings for the PCA in cases have very wide confidence intervals. This indicates that (with the exception of component one), this factor structure may not be reproducible. The higher variance in the loadings of the cases PCA is probably due to its much smaller sample size. The first component, accounting for 32% of the variance, reflects Global Psychological Distress, based on positive loadings from all SCL-90-R scales, all measures of affective distress (e.g. State & Trait Anxiety, POMS) as well as the PILL, Perceived Stress Scale and the EPQ-R Neuroticism scale. The second component accounted for an additional 16% of the variance and reflected Passive Coping and Reactivity. This component included positive loadings from all three PCS subscales as well as the Praying and Hoping subscale of the CSQ-R. Also, the Kohn score, a measure of reactivity to somatic experiences, loaded positively on this component. The third component, Active Pain Coping, accounted for an additional 10% of the variance and includes positive loadings from the remaining CSQ-R subscales. The final component accounted for an additional 6% of the variance, with positive loadings from POMS Positive Affect as well as the EPQ-R Extraversion Scale. This component may reflect Psychological Resilience.

We calculated Cronbach's alpha for each component (using items with a loading of 0.5 or higher). The values of alpha ranged from 0.54 to 0.87 for the PCA in controls and from 0.45 to 0.93 for the PCA in cases.

Discussion

The findings above present the baseline psychosocial data from a large cohort of TMD-free controls compared to a smaller group of individuals meeting research diagnostic criteria for chronic TMD (i.e., cases). Significant case-control differences emerged across multiple psychosocial content domains, such that TMD cases reported higher levels of psychological and affective distress, greater perceived stress and catastrophizing, and increased somatic awareness compared to controls. The case-control differences of greatest magnitude emerged for measures of somatic awareness (i.e., PILL, SCL-90R Somatization), with standardized odds ratios exceeding 2.0. In general, case-control differences for other psychosocial variables were of more modest magnitude. These findings are consistent with previous results of case-control studies examining psychosocial variables among individuals with TMD[8–10,13,29,47,48,50,51,61,67], as well as in studies of people with other types of chronic pain.[20,31,35] Less well-documented are the differences in pain coping that were observed in this sample. Specifically, consistent with recent evidence[8,67] TMD cases reported higher levels of catastrophizing, a form of maladaptive cognitive appraisal that has been associated with increased pain and disability across multiple chronic pain conditions.[22,79] Surprisingly, controls reported greater use of praying and hoping, which is typically construed as a passive coping strategy, as supported by its loading with catastrophizing in our PCA. It should be noted that the magnitude of this difference was quite small, and the practical significance of this finding requires further investigation.

From a methodological perspective, it is notable that cases and controls in the present study were recruited from the same communities using similar recruitment approaches. This avoids the sampling biases that can occur when cases are recruited from clinical settings and controls are recruited from non-clinical sources. Such biases tend to inflate case-control differences in psychological functioning, as many of these psychosocial variables are associated with health care seeking in chronic pain populations.[36,85] This may also explain the modest magnitude of many of our case-control differences in psychological functioning, as compared to what might be expected when contrasting community-based controls to clinic cases.

Within controls, the association of demographic factors with psychosocial variables was examined independently and without adjustment for other factors. In general, age-related differences were small in magnitude and failed to follow a consistent pattern, perhaps owing to the relatively restricted age range in our cohort. However, gender differences emerged for multiple variables, with women reporting higher levels of psychological distress across several SCL90-R scales, along with greater neuroticism, perceived stress, negative affect, and somatic awareness. Women also reported greater use of catastrophizing, distraction and praying & hoping, while men reported higher scores on coping self statements, and ignoring and reinterpreting pain sensations. These gender differences are consistent with previously published results,[3,4,23,76] and the increased expression of these potential psychosocial risk factors may contribute to the greater prevalence of TMD among women.[42] Ethnic group differences were also observed for several psychosocial measures, such that non-whites reported higher psychological distress across all examined SCL90R subscales. Consistent with previous findings[32], ethnic differences in pain coping emerged, with significantly greater use of catastrophizing, praying & hoping, distraction, and reinterpreting pain sensations among non-whites. Regarding somatic awareness, ethnic differences emerged for both the PILL and the Kohn, similar to previous results.[68]

That psychosocial factors differed significantly between cases and controls raises the possibility that such psychosocial variables may represent predisposing risk factors for the development of chronic pain. Of course, our present cross-sectional analysis cannot determine the direction of the association; however, a limited literature has addressed whether premorbid psychosocial factors are associated with subsequent development of TMD. Members of this research group have reported that premorbid measures of depression, perceived stress, and mood state obtained at baseline were significant predictors of new onset TMD in a sample of healthy young females.[75] Also, Aggarwal and colleagues[1] found that preexisting health anxiety predicted future development of chronic orofacial pain. Similar findings supporting psychosocial variables as premorbid risk factors for development of chronic pain have been reported for other chronic pain conditions, including chronic widespread pain [53], regional musculoskeletal pain,[57] and low back pain.[43,44,57] While we are not able to address this issue in the present analysis of our baseline data, the design of the OPPERA study will permit such analyses at the conclusion of the follow-up period. For example, Aggarwal and colleagues reported that four psychosocial measures distinguished cases from controls in univariate analyses; however, only health anxiety remained a significant predictor in the multivariate approach.[1] Therefore, our future analyses to identify risk factors for new onset TMD will include multivariate approaches that concurrently take into account multiple psychosocial measures, as well as potential predictors from other domains.

Principal Component Analysis was implemented as an approach to operationalizing the psychosocial component of the OPPERA heuristic model[17] PCA revealed multiple latent constructs based on the extensive battery of psychosocial measures. Among controls, the four components reflected: (1) stress, anxiety, and neuroticism; (2) global psychological

symptoms; (3) passive pain coping; and (4) active pain coping. The model was quite stable, and the underlying constructs may reflect important sub-domains of the "enhanced psychological distress" intermediate phenotype previously proposed in the OPPERA conceptual model.[49] Specifically, the PCA model indicates the presence of multiple aspects of psychosocial function, which go beyond the unitary concept of "psychological distress." Indeed, the first two components seem to suggest that there are two different aspects of "psychological distress," one comprised primarily of personality characteristics and affective distress (including anxiety and stress), and the other comprised of more global psychological adjustment, including both somatic and psychological symptoms. That state and trait anxiety load on the first component, while SCL90R Anxiety loads on the second component is likely due to method variance, given that all of the other SCL90R scales also loaded on component two. The last two components from the PCA model go beyond "distress" and reflect different aspects of pain coping. In contrast to the psychological symptoms reflected by the first two components, these coping components represent cognitive and behavioral processes that may modulate the experience of pain and associated psychological distress. Thus, the multiple constructs reflected by these components may reflect important psychosocial domains for identifying individuals at risk for new onset TMD pain. Among TMD cases, the underlying latent constructs identified in the present study included: (1) global psychological distress; (2) passive pain coping and reactivity; (3) active pain coping; and (4) psychological resilience. Given the difference in constructs identified within the controls versus the cases, it is tempting to speculate that the underlying structure of psychosocial constructs, as reflected by the component measures used in this study, differs for individuals with chronic pain compared to those without. However, given the instability of the PCA model in TMD cases, any potential case-control differences should be interpreted with caution. Also, the latent variables identified by our PCA should be viewed as exploratory, and further confirmation of these results will be needed. A logical next step would be to determine the replicability of these findings in another sample. Successful replication would increase confidence in the validity of the latent constructs reflected by these components, such that indices representing these underlying constructs could then be derived and used in future analyses designed to identify risk factors for new onset TMD.

The limitations of this study deserve mention. First, relative to our large control population, the sample size for cases is quite modest, which contributed to the instability of the PCA model in cases and limited our ability to compare PCA models for cases versus controls. Second, the analyses herein are restricted to the psychosocial data and do not examine potentially important associations between psychosocial variables and other phenotypic measures, such as those presented in other manuscripts in this volume. However, such analyses are planned for future manuscripts. Third, given the large number of analyses already presented, only univariate demographic comparisons were conducted, and the pattern of findings from psychosocial variables may change when multiple psychosocial measures are simultaneously considered using a multivariate approach. In future analyses, it also will be informative to examine psychosocial measures stratified across multiple demographic variables (e.g., age differences within each gender). Moreover, our analysis of ethnic differences only considered whites versus non-whites, and future investigation of different ethnic subgroups is warranted.

These limitations notwithstanding, the current findings demonstrate case-control differences across multiple domains of psychosocial functioning, with the most robust differences emerging for indices of somatic awareness. Moreover, our large sample of controls provided statistical power for demographic comparisons, which revealed gender and ethnic group differences for multiple psychosocial variables. Similar to recently reported findings from healthy subjects,[56] our PCA suggests that these multiple psychosocial measures can be

reduced to a smaller subset of latent constructs. Future analyses will examine associations of these psychosocial variables with other important phenotypes, such as quantitative sensory testing data and clinical examination findings. Further, genetic associations with psychosocial variables will be explored in future work. Finally, the ultimate goal of collecting these psychosocial measures is to determine their ability to predict new onset of TMD, and these analyses will be conducted upon completion of the follow-up phase of the OPPERA Study.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Reference List

1. Aggarwal VR, Macfarlane GJ, Farragher TM, McBeth J. Risk factors for onset of chronic oro-facial pain--results of the North Cheshire oro-facial pain prospective population study. Pain. 2010; 149:354–9. [PubMed: 20304556]
2. Almansa C, Rey E, Sanchez RG, Sanchez AA, Diaz-Rubio M. Prevalence of functional gastrointestinal disorders in patients with fibromyalgia and the role of psychologic distress. Clin Gastroenterol Hepatol. 2009; 7:438–45. [PubMed: 19138763]
3. Barsky AJ, Peekna HM, Borus JF. Somatic symptom reporting in women and men. J Gen Intern Med. 2001; 16:266–75. [PubMed: 11318929]
4. Becker JB, Monteggia LM, Perrot-Sinal TS, Romeo RD, Taylor JR, Yehuda R, Bale TL. Stress and disease: is being female a predisposing factor? J Neurosci. 2007; 27:11851–5. [PubMed: 17978023]
5. Boyce PM, Koloski NA, Talley NJ. Irritable bowel syndrome according to varying diagnostic criteria: are the new Rome II criteria unnecessarily restrictive for research and practice? Am J Gastroenterol. 2000; 95:3176–83. [PubMed: 11095338]
6. Burris JL, Cyders MA, De LR, Smith GT, Carlson CR. Posttraumatic stress disorder symptoms and chronic orofacial pain: an empirical examination of the mutual maintenance model. J Orofac Pain. 2009; 23:243–52. [PubMed: 19639104]
7. Buysse DJ, Reynolds CF III, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res. 1989; 28:193–213. [PubMed: 2748771]
8. Campbell CM, Kronfli T, Buenaver LF, Smith MT, Berna C, Haythornthwaite JA, Edwards RR. Situational versus dispositional measurement of catastrophizing: associations with pain responses in multiple samples. J Pain. 2010; 11:443–53. [PubMed: 20439057]
9. Carlson CR. Psychological considerations for chronic orofacial pain. Oral Maxillofac Surg Clin North Am. 2008; 20:185–95. vi. [PubMed: 18343324]
10. Carlson CR, Okeson JP, Falace DA, Nitz AJ, Curran SL, Anderson D. Comparison of psychologic and physiologic functioning between patients with masticatory muscle pain and matched controls. J Orofacial Pain. 1993; 7:15–22. [PubMed: 8467294]
11. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. J Health Soc Behav. 1983; 24:385–96. [PubMed: 6668417]
12. Cook AJ, Chastain DC. The classification of patients with chronic pain: age and sex differences. Pain Res Manag. 2001; 6:142–51. [PubMed: 11854777]
13. de Leeuw R, Bertoli E, Schmidt JE, Carlson CR. Prevalence of traumatic stressors in patients with temporomandibular disorders. J Oral Maxillofac Surg. 2005; 63:42–50. [PubMed: 15635556]

14. de DP, Del BE, Gori-Savellini S, Manzoni GC, Martucci N, Nappi G, Savoldi F. Personality patterns of headache sufferers. Cephalalgia. 1983; 3(Suppl 1):195–214. 195–214. [PubMed: 6616603]
15. De LR, Studts JL, Carlson CR. Fatigue and fatigue-related symptoms in an orofacial pain population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005; 99:168–74. [PubMed: 15660087]
16. Derogatis, L. The SCL-90-R. administration, scoring and procedures manual. Minneapolis, MN: National Computer Systems, Inc; 1994.
17. Diatchenko L, Nackley AG, Slade GD, Fillingim RB, Maixner W. Idiopathic pain disorders--pathways of vulnerability. Pain. 2006; 123:226–30. [PubMed: 16777329]
18. Drangsholt, M.; LeResche, L. Temporomandibular disorder pain. In: Crombie, IK.; Croft, PR.; Linton, SJ.; LeResche, L.; Von Korff, M., editors. Epidemiology of Pain. Seattle: IASP Press; 1999. p. 203-33.
19. Dubreuil DL, Kohn PM. Reactivity and response to pain. Pers Indiv Diff. 1986; 7:907–9.
20. Dworkin, RH.; Breitbart, WS. Psychosocial aspects of pain: a handbook for health care providers. Seattle: IASP Press; 2004.
21. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders. J Craniomandib Disord. 1992; 6:302–55.
22. Edwards RR, Bingham CO III, Bathon J, Haythornthwaite JA. Catastrophizing and pain in arthritis, fibromyalgia, and other rheumatic diseases. Arthritis Rheum. 2006; 55:325–32. [PubMed: 16583384]
23. Edwards RR, Haythornthwaite JA, Sullivan MJ, Fillingim RB. Catastrophizing as a mediator of sex differences in pain: differential effects for daily pain versus laboratory-induced pain. Pain. 2004; 111:335–41. [PubMed: 15363877]
24. Efron, B.; Tibshirani, R. An introduction to bootstrap. Boca Raton, FL: Chapman & Hall/CRC; 1994.
25. Eli I. Psychosocial factors in the etiology, diagnosis and management of temporomandibular disorders. Alpha Omegan. 2003; 96:20–3. [PubMed: 12955778]
26. Epker J, Gatchel RJ. Coping profile differences in the biopsychosocial functioning of patients with temporomandibular disorder. Psychosom Med. 2000; 62:69–75. [PubMed: 10705913]
27. Eysenck, HJ.; Eysenck, SBG. Manual of the ysenck Personality Questionnaire. San Diego, CA: Educational and Industrial Testing Service; 1994.
28. Eysenck SBG, Eysenck HJ, Barrett P. A revised version of the psychoticism scale. Personality and Indivdual Differences. 1985; 6:21–9.
29. Ferrando M, Andreu Y, GaldOn MJ, Dura E, Poveda R, Bagan JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004; 98:153–60. [PubMed: 15316541]
30. Garofalo JP, Gatchel RJ, Wesley AL, Ellis E. Predicting chronicity in acute temporomandibular joint disorders using the research diagnostic criteria. Journal of the American Dental Association. 1998; 129:438–47. [PubMed: 9573694]
31. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull. 2007; 133:581–624. [PubMed: 17592957]
32. Hastie BA, Riley JL III, Fillingim RB. Ethnic differences in pain coping: factor structure of the coping strategies questionnaire and coping strategies questionnaire-revised. J Pain. 2004; 5:304–16. [PubMed: 15336635]
33. Hazlett-Stevens H, Craske MG, Mayer EA, Chang L, Naliboff BD. Prevalence of irritable bowel syndrome among university students. The roles of worry, neuroticism, anxiety sensitivity and visceral anxiety. J Psychosom Res. 2003; 55:501–5. [PubMed: 14642979]
34. Isong U, Gansky SA, Plesh O. Temporomandibular joint and muscle disorder-type pain in U.S. adults: the National Health Interview Survey. J Orofac Pain. 2008; 22:317–22. [PubMed: 19090404]
35. Keefe FJ, Rumble ME, Scipio CD, Giordano LA, Perri LM. Psychological aspects of persistent pain: current state of the science. J Pain. 2004; 5:195–211. [PubMed: 15162342]

36. Kersh BC, Bradley LA, Alarcon GS, Alberts KR, Sotolongo A, Martin MY, Aaron LA, Dewaal DF, Domino ML, Chaplin WF, Palardy NR, Cianfrini LR, Triana-Alexander M. Psychosocial and health status variables independently predict health care seeking in fibromyalgia. Arthritis Rheum. 2001; 45:362–71. [PubMed: 11501724]
37. Kim HI, Lee JY, Kim YK, Kho HS. Clinical and psychological characteristics of TMD patients with trauma history. Oral Dis. 2010; 16:188–92. [PubMed: 19758407]
38. Kino K, Sugisaki M, Haketa T, Amemori Y, Ishikawa T, Shibuya T, Sato F, Amagasa T, Shibuya T, Tanabe H, Yoda T, Sakamoto I, Omura K, Miyaoka H. The comparison between pains, difficulties in function, and associating factors of patients in subtypes of temporomandibular disorders. J Oral Rehabil. 2005; 32:315–25. [PubMed: 15842238]
39. Kino K, Sugisaki M, Ishikawa T, Shibuya T, Amagasa T, Miyaoka H. Preliminary psychologic survey of orofacial outpatients. Part 1: Predictors of anxiety or depression. J Orofac Pain. 2001; 15:235–44. [PubMed: 11575194]
40. Kohn, PM. Sensation-seeking, augmenting-reducing, and strength of the nervous system. In: Spence, JT.; Izard, DE., editors. Motivation, Emotion, and Personality. Amsterdam: Elsevier; 1985. p. 167-73.
41. Krogstad BS, Jokstad A, Dahl BL, Soboleva U. Somatic complaints, psychologic distress, and treatment outcome in two groups of TMD patients, one previously subjected to whiplash injury. J Orofac Pain. 1998; 12:136–44. [PubMed: 9656891]
42. LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. Critical Reviews in Oral Biology & Medicine. 1997; 8:291–305. [PubMed: 9260045]
43. Linton SJ. A review of psychological risk factors in back and neck pain. Spine. 2000; 25:1148–56. [PubMed: 10788861]
44. Linton SJ. Do psychological factors increase the risk for back pain in the general population in both a cross-sectional and prospective analysis? Eur J Pain. 2005; 9:355–61. [PubMed: 15979015]
45. Lipton RB, Hamelsky SW, Kolodner KB, Steiner TJ, Stewart WF. Migraine, quality of life, and depression: a population-based case-control study. Neurology. 2000; 55:629–35. [PubMed: 10980724]
46. Lorr, M.; McNair, DM. Profile of Mood States: Bipolar Form. San Diego: Educational and Industrial Testing Service; 1988.
47. Macfarlane TV, Blinkhorn AS, Davies RM, Ryan P, Worthington HV, Macfarlane GJ. Orofacial pain: just another chronic pain? Results from a population-based survey. Pain. 2002; 99:453–8. [PubMed: 12406520]
48. Macfarlane TV, Kenealy P, Kingdon HA, Mohlin B, Pilley JR, Mwangi CW, Hunter L, Richmond S, Shaw WC. Orofacial pain in young adults and associated childhood and adulthood factors: results of the population study, Wales, United Kingdom. Community Dent Oral Epidemiol. 2009; 37:438–50. [PubMed: 19624699]
49. Maixner W, Diatchenko L, Dubner R, Fillingim RB, Greenspan JD, Knott C, Ohrbach R, Weir B, Glade GD. Orofacial pain prospective evaluation and risk assessment study - the OPPERA study. J Pain Submitted. 2011; XX:XXX–XXX.
50. Manfredini D, Landi N, Bandettini di PA, Dell'Osso L, Bosco M. A critical review on the importance of psychological factors in temporomandibular disorders. Minerva Stomatol. 2003; 52:321–30. [PubMed: 12874536]
51. Manfredini D, Winocur E, Ahlberg J, Guarda-Nardini L, Lobbezoo F. Psychosocial impairment in temporomandibular disorders patients. RDC/TMD axis II findings from a multicentre study. J Dent. 2010; 38:765–72. [PubMed: 20600559]
52. Mas AJ, Carmona L, Valverde M, Ribas B. Prevalence and impact of fibromyalgia on function and quality of life in individuals from the general population: results from a nationwide study in Spain. Clin Exp Rheumatol. 2008; 26:519–26. [PubMed: 18799079]
53. McBeth J, Macfarlane GJ, Benjamin S, Silman AJ. Features of somatization predict the onset of chronic widespread pain: results of a large population-based study. Arthritis Rheum. 2001; 44:940–6. [PubMed: 11315933]

54. McDermid AJ, Rollman GB, McCain GA. Generalized hypervigilance in fibromyalgia: evidence of perceptual amplification. Pain. 1996; 66:133–44. [PubMed: 8880834]
55. Moayedi M, Weissman-Fogel I, Crawley AP, Goldberg MB, Freeman BV, Tenenbaum HC, Davis KD. Contribution of chronic pain and neuroticism to abnormal forebrain gray matter in patients with temporomandibular disorder. Neuroimage. 2011; 55:277–86. [PubMed: 21156210]
56. Mounce C, Keogh E, Eccleston C. A principal components analysis of negative affect-related constructs relevant to pain: evidence for a three component structure. J Pain. 2010; 11:710–7. [PubMed: 20015698]
57. Nahit ES, Pritchard CM, Cherry NM, Silman AJ, Macfarlane GJ. The influence of work related psychosocial factors and psychological distress on regional musculoskeletal pain: a study of newly employed workers. J Rheumatol. 2001; 28:1378–84. [PubMed: 11409134]
58. Ohrbach R, Dworkin SF. Five-year outcomes in TMD. relationship of changes in pain to changes in physical and psychological variables. Pain. 1998; 74:315–26. [PubMed: 9520246]
59. Ohrbach R, Sherman J, Beneduce C, Zittel-Palamara K, Pak Y. Extraction of RDC/TMD subscales from the symptom check list-90: does context alter respondent behavior? J Orofac Pain. 2008; 22:331–9. [PubMed: 19090406]
60. Osman A, Barrios FX, Gutierrez PM, Kopper BA, Merrifield T, Grittmann L. The Pain Catastrophizing Scale: further psychometric evaluation with adult samples. J Behav Med. 2000; 23:351–65. [PubMed: 10984864]
61. Pallegama RW, Ranasinghe AW, Weerasinghe VS, Sitheeque MA. Anxiety and personality traits in patients with muscle related temporomandibular disorders. J Oral Rehabil. 2005; 32:701–7. [PubMed: 16159346]
62. Paul B, Leitner C, Vacariu G, Wick F, Zehetmayer S, Matzner M, Mittermaier C, Vanecek E, Ebenbichler G. Low-back pain assessment based on the Brief ICF Core Sets: diagnostic relevance of motor performance and psychological tests. Am J Phys Med Rehabil. 2008; 87:452–60. [PubMed: 18496247]
63. Pennebaker, JW. The psychology of physical symptoms. New York: Springer-Verlag; 1982.
64. Persson LO, Larsson BM, Nived K, Eberhardt K. The development of emotional distress in 158 patients with recently diagnosed rheumatoid arthritis: a prospective 5-year follow-up study. Scand J Rheumatol. 2005; 34:191–7. [PubMed: 16134724]
65. Pett, MA.; Lackey, NR.; Sullivan, JJ. Making sense of factor analysis: the use of factor analysis for insturment development in health care research. Thousand Oaks, CA: Sage Publications; 2003.
66. Plesh O, Crawford PB, Gansky SA. Chronic pain in a biracial population of young women. Pain. 2002; 99:515–23. [PubMed: 12406528]
67. Quartana PJ, Buenaver LF, Edwards RR, Klick B, Haythornthwaite JA, Smith MT. Pain catastrophizing and salivary cortisol responses to laboratory pain testing in temporomandibular disorder and healthy participants. J Pain. 2010; 11:186–94. [PubMed: 19853521]
68. Rahim-Williams FB, Riley JL III, Campbell CM, Herrera D, Hastie BA, Fillingim RB. Association of hypervigilance with perceptual and cardiovascular responses to experimental pain among african americans and non-hispanic whites. Journal of Pain. 2008; 9:52.
69. Riley JL III, Robinson ME. CSQ. five factors or fiction? Clin J Pain. 1997; 13:156–62. [PubMed: 9186023]
70. Rollman GB, Gillespie JM. The role of psychosocial factors in temporomandibular disorders. Current Review of Pain. 2000; 4:71–81. [PubMed: 10998718]
71. Rosenstiel AK, Keefe FJ. The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. Pain. 1983; 17:33–44. [PubMed: 6226916]
72. Sarason IG, Johnson JH, Siegel JM. Assessing the impact of life changes: development of the Life Experiences Survey. J Consult Clin Psychol. 1978; 46:932–46. [PubMed: 701572]
73. Schiphorst Preuper HR, Reneman MF, Boonstra AM, Dijkstra PU, Versteegen GJ, Geertzen JH. The relationship between psychosocial distress and disability assessed by the Symptom Checklist-90-Revised and Roland Morris Disability Questionnaire in patients with chronic low back pain. Spine J. 2007; 7:525–30. [PubMed: 17905314]

74. Slade GD, Bair E, By K, Mulkey F, Baraian C, Rothwell R, Reynolds M, Miller V, Gonzalez Y, Gordon S, Ribeiro-Dasilva M, Feng Lim P, Greenspan JD, Dubner R, Fillingim RB, Diatchenko L, Maixner W, Dampier D, Knott C, Ohrbach R. Study methods, recruitment, socio-demographic findings and demographic representativeness in the OPPERA study. J Pain. 2011; XX:XXX–XXX. Submitted.
75. Slade GD, Diatchenko L, Bhalang K, Sigurdsson A, Fillingim RB, Belfer I, Max MB, Goldman D, Maixner W. Influence of psychological factors on risk of temporomandibular disorders. J Dent Res. 2007; 86:1120–5. [PubMed: 17959908]
76. Solomon MB, Herman JP. Sex differences in psychopathology: of gonads, adrenals and mental illness. Physiol Behav. 2009; 97:250–8. [PubMed: 19275906]
77. Spielberger, CD.; Gorusch, RL.; Lushene, R.; Vagg, PR.; Jacobs, GA. Manual for the State-Trait Anxiety Inventory (Form Y 1). Palo Alto, CA: Consulting Psychologists Press; 1983.
78. Sullivan MJ, Bishop S, Pivik J. The Pain Catastrophizing Scale: development and validation. Psychological Assessment. 1995; 7:524–32.
79. Sullivan MJ, Thorn B, Haythornthwaite JA, Keefe F, Martin M, Bradley LA, Lefebve JC. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain. 2001; 17:52–64. [PubMed: 11289089]
80. Talley NJ, Boyce PM, Jones M. Predictors of health care seeking for irritable bowel syndrome: a population based study [see comments]. Gut. 1997; 41:394–8. [PubMed: 9378398]
81. Thompson B, Daniel LG. Factor analytic evidence for the construct validity of scores: a historical overview and some guidelines. Educational and Psychological Measurement. 1996; 56:197–208.
82. Velly AM, Gornitsky M, Philippe P. Contributing factors to chronic myofascial pain: a case-control study. Pain. 2003; 104:491–9. [PubMed: 12927621]
83. Ware J Jr, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. Med Care. 1996; 34:22–33.
84. Weathers, FW.; Litz, BT.; Herman, DS.; Huska, JA.; Keane, TM. The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility. Paper presented at the Meeting of the International Society for Traumatic Studies; San Antonio, TX. 1993.
85. Whitehead WE, Bosmajian L, Zonderman AB, Costa PT Jr, Schuster MM. Symptoms of psychologic distress associated with irritable bowel syndrome. Comparison of community and medical clinic samples. Gastroenterology. 1988; 95:709–14. [PubMed: 3396818]
86. Yatani H, Studts J, Cordova M, Carlson CR, Okeson JP. Comparison of sleep quality and clinical and psychologic characteristics in patients with temporomandibular disorders. J Orofac Pain. 2002; 16:221–8. [PubMed: 12221738]
87. Zuckerman LA, Oliver JM, Hollingsworth HH, Austrin HR. A comparison of life events scoring methods as predictors of psychological symptomatology. J Human Stress. 1986; 12:64–70.

Perspective

This article reports baseline psychosocial findings from the OPPERA Study, a large prospective cohort study designed to discover causal determinants of TMD pain. Findings indicate significant differences between TMD cases and TMD-free controls across multiple psychosocial constructs, and future analyses will determine whether these psychosocial factors increase risk for new onset TMD.

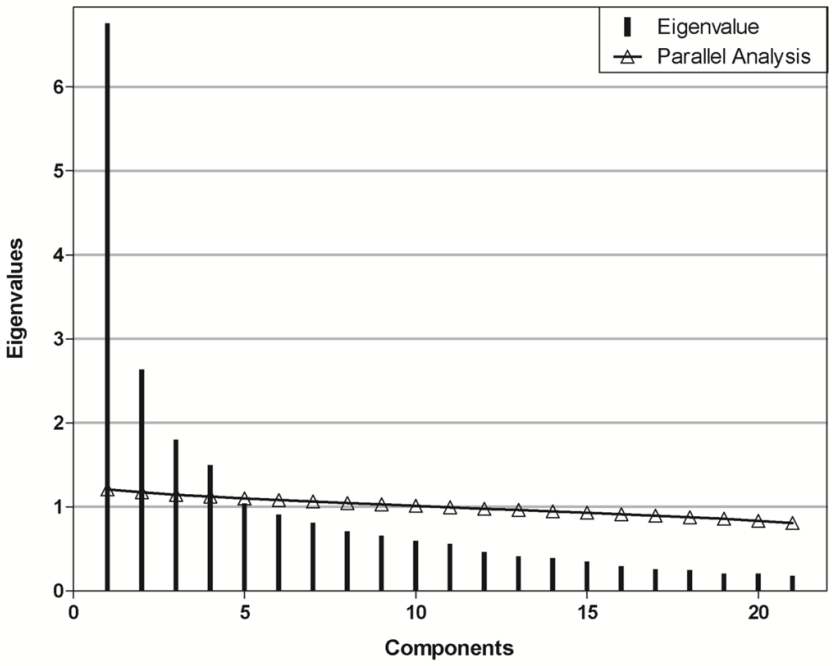

Figure 1.

Scree plot and parallel analysis from PCA. The vertical lines depict the eigenvalues for each scomponent, showing a significant drop off after component 4. Also, the open triangles and solid black line show the upper bound of the 95% confidence interval for eigenvalues that would be expected for each component from a PCA computed on a random data set. As can be seen, the observed eigenvalues for the first 4 components exceed the chance line, further supporting the selection of a 4 component model.

Table 1

Global Measures of Psychological Function for TMD Cases and Controls

									Non-imputed effect estimates[2]				Imputed effect estimates		
		Controls			TMD cases				Site-adjusted effect[3]		Fully adjusted effect[4]		Fully adjusted effect		
Putative risk factor	**Units and potential range**	**n**	**mean**	**se**	**n**	**mean**	**se**	**P-value[1]**	**SOR[5]**	**95% CI[6]**	**SOR**	**95%CI**	**n**	**SOR**	**95%CI**
SCL 90R Depression	(0–4 scale)	1594	0.37	0.01	181	0.64	0.05	<0.0001	1.5	1.3, 1.7	1.6	1.4, 1.8	1807	1.6	1.4, 1.8
SCL 90R Somatization	(0–4 scale)	1604	0.25	0.01	182	0.64	0.04	<0.0001	2.1	1.8, 2.4	2.1	1.8, 2.4	1807	2.1	1.8, 2.4
SCL 90R Anxiety	(0–4 scale)	1608	0.19	0.01	182	0.35	0.03	<0.0001	1.4	1.2, 1.6	1.5	1.3, 1.7	1807	1.4	1.3, 1.7
SCL 90R Hostility	(0–4 scale)	1617	0.27	0.01	182	0.46	0.04	<0.0001	1.4	1.2, 1.6	1.5	1.3, 1.7	1807	1.5	1.3, 1.7
EPQ-R Extraversion	(0–12 scale)	1606	8.44	0.08	180	8.37	0.25	0.7642	1.0	0.8, 1.1	1.0	0.8, 1.2	1807	1.0	0.8, 1.2
EPQ-R Neuroticism	(0–12 scale)	1611	4.20	0.08	180	5.60	0.26	<0.0001	1.6	1.3, 1.8	1.4	1.2, 1.7	1807	1.4	1.2, 1.7

[1] P-values are from analysis variance model comparing mean values of putative risk factor between cases and controls, with adjustment for study site.

[2] Non-imputed effect estimates are for complete case analysis using numbers of subjects in columns headed n.

[3] Site-adjusted effects were computed in logistic regression models where the putative risk factor as the main explanatory variable and study site as covariate.

[4] Fully-adjusted effects were computed in logistic regression models that additionally include covariates of age group, gender and race/ethnicity.

[5] SOR is standardized odds ratio from a logistic regression model evaluating the linear effect of the risk factor that was standardized by z-score transformation

[6] 95% confidence interval for SOR

Table 2

Measures of Affective Distress and Stress for TMD Cases and Controls

									Non-imputed effect estimates[2]				Imputed effect estimates		
		Controls			TMD cases				Site-adjusted effect[3]		Fully adjusted effect[4]		Fully adjusted effect		
Putative risk factor	**Units and potential range**	**n**	**mean**	**se**	**n**	**mean**	**se**	**P-value[1]**	**SOR[5]**	**95% CI[6]**	**SOR**	**95%CI**	**n**	**SOR**	**95%CI**
State Anxiety Inventory	(20–80 scale)	1598	30.90	0.24	181	32.69	0.73	0.0162	1.2	1.1, 1.4	1.3	1.1, 1.5	1809	1.3	1.1, 1.5
Trait Anxiety Inventory	(20–80 scale)	1585	35.50	0.24	180	38.59	0.81	<0.0001	1.4	1.2, 1.7	1.5	1.3, 1.7	1812	1.5	1.3, 1.7
POMS-Bi Positive Affect	(30–120 scale)	1547	87.18	0.4	178	82.62	1.23	0.0002	0.7	0.6,0.8	0.7	0.6, 0.8	1809	0.7	0.6, 0.8
POMS-Bi Negative Affect	(30–120 scale)	1565	49.47	0.4	180	56.34	1.29	<0.0001	1.5	1.3, 1.7	1.5	1.3, 1.7	1809	1.4	1.2, 1.7
Perceived Stress Scale	(0–40 scale)	1603	14.66	0.16	183	16.81	0.51	<0.0001	1.4	1.2, 1.7	1.5	1.3, 1.8	1807	1.5	1.3, 1.8

[1] P-values are from analysis variance model comparing mean values of putative risk factor between cases and controls, with adjustment for study site.

[2] Non-imputed effect estimates are for complete case analysis using numbers of subjects in columns headed n.

[3] Site-adjusted effects were computed in logistic regression models where the putative risk factor as the main explanatory variable and study site as covariate.

[4] Fully-adjusted effects were computed in logistic regression models that additionally include covariates of age group, gender and race/ethnicity.

[5] SOR is standardized odds ratio from a logistic regression model evaluating the linear effect of the risk factor that was standardized by z-score transformation

[6] 95% confidence interval for SOR

Table 3

Measures of Somatic Awareness and Coping for TMD Cases and Controls

									Non-imputed effect estimates[2]				Imputed effect estimates		
		Controls			TMD cases				Site-adjusted effect[3]		Fully adjusted effect[4]		Fully adjusted effect		
Putative risk factor	**Units and potential range**	**n**	**mean**	**se**	**n**	**mean**	**se**	**P-value[1]**	**SOR[5]**	**95% CI[6]**	**SOR**	**95%CI**	**n**	**SOR**	**95%CI**
Pill Global Score	(1–270 scale)	1542	88.25	0.55	171	114.09	2.37	<0.0001	2.5	2.2, 3.0	2.5	2.1, 2.9	1810	2.4	2.0, 2.8
Kohn Global Score	(24–120)	1567	71.34	0.30	177	75.20	0.89	<0.0001	1.4	1.2, 1.7	1.3	1.1, 1.5	1807	1.3	1.1, 1.5
PCS Helplessness	(0–24 scale)	1617	3.47	0.10	183	5.61	0.37	<0.0001	1.6	1.4, 1.8	1.5	1.3, 1.8	1811	1.5	1.3, 1.8
PCS Magnification	(0–12 scale)	1624	1.89	0.05	182	2.84	0.19	<0.0001	1.5	1.3, 1.7	1.5	1.3, 1.7	1811	1.5	1.3, 1.7
PCS Rumination	(0–16 scale)	1624	4.15	0.10	184	5.67	0.32	<0.0001	1.5	1.3, 1.7	1.4	1.2, 1.7	1811	1.4	1.2, 1.7
CSQ Coping Statements	(0–6 scale)	1616	3.57	0.04	182	3.58	0.10	0.9469	1.0	0.9, 1.2	1.0	0.9, 1.2	1812	1.1	0.9, 1.2
CSQ Reinterpreting Pain	(0–6 scale)	1618	1.23	0.03	183	1.06	0.10	0.0991	0.9	0.7, 1.0	1.0	0.8, 1.2	1812	1.0	0.8, 1.2
CSQ Distraction	(0–6 scale)	1618	2.42	0.04	181	2.06	0.11	0.0018	0.8	0.7, 0.9	0.8	0.7, 1.0	1812	0.8	0.7, 1.0
CSQ Ignoring Pain	(0–6 scale)	1610	2.62	0.04	182	2.58	0.10	0.7154	1.0	0.8, 1.1	1.0	0.9, 1.2	1812	1.0	0.9, 1.2
CSQ Praying & Hoping	(0–6 scale)	1622	2.32	0.05	182	1.86	0.14	0.0039	0.8	0.7, 0.9	0.9	0.7, 1.0	1812	0.8	0.7, 1.0

[1] P-values are from analysis variance model comparing mean values of putative risk factor between cases and controls, with adjustment for study site.

[2] Non-imputed effect estimates are for complete case analysis using numbers of subjects in columns headed n.

[3] Site-adjusted effects were computed in logistic regression models where the putative risk factor as the main explanatory variable and study site as covariate.

[4] Fully-adjusted effects were computed in logistic regression models that additionally include covariates of age group, gender and race/ethnicity.

[5] SOR is standardized odds ratio from a logistic regression model evaluating the linear effect of the risk factor that was standardized by z-score transformation

[6] 95% confidence interval for SOR

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Table 4

Component Loadings for PCA Model in Controls who do not have TMD (n=1633)

	Component 1	Component 2	Component 3	Component
SCL 90R Depression	0.30	**0.67**	0.05	0.02
SCL 90R Somatization	−0.12	**0.85**	0.11	0.01
SCL 90R Anxiety	0.10	**0.81**	0.04	0.01
SCL 90R Hostility	0.08	**0.74**	0.01	0.00
EPQ-R Extraversion	**−0.62**	0.24	0.12	−0.05
EPQ-R Neuroticism	**0.56**	0.15	0.17	−0.01
State Anxiety Inventory	**0.80**	0.11	−0.10	0.00
Trait Anxiety Inventory	**0.80**	0.11	0.03	0.02
POMS-Bi Positive Affect	**−0.87**	0.13	0.12	0.06
POMS-Bi Negative Affect	**0.57**	0.32	−0.06	0.05
PSS Perceived Stress Scale	**0.70**	0.13	0.06	0.00
PILL Global Score	−0.09	**0.66**	0.12	0.03
Global Kohn Score	0.25	−0.17	**0.46**	−0.26
PCS Helplessness	−0.01	0.12	**0.83**	−0.05
PCS Magnification	−0.05	0.15	**0.78**	0.01
PCS Rumination	−0.10	0.08	**0.88**	−0.07
CSQ Coping Statements	−0.11	0.07	−0.01	**0.79**
CSQ Reinterpreting Pain	0.13	−0.03	0.02	**0.72**
CSQ Distraction	0.04	−0.18	0.32	**0.66**
CSQ Ignoring Pain	−0.03	0.12	−0.33	**0.79**
CSQ Praying & Hoping	0.00	−0.19	**0.56**	0.31
Cumulative Variance	0.19	0.35	0.49	0.61
Cronbach's Alpha	0.87	0.86	0.54	0.74