Impact of Catastrophizing in Patients with Temporomandibular Disorders—A Systematic Review

Birgitta Häggman-Henrikson, DDS, PhD
Christian Bechara, DDS, MSc
Brousk Pishdari, DDS, MSc
Department of Orofacial Pain and Jaw Function
Faculty of Odontology
Malmö University, Malmö, Sweden

Corine M. Visscher, PT, PhD
Department of Orofacial Pain and Dysfunction
Academic Centre for Dentistry Amsterdam
University of Amsterdam and Vrije Universiteit
Amsterdam, The Netherlands

EwaCarin Ekberg, DDS, Odont Dr
Department of Orofacial Pain and Jaw Function
Faculty of Odontology
Malmö University, Malmö, Sweden

Correspondence to:
Prof EwaCarin Ekberg
Department of Orofacial Pain and Jaw Function
Malmö University, SE-205 06
Malmö, Sweden
Email: ewacarin.ekberg@mau.se

Submitted December 17, 2019;
accepted August 3, 2020.

Aims: To assess the prevalence of catastrophizing in patients with temporomandibular disorders (TMD) and the possible associations between catastrophizing and treatment outcome. **Methods:** This review was registered in the Prospero database (CRD42018114233). Electronic searches were performed in PubMed, Scopus, and PsycINFO from the inception of each database up to October 26, 2018, and were combined with a hand search. Articles focusing on levels of catastrophizing and how catastrophizing affects pain levels and treatment outcomes for patients diagnosed with TMD were included, as well as studies reporting how treatment outcomes were affected by cognitive behavioral treatment as an addition to standard treatment for TMD. Reviews and case reports were excluded. Risk of bias was assessed with the Newcastle-Ottawa scale. **Results:** The literature search identified 266 articles. After screening of abstracts, the full texts of 59 articles were assessed. Of these, 37 articles, including 4,789 patients with TMD and 6,617 controls, met the inclusion criteria. Higher levels of pain catastrophizing were reported in patients with TMD, with a large effect size (Hedges' g = 0.86) compared to pain-free controls. Furthermore, associations of higher levels of catastrophizing with higher symptom severity and with poorer treatment outcome were reported together with indications of positive effects from cognitive behavioral therapy. **Conclusion:** The results suggest an association between catastrophizing and TMD that may affect not only symptom severity but also treatment outcome. Assessing levels of pain catastrophizing might therefore be valuable in the assessment and management of patients with TMD. *J Oral Facial Pain Headache 2020;34:379–397. doi: 10.11607/ofph.2637*

Keywords: *catastrophizing, cognitive behavioral therapy, pain, temporomandibular disorders, treatment outcome*

Catastrophizing is a mental, out-of-proportion exaggeration of an event, stimulus, or emotion; in general, it is described as expecting the worst possible outcome of future events or focusing on the negative aspects of past events. Pain catastrophizing is defined as a maladaptive cognitive-affective response to pain that involves negative thinking regarding the pain experience[1] and is believed to be a multidimensional construct consisting of rumination (not being able to direct attention away from pain), magnification (worry or exaggeration of the seriousness of something), and helplessness (feeling nothing can be done to reduce the pain).[2] Pain catastrophizing can predict pain intensity and disability[3] and has been associated with increased affective distress,[4] muscle and joint tenderness, and pain-related disability.[5,6] It has been suggested that early treatment for pain catastrophizing may serve as a prevention of chronic pain.[7] Reductions in pain catastrophizing are associated with improvements in pain and pain treatment outcome.[8] Taken together, the relationship between catastrophizing and pain has been demonstrated in different pain conditions, and it is therefore important to evaluate the level of pain catastrophizing in chronic pain patients.

The most common cause of chronic pain in the orofacial region are temporomandibular disorders (TMD), an umbrella term for musculoskeletal conditions that include orofacial pain and jaw dysfunction. TMD is usually classified into subgroups depending on whether it is related to the temporomandibular joint or to the masticatory muscles.[9] The preva-

lence of TMD pain is 10% to 15% in the general population worldwide, higher in the 20- to 50-year age group, and twice as common in women as in men. Notable with regard to the prevalence of TMD pain is that TMD is more common among younger people when compared to other chronic pain conditions.[10–12] However, in common with other chronic pain conditions, pain catastrophizing has been suggested to be more prevalent among TMD patients compared to healthy subjects.[13]

In 1992, the Research Diagnostic Criteria for TMD (RDC/TMD) were introduced to standardize the diagnostic process for TMD. These criteria were updated to improve validity and clinical utility in the Diagnostic Criteria for TMD (DC/TMD), which was intended for both research and clinical use.[14] This dual-axis system is based on Axis I, which provides a diagnosis of the physical condition, and Axis II, which assesses a patient's psychologic status and pain-related disability. Thus, the DC/TMD is intended to assist dentists in both TMD diagnosis and assessment of the prognosis.[14] The importance of incorporating psychosocial assessment in both prognosis and treatment planning has been emphasized.[15]

As with many other chronic pain conditions, TMD is seen with a variety of comorbid conditions, such as fibromylagia, rheumatism, and psoriatic arthritis.[16] Furthermore, psychologic comorbidities have been reported, with high levels of clinical depression in TMD patients as well as a positive correlation between psychologic distress and TMD severity.[17] Several studies have reported a higher prevalence of posttraumatic stress disorder in TMD patients compared to healthy controls.[18,19] Some of these comorbidities may also affect the prognosis and treatment outcome for the individual patient.[20]

Many different treatment modalities are used separately or in combination for patients with TMD. Patient information and counseling are important and essential components of treatment in order to reduce pain and anxiety. In addition to different behavioral treatment modalities, occlusal appliances are commonly used, sometimes in combination with jaw exercises and pharmacologic treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants.[21,22] Although these commonly used treatment modalities can achieve positive outcomes, there are several factors affecting treatment prognosis, with psychosocial factors identified as being particularly important. There is also a subgroup of TMD patients who do not respond well to conventional treatments alone.[23]

Chronic pain is complex and is affected not only by pathophysiology, but also by a patient's emotional and cognitive response. The purpose of cognitive behavioral therapy (CBT) is to create knowledge and understanding of pain, self-management of pain, and how to reduce the associated negative effects on quality of life.[24] CBT combines the treatment principles of basic cognitive and behavioral treatment to treat conditions such as depression, anxiety, and catastrophizing. CBT focuses on active coping strategies, such as behavioral activation focusing on specific problems within the patient that are affected by internal or external stimuli—therapists guide the patient into detecting behaviors the patients may be unaware of so that they instead process these behaviors, such as avoidance-response and fear, consciously. Pain catastrophizing is thereby treated by emphasizing awareness of the role of cognition in the experience of pain and the patient's coping ability.[25]

The general aim of this systematic review was to evaluate the importance of catastrophizing in patients with TMD. Specific aims were to assess the prevalence of catastrophizing in patients with TMD and the association between catastrophizing and the outcomes of TMD treatment.

Materials and Methods

This systematic review was registered with PROSPERO (International Prospective Register of Systematic Reviews; CRD42018114233) and was conducted in accordance with PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) guidelines.[26] Eligibility criteria were formulated using the PICO (population, intervention/exposure, comparison, and outcome) approach to identify articles reporting levels of catastrophizing for patients with TMD in studies with or without control groups. The components of the PICO question were as follows:

- Population: Patients diagnosed with TMD according to the RDC/TMD or DC/TMD criteria, and study size $\geq$ 10
- Intervention/exposure: Levels of catastrophizing or CBT as intervention
- Comparison: Control group without TMD or no comparison
- Primary outcome: Catastrophizing assessed with the Pain Catastrophizing Scale (PCS)
- Secondary outcomes: Catastrophizing assessed with other instruments and levels of pain in relation to catastrophizing

Studies in English, Swedish, or Dutch languages were included. Letters to the editor, conference proceedings, meeting abstracts, and review articles were excluded.

The PCS was developed in 1995 and is the most commonly used instrument to assess pain catastro-

Table 1 Search Strategies for the Different Databases and Number of Identified Records

Database	Search strategy	Records
PubMed	(((((((((tmjd) OR temporomandibular disorder*) OR temporomandibular joint disorder*) OR tmd) OR tmj disorder*) OR craniomandibular disorder*) OR (((facial OR jaw OR orofacial OR craniofacial OR trigem*)) AND pain)) OR "Craniomandibular Disorders"[Mesh] OR "temporomandibular joint disorders"[MeSH])) AND (((((((("Catastrophization"[Mesh]) OR Catastrophization) OR Catastrophisation) OR Catastrophizing*) OR Catastrophising*) OR Pain catastrophizing scale*) OR PCS) OR ((((catastrophic) OR catastrophe)) AND (((((("Thinking"[Mesh]) OR "Emotions"[Mesh]) OR thinking) OR thoughts) OR feelings)))	183
Scopus	(TITLE-ABS-KEY ("craniomandibular disorder*" OR "temporomandibular joint disorder*" OR "temporomandibular disorder*" OR tmjd OR tmd OR "tmj disorder*" OR ((facial OR jaw OR orofacial OR craniofacial OR trigem*) AND pain))) AND (TITLE-ABS-KEY (pcs OR "Pain catastrophizing scale" OR catastrophizing* OR catastrophisation OR catastrophization OR catastrophising* OR ((thinking OR thought OR feeling) AND (catastrophe OR catastrophic)))))	145
PsycINFO	(MAINSUBJECT.EXACT("Bruxism") OR (temporomandibular disorder*) OR (temporomandibular joint disorder*) OR (craniomandibular disorder*) OR (tmj disorder*) OR tmd OR tmjd OR ((facial OR jaw OR orofacial OR craniofacial OR trigem*) AND pain)) AND (MAINSUBJECT.EXACT("Catastrophizing") OR Catastrophization OR Catastrophisation OR Catastrophizing* OR Catastrophising* OR ((Catastrophe OR Catastrophic) AND (MAINSUBJECT.EXACT.EXPLODE("Thinking") OR MAINSUBJECT.EXACT.EXPLODE("Emotions") OR thinking OR thoughts OR feelings)) OR PCS OR (Pain catastrophizing scale))	106

phizing. This instrument measures three domains: helplessness, rumination, and magnification. The PCS seems to be invariant across genders and across patient vs nonpatient status in the context of pain.[27] The PCS includes 13 questions about thoughts or feelings that arise when experiencing pain. The variables are ranked on an ordinal scale with five different answer options ranging from 0 (not at all) to 4 (all the time), providing a total score of 0 to 52 points. The PCS has been shown to have good validity and reliability and is therefore considered to have a moderate to excellent quality.[28]

Levels of pain could be reported as pain intensity (eg, numeric rating scale [NRS], visual analog scale [VAS]) or other measures.

Search Strategy

An electronic literature search was performed in PubMed, Scopus, and PsycINFO from the inception of each database up to October 26, 2018. The main search strategy was developed for PubMed and then adapted for the other databases. The search strategy was developed in collaboration with two information specialists at Malmö University, Malmö, Sweden. One librarian developed the search strategy, and a second librarian did a peer review. The full search strategy for PubMed is provided in Table 1. There was no language restriction in the literature search stage, and any exclusion due to language was documented in the full-text assessment stage. The electronic search was combined with a hand search of the reference lists of included articles. Gray literature was not included, and authors were not contacted for additional information.

Procedure

Two authors (B.H.H. and EC.E.) independently read all titles and abstracts to identify potentially eligible articles for inclusion. If one of these reviewers deemed an article as potentially of interest, it was included for full-text assessment. All potentially eligible articles were then retrieved as full-text articles to determine whether they met the inclusion criteria. Any disagreement was resolved by discussion among the two reviewers, and, if needed, with a third reviewer (C.M.V.).

Data extraction was carried out with a predesigned data extraction form. The following data were extracted: author; publication year, country, description of setting, patient characteristics, prevalence of catastrophizing, pain intensity levels, and outcome of TMD treatment. Data extraction was conducted by one author (B.H.H.), and the data extracted were reviewed by a second author (C.M.V.). Risk of bias was assessed by two independent reviewers (B.H.H. and C.M.V.) with the Newcastle-Ottawa Scale (NOS) for case-control and cohort studies,[29] and any disagreement was resolved by discussion.

In addition to a qualitative synthesis based on the extracted tabulated data, eligible studies were included in a quantitative meta-analysis. Thus, for the included primary studies that reported levels of catastrophizing according to the PCS and measures of center and spread, a meta-analysis was conducted, including corrected effect size analysis (Hedges' g). The effect size was interpreted as small (0.20 to 0.49), moderate (0.50 to 0.79), or large (≥ 0.80).[30] To account for heterogeneity between studies, a random-effects analysis was performed with Review Manager software (RevMan version 5.3, the Cochrane Collaboration) and Meta-Essentials (version 1.4, Erasmus Research Institute),[31] in combination with graphs generated by DistillerSR Forest Plot Generator (Evidence Partners). In the qualitative synthesis of the quantitative data, the strength of associations based on absolute values of *r* was interpreted as very weak (≤ 0.19), weak (0.20 to 0.39), moderate (0.40 to 0.59), or strong (≥ 0.60).[32]

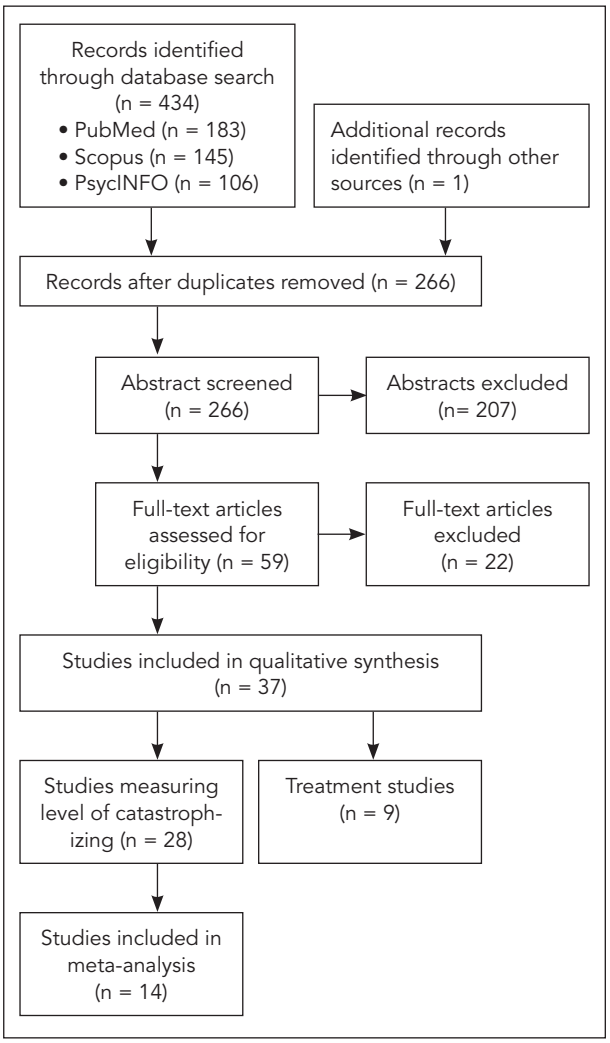

Fig 1 PRISMA flowchart of included and excluded studies.

Results

The electronic search in PubMed, Scopus, and PsycINFO up to October 26, 2018, together with a hand search, identified a total of 435 articles (Fig 1). After removal of duplicates and screening of 266 abstracts, 59 full texts were reviewed. Of these, 22 articles were excluded due to not fulfilling the inclusion criteria (see Appendix 1 in the online version of this article at www.quintpub.com/journals), and 37 articles[13,33–68] published between 2001 and 2018 that included 4,789 patients with TMD and 6,617 controls remained for the qualitative synthesis.

Risk of bias was assessed with the NOS for case-control and cohort studies (Tables 2a to 2c).

In total, 28 articles concerned level of catastrophizing in patients with TMD and associations with other factors (Table 3a). Of these, 14 were case-series studies, 11 were case-control studies, and 3 were cohort studies. A majority of these studies (17 articles) were based in study populations from the US. A total of 9 studies reported treatment outcomes; all of these were case-control studies, and 7 had been conducted in the US (Table 3b).

In total, 14 studies were included in the random-effects meta-analysis. Nine of these included healthy control groups with a total of 2,072 subjects, providing an overall score of 9.5 on the PCS scale and with no heterogeneity (I^2 score: 0%; Fig 2a). For the studies with TMD groups (n = 14), with a total of 1,163 subjects, a significantly higher PCS score of 17.6 was seen (Fig 2b), but with considerable heterogeneity (I^2 score: 96%). The combined effect size for the 9 studies that could be included in the effect size analysis was large (Hedges' g = 0.86; Fig 2c).

Pain catastrophizing was positively associated with TMD pain–related factors, such as pain intensity,[33] pain interference,[34] pain on palpation,[35] fatigue and pain in a provocation chewing test,[36] and neck disability.[37] With regard to correlations with such pain outcomes, there was a moderate to strong positive correlation between catastrophizing and pain intensity (r = 0.3 to 0.68),[34,38–40] and moderate positive correlations of catastrophizing with pain severity (r = 0.36 to 0.47),[33,41] pain interference (r = 0.38 to 0.52),[34,35,39,41,42] and pain and disability (r = 0.43 to 0.46).[37,43] Significant correlations of varying strengths were also reported in relation to neck disability (r = 0.61),[37] pain from a provocation chewing test (r = 0.41),[36] and experimental pain responses in terms of pain thresholds (r = −0.31) and suprathresholds (r = 0.43)[44] (Table 3a).

Catastrophizing was also associated with higher pain ratings[45]; high-impact pain (OR = 1.59)[46]; onset (OR = 1.98) and progression (OR = 2.17) of clinically significant pain[47]; and pain persistence (OR = 6.71).[48] Catastrophizing also explained 14% of the variance in pain-related activity interference[42] and in pain response from a provocation chewing test[36] (Table 3a).

Nine studies evaluated treatment outcome in relation to catastrophizing and generally reported reduced levels of catastrophizing, pain intensity, and activity interference after CBT. Litt et al evaluated the effect of adding CBT to standard treatment in three studies with partly overlapping study samples,[49–51] reporting that catastrophizing decreased after CBT, predicted momentary pain (estimated effect = 1.23, F = 18.91, P < .001), and moderated treatment effects for pain (β = 0.64, F = 32.07, P < .007) and pain interference (β = 0.62, F = 25.72, P < .007). Durà-Ferrandis et al reported that catastrophizing modified the effect (0.3, P < .05) of treatment on pain intensity.[52] Turner et al evaluated the effect of CBT in four studies with partly overlapping study samples[40,53–55] and reported that, compared to control groups, CBT groups showed

Table 2a Risk of Bias Assessment of Included Case Series (n = 11) and Case-Control Studies (n = 14)

	Selection				Comparability		Exposure			
Study, y	S1	S2	S3	S4	C1a	C1b	E1	E2	E3	Total
Brandini et al,[57] 2011	+			+	+		+	+		5
Buenaver et al,[41] 2012*	+									1
Campbell et al,[44] 2010	+			+	+	+	+	+		6
Castrillon et al,[38] 2008	+			+		+	+	+	+	6
Chen et al,[58] 2012	+		+	+	+	+	+	+	+	8
Chen et al,[59] 2013	+		+	+	+	+	+	+	+	8
Costa et al,[60] 2017	+			+			+	+		4
Davis et al,[34] 2014*	+	+								2
Fillingim et al,[61] 2011	+		+	+	+	+	+	+	+	8
Gil-Martínez et al,[37] 2017	+	+		+			+	+	+	6
Gustin et al,[62] 2011	+						+	+	+	4
Hollins et al,[63] 2009	+			+	+	+		+	+	6
Jerjes et al,[64] 2007	+						+	+		3
Kothari et al,[65] 2017	+		+	+			+	+	+	6
Kotiranta et al,[66] 2015*	+	+								2
La Touche et al,[43] 2014*	+						+			2
La Touche et al,[36] 2015	+			+			+	+	+	5
Lerman et al,[33] 2018*	+									1
Litt et al,[45] 2004*	+						+			2
Miller et al,[46] 2018*	+	+								2
Quartana et al,[67] 2010	+						+	+	+	4
Reiter et al,[48] 2018*	+	+								2
Turner et al,[42]2001*	+						+			2
Turner et al,[39] 2004*	+						+			2
Turner et al,[35] 2005a*	+						+			2

The Newcastle-Ottawa Scale for Case-Control Studies was used for assessment. S1 = definition of cases; S2 = representativeness of cases; S3: selection of controls; S4 = definition of controls; C1a = age; C1b = other factors; E1 = assessment; E2 = same method was used for cases and controls; E3 = nonresponse rate.
*Case series studies. Please note that per the definitions of the criteria in the Newcastle-Ottawa scale, case series studies (without control group) cannot achieve scores for items S3, S4, C1a, C1b, E2, and E3.

Table 2b Risk of Bias Assessment of Included Cohort Studies (n = 3)

	Selection				Comparability		Outcome			
Study, y	S1	S2	S3	S4	C1	C2	O1	O2	O3	Total
Bair et al,[56] 2013		+	+	+	+	+	+	+	+	8
Fillingim et al,[13] 2013		+	+	+	+	+	+	+	+	8
Velly et al,[47] 2011	+			+	+	+	+	+		6

The Newcastle-Ottawa Scale for Cohort Studies was used for assessment. S1 = representativeness of cohort; S2 = selection of nonexposed cohort; S3 = ascertainment of exposure; S4 = outcome not present at start; C1 = age; C2 = other factors; O1 = assessment; O2 = length of follow-up; O3 = follow-up rate.

Table 2c Risk of Bias Assessment of Included Treatment Studies (n = 9)

	Selection				Comparability		Exposure			
Study, y	S1	S2	S3	S4	C1	C2	E1	E2	E3	Total
Costa et al,[68] 2015	+		+		+		+	+		5
Durá-Ferrandis et al,[52] 2017	+	+	+		+	+	+	+		7
Litt et al,[50] 2009	+				+			+	+	4
Litt et al,[51] 2010	+				+			+	+	4
Litt and Porto,[49] 2013	+				+			+	+	4
Turner et al,[40] 2005b	+					+		+	+	4
Turner et al,[54] 2006	+				+	+		+	+	5
Turner et al,[53] 2007	+				+	+		+	+	5
Turner et al,[55] 2011	+				+	+		+		4

The Newcastle-Ottawa Scale for Case-Control Studies was used for assessment. S1 = definition of cases; S2 = representativeness of cases; S3 = selection of controls; S4 = definition of controls; C1 = age; C2 = other factors; E1 = assessment; E2 = same method for cases and controls; E3 = nonresponse rate.

reduced pain intensity (CPI: 3.9 vs 4.7)[54] and larger proportions of participants with reduced activity interference (34% to 35% vs 13%).[40,54] Furthermore, catastrophizing was a mediator of CBT effects on both activity interference (–0.59), explaining 46% of the total effect, and pain interference (–0.44), explaining 30% of the total treatment effects[53] (Table 3b).

Table 3a Summary of Results for Levels of Catastrophizing and Associations with Other Factors (n = 28)

Study (y), country	Setting, study design	Participants (% F), mean age (range)	Methods	Results
Bair et al[56] (2013), USA	Population sample, prospective cohort	2,737 healthy individuals (18–44 y)	PCS, variable importance score	A total of 260 individuals developed TMD. Catastrophizing was a weak predictor for TMD (variable importance score: 10.4)
Brandini et al[57] (2011), Australia	Hospital staff and patients, case-control	15 (100%) TMD patients, 31 y; 14 (100%) healthy individuals, 29 y	PCS	There was no significant difference in mean (SD) PCS score between TMD group (12.7 [10.6]) and healthy participants (11.0 [8.4]). There was no significant correlation between catastrophizing and kinematic variables during chewing.
Buenaver et al[41] (2012), USA	Orofacial pain clinic + population sample,-case series	214 (74%) TMD patients, 34 y (18–65)	PCS, PSQI, BPI	The PCS was associated with sleep disturbances (r = 0.37), pain severity (r = 0.36), and pain interference (r = 0.52) (all P < .001). A significant portion of the variance in clinical pain severity and pain-related interference attributable to pain catastrophizing (ie, rumination) was mediated by sleep disturbance.
Campbell et al[44] (2010) USA	Hospital advertising, case-control	84 (38%) healthy individuals; 48 (85%) TMD patients; 43 (62%) arthritis patients	PCS, SCQ, HPT	There was a higher mean PCS (P = .01) in the TMD group (14.3) compared to healthy (9.5) participants. TMD patients showed negative correlations of the PCS (r = −0.31, P < .05) and SCQ (r = −0.30, P < .05) with HPT and positive correlation with suprathresholds of heat stimuli (both r = 0.43, P < .01) and painful aftersensation (r = 0.46 and 0.50, respectively; both P < .01).
Castrillon et al[38] (2008), Denmark	University students, experimental-case-control	10 (100%) TMD patients, 24 y; 47 (100%) healthy individuals, 29 y	CSQ, VAS for pain	There was no difference in CSQ between the TMD and control groups. In the TMD group, there was a significant correlation (r = 0.68, P < .03) between catastrophizing and VAS pain.
Chen et al[58] (2012), USA	Orofacial pain clinic and university advertising, case-control	83 TMD patients, 33 y (18–60); 76 TMD + WPT, 40 y (18–60); 181 healthy individuals, 30 y (18–60)	PCS, CPSQ, GCPS	Controls had a significantly (P < .004) lower mean PCS (6.8) compared to both TMD (10.7) and TMD + WPT (11.8) patients.
Chen et al[59] (2013), USA	Orofacial pain clinic and university advertising, case-control	159 (100%) TMD patients, 36 y (18–60); 131 (100%), healthy individuals, 30 y (18–60)	PCS, CPSQ, GCPS	Controls had a significantly (P < .004) lower mean PCS (6.0) compared to the TMD group (11.2). TMD patients with pain comorbidity reported higher PCS.
Costa et al[60] (2017), Brazil	Hospital orofacial pain clinic, case control	47 (80%) TMD patients, 28 y; 50 (88%) TMD + headache patients, 29 y	PRSS subscale, VAS	The TMD + headache group had higher mean (SD) PRSS (2.1 [1.2]) than the TMD-only group (1.6 [1.4]) (P = .048).
Davis et al[34] (2014), USA	Orofacial pain clinic, case series	50 (90%) TMD patients, 41 y (18–80)	PSWQ, STAI, PCS, NRS (0–10) for pain (current, worst, least, average, interference), disability score (0–10)	Mean (SD) PCS: Total: 15.7 (11.7) Rumination: 5.2 (3.8) Magnification: 3.3 (2.7) Helplessness: 7.3 (6.1) PCS was correlated with worst (r = 0.39, P < .01) and least (r = 0.32, P < .05) pain intensity, as well as pain interference (r = 0.38, P < .01).
Fillingim et al[61] (2011), USA	Population sample, cross-sectional case-control	1,633 TMD-free controls; 185 TMD patients; (18–44 y)	STAI, PCS	TMD cases had higher levels of catastrophizing than controls (P < .0001). PCS: Helplessness: 5.61 vs 3.47 Magnification: 2.84 vs 1.89, Rumination: 5.67 vs 4.15

BPI = Brief Pain Inventory; CSQ = Coping Strategies Questionnaire; GCPS = Graded Chronic Pain Scale; HPT = heat pain threshold; PCS = Pain Catastrophizing Scale; PSQI = Pittsburgh Sleep Quality Index; PSWQ = Penn State Worry Questionnaire; STAI = State Trait Anxiety Inventory; VAS = visual analog scale; WPT = widespread body palpation tenderness.

Conclusions	Limitations
Multivariable methods were used to identify the most important predictors of first-onset TMD in the OPPERA study. Important variables included comorbid pain conditions, preexisting pain, and somatic awareness. Demographic characteristics, which probably reflect environmental variables not measured in OPPERA, also appear to play an important role in the etiology of TMD.	
This exploratory study provided data suggesting that psychologic factors, manifesting as depression and stress, play a role in influencing the association between pain and motor activity.	
These results suggest that rumination on pain may contribute to clinical pain indirectly through alterations in sleep. Prospective studies are needed to examine the associations between these constructs. These findings have important theoretical and clinical implications. Critically, interventions that reduce pain catastrophizing may concurrently improve sleep and clinical pain.	
This study adds to a growing body of literature examining catastrophizing. These findings highlight the potential importance of the multidimensional assessment of pain-related catastrophizing and suggest a role for measuring catastrophizing related to specific, definable events.	There were gender and age differences between groups; but gender- and age-adjusted analyses for associations.
Glutamate-evoked pain responses in healthy subjects and persistent myofascial TMD pain have similar sensory-discriminative and affective-unpleasantness components, but differ in psychosocial features. This study suggests that experimental designs based on intramuscular glutamate injection can provide an appropriate model for elucidating persistent myofascial pain conditions.	The TMD group had a significantly higher age.
TMD subjects with WPT experience a greater level of multiple comorbid pain conditions compared to TMD subjects without WPT and non-TMD controls. Integration of bodily pain assessments can be informative for the evaluation, diagnosis, and management of TMD.	The TMD group had a significantly higher age.
The concurrent assessment of multiple physiologic and psychologic systems is critical to our understanding of the pathophysiologic processes that contribute to painful TMD and associated comorbid conditions, which will ultimately guide and inform appropriate treatment strategies that address the multisystem dysregulation associated with complex and common persistent pain conditions.	The same study population was included as in Chen et al[87] (2012). The TMD group had a significantly higher age.
Coexistence of headache further exacerbates clinical characteristics in patients with painful TMD, which implies involvement of common mechanisms and pathways of vulnerability in these patients.	
Participants with chronic orofacial pain reported experiencing substantial levels of trait worry, anxiety, pain catastrophizing, and worry about pain that related to pain ratings directly and indirectly.	
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.	

Table 3a *(cont)* Summary of Results for Levels of Catastrophizing and Associations with Other Factors (n = 28)

Study (y), country	Setting, study design	Participants (% F), mean age (range)	Methods	Results
Fillingim et al[13] (2013), USA	Population sample, prospective cohort	Baseline: 3,263 (60%), TMD-free individuals 27 y (18–44) Follow-up: 260 first-onset TMD patients	STAI, PCS	Global psychologic and somatic symptoms, but not PCS, emerged as the most robust risk factors for incident TMD. PCS was not a predictor for TMD onset.
Gil-Martínez et al[37] (2017), Spain	University TMD/neurology clinic, cross-sectional case-control	50 (78%) TMD patients, 46 y; 50 (92%) migraine patients, 49 y	PCS, VAS pain intensity (0–100), NDI, CF-PDI	Compared to the migraine group, the TMD group had lower levels (mean [SD]) of rumination 9.2 (2.5) and helplessness 10.8 (4.1), but a similar level of magnification 6.4 (2.1). For the TMD group, but not the migraine group, there were correlations of the PCS with NDI (r = 0.61, P < .01) and CF-PDI (r = 0.43, P < .01), but no significant correlation with pain intensity.
Gustin et al[62] (2011), Australia	University clinic, case-control	21 (76%) TMD patients, 45 y (24–71); 24 (75%) TNP, 56 y (42–75); 38 (76%) controls, 49 y (23–81)	PCS	TMD patients showed a higher mean (SD) PCS compared to controls (18.7 [10.9] vs 10.1 [8.9]) (P = .002), but no difference compared to TNP (23.6 [11.5]) patients.
Hollins et al[63] (2009), USA	University orofacial pain clinic and advertising, case-control	22 (100%) TMD patients, 38 y; 12 (100%) fibromyalgia patients, 35 y; 20 (100%) healthy individuals, 38 y	PCS	There were significant differences in mean (SD) PCS (P = .01) among groups: Healthy: 8.4 (7.2) TMD patients: 12.6 (9.2) Fibromyalgia patients: 17.8 (7.9)
Jerjes et al[64] (2007), UK	University clinic, case-control	51 (76%) TMD patients, 37 y (18–70); 51 (67%) chronic daily headache patients, 40 y (18–70)	CSQ, IPQ	Compared to TMD patients, patients with chronic daily headache had higher mean (SD) CSQ (7.8 [8.0] vs 14.0 [10.2]; P < .01) at baseline. There was no significant difference at the 6-month follow-up (8.0 [9.6[vs 12.0 [10.6]).
Kothari et al[65] (2017), Denmark	University orofacial pain clinic, case-control	58 (83%) TMD patients, 37 y (20–74); 41 (73%) controls, 32 y (20–61)	PCS	TMD patients had higher mean (SD) PCS than controls: Total: 20.7 (11.0) vs 10.3 (9.9), P < .001 Rumination: 7.0 (4.8) vs 4.5 (4.4), P = .005 Magnification: 4.1 (2.9) vs 1.8 (1.9), P < .001 Helplessness: 9.6 (5.3) vs 3.8 (4.1), P < .001
Kotiranta et al[66] (2015), Finland	Primary dental care clinic, case series	399 (83%) TMD patients, 40 y (18–70)	PCS, GCPS (grouped into no, low, and high disability)	There were significant differences in CPI (P < .000) among the GCPS groups, with 3.7 in no disability, 6.0 in low disability, and 7.7 in high disability.
La Touche et al[43] (2014), Spain	Hospital and private TMD clinics, case series	192 (69%) TMD patients, 46 y (19–78)	PCS, CF-PDI	TMD patients reported a high mean (SD) PCS: (23.7 [8.9]). There was a significant correlation between PCS and CF-PDI (r = 0.46, P < .01).
La Touche et al[36] (2015), Spain	Public health and private TMD clinics, experimental case-control	83 (61%) TMD patients, 43 y (19–60), with subgroups: Mild neck disability (NDI < 15; n = 41); moderate neck disability (NDI ≥ 15; n = 42); 39 (67%) controls, 41 y (30–65)	PCS, NDI, VAS for pain and for fatigue, PPT pain-free maximum mouth opening	TMD patients had a higher PCS than controls (16.4 [3.9] vs 5.5 [1.8]; P = .01). For TMD patients with mild neck disability, there was a correlation between PCS and pain (r = 0.40, P < .01), and PCS predicted fatigue (r^2 = 0.12, P = .01) and pain (r^2 = 0.14, P = .01) 24 hours after a provocation chewing test. For TMD patients with moderate neck disability, there was a correlation between PCS and fatigue (r = 0.44, P < .01), and PCS predicted fatigue (r^2 = 0.17, P = .004) 24 hours after a provocation chewing test.

CF-PDI = Craniofacial Pain and Disability Inventory; CSQ = Coping Strategies Questionnaire; IPQ = Illness Perception Questionnaire; NDI = Neck Disability Inventory; PCS = Pain Catastrophizing Scale; PPT = pressure pain threshold; STAI = State Trait Anxiety Inventory; TNP = trigeminal neuropathic pain.

Conclusions	Limitations
Measures of somatic symptoms were most strongly associated with TMD onset, but perceived stress, previous life events, and negative affect also predicted TMD incidence.	
Differences between the migraine group and the chronic TMD group were found in craniofacial pain and disability, pain catastrophizing, and headache impact, but they were similar for pain intensity, neck disability, and kinesiophobia. Neck disability and kinesiophobia were covariates of craniofacial pain and disability (34% of variance explained) for chronic TMD. In the migraine group, neck disability was a predictive factor for headache impact (19.3% of variance explained).	
These findings support growing evidence that the negative affective, cognitive, and psychosocial state of chronic pain is universal, regardless of a neuropathic or nociceptive nature. Further characterization of these four dimensions of the pain experience in different chronic pain subtypes may improve the effectiveness of cognitive-behavioral therapy.	
Pain patients showed robust perceptual amplification of cutaneous pressure stimuli and modest amplification of auditory stimuli. In both cases, perceptual amplification extended to even the lowest stimulus intensities, a result that is not consistent with the predictions of the generalized hypervigilance hypothesis. An alternative formulation, the attentional gain control model of hypervigilance, is proposed, according to which those types of stimuli that are associated with pain are amplified because of the attention that is habitually directed toward them.	
This study suggests that differences in cognitive findings between these two groups of patients are not sustained over time. Initially, the headache patients displayed more catastrophizing, were more distressed, and were more depressed. However, these differences disappeared at follow-up. Significant correlations between perceived performance (timeline subscale IPQ); disability and anxious mood; perceived consequence with disability and depressed mood; and catastrophizing with pain, disability, and anxious mood present possible targets for therapeutic intervention.	
TMD pain patients had elevated scores of depressive symptoms, somatization, sleep dysfunction, and increased levels of catastrophic thoughts, which is consistent with previous findings. Thus, these findings support the current perspective that TMD is multidimensional, with a combination of physical, psychologic, and social factors contributing to the overall presentation of this disorder.	
The results suggest that GCPS-related disability scoring can be used as a simple screening instrument in primary care settings to identify individuals with different, clinically relevant psychosocial subtypes.	
The CF-PDI showed good psychometric properties. Based on the findings of this study, the CF-PDI can be used in research and clinical practice for the assessment of patients with craniofacial pain.	This study was in a convenience sample.
Neck-pain–related disability and pain catastrophizing have an influence on the sensory-motor variables evaluated in patients with headache attributed to TMD.	

Table 3a *(cont)* Summary of Results for Levels of Catastrophizing and Associations with Other Factors (n = 28)

Study (y), country	Setting, study design	Participants (% F), mean age (range)	Methods	Results
Lerman et al[33] (2018), US	University clinic and advertising, case series	156 (100%) TMD patients, 37 y (18–60)	PCS, BPI pain severity	The mean (SD) reported PCS was 21.4 (9.8) for total score, 7.9 (3.8) for rumination, 4.2 (2.9) for magnification, and 9.2 (4.6) for helplessness. There were higher levels in African American compared to Caucasian patients (25.2 [10.9] vs 20.3 [9.2]). PCS correlated to pain severity (r = 0.47, P < .01)
Litt et al[45] (2004), USA	Population sample, case series	30 (87%) TMD patients, 36 y	PRSS catastrophizing subscale (0–5), current pain (0–10)	The mean (SD) PRSS catastrophizing score was 1.6 (0.8). Momentary catastrophizing was a predictor for higher pain (P < .01), and higher catastrophizing scores were predictive of higher mean pain ratings (PRSS score t(114.46) = 5.87, P < .001).
Miller et al[46] (2018), USA	Cross-sectional case series	846 (77%) TMD patients, 28.0 y (18–44), with pain subgroups: low-impact GCPS (I + II-low), high-impact GCPS (II-high, III, IV)	CSQ-Revised catastrophizing subscale (0–6), GCPS	TMD patients with high-impact pain showed higher catastrophizing (1.0 [0.7]) compared to the low-impact pain group (0.6 [0.5]). Catastrophizing was significant in the regression model (OR 1.46, 1.25–1.7).
Quartana et al[67] (2010), USA	Orofacial pain clinic and advertising, cross-sectional case-control	39 (82%) TMD patients, 34 y; 22 (96%) controls 26 y	PCS, PPT, HPT, cold pain rating	TMD cases had higher levels of catastrophizing (PCS mean [SD]: 14.0 [8.8]) than controls (8.9 [6.8]) (P < .05). Higher PCS was associated with flattened morning salivary cortisol profile. There were no correlations of PCS with PPT, HPT, or cold pain rating.
Reiter et al[48] (2018), Israel	University orofacial pain clinic, case series	163 (66%) TMD patients, 36 y (18–60)	PCS, PHQ-9, GAD-7, PHQ-15	Higher PCS was associated with a higher prevalence of myofascial pain with referral (P < .05); lower prevalence of myalgia (P < .02); and higher pain persistence, GCPS, depression, anxiety, and nonspecific physical symptoms (all P < .001). Catastrophizing was associated with pain persistence (OR: 6.71, 95% CI: 1.58–28.41; P = .01).
Turner et al[42] (2001), USA	TMD clinic, cross-sectional case series	118 (83%) TMD patients, 39 y (21–67)	CPI, CSQ catastrophizing subscale (0–6), MPI interference scale	Mean (SD) catastrophizing score (2.2 [1.5]) was correlated with pain-related activity interference (r = 0.45, P < .0001). Catastrophizing explained variance in pain-related activity interference (r^2 = 0.14, P < .0001), nonmasticatory jaw activity limitation (change in r^2 = 0.08, P < .001), and depression (change in r^2 = 0.33, P < .0001).
Turner et al[39] (2004), USA	TMD clinic, cross-sectional case series	100 (87%) TMD patients, 39 y (16–67)	Catastrophizing assessed by 3 questions adapted from the CSQ, PCS rumination subscale (0–10)	Catastrophizing was low (mean [SD]: 2.7 [2.4]) and stable over a 2-week period. Higher levels of catastrophizing were seen among younger people and were correlated with characteristic pain intensity (r = 0.55; P < .0001) and pain-related disability (r= 0.43, P < .0001).
Turner et al[35] (2005a), USA	TMD clinic, cross-sectional case series	338 (87%) TMD patients, 37 y	GCPS, CPI disability score, CSQ catastrophizing subscale (0–6), MFIQ	After correction for age, gender, and education, catastrophizing (mean [SD]: 1.7 [1.3]) explained 10% of muscle palpation (P < .001), 3% of TMJ pain on palpation (P < .01), 18% of pain intensity (r = 0.42, P < .001), and 25% of pain-related disability (r = 0.5, P < .001).
Velly et al[47] (2011), USA	Population sample, prospective cohort	570 (88%) TMD patients	CSQ catastrophizing subscale, GCPS (CPI + disability score)	Catastrophizing at baseline, corrected for age, gender, and pain intensity, contributed to an increase in pain intensity and disability at the 18-month follow-up (β = 3.79, P < .0001). Catastrophizing, corrected for depression, pain intensity, age, gender, and widespread pain, was a predictor for onset (OR: 1.71, 95% CI: 1.09–2.30; P = .02) and progression (OR: 2.16, 95% CI: 1.62–2.87; P < .0001) of clinically significant pain.

BPI = Brief Pain Inventory; CF-PDI = Craniofacial Pain and Disability Inventory; CPI = characteristic pain intensity; CPSQ = Comprehensive Pain Symptom Questionnaire; CSQ = Coping Strategies Questionnaire; GAD-7 = General Anxiety Disorder-7; GCPS = Graded Chronic Pain Scale; HPT = heat pain threshold; MPI = Multidimensional Pain Inventory; PCS = Pain Catastrophizing Scale; PHQ-9/-15: Patient Health Questionnaire-9/-15; PPT = pressure pain threshold; PRSS = Pain-Related Self-Statement Scale.

Conclusions	Limitations
These findings identify pain catastrophizing as a potentially important link between ethnicity and clinical pain and suggest that interventions targeting pain-related helplessness could improve both sleep and pain, especially for African American patients.	
Hierarchal linear regression models using both dispositional and momentary predictors indicated that momentary pain was a function of both dispositional tendency to catastrophize and momentary measures of catastrophizing, self-efficacy, and mood states.	
This article presents the results of a multivariable model designed to discriminate between people with high- and low-impact pain in a community-based sample of painful chronic TMD. The findings emphasize the importance of catastrophizing, jaw limitation, and painful body sites associated with pain-related impact.	
Neurophysiologic mechanisms by which pain catastrophizing is related to acute and chronic pain recently have come under empirical study. Understanding of these mechanisms has the unique potential to shed light on key central nervous system factors that mediate catastrophizing pain relations and therapeutic benefits associated with changes in catastrophizing and related cognitive processes.	
High pain catastrophizing TMD patients were similar to patients with other chronic pain conditions, but differed from TMD patients as a group. The findings of this study support the addition of an assessment for pain catastrophizing to the DC/TMD for early identification of TMD patients who might be at higher risk for developing chronic pain.	
The results suggest that for patients with moderate or high levels of TMD pain and dysfunction, beliefs about pain play an important role in physical and psychosocial functioning.	Of 187 eligible patients, only 118 (63%) enrolled in the study.
Catastrophizing is stable over short periods of time in the absence of a substantial change in pain within patients, and times of greater catastrophizing are associated with worse pain, disability, and mood.	Of 244 eligible patients, only 110 (45%) enrolled. There was likely some overlap with the study population of Turner et al[42] (2001).
TMD patients who catastrophize have higher scores on clinical examination measures, reflecting more widely dispersed and severe pain upon palpation of TMD-related facial activity interference and health care use.	Of 722 eligible patients, only 338 (47%) enrolled. There was an overlap with the study populations of Turner et al[42] (2001) and Turner et al[39] (2004).
Results indicate that catastrophizing and depression contribute to the progression of chronic TMD pain and disability, and therefore should be considered as important factors when evaluating and developing treatment plans for patients with TMD.	

Table 3b Summary of Treatment Outcomes in Relation to Catastrophizing (n = 9)

Study (y), country	Setting, study design	Patients, n (% W), mean age (range)	Evaluation and treatment methods	Association/treatment outcome
Costa et al[75] (2015), Brazil	Hospital orofacial pain clinic, RCT	60 (90%) TMD patients, 32 y T1: n = 30 T2: n = 30	PRSS **catastrophizing** subscale T1: Counseling T2: Counseling + splint	Baseline mean (SD) PRSS: 2.1 (1.3) Follow-up: T1: 1.1 (1.3); T2: 0.8 (0.8) There was a significant reduction in pain catastrophizing in both groups ($P < .05$). Dropouts: T1: 13/30 (43%); T2: 6/30 (20%)
Durá-Ferrandis et al[52] (2017), Spain	Orofacial pain clinic, case-control	72 TMD patients, 39 y (17–75 y) T1: 41 (87%) T2: 31 (91%)	PCS, GCPS T1: CBT T2: ST (splint, jaw exercises, NSAIDs)	Dropouts: CBT: 11/41 (27%) ST: 2/31 (6.5%) CBT reduced PCS (estimated effect: **0.39**, $P < .05$). PCS modified effect of treatment on pain intensity (estimated effect: **0.3**, $P < .05$).
Litt et al[50] (2009), USA	Dental clinic + advertising,RCT	54 (85%) TMD patients, 41 y T1: 22 T2: 32	CSQ **catastrophizing** subscale (2 items), MPI pain T1: ST (splint, NSAIDs) T2: ST + CBT	Patients with ST + CBT reported a greater but nonsignificant decrease in pain compared to ST (1.4 vs 0.6). The CBT group showed a greater decrease in catastrophizing ($F = 7.44$, $P < .01$). Catastrophizing predicted momentary pain (estimated effect: **1.23**, $F = 18.91$, $P < .001$).
Litt et al[51] (2010), USA	Dental clinic + advertising,RCT	101 (84%) TMD patients, 41 y T1: 49 T2: 52	CSQ **catastrophizing** subscale (2 items), MPI pain T1: ST (splint, NSAIDs) T2: ST + CBT	Patients with ST + CBT showed steeper decreases in pain over time compared to the ST ($F = 6.57$; $P < .01$). Catastrophizing moderated the treatment effect for pain ($\beta = 0.64$, SE = 0.12, $F = 32.07$, $P < .007$) and pain interference ($\beta = 0.62$, SE = 0.14, $F = 25.72$, $P < .007$).
Litt et al[49] (2013), USA	Dental clinic + advertising, RCT	101 (84%) TMD patients with pain > 3 mo, 39 y	PRSS catastrophizing subscale (0–5), MPI pain T1: Splint + NSAIDs T2: Splint + NSAIDS + CBT	Nonresponders (16%) reported more psychiatric symptoms, poorer coping, and higher levels of catastrophizing. Predictors for treatment responders included addition of CBT to ST, higher treatment attendance, and larger decrease in catastrophizing.
Turner et al[40] (2005b), USA	Hospital orofacial pain clinic, RCT	126 (88%) TMD patients, 37 y T1: 61 T2: 65	Catastrophizing: three questions adapted from CSQ and PCS rumination subscales (0–10) T1: CBT T2: Self-care	The CBT group showed a significant ($P < .0001$) decrease in catastrophizing compared to self-care (2.5 [2.4] vs 1.8 [2.2]). A larger proportion (34% vs 13%, $P < .05$) of the patients who received CBT showed clinically important (50%) improvement in activity interference and jaw limitations.
Turner et al[54] (2006), USA	Hospital orofacial pain clinic, RCT	158 (87%) TMD patients T1: 79 T2: 79	CSQ catastrophizing subscale (0–6), PCS rumination subscale, CPI, GCPS T1: CBT T2: Self-care	After 12 mo, the CBT group showed a greater decrease of activity interference, with 35% vs 13% reporting no interference ($P = .004$), mean CPI 3.9 vs 4.7 ($P = .02$), and no catastrophizing in 29% vs 4% of patients ($P < .0001$).
Turner et al[53] (2007), USA	Hospital orofacial pain clinic, RCT	115 (87%) TMD patients T1: 55 T2: 60	CSQ catastrophizing subscale (0–6) PCS rumination subscale, CPI, GCPS T1: CBT T2: Self-care	Mediators for treatment outcome of CBT vs self care were evaluated. Patients who reported more pain sites, depressive symptoms, and catastrophizing at baseline had greater activity interference at 1 y. Catastrophizing was a mediator of CBT effects on activity interference (–0.59; 95% CI: –1.11,–0.31) which explained 46% of the total effect, and pain intensity (–0.44; 95% CI: –0.94, –0.12), which explained 30% of the total effect.
Turner et al[55] (2011), USA	Hospital orofacial pain clinic + advertising,RCT	171 (100)% TMD patients T1: 59 T2: 55 T3: 57	CSQ, GCPS T1: Self-care T2: Modified self-care T3: Oral contraceptives	Targeting self-management to menstrual cycle–related symptoms did not increase the treatment efficacy. Pain was lower with self-management ($P = .003$) compared to oral contraceptives at 12 mo, but not at 6 mo. CSQ was lower in the oral contraceptives group at 6 mo, ($P = .04$), but not at 12 mo.

CBT = cognitive-behavioral treatment; CPI = characteristic pain intensity; CSQ = Coping Strategies Questionnaire; GCPS = Graded Chronic Pain Scale; PCS = Pain Catastrophizing Scale; PRSS = pain-related self-statement scale; MPI = Multidimensional Pain Inventory; NSAIDs = nonsteroidal anti-inflammatory drugs; ST = standard treatment.

Conclusion	Limitations
Minimally invasive strategies could provide an improvement in the psychologic aspects of TMD patients, and the use of an occlusal splint seems to hasten the manifestation of these effects.	There was a large dropout.
The results could set the principles for the development of more efficient and effective cognitive behavioral interventions for chronic pain.	There was a large dropout in the CBT group.
The results suggest that CBT for TMD pain can help patients alter their coping behaviors and that these changes translate into improved outcomes.	
It was concluded that brief treatments can yield significant reductions in pain, life interference, and depressive symptoms in TMD patients and that the addition of cognitive-behavioral coping skills will add to treatment efficacy, especially for those low in somatization or high in readiness or self-efficacy.	There was a possible overlap of the study population with Litt et al,[50] 2009.
It was concluded that CBT may be made more efficacious for TMD patients by placing further emphasis on decreasing catastrophizing and on individualizing care. This article provides evidence that the TMD chronic pain population is heterogenous and that a subsample of patients will be unresponsive to standard or psychosocial approaches. The addition of CBT to treatment may be helpful for this group, but new, individualized approaches will be needed to treat all patients effectively.	The study population was the same as in Litt et al,[51] 2010.
The brief CBT was efficacious in decreasing catastrophizing, increasing perceived control over pain, and improving activity interference and jaw use limitations for a subgroup of patients.	Of 366 eligible patients, only 158 (43%) enrolled in the study. A further 32 (20%) were not part of the follow-up analysis.
A brief CBT intervention improves 1-y clinical outcomes of TMD clinical patients, and these effects appear to result from specific CBT interventions.	Of 366 eligible patients, only 158 (43%) enrolled in the study. The study population was similar to Turner et al, 2005.[40]
The results provide further support for cognitive-behavioral models of chronic pain and point to the potential benefits of interventions to modify specific pain-related beliefs in CBT and other health care encounters.	Subset of the study population in Turner et al,[54] 2006.
The study provides further support for long-term benefits of a safe, low-intensity (two in-person sessions and six brief telephone calls), dental hygienist–delivered self-management treatment for TMD pain.	Large dropout (> 50%) in the oral contraceptives group.

Study, y	Mean	95% CI	Weight (%)
Brandini et al,[56] 2011	11.0	6.69, 15.31	1.1
Campbell et al,[44] 2010	9.5	7.54, 11.46	5.3
Chen et al,[58] 2012	6.8	−3, 16.6	0.2
Fillingim et al,[61] 2011	9.5	9, 9.99	84.7
Gustin et al,[62] 2011	10.1	7.36, 12.84	2.7
Hollins et al,[63] 2009	8.4	5.26, 11.54	2.1
Kothari et al,[65] 2017	10.3	7.36, 13.24	2.4
La Touche et al,[36] 2015	5.5	4.95, 6.05	0.1
Quartana et al,[67] 2010	8.9	5.2, 12.62	1.5
Total	**9.51**	**9.06, 9.96**	**100.00**

Levels of catastrophizing (random effects)

1 5 10 15
9.51

a I^2 = 0%.

Study, y	Mean	95% CI	Weight (%)
Brandini et al,[57] 2011	12.7	7.41, 17. 99	6.2
Campbell et al,[44] 2010	14.3	11.75, 16.85	7.8
Chen et al,[58] 2012	10.7	8.54, 12.86	8
Davis et al,[34] 2014	15.7	12.37, 19.03	6.9
Fillingim et al,[61] 2011	14.1	12.34, 15.86	8.1
Gil-Martínez et al,[37] 2017	26.4	24.05, 28.75	7.9
Gustin et al,[62] 2011	18.7	14, 23.4	6.6
Hollins et al,[63] 2009	12.6	8.68, 16.52	7.1
Kothari et al,[65] 2017	20.7	17.96, 23.44	7.7
La Touche et al,[43] 2014	23.7	22.52, 24.88	8.3
La Touche et al,[36] 2015	16.4	15.62, 17.18	8.4
Lerman et al,[33] 2018	21.4	19.83, 22.97	8.2
Quartana et al,[67] 2010	14.0	11.26, 16.74	7.7
Reiter et al,[48] 2018	21.4	18.88, 23.98	7.8
Total	**17.6**	**15, 20.2**	**100.00**

Levels of catastrophizing (random effects)

10 15 17.6 20 25

b I^2 = 96%.

Study, y	Mean	95% CI	Weight (%)
Brandini et al,[57] 2011	0.17	−0.57, 0.92	9.5
Campbell et al,[44] 2010	0.53	0.17, 0.89	11.8
Chen et al,[58] 2012	0.51	0.25, 0.78	12.3
Fillingim et al,[61] 2011	0.45	0.29, 0.6	12.6
Gustin et al,[62] 2011	0.88	0.33, 1.45	10.6
Hollins et al,[63] 2009	0.5	−0.12, 1.13	10.3
Kothari et al,[65] 2017	0.98	0.56, 1.41	11.5
La Touche et al,[36] 2015	3.21	2.67, 3.78	10.6
Quartana et al,[67] 2010	0.62	0.09, 1.16	10.8
Total	**0.86**	**0.18, 1.54**	**100.00**

Effect size (random effects)

−0.5 0 0.5 0.86 1 1.5 2 2.5 3 3.5

z value = 2.91, *P* = .004, I^2 = 91.6%.

c

Fig 2 Forest plots based on random-effects meta-analysis for levels of catastrophizing on the Pain Catastrophizing Scale for **(a)** healthy control groups (n = 9) and **(b)** TMD groups (n = 14), together with **(c)** the effect size (Hedges' g) in studies including both a TMD group and a control group (n = 9).

Discussion

The main finding from this systematic review was that patients with TMD report higher levels of catastrophizing compared to controls. Furthermore, an association was seen between higher levels of catastrophizing and higher TMD symptom severity, as well as between higher levels of catastrophizing and poorer TMD treatment outcome. In addition, the included studies suggested positive effects of CBT treatment on the catastrophizing levels in patients with TMD.

The etiology of TMD is considered to be multifactorial, where contributing factors such as parafunctional habits, trauma, pain in other parts of the body, stress, and emotional distress are among those that

can increase risk and initiate and perpetuate the condition in the biopsychosocial model of pain.[9] Thus, psychosocial factors play an important role in chronic pain conditions, including chronic TMD pain.[69] Comorbidity with a range of psychosocial factors, such as depression, anxiety, and catastrophizing, has been investigated in patients with TMD.[70] Such comorbidities may affect the existing situation for the patients and the outcome of any self-management or treatment prescribed by the care provider.[20]

The strength of results from a meta-analysis is highly dependent on the quality of the included primary studies. While robust risk of bias instruments exist for randomized and nonrandomized controlled trials, quality assessment of case-control studies, cohort studies, and case series is more challenging. As the NOS covers both case-control and cohort studies and has also been used for case series studies, it was the tool chosen for the present review, although low interrater reliability has been reported for the NOS when used for cohort studies.[71]

In the present quality assessment, a majority of the studies that received a low score were case-series studies or case-control studies without TMD-free control groups. Thus, the low-quality scores were largely related to study design. By design, a case-control study cannot differentiate between psychosocial symptoms as a risk factor for or as a consequence of developing a chronic TMD pain condition. The risk of bias assessment in the present review was based on the specific aim to compare TMD patients to individuals without TMD. This means that the quality scores of the included case-control studies could be higher when risk of bias is assessed in relation to the specific aim of the respective studies.

For the meta-analysis, the PCS total score was chosen as the primary outcome measure, and this scale was also used in a majority of the studies included in the present review. Good internal reliability and test-retest reliability have been reported for PCS total scores, but not for the subscales.[72] The main reason for studies being excluded from the meta-analysis was due to reporting catastrophizing with other outcome measures, such as the Coping Strategies Questionnaire or the Pain-Related Self-Statements Scale, or because they did not report both central measures and dispersion values, which are necessary for performing a meta-analysis.

For the control groups included in the meta-analysis, there was little variation in catastrophizing levels among the studies, and the calculated mean values were within the normal reference cut-offs. This indicates that the control groups were homogenous and representative of the general population and of pain-free individuals.[27]

The levels of catastrophizing found in patients with TMD are in line with those reported in a recent systematic review that examined patients with different pain conditions, including head and neck pain and generalized pain.[72] The mean overall outcome for catastrophizing (17.6 on the PCS) found in the meta-analysis in the present study is still categorized as a relatively low level of catastrophizing. A cut-off of 23 has been suggested for high catastrophizing, and scores > 30 proposed as clinically relevant.

The finding of higher, albeit varying, levels of catastrophizing in TMD groups compared to TMD-free controls in the primary studies included in the present review is in line with reports for other pain conditions.[72] One explanation for the different levels of catastrophizing found for different TMD groups in the present review could be the considerable heterogeneity between studies with regard to study population. Although the diagnosis of TMD was standardized, only study populations defined by the RDC/TMD or DC/TMD criteria were included; the patient groups still differed with regard to other aspects, such as distribution of gender and age; comorbidity with other psychologic conditions or with presence of headache, migraine, and pain in other areas of the body; and pain chronicity. All of these factors are proposed to influence levels of catastrophizing.[72] Gender differences in chronic pain conditions, including chronic TMD pain, are assumed to be a result of differences in behavioral, hormonal, and psychosocial factors, although this complex interplay is not fully understood.[73] However, even though some studies have indicated higher levels of catastrophizing in women compared to men, the recent systematic review by Wheeler et al based on a meta-analysis of 220 primary studies concluded that levels of catastrophizing were not related to age or gender, but rather related to the type of pain condition, with the highest levels in individuals with generalized pain.[72] It is therefore reasonable to assume that the differences between the TMD groups in the present review are attributed to differences in pain comorbidity, such as migraine, pain in other areas of the body, and presence of generalized pain.

It was possible to conduct a meta-analysis in a subgroup of the included studies, providing additional statistical strength of evidence for the relatively higher levels of catastrophizing in patients with TMD compared to control groups without TMD pain. A majority of the primary studies included in the meta-analysis also included control groups without TMD pain, demonstrating significantly lower levels of catastrophizing in pain-free controls compared to patients with TMD pain. This was confirmed by the overall large effect size when the TMD groups were compared to the control groups.

In addition to the findings of a higher level of catastrophizing in TMD patients, the qualitative synthesis of the reported quantitative data suggested an association between levels of catastrophizing and severity of TMD. Pain catastrophizing was positively associated with TMD pain–related factors, such as pain intensity,[33] pain interference,[34] pain on palpation,[35] fatigue and pain in a provocation chewing test,[36] and neck disability.[37] Furthermore, an association between level of catastrophizing and number of health care visits[35] was reported. The included primary studies generally demonstrated moderate correlations between catastrophizing and pain outcomes. Catastrophizing was also associated with higher pain[45,46] and with onset, progression, and persistence of pain.[47,48]

Thus, levels of catastrophizing were related to pain intensity and pain interference, pain on palpation, fatigue and pain in a provocation chewing test, and number of health care visits. Furthermore, higher levels of catastrophizing before treatment were related to being a nonresponder to treatment and reporting higher activity interference 1 year later. Taken together, these findings are in line with other studies in patients with TMD and other pain conditions[36,48] that showed an association between catastrophizing and a range of symptom severity, affecting both patient suffering and health care utilization. This highlights the costs of chronic orofacial pain both for the individual patient and for society.

There were also a number of primary studies in the present review evaluating the outcome of CBT in patients with TMD, mainly reporting positive effects on outcome measures such as pain intensity, pain interference, and coping.[40,50–52] A meta-analysis was not deemed appropriate for these treatment studies due to differences in outcome measures, treatment modalities, assessment, definitions of patient groups, and overlap in study populations among the studies. Of the nine primary treatment studies, a possible overlap in patient samples was found in six.[40,49–51,53,54] The results from the studies suggested that CBT, as the only treatment provided or compared to self-care or standard treatment (splint, NSAIDs, etc), can reduce catastrophizing and pain in TMD patients, thereby improving treatment prognosis and outcome. These findings are in line with a previous study showing that a chronic pain trajectory can be modified favorably by a treatment that includes cognitive-behavioral skill training and biofeedback. At a 1-year follow-up, patients in the intervention group showed lower pain intensity, less severe depression, and better coping strategies than patients from the nonintervention group.[74]

Taken together, the findings in the present systematic review highlight the importance of psychosocial screening of patients with TMD, as was suggested by Dworkin and LeReche in 1992 with the introduction of the RDC/TMD. With the DC/TMD criteria, the instruments for psychosocial assessment have been further refined and are also provided on two levels: a screening level and a comprehensive level.[14] The benefit of this is that it provides instruments for a more comprehensive assessment in orofacial pain clinics, as well as at a more basic screening level suitable for general dental practioners.[15,75] Catastrophizing is not, however, currently part of the comprehensive DC/TMD Axis II assessment, but could be suggested to be an additional component when an extended comprehensive assessment is deemed necessary. The associations between catastrophizing and poorer treatment outcome, together with indications of positive effects from CBT, might be of clinical significance. Future studies should investigate whether catastrophizing is a relevant indicator for treatment outcomes in patients with TMD and therefore valuable for tailored treatment decisions in the dental clinic.[15]

In order to evaluate the possible impact of catastrophizing as a risk factor for development and chronification of TMD pain, it would be imperative to use a longitudinal cohort study design including patients with and without TMD pain, to use specified diagnostic criteria according to the DC/TMD, and instruments with a good reliability—such as the PCS—for assessing catastrophizing. In addition, treatment effectiveness studies in patients with TMD should focus on specific psychologic variables, such as catastrophizing, which could be used to tailor the treatment to the specific demands of the patient. For example, studies that examine individual data for response trajectories are recommended.[72]

Conclusions

The results from the studies included in the present review suggest an association between catastrophizing and TMD that may affect not only symptom severity but also treatment outcome. These findings suggest that assessment of levels of pain catastrophizing might be valuable in the assessment and management of patients with TMD.

Clinical Implications

- Patients with higher levels of catastrophizing often have higher pain intensity and more pain interference.
- For assessment of pain catastrophizing, the PCS can be included in the psychosocial assessment of chronic pain patients.
- For patients with high levels of catastrophizing, the clinician may consider referral for CBT.

Acknowledgments

This study was conducted without any funding. The authors declare no conflicts of interest in connection with this article. Preliminary results from this paper were presented as an oral presentation at the International Association for Dental Research meeting, Vancouver, Canada on 21st June 2019.

E.C.E., B.C., B.B.P. and B.H.H. together created the original concept and design. Abstract screening and full text assessment were carried out by BHH, ECE, BC and BBP. Data extraction, quality assessment and analysis were carried out by BHH and CMV. Interpretation of data was done by all authors. All authors revised the manuscript for intellectual content and approved the final version of the manuscript.

References

1. Sullivan MJ, Lynch ME, Clark AJ. Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions. Pain 2005;113:310–315.
2. Sullivan MJ, Thorn B, Haythornthwaite JA, et al. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain 2001;17:52–64.
3. Wertli MM, Burgstaller JM, Weiser S, Steurer J, Kofmehl R, Held U. Influence of catastrophizing on treatment outcome in patients with nonspecific low back pain: A systematic review. Spine (Phila Pa 1976) 2014;39:263–273.
4. Spinhoven P, Ter Kuile M, Kole-Snijders AM, et al. Catastrophizing and internal pain control as mediators of outcome in the multidisciplinary treatment of chronic low back pain. Eur J Pain 2004;8:211–219.
5. Severeijns R, Vlaeyen JW, van den Hout MA, Picavet HS. Pain catastrophizing is associated with health indices in musculoskeletal pain: A cross-sectional study in the Dutch community. Health Psychol 2004;23:49–57.
6. Severeijns R, Vlaeyen JW, van den Hout MA, Weber WE. Pain catastrophizing predicts pain intensity, disability, and psychological distress independent of the level of physical impairment. Clin J Pain 2001;17:165–172.
7. Sullivan MJ, Feuerstein M, Gatchel R, Linton SJ, Pranksy G. Integrating psychosocial and behavioral interventions to achieve optimal rehabilitation outcomes. J Occup Rehabil 2005;15:475–489.
8. Burns JW, Glenn B, Bruehl S, Harden RN, Lofland K. Cognitive factors influence outcome following multidisciplinary chronic pain treatment: A replication and extension of a cross-lagged panel analysis. Behav Res Ther 2003;41:1163–1182.
9. Dworkin SF, LeResche L. Research Diagnostic criteria for Temporomandibular Disorders: Review, criteria, examinations and specifications, critique. J Craniomand Disord 1992;6:301–355.
10. Gillborg S, Åkerman S, Lundegren N, Ekberg EC. Temporomandibular disorder pain and related factors in an adult population: A cross-sectional study in Southern Sweden. J Oral Facial Pain Headache 2017;31:37–45.
11. LeResche L. Epidemiology of temporomandibular disorders: Implications for the investigation of etiologic factors. Crit Rev Oral Biol Med 1997;8:291–305.
12. Häggman-Henrikson B, Ilgunas A, Visscher C, et al. Increasing gender differences in the prevalence and chronification of orofacial pain in the population. Pain 2020;161:1768–1775.
13. Fillingim RB, Ohrbach R, Greenspan JD, et al. Psychological factors associated with development of TMD: The OPPERA prospective cohort study. J Pain 2013;14(12 suppl):T75–T90.
14. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for clinical and research applications: Recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache 2014;28:6–27.
15. Visscher CM, Baad-Hansen L, Durham J, et al. Benefits of implementing pain-related disability and psychological assessment in dental practice for patients with temporomandibular pain and other oral health conditions. J Am Dent Assoc 2018;149:422–431.
16. Chen H, Slade G, Lim PF, Miller V, Maixner W, Diatchenko L. Relationship between temporomandibular disorders, widespread palpation tenderness, and multiple pain conditions: A case-control study. J Pain 2012;13:1016–1027.
17. De La Torre Canales G, Câmara-Souza MB, Muñoz Lora VRM, et al. Prevalence of psychosocial impairment in temporomandibular disorder patients: A systematic review. J Oral Rehabil 2018;45:881–889.
18. Aghabeigi B, Feinmann C, Harris M. Prevalence of post-traumatic stress disorder in patients with chronic idiopathic facial pain. Br J Oral Maxillofac Surg 1992;30:360–364.
19. Carlson CR, Reid KI, Curran SL. Psychological and physiological parameters of masticatory muscle pain. Pain 1998;76:297–307.
20. Huttunen J, Qvintus V, Suominen AL, Sipilä K. Role of psychosocial factors on treatment outcome of temporomandibular disorders. Acta Odontol Scand 2019;77:119–125.
21. Häggman-Henrikson B, Alstergren P, Davidson T, et al. Pharmacological treatment of oro-facial pain—Health technology assessment including a systematic review with network meta-analysis. J Oral Rehabil 2017;44:800–826.
22. List T, Axelsson S. Management of TMD: Evidence from systematic reviews and meta-analyses. J Oral Rehabil 2010;37:430–451.
23. Litt MD, Porto FB. Determinants of pain treatment response and nonresponse: Identification of TMD patient subgroups. J Pain 2013;14:1502–1513.
24. Williams AC, Eccleston C, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults. Cochrane Database Syst Rev 2012;11:CD007407.
25. Waters SJ, McKee DC, Keefe FJ. Cognitive behavioral approaches to the treatment of pain. Psychopharmacol Bull 2007;40:74–88.
26. Moher D, Liberati A, Tetzlaff J, Altman DG, PRISMA group. Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. PLoS Med 2009;6:e1000097.
27. Osman A, Barrios FX, Kopper BA, Hauptmann W, Jones J, O'Neill E. Factor structure, reliability, and validity of the Pain Catastrophizing Scale. J Behav Med 1997;20:589–605.
28. Darnall BD, Sturgeon JA, Cook KF, et al. Development and validation of a daily Pain Catastrophizing Scale. J Pain 2017;18:1139–1149.
29. Stang A. Critical evaluation of the Newcastle-Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. Eur J Epidemiol 2010;25:603–605.
30. Cohen J. Statistical Power Analysis for the Behavioral Sciences, ed 2. Hillsdale, NJ: Lawrence Erlbaum, 1988.
31. Suurmond R, van Rhee H, Hak T. Introduction, comparison, and validation of meta-essentials: A free and simple tool for meta-analysis. Res Synth Methods 2017;8:537–553.
32. Evans JD. Straightforward Statistics for the Behavioral Sciences. Pacific Grove, CA: Brooks/Cole, 1996.

33. Lerman SF, Campbell CM, Buenaver LF, et al. Exploring the role of negative cognitions in the relationship between ethnicity, sleep, and pain in women with temporomandibular joint disorder. J Pain 2018;19:1342–1351.
34. Davis CE, Stockstill JW, Stanley WD, Qu Q. Pain-related worry in patients with chronic orofacial pain. J Am Dent Assoc 2014;145:722–730.
35. Turner JA, Brister H, Huggins K, Mancl L, Aaron LA, Truelove EL. Catastrophizing is associated with clinical examination findings, activity interference, and health care use among patients with temporomandibular disorders. J Orofac Pain 2005;19:291–300.
36. La Touche R, Paris-Alemany A, Gil-Martínez A, et al. Masticatory sensory-motor changes after an experimental chewing test influenced by pain catastrophizing and neck-pain–related disability in patients with headache attributed to temporomandibular disorders. J Headache Pain 2015;16:20.
37. Gil-Martínez A, Navarro-Fernández G, Mangas-Guijarro MA, et al. Comparison between chronic migraine and temporomandibular disorders in pain-related disability and fear-avoidance behaviors. Pain Med 2017;18:2214–2223.
38. Castrillon EE, Cairns BE, Ernberg M, et al. Glutamate-evoked jaw muscle pain as a model of persistent myofascial TMD pain? Arch Oral Biol 2008;53:666–676.
39. Turner JA, Mancl L, Aaron LA. Pain-related catastrophizing: A daily process study. Pain 2004;110:103–111.
40. Turner JA, Mancl L, Aaron LA. Brief cognitive-behavioral therapy for temporomandibular disorder pain: Effects on daily electronic outcome and process measures. Pain 2005;117:377–387.
41. Buenaver LF, Quartana PJ, Grace EG, et al. Evidence for indirect effects of pain catastrophizing on clinical pain among myofascial temporomandibular disorder participants: The mediating role of sleep disturbance. Pain 2012;153:1159–1166.
42. Turner JA, Dworkin SF, Mancl L, Huggins KH, Truelove EL. The roles of beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorders. Pain 2001;92:41–51.
43. La Touche R, Pardo-Montero J, Gil-Martínez A, et al. Craniofacial pain and disability inventory (CF-PDI): Development and psychometric validation of a new questionnaire. Pain Physician 2014;17:95–108.
44. Campbell CM, Kronfli T, Buenaver LF, et al. Situational versus dispositional measurement of catastrophizing: Associations with pain responses in multiple samples. J Pain 2010;11:443–453.e2.
45. Litt MD, Shafer D, Napolitano C. Momentary mood and coping processes in TMD pain. Health Psychol 2004;23:354–362.
46. Miller VE, Poole C, Golightly Y, et al. Characteristics associated with high-impact pain in people with temporomandibular disorder: A cross-sectional study. J Pain 2018;20:288–300.
47. Velly AM, Look JO, Carlson C, et al. The effect of catastrophizing and depression on chronic pain—A prospective cohort study of temporomandibular muscle and joint pain disorders. Pain 2011;152:2377–2383.
48. Reiter S, Eli I, Mahameed M, et al. Pain catastrophizing and pain persistence in temporomandibular disorder patients. J Oral Facial Pain Headache 2018;32:309–320.
49. Litt MD, Porto FB. Determinants of pain treatment response and nonresponse: Identification of TMD patient subgroups. J Pain 2013;14:1502–1513.
50. Litt MD, Shafer DM, Ibanez CR, Kreutzer DL, Tawfik-Yonkers Z. Momentary pain and coping in temporomandibular disorder pain: Exploring mechanisms of cognitive behavioral treatment for chronic pain. Pain 2009;145:160–168.
51. Litt MD, Shafer DM, Kreutzer DL. Brief cognitive-behavioral treatment for TMD pain: Long-term outcomes and moderators of treatment. Pain 2010;151:110–116.
52. Durá-Ferrandis E, Ferrando-García M, Galdón-Garrido J, Andreu-Vaillo Y. Confirming the mechanisms behind cognitive-behavioural therapy effectiveness in chronic pain using structural equation modeling in a sample of patients with temporomandibular disorders. Clin Psychol Psychother 2017;24:1377–1383.
53. Turner JA, Holtzman S, Mancl L. Mediators, moderators, and predictors of therapeutic change in cognitive-behavioral therapy for chronic pain. Pain 2007;127:276–286.
54. Turner JA, Mancl L, Aaron LA. Short- and long-term efficacy of brief cognitive-behavioral therapy for patients with chronic temporomandibular disorder pain: A randomized, controlled trial. Pain 2006;121:181–194.
55. Turner JA, Mancl L, Huggins KH, Sherman JJ, Lentz G, LeResche L. Targeting temporomandibular disorder pain treatment to hormonal fluctuations: A randomized clinical trial. Pain 2011;152:2074–2084.
56. Bair E, Ohrbach R, Fillingim RB, et al. Multivariable modeling of phenotypic risk factors for first-onset TMD: The OPPERA prospective cohort study. J Pain 2013;14(12 suppl):T102–T115.
57. Brandini DA, Benson J, Nicholas MK, Murray GM, Peck CC. Chewing in temporomandibular disorder patients: An exploratory study of an association with some psychological variables. J Orofac Pain 2011;25:56–67.
58. Chen H, Slade G, Lim PF, Miller V, Maixner W, Diatchenko L. Relationship between temporomandibular disorders, widespread palpation tenderness, and multiple pain conditions: A case-control study. J Pain 2012;13:1016–1027.
59. Chen H, Nackley A, Miller V, Diatchenko L, Maixner W. Multisystem dysregulation in painful temporomandibular disorders. J Pain 2013;14:983–996.
60. Costa YM, Alves da Costa DR, de Lima Ferreira AP, et al. Headache exacerbates pain characteristics in temporomandibular disorders. J Oral Facial Pain Headache 2017;31:339–345.
61. Fillingim RB, Ohrbach R, Greenspan JD, et al. Potential psychosocial risk factors for chronic TMD: Descriptive data and empirically identified domains from the OPPERA case-control study. J Pain 2011;12(11 suppl):T46–T60.
62. Gustin SM, Wilcox SL, Peck CC, Murray GM, Henderson LA. Similarity of suffering: Equivalence of psychological and psychosocial factors in neuropathic and non-neuropathic orofacial pain patients. Pain 2011;152:825–832.
63. Hollins M, Harper D, Gallagher S, et al. Perceived intensity and unpleasantness of cutaneous and auditory stimuli: An evaluation of the generalized hypervigilance hypothesis. Pain 2009;141:215–221.
64. Jerjes W, Madland G, Feinmann C, et al. A psychological comparison of temporomandibular disorder and chronic daily headache: Are there targets for therapeutic interventions? Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:367–373.
65. Kothari SF, Baad-Hansen L, Svensson P. Psychosocial profiles of temporomandibular disorder pain patients: Proposal of a new approach to present complex data. J Oral Facial Pain Headache 2017;31:199–209.
66. Kotiranta U, Suvinen T, Kauko T, et al. Subtyping patients with temporomandibular disorders in a primary health care setting on the basis of the Research Diagnostic Criteria for Temporomandibular Disorders Axis II pain-related disability: A step toward tailored treatment planning? J Oral Facial Pain Headache 2015;29:126–134.

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;11:186–194.
68. Costa YM, Porporatti AL, Stuginski-Barbosa J, Bonjardim LR, Conti PC. Additional effect of occlusal splints on the improvement of psychological aspects in temporomandibular disorder subjects: A randomized controlled trial. Arch Oral Biol 2015;60:738–744.
69. Manfredini D, Landi N, Bandettini Di Poggio A, Dell'Osso L, Bosco M. A critical review on the importance of psychological factors in temporomandibular disorders. Minerva Stomatol 2003;52:321–326, 327–330.
70. De Laat A, Meuleman H, Stevens A, Verbeke G. Correlation between cervical spine and temporomandibular disorders. Clin Oral Investig 1998;2:54–57.
71. Hartling L, Milne A, eHamm MP, et al. Testing the Newcastle Ottawa Scale showed low reliability between individual reviewers. J Clin Epidemiol 2013;66:982–993.
72. Wheeler CHB, Williams ACC, Morley SJ. Meta-analysis of the psychometric properties of the Pain Catastrophizing Scale and associations with participant characteristics. Pain 2019;160:1946–1953.
73. Visscher CM, Lobbezoo F. TMD pain is partly heritable. A systematic review of family studies and genetic association studies. J Oral Rehabil 2015;42:386–399.
74. Gatchel RJ, Stowell AW, Wildenstein L, Riggs R, Ellis E 3rd. Efficacy of an early intervention for patients with acute temporomandibular disorder-related pain: A one-year outcome study. J Am Dent Assoc 2006;137:339–347.
75. Häggman Henrikson B, Ekberg E, Ettlin DA, et al. Mind the gap: A systematic review of implementation of screening for psychological comorbidity in dental and dental hygiene education. J Dent Educ 2018;82:1065–1076.

Appendix 1 Articles Excluded During Full-Text Assessment and Main Reasons for Exclusion (n = 22)

Main reason for exclusion	Studies, n	Study, y
Did not use RDC/TMD or DC/TMD for diagnosis	9	Flor et al,[1] 1993 Greco et al,[2] 1997 Jang et al,[3] 2018 Kucyi et al,[4] 2014 Madland et al,[5] 2000 Roditi et al,[6] 2009 Rollman et al,[7] 2013 Rollman et al,[8] 2012 Van Damme et al,[9] 2018
Not TMD population	2	Dagsdóttir et al,[10] 2016 de Boer et al,[11] 2014
No specific data on catastrophizing for TMD group	8	Aguiar et al,[12] 2017 Conti et al,[13] 2012 Dagsdóttir et al,[14] 2015 Diernberger et al,[15] 2008 Harfeldt et al,[16] 2018 Ingram et al,[17] 2011 Kapos et al,[18] 2018 Muñoz-García et al,[19] 2017
Same study population as another included study	1	Gil-Martínez et al,[20] 2016
Commentary or letter to editor	1	Lautenbacher,[21] 2012
Review	1	Maísa Soares and Rizzatti-Barbosa,[22] 2015

References

1. Flor H, Behle DJ, Birbaumer N. Assessment of pain-related cognitions in chronic pain patients. Behav Res Ther 1993;31:63–73.
2. Greco CM, Rudy TE, Turk DC, Herlich A, Zaki HH. Traumatic onset of temporomandibular disorders: Positive effects of a standardized conservative treatment program. Clin J Pain 1997;13:337–347.
3. Jang HH, Kim ME, Kim HK. Pain catastrophizing mediates the effects of psychological distress on pain Interference in patients with orofacial pain: A cross-sectional study. J Oral Facial Pain Headache 2018;32:409–417.
4. Kucyi A, Moayedi M, Weissman-Fogel I, et al. Enhanced medial prefrontal-default mode network functional connectivity in chronic pain and its association with pain rumination. J Neurosci 2014;34:3969–3975.
5. Madland G, Feinmann C, Newman S. Factors associated with anxiety and depression in facial arthromyalgia. Pain 2000;84:225–232.
6. Roditi D, Robinson ME, Litwins N. Effects of coping statements on experimental pain in chronic pain patients. J Pain Res 2009;2:109–116.
7. Rollman A, Gorter RC, Visscher CM, Naeije MM. Why seek treatment for temporomandibular disorder pain complaints? A study based on semi-structured interviews. J Orofac Pain 2013;27:227–234.
8. Rollman A, Visscher CM, Gorter RC, Naeije M. Care seeking for orofacial pain. J Orofac Pain 2012;26:206–214.
9. Van Damme S, Vanden Bulcke C, Van Den Berghe L, Poppe L, Crombez G. Do patients with chronic unilateral orofacial pain due to a temporomandibular disorder show increased attending to somatosensory input at the painful side of the jaw? PeerJ 2018;6:e4310.
10. Dagsdóttir LK, Skyt I, Vase L, Baad-Hansen L, Castrillon E, Svensson P. Reports of perceptual distortion of the face are common in patients with different types of chronic oro-facial pain. J Oral Rehabil 2016;43:409–416.
11. de Boer MJ, Versteegen GJ, Vermeulen KM, Sanderman R, Struys MM. A randomized controlled trial of an Internet-based cognitive-behavioural intervention for non-specific chronic pain: An effectiveness and cost-effectiveness study. Eur J Pain 2014;18:1440–1451.
12. Aguiar AS, Bataglion C, Visscher CM, Bevilaqua Grossi D, Chaves TC. Cross-cultural adaptation, reliability and construct validity of the Tampa scale for kinesiophobia for temporomandibular disorders (TSK/TMD-Br) into Brazilian Portuguese. J Oral Rehabil 2017;44:500–510.
13. Conti PC, Pinto-Fiamengui LM, Cunha CO, Conti AC. Orofacial pain and temporomandibular disorders: The impact on oral health and quality of life. Braz Oral Res 2012;26(suppl 1):s120–s123.
14. Dagsdóttir LK, Skyt I, Vase L, Baad-Hansen L, Castrillon E, Svensson P. Experimental orofacial pain and sensory deprivation lead to perceptual distortion of the face in healthy volunteers. Exp Brain Res 2015;233:2597–2606.
15. Diernberger S, Bernhardt O, Schwahn C, Kordass B. Self-reported chewing side preference and its associations with occlusal, temporomandibular and prosthodontic factors: Results from the population-based Study of Health in Pomerania (SHIP-o). J Oral Rehabil 2008;35:613–620.
16. Harfeldt K, Alexander L, Lam J, et al. Spectroscopic differences in posterior insula in patients with chronic temporomandibular pain. Scand J Pain 2018;18:351–361.

17. Ingram M, Choi YH, Chiu CY, et al. Use of the minimal clinically important difference (MCID) for evaluating treatment outcomes with TMJMD patients: A preliminary study. J Appl Biobehav Res 2011;16:148–166.
18. Kapos FP, Look JO, Zhang J, Hodges JS, Schiffman EL. Predictors of long-term temporomandibular disorder pain intensity: An 8-year cohort study. J Oral Facial Pain Headache 2018;32:113–122.
19. Muñoz-García D, López-de-Uralde-Villanueva I, Beltrán-Alacreu H, La Touche R, Fernández-Carnero J. Patients with concomitant chronic neck pain and myofascial pain in masticatory muscles have more widespread pain and distal hyperalgesia than patients with only chronic neck pain. Pain Med 2017;18:526–537.
20. Gil-Martínez A, Grande-Alonso M, La Touche R, et al. Psychosocial and somatosensory factors in women with chronic migraine and painful temporomandibular disorders. Pain Res Manag 2016;2016:3945673.
21. Lautenbacher S. Pain, sleeping problems and their many relatives. Pain 2012;153:1138.
22. Maísa Soares G, Rizzatti-Barbosa CM. Chronicity factors of temporomandibular disorders: A critical review of the literature. Braz Oral Res 2015;29:S1806–83242015000100300.