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

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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 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). Pain catastrophizing can predict pain intensity and disability and has been associated with increased affective distress, muscle and joint tenderness, and pain-related disability. It has been suggested that early treatment for pain catastrophizing may serve as a prevention of chronic pain. Reductions in pain catastrophizing are associated with improvements in pain and pain treatment outcome. 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. The preva-
lence of TMD pain is 10% to 15% in the general popu-
lation 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 condi-
tions, 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 treat-
ment planning has been emphasized.15

As with many other chronic pain conditions, TMD
is seen with a variety of comorbid conditions, such
as fibromyalgia, rheumatism, and psoriatic arthri-
tis.16 Furthermore, psychologic comorbidities have
been reported, with high levels of clinical depres-
sion 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 com-
pared to healthy controls.18,19 Some of these comor-
bidities may also affect the prognosis and treatment
outcome for the individual patient.20

Many different treatment modalities are used sep-
arately 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-in-
flammatory drugs (NSAIDs) and muscle relaxants.21,22
Although these commonly used treatment modalities
can achieve positive outcomes, there are several fac-
tors affecting treatment prognosis, with psychoso-
cial 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 emotion-
al 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 treat-
ment 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 beh-
aviors, such as avoidance-response and fear, con-
sciously. 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 pa-
ients 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 for-
mulated 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 languag-
es 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-
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 E.C.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 pre-designed 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

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

<table>
<thead>
<tr>
<th>Database</th>
<th>Search strategy</th>
<th>Records</th>
</tr>
</thead>
<tbody>
<tr>
<td>PubMed</td>
<td>(((((((tmjd) OR temporomandibular disorder*) OR temporomandibular joint disorder*) OR tmj disorder*) OR craniomandibular disorder*) OR (((facial OR jaw OR orofacial OR craniofacial OR trigem) AND pain)) OR “Craniofacial Disorders”[Mesh] OR “temporomandibular joint disorders”[MeSH]) AND (((“Catastrophizing”[Mesh]) OR Catastrophization) OR Catastrophisation) OR Catastrophising*) OR Catastrophising*) OR Pain catastrophizing scale*) OR PCS) OR (((catastrophic) OR catastrophic) AND (((“Thinking”[Mesh]) OR “Emotions”[Mesh]) OR thinking) OR thoughts) OR feelings))</td>
<td>183</td>
</tr>
<tr>
<td>Scopus</td>
<td>( TITLE-ABS-KEY (“craniofacial disorder” OR “temporomandibular disorder” OR “temporomandibular joint disorder” OR tmj disorder) OR tmjd OR tmd OR ((facial OR jaw OR orofacial OR craniofacial OR trigem) AND pain )) AND ( TITLE-ABS-KEY ( pcs OR “Pain catastrophizing scale” OR Catastrophising*) OR Catastrophisation OR catastrophe OR catastrophising OR ( (( thinking OR thought OR feeling ) AND ( catastrophe OR catastrophic )))</td>
<td>145</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>( MAINSUBJECT.EXACT(“Bruxism”) OR (temporomandibular disorder*) OR (temporomandibular joint disorder*) OR (tmj disorder*) OR tmjd OR tmd OR ((facial OR jaw OR orofacial OR craniofacial OR trigem) AND pain)) AND ( MAINSUBJECT.EXACT(“Catastrophizing”) OR Catastrophization OR Catastrophisation OR Catastrophising*) OR ((Catastrophe OR Catastrophic) AND ( MAINSUBJECT.EXACT.EXplode(&quot;Thinking&quot;) OR MAINSUBJECT.EXACT.EXplode(&quot;Emotions&quot;) OR thinking OR thoughts OR feelings)) OR PCS OR (Pain catastrophizing scale))</td>
<td>106</td>
</tr>
</tbody>
</table>
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 were excluded due to not fulfilling the inclusion criteria (Table 3a). 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 ratings35; high-impact pain (OR = 1.59)46; onset (OR = 1.98) and progression (OR = 2.17) of clinically significant pain47; and pain persistence (OR = 6.11).48 Catastrophizing also explained 14% of the variance in pain-related activity interference42 and in pain response from a provocation chewing test36 (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 ($\beta = 0.64$, $F = 32.07$, $P < .007$) and pain interference ($\beta = 0.62$, $F = 25.72$, $P < .007$). 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 samples40,53–55 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)

<table>
<thead>
<tr>
<th>Study, y</th>
<th>Selection</th>
<th>Comparability</th>
<th>Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S1 S2 S3 S4</td>
<td>C1a C1b</td>
<td>E1 E2 E3</td>
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<tr>
<td>Brandini et al, 2011</td>
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<td>+ +</td>
<td>+ +</td>
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<tr>
<td>Buenaver et al, 2012*</td>
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<td>La Touche et al, 2015</td>
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<td>Reiter et al, 2018*</td>
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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)

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<td>S1 S2 S3 S4</td>
<td>C1 C2</td>
<td>O1 O2 O3</td>
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<td>Fillingim et al, 2013</td>
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<tr>
<td>Velly et al, 2011</td>
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<td></td>
</tr>
</tbody>
</table>

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)

<table>
<thead>
<tr>
<th>Study, y</th>
<th>Selection</th>
<th>Comparability</th>
<th>Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S1 S2 S3 S4</td>
<td>C1 C2</td>
<td>E1 E2 E3</td>
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<td>Turner et al, 2005b</td>
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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)

<table>
<thead>
<tr>
<th>Study (y), country</th>
<th>Setting, study design</th>
<th>Participants (% F), mean age (range)</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bair et al</strong>&lt;sup&gt;56&lt;/sup&gt; (2013), USA</td>
<td>Population sample, prospective cohort</td>
<td>2,737 healthy individuals (18–44 y)</td>
<td>PCS, variable importance score</td>
<td>A total of 260 individuals developed TMD. Catastrophizing was a weak predictor for TMD (variable importance score: 10.4)</td>
</tr>
<tr>
<td><strong>Brandini et al</strong>&lt;sup&gt;57&lt;/sup&gt; (2011), Australia</td>
<td>Hospital staff and patients, case-control</td>
<td>15 (100%) TMD patients, 31 y; 14 (100%) healthy individuals, 29 y</td>
<td>PCS</td>
<td>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.</td>
</tr>
<tr>
<td><strong>Buenaver et al</strong>&lt;sup&gt;41&lt;/sup&gt; (2012), USA</td>
<td>Orofacial pain clinic + population sample, case series</td>
<td>214 (74%) TMD patients, 34 y (18–65)</td>
<td>PCS, PSQI, BPI</td>
<td>The PCS was associated with sleep disturbances (r = 0.37), pain severity (r = 0.36), and pain interference (r = 0.52) (all P &lt; .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.</td>
</tr>
<tr>
<td><strong>Campbell et al</strong>&lt;sup&gt;44&lt;/sup&gt; (2010), USA</td>
<td>Hospital advertising, case-control</td>
<td>84 (38%) healthy individuals; 48 (85%) TMD patients; 43 (62%) arthritic patients</td>
<td>PCS, SCQ, HPT</td>
<td>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 &lt; .05) and SCQ (r = −0.30, P &lt; .05) with HPT and positive correlation with suprathresholds of heat stimuli (both r = 0.43, P &lt; .01) and painful aftereffects (r = 0.46 and 0.50, respectively; both P &lt; .01).</td>
</tr>
<tr>
<td><strong>Castrillon et al</strong>&lt;sup&gt;58&lt;/sup&gt; (2008), Denmark</td>
<td>University students, experimental-case-control</td>
<td>10 (100%) TMD patients, 24 y; 47 (100%) healthy individuals, 9 y</td>
<td>CSQ, VAS for pain</td>
<td>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 &lt; .03) between catastrophizing and VAS pain.</td>
</tr>
<tr>
<td><strong>Chen et al</strong>&lt;sup&gt;59&lt;/sup&gt; (2012), USA</td>
<td>Orofacial pain clinic and university advertising, case-control</td>
<td>83 TMD patients, 33 y (18–60); 76 TMD + WPT, 40 y (18–60); 181 healthy individuals, 30 y (18–60)</td>
<td>PCS, CPSQ, GCPS</td>
<td>Controls had a significantly (P &lt; .004) lower mean PCS (6.8) compared to both TMD (10.7) and TMD + WPT (11.8) patients.</td>
</tr>
<tr>
<td><strong>Chen et al</strong>&lt;sup&gt;59&lt;/sup&gt; (2013), USA</td>
<td>Orofacial pain clinic and university advertising, case-control</td>
<td>159 (100%) TMD patients, 36 y (18–60); 131 (100%), healthy individuals, 30 y (18–60)</td>
<td>PCS, CPSQ, GCPS</td>
<td>Controls had a significantly (P &lt; .004) lower mean PCS (6.0) compared to the TMD group (11.2). TMD patients with pain comorbidity reported higher PCS.</td>
</tr>
<tr>
<td><strong>Costa et al</strong>&lt;sup&gt;70&lt;/sup&gt; (2017), Brazil</td>
<td>Hospital orofacial pain clinic, case control</td>
<td>47 (80%) TMD patients, 28 y; 50 (88%) TMD + headache patients, 29 y</td>
<td>PRSS subscale, VAS</td>
<td>The TMD + headache group had higher mean (SD) PRSS (2.1 [1.2]) than the TMD-only group (1.6 [1.4]) (P = .048).</td>
</tr>
<tr>
<td><strong>Davis et al</strong>&lt;sup&gt;34&lt;/sup&gt; (2014), USA</td>
<td>Orofacial pain clinic, case series</td>
<td>50 (90%) TMD patients, 41 y (18–80)</td>
<td>PSWQ, STAI, PCS, NRS (0–10) for pain (current, worst, least, average, interference), disability score (0–10)</td>
<td>Mean (SD) PCS: Total: 15.7 (11.7) Ruminations: 5.2 (3.8) Magnification: 3.3 (2.7) Helplessness: 7.3 (6.1) PCS was correlated with worst (r = 0.39, P &lt; .01) and least (r = 0.32, P &lt; .05) pain intensity, as well as pain interference (r = 0.38, P &lt; .01).</td>
</tr>
<tr>
<td><strong>Fillingim et al</strong>&lt;sup&gt;51&lt;/sup&gt; (2011), USA</td>
<td>Population sample, cross-sectional case-control</td>
<td>1,533 TMD-free controls; 185 TMD patients; 18 (14–44 y)</td>
<td>STAI, PCS</td>
<td>TMD cases had higher levels of catastrophizing than controls (P &lt; .0001). PCS: Helplessness: 5.61 vs 3.47 Magnification: 2.84 vs 1.89, Ruminations: 5.67 vs 4.15</td>
</tr>
</tbody>
</table>

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.
<table>
<thead>
<tr>
<th>Conclusions</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
<td>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.</td>
</tr>
<tr>
<td>There were gender and age differences between groups; but gender- and age-adjusted analyses for associations.</td>
<td>The TMD group had a significantly higher age.</td>
</tr>
<tr>
<td>The TMD group had a significantly higher age.</td>
<td>The same study population was included as in Chen et al(^8) (2012). The TMD group had a significantly higher age.</td>
</tr>
<tr>
<td>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.</td>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
<td>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.</td>
</tr>
</tbody>
</table>
Table 3a (cont) Summary of Results for Levels of Catastrophizing and Associations with Other Factors (n = 28)

<table>
<thead>
<tr>
<th>Study (y), country</th>
<th>Setting, study design</th>
<th>Participants (% F), mean age (range)</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fillingim et al13 (2013), USA</td>
<td>Population sample, prospective cohort</td>
<td>Baseline: 3,263 (60%), TMD-free individuals 27 y (18–44) Follow-up: 260 first-onset TMD patients 50 (78%) TMD patients, 46 y; 50 (92%) migraine patients, 49 y</td>
<td>STAI, PCS</td>
<td>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.</td>
</tr>
<tr>
<td>Gil-Martínez et al62 (2017), Spain</td>
<td>University TMD/neurology clinic, cross-sectional case-control</td>
<td>PCS, VAS, pain intensity (0–100); NDI, CF-PDI TMD patients reported a high mean (SD) PCS: (23.7 [8.9]). There was a significant correlation between PCS and CF-PDI (&lt; .01) and pain (&lt; .001). However, the highest mean (SD) CSQ (7.8 [5.2]) and pain (24.6 [9.1]) TMD patients compared to controls (16.4 [5.3] vs 10.1 [8.9]; P = .002), but no difference compared to TNP (23.6 [11.5]) patients.</td>
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<tr>
<td>Gustin et al62 (2011), Australia</td>
<td>University clinic, case-control</td>
<td>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.</td>
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<tr>
<td>Hollins et al63 (2009), USA</td>
<td>University orofacial pain clinic and advertising, case-control</td>
<td>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)</td>
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<tr>
<td>Jerjes et al64 (2007), UK</td>
<td>University clinic, case-control</td>
<td>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 &lt; .01) at baseline. There was no significant difference at the 6-month follow-up (8.0 [9.6] vs 12.0 [10.6])</td>
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<tr>
<td>Kothari et al65 (2017), Denmark</td>
<td>University orofacial pain clinic, case-control</td>
<td>PCS TMD patients had higher mean (SD) PCS than controls: Total: 20.7 (11.0) vs 10.3 (9.9); P &lt; .001 Ruminations: 7.0 (4.8) vs 4.5 (4.4); P = .005 Magnification: 4.1 (2.9) vs 1.8 (1.9); P &lt; .001 Helplessness: 9.6 (5.3) vs 3.8 (4.1); P &lt; .001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kotiranta et al66 (2015), Finland</td>
<td>Primary dental care clinic, case series</td>
<td>PCS, GCPS There were significant differences in CPI (P &lt; .000) among the GCPS groups, with 3.7 in no disability, 6.0 in low disability, and 7.7 in high disability.</td>
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</tr>
<tr>
<td>La Touche et al67 (2014), Spain</td>
<td>Hospital and private TMD clinics, case series</td>
<td>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 &lt; .01).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>La Touche et al68 (2015), Spain</td>
<td>Public health and private TMD clinics, experimental case-control</td>
<td>PCS, NDI, VAS for pain and 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 &lt; .01), and PCS predicted fatigue (r² = 0.12, P = .01) and pain (r² = 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 &lt; .01), and PCS predicted fatigue (r² = 0.17, P = .004) 24 hours after a provocation chewing test.</td>
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</tbody>
</table>

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

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)

<table>
<thead>
<tr>
<th>Study (y), country</th>
<th>Setting, study design</th>
<th>Participants (% F), mean age (range)</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lerman et al33 (2018), US</td>
<td>University clinic and advertising, case series</td>
<td>156 (100%) TMD patients, 37 y (18–60)</td>
<td>PCS, BPI pain severity</td>
<td>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 &lt; .01).</td>
</tr>
<tr>
<td>Litt et al45 (2004), USA</td>
<td>Population sample, case series</td>
<td>30 (87%) TMD patients, 36 y</td>
<td>PRSS catastrophizing subscale (0–5), current pain (0–10)</td>
<td>The mean (SD) PRSS catastrophizing score was 1.6 (0.8). Momentary catastrophizing was a predictor for higher pain (P &lt; .01), and higher catastrophizing scores were predictive of higher mean pain ratings (PRSS score f[11.46] = 5.87, P &lt; .001).</td>
</tr>
<tr>
<td>Miller et al46 (2018), USA</td>
<td>Cross-sectional case series</td>
<td>846 (77%) TMD patients, 28.0 y (18–44), with pain subgroups: low-impact GCPS (I + II-high), high-impact GCPS (II-high, III, IV)</td>
<td>CSQ-Revised catastrophizing subscale (0–6), GCPS</td>
<td>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).</td>
</tr>
<tr>
<td>Quartana et al47 (2010), USA</td>
<td>Orofacial pain clinic and advertising, cross-sectional case-control</td>
<td>39 (82%) TMD patients, 34 y, 22 (96%) controls 26 y</td>
<td>PCS, PPT, HPT, cold pain rating</td>
<td>TMD cases had higher levels of catastrophizing (PCS mean [SD]: 14.0 [8.8]) than controls (8.9 [6.8]) (P &lt; .05). Higher PCS was associated with flattened morning salivary cortisol profile. There were no correlations of PCS with PPT, HPT, or cold pain rating.</td>
</tr>
<tr>
<td>Reiter et al48 (2018), Israel</td>
<td>University orofacial pain clinic, case series</td>
<td>163 (66%) TMD patients, 36 y (18–60)</td>
<td>PCS, PHQ-9, GAD-7, PHQ-15</td>
<td>Higher PCS was associated with a higher prevalence of myofascial pain with referral (P &lt; .05); lower prevalence of myalgia (P &lt; .02); and higher pain persistence, GCPS, depression, anxiety, and nonspecific physical symptoms (all P &lt; .001). Catastrophizing was associated with pain persistence (OR: 6.71, 95% CI: 1.58–28.41; P = .01).</td>
</tr>
<tr>
<td>Turner et al42 (2001), USA</td>
<td>TMD clinic, cross-sectional case series</td>
<td>118 (83%) TMD patients, 39 y (21–67)</td>
<td>CPI, CSQ catastrophizing subscale (0–6), MPI interference scale</td>
<td>Mean (SD) catastrophizing score (2.2 [1.5]) was correlated with pain-related activity interference (r = 0.45, P &lt; .0001). Catastrophizing explained variance in pain-related activity interference (r² = 0.14, P &lt; .0001), nonnasticatory jaw activity limitation (change in r² = 0.08, P &lt; .001), and depression (change in r² = 0.33, P &lt; .0001).</td>
</tr>
<tr>
<td>Turner et al49 (2004), USA</td>
<td>TMD clinic, cross-sectional case series</td>
<td>100 (87%) TMD patients, 39 y (16–67)</td>
<td>Catastrophizing assessed by 3 questions adapted from the CSQ, PCS rumination subscale (0–10)</td>
<td>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 = 0.58; P &lt; .0001) and pain-related disability (r = 0.43, P &lt; .0001).</td>
</tr>
<tr>
<td>Turner et al45 (2005a), USA</td>
<td>TMD clinic, cross-sectional case series</td>
<td>338 (87%) TMD patients, 37 y</td>
<td>GCPS, CPI disability score, CSQ catastrophizing subscale (0–6), MFOQ</td>
<td>After correction for age, gender, and education, catastrophizing (mean [SD]: 1.7 [1.3]) explained 10% of muscle palpation (P &lt; .001), 3% of TMJ pain on palpation (P &lt; .01), 18% of pain intensity (r = 0.42, P &lt; .001), and 25% of pain-related disability (r = 0.5, P &lt; .001).</td>
</tr>
<tr>
<td>Velly et al47 (2011), USA</td>
<td>Population sample, prospective cohort</td>
<td>570 (88%) TMD patients</td>
<td>CSQ catastrophizing subscale, GCPS (CPI + disability score)</td>
<td>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 &lt; .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 &lt; .0001) of clinically significant pain.</td>
</tr>
</tbody>
</table>

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; PRS = Pain Catastrophizing Scale; PHQ-9/15 = Patient Health Questionnaire-9/15; PPT = pressure pain threshold; PRSS = Pain-Related Self-Statement Scale.
### Conclusions

<table>
<thead>
<tr>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>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).</td>
</tr>
<tr>
<td>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).</td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td>Study (y), country</td>
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<tr>
<td>Costa et al(^{55}) (2015), Brazil</td>
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<tr>
<td>Durá-Ferrandis et al(^{52}) (2017), Spain</td>
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<tr>
<td>Litt et al(^{50}) (2009), USA</td>
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<tr>
<td>Litt et al(^{51}) (2010), USA</td>
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<tr>
<td>Litt et al(^{59}) (2013), USA</td>
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<tr>
<td>Turner et al(^{50}) (2005b), USA</td>
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<td>Turner et al(^{53}) (2007), USA</td>
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<tr>
<td>Turner et al(^{55}) (2011), USA</td>
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</tbody>
</table>

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

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.

The results could set the principles for the development of more efficient and effective cognitive behavioral interventions for chronic pain.

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.

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 heterogeneous 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 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.

A brief CBT intervention improves 1-y clinical outcomes of TMD clinical patients, and these effects appear to result from specific CBT interventions.

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.

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.

### Limitations

There was a large dropout.

There was a large dropout in the CBT group.

There was a possible overlap of the study population with Litt et al, 2009.

The study population was the same as in Litt et al, 2010.

Of 366 eligible patients, only 158 (43%) enrolled in the study. A further 32 (20%) were not part of the follow-up analysis.

Of 366 eligible patients, only 158 (43%) enrolled in the study. The study population was similar to Turner et al, 2005.

Subset of the study population in Turner et al, 2006.

Large dropout (> 50%) in the oral contraceptives group.
Study, y | Mean | 95% CI | Weight (%) | Levels of catastrophizing (random effects)
---|---|---|---|---
Brandini et al, 2011 | 11.0 | 6.69, 15.31 | 1.1 |
Campbell et al, 2010 | 9.5 | 7.54, 11.46 | 5.3 |
Chen et al, 2012 | 6.8 | -3, 16.6 | 0.2 |
Fillingim et al, 2011 | 9.5 | 9, 9.99 | 84.7 |
Gustin et al, 2011 | 10.1 | 7.26, 12.84 | 2.7 |
Hollins et al, 2009 | 8.4 | 5.26, 11.54 | 2.1 |
Kothari et al, 2017 | 10.3 | 7.36, 13.24 | 2.4 |
La Touche et al, 2015 | 5.5 | 4.95, 6.05 | 0.1 |
Quartana et al, 2010 | 8.9 | 5.2, 12.62 | 1.5 |
Total | 9.51 | 9.06, 9.96 | 100.00 |

Study, y | Mean | 95% CI | Weight (%) | Levels of catastrophizing (random effects)
---|---|---|---|---
Brandini et al, 2011 | 12.7 | 7.41, 17.99 | 6.2 |
Campbell et al, 2010 | 14.3 | 11.75, 16.85 | 7.8 |
Chen et al, 2012 | 10.7 | 8.54, 12.86 | 8 |
Davis et al, 2014 | 15.7 | 12.37, 19.03 | 6.9 |
Fillingim et al, 2011 | 14.1 | 12.34, 15.86 | 8.1 |
Gil-Martínez et al, 2017 | 26.4 | 24.05, 28.75 | 7.9 |
Gustin et al, 2011 | 18.7 | 14, 23.4 | 6.6 |
Hollins et al, 2009 | 12.6 | 8.68, 16.52 | 7.1 |
Kothari et al, 2017 | 20.7 | 17.96, 23.44 | 7.7 |
La Touche et al, 2014 | 23.7 | 22.52, 24.88 | 8.3 |
La Touche et al, 2015 | 16.4 | 15.62, 17.18 | 8.4 |
Lerman et al, 2018 | 21.4 | 19.83, 22.97 | 8.2 |
Quartana et al, 2010 | 14.0 | 11.26, 16.74 | 7.7 |
Reiter et al, 2018 | 21.4 | 18.88, 23.98 | 7.8 |
Total | 17.6 | 15, 20.2 | 100.00 |

Study, y | Mean | 95% CI | Weight (%) | Effect size (random effects)
---|---|---|---|---
Brandini et al, 2011 | 0.17 | -0.57, 0.92 | 9.5 |
Campbell et al, 2010 | 0.53 | 0.17, 0.89 | 11.8 |
Chen et al, 2012 | 0.51 | 0.25, 0.78 | 12.3 |
Fillingim et al, 2011 | 0.45 | 0.29, 0.6 | 12.6 |
Gustin et al, 2011 | 0.88 | 0.33, 1.45 | 10.6 |
Hollins et al, 2009 | 0.5 | -0.12, 1.13 | 10.3 |
Kothari et al, 2017 | 0.98 | 0.56, 1.41 | 11.5 |
La Touche et al, 2015 | 3.21 | 2.67, 3.78 | 10.6 |
Quartana et al, 2010 | 0.62 | 0.09, 1.16 | 10.8 |
Total | 0.86 | 0.18, 1.54 | 100.00 |

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...
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. 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. 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. 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. 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. 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,\textsuperscript{39} pain interference,\textsuperscript{34} pain on palpation,\textsuperscript{35} fatigue and pain in a provocation chewing test,\textsuperscript{36} and neck disability.\textsuperscript{37} Furthermore, an association between level of catastrophizing and number of health care visits\textsuperscript{35} was reported. The included primary studies generally demonstrated moderate correlations between catastrophizing and pain outcomes. Catastrophizing was also associated with higher pain\textsuperscript{45,46} and with onset, progression, and persistence of pain.\textsuperscript{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\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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 practitioners.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.
References


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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, E.C.E., 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.


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

<table>
<thead>
<tr>
<th>Main reason for exclusion</th>
<th>Studies, n</th>
<th>Study, y</th>
</tr>
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<tbody>
<tr>
<td>Did not use RDC/TMD or DC/TMD for diagnosis</td>
<td>9</td>
<td>Flor et al, 1993</td>
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<td>Greco et al, 1997</td>
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<td>Jang et al, 2018</td>
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<td>Kucyi et al, 2014</td>
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<td></td>
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<td>Madland et al, 2000</td>
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<td>Roditi et al, 2009</td>
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<td>Rollman et al, 2013</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td>Van Damme et al, 2018</td>
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<tr>
<td>Not TMD population</td>
<td>2</td>
<td>Dagsdottir et al, 2016</td>
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<td></td>
<td></td>
<td>de Boer et al, 2014</td>
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<tr>
<td>No specific data on catastrophizing for TMD group</td>
<td>8</td>
<td>Aguiar et al, 2017</td>
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<td></td>
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<td>Conti et al, 2012</td>
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<td>Dagsdottir et al, 2015</td>
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<td>Muniz-Garcia et al, 2017</td>
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<td>Same study population as another included study</td>
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<td>Gil-Martinez et al, 2016</td>
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<td>Commentary or letter to editor</td>
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<td>Lautenbacher, 2012</td>
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<tr>
<td>Review</td>
<td>1</td>
<td>Maísa Soares and Rizzatti-Barbosa, 2015</td>
</tr>
</tbody>
</table>

**References**

9. Van Damme S, Vanden Bulcke C, Van Den Bergh 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? Peed 2018;6:4310.


