



Oral behaviors mediate the relationship between anxiety and painful temporomandibular disorders

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ABSTRACT

Objectives: Anxiety is strongly associated with chronic musculoskeletal pain, including painful temporomandibular disorders (p-TMD). Non-functional oral behaviors—such as wake-time tooth clenching or keeping the lower jaw in a tense position—are prevalent stress-related behaviors among individuals with elevated anxiety. These behaviors are thought to act as maladaptive coping strategies increasing strain on the masticatory muscles and the temporomandibular joint, thereby enhancing peripheral nociceptive input and contributing to the onset or persistence of p-TMD in individuals with high anxiety. While this behavioral pathway is theoretically supported, it has yet to be empirically verified. This study investigated whether non-functional oral behaviors mediate the relationship between anxiety and p-TMD.

Design: We recruited 299 adults with p-TMD (myofascial pain and/or arthralgia) and 374 pain-free controls. Anxiety levels and the frequency of non-functional oral behaviors were assessed using the Generalized Anxiety Disorder scale and the Oral Behavior Checklist, respectively. Mediation analysis was conducted to examine both the direct (anxiety → p-TMD) and indirect (anxiety → oral behaviors → p-TMD) pathways.

Results: Mediation analysis revealed that non-functional oral behaviors fully mediate the relationship between anxiety and p-TMD.

Conclusion: Non-functional oral behaviors are a key behavioral mechanism linking anxiety to p-TMD. These findings highlight the importance of targeting oral behaviors in interventions for TMD pain, particularly among individuals with high anxiety, and provide a foundation for future research into behavioral and neural mechanisms underlying TMD.

1. Introduction

Temporomandibular disorders (TMD) are the most common cause of chronic orofacial pain and represent the second most frequent painful musculoskeletal condition after chronic low back pain. TMD are highly prevalent and affect 5–12 % of the global population (NIDCR (National Institute of Dental and Craniofacial Research), 2024) with an annual incidence rate of approximately 4 % (Slade et al., 2016). TMD cause substantial personal suffering and impacting negatively on quality of life (Almoznino et al., 2015; Cioffi et al., 2014). To date, the etiology and mechanisms of TMD remain not fully understood (Svensson, 2024).

Evidence from the multisite NIH-funded Orofacial Pain Perspective Evaluation and Risk Assessment (OPPERA) study has shown that genetic, environmental, and psychosocial factors contribute to the onset of painful TMD (p-TMD) (Slade et al., 2016). Notably, oral behaviors—jaw motor activities such as wake-time tooth clenching or holding the jaw tense, gum chewing, or nail biting (Markiewicz et al., 2006)—are among the strongest predictors of TMD incidence (Ohrbach et al., 2013). These activities, particularly sustained tooth clenching, are thought to increase strain on the muscles of mastication and surrounding tissues, leading to local inflammation (Louca Jounger et al., 2017). Results from a recent study indicate that not all oral behaviors play a similar role. Specifically,

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a factor analysis identified a subset of specific oral activities, termed non-functional oral behaviors, that are predictive of p-TMD. These behaviors involve sustained tooth contact or contraction of the jaw muscles, such as or holding the jaw in a tense position (Donnarumma et al., 2021) or tooth clenching, and differ from other oral behaviors having a functional purpose.

Anxiety plays a significant role in TMD (Boscato et al., 2013; Dos Santos et al., 2022). First, as in other painful musculoskeletal disorders, anxiety amplifies pain perception by heightening somatic sensations (*direct pathway*) (Barsky et al., 1988; Cioffi et al., 2016; Chow et al., 2022; Costello et al., 2019). Second, anxiety is linked to an increased frequency of oral behaviors (Chow & Cioffi, 2019; van Selms & Lobbezoo, 2024) which can lead to muscle overload and peripheral sensitization (Farella et al., 2010; Louca Jounger et al., 2017) further driving nociceptive input and p-TMD (*indirect pathway*).

Although studies have demonstrated positive associations between anxiety, heightened masticatory muscle activity, and self-reported oral behaviors (Rofaeel et al., 2020; van Selms & Lobbezoo, 2024) as well as between oral behaviors and p-TMD (Donnarumma et al., 2021; Michelotti et al., 2010; Ohrbach et al., 2013), the mechanistic pathways linking these factors remain unclear. Specifically, whether anxiety contributes to p-TMD through a direct pathway or via non-functional oral behaviors (indirect pathway) has been minimally explored. Mediation models provide a useful framework for investigating different pathways of relationship between anxiety, oral behaviors, and p-TMD.

This study aimed to determine whether non-functional oral behaviors mediate the relationship between anxiety and p-TMD using behavioral data retrieved from a previously collected cohort including over seven hundred participants (Donnarumma et al., 2021). We hypothesized that self-reported non-functional oral behaviors mediate the relationship between anxiety and p-TMD.

2. Methods

2.1. Study sample

The study was reviewed and approved by the Research Ethics Committee at the University of Naples Federico II, Italy (protocol 48/18). The study sample included in this investigation has been described previously (Donnarumma et al., 2021). Participants were selected from a cohort of 785 consecutive patients referred to the TMD Clinic at the University of Naples Federico II, Italy, for a TMD consultation (from May 2015 to May 2017). Individuals aged 18 years or older with a diagnosis of p-TMD (i.e., TMD myalgia, myofascial pain, myofascial pain with referral, or arthralgia) established according to the Diagnostic Criteria for TMD (DC/TMD) (Schiffman et al., 2014) were recruited. Exclusion criteria included neuropathic or generalized pain conditions, such as trigeminal neuralgia, fibromyalgia, burning mouth syndrome, persistent idiopathic facial pain, post-traumatic trigeminal neuropathic pain, migraine, or neck pain. Participants were examined by a single trained operator (AM).

The study group included a cohort of 299 individuals with p-TMD (P-TMD: 83 males, 216 females; mean age \pm SD = 37.6 \pm 16.0 years). A control group of similar age was also recruited (CTR group; n = 444, 123 males, 321 females; mean age \pm SD = 37.4 \pm 16.4 years). This cohort of participants included individuals without TMD or with non-painful TMD (i.e., non-painful TMJ disc displacement with or without reduction), recruited from individuals accompanying patients to local dental clinics. Exclusion criteria for the CTR group were the same as for the P-TMD group. Controls were screened for signs and symptoms of TMD using the Symptom Questionnaire from the DC/TMD. All control participants were asked whether they had experienced pain in the jaw, temple, ear, or in front of the ear within the past 30 days. Only individuals who did not report any such pain were eligible for inclusion in the control group. For participants who reported symptoms suggestive of non-p-TMD (e.g., TMJ clicking), a clinical examination was conducted to assess for the

presence of non-painful TMD conditions.

2.2. Assessment of oral behaviors, anxiety, and pain characteristics

All participants were invited to fill in the Italian version of the Oral Behavior Checklist (OBC), a 21-item instrument assessing the self-reported frequency of oral behaviors in the preceding 30 days (Markiewicz et al., 2006; Ohrbach et al., 2008). Participants reported the frequency for each oral behavior listed in the instrument by choosing among the following options: “none of the time,” “a little of the time,” “some of the time,” “most of the time,” or “all of the time”. Each response was assigned a score from 0 to 4. Non-functional (NF) oral behaviors were scored using 6 OBC items (grind teeth during waking hours #3; clench teeth together during waking hours #4; press, touch or hold teeth together #5; hold, tighten, or tense muscles #6; hold or jut jaw forward or to the side #7; hold jaw in rigid or tense position #11). In a previous study, this subset of behaviors was shown to be associated with TMD through factor analysis (Donnarumma et al., 2021). A previous study showed that the test–retest reliability of OBC items, assessed against surface electromyography for 10 performed oral behaviors, ranged from 0.60 to 0.90 (Markiewicz et al., 2006). The test-retest reliability of the Italian version of the OBC ranges between 0.87 and 0.95 (Donnarumma et al., 2018).

Participants’ anxiety was measured using the Italian version of the Generalized Anxiety Disorder Scale 7 (GAD-7) included in the DC/TMD, a 7-item instrument which evaluates the presence of specific behaviors (e.g. trouble relaxing, being restless, worrying too much about things, etc.) in the preceding two weeks (Spitzer et al., 2006). Participants reported the frequency for each behavior listed in the instrument by choosing among the following options: “not at all”, “several days”, “more than half the days”, and “nearly every day”. Each answer was assigned a score from 0 to 4. Threshold scores of 5, 10 and 15 indicate mild, moderate, and severe anxiety respectively. The validity and reliability of the Italian version of the GAD-7 have been previously tested (Bolgeo et al., 2023).

The Graded Chronic Pain Scale was used to calculate the characteristic pain intensity and Graded Chronic Pain Score (GCPS) for each participant with p-TMD. The latter was derived from the participant’s characteristic pain intensity and pain-related disability—a composite measure based on pain interference with daily, recreational, and social activities, as well as the number of days facial pain was experienced over the past six months (Von Korff et al., 1992). Each participant with TMD was assigned a GCPS score, i.e.: 1: low intensity pain, without disability; 2: High intensity pain, without disability; 3: Moderately limiting; 4: Severely limiting.

2.3. Statistical analysis

Continuous variables were reported as means and standard deviations. Prior to perform the mediation analysis, a linear regression model was used to test whether GAD-7 scores (independent variable) were associated with NF scores (dependent variable). Thereafter, binomial logistic regression was used to test whether NF scores (independent variable) were associated with P-TMD (dependent variable, P-TMD or CTR). Structural equation modelling (SEM), and in particular generalized SEM (GSEM) having a binary outcome, were used to evaluate the mediation effect of NF scores on the association between anxiety and P-TMD. Sobel’s test and bootstrapping approach were used to test the mediation effect. Unpaired t-tests were used to test between group (P-TMD vs CTR) differences in NF and GAD-7 scores. A p-value of < 0.05 was defined statistically significant. All statistical analyses were performed using Stata version 16 (Stata Corp., College Station, TX, USA).

3. Results

Participants in the P-TMD group had diagnosis of myogenous

($n = 193$, 64.5 %), arthrogenous ($n = 15$; 5.0 %) TMD, or both ($n = 91$; 30.5 %). The mean \pm SD characteristic pain intensity score in the P-TMD group was 40.9 ± 29.3 . One-hundred and four participants had GCPS 1, 57 had GCPS 2, 69 had GCPS 3, and 70 had GCPS 4. Mean \pm SD NF behaviors scores in the P-TMD and the CTR group were 7.0 ± 5.4 and 4.1 ± 3.8 , respectively, and were significantly higher in the P-TMD than in CTR group ($p < 0.001$). Mean \pm SD GAD-7 scores in the P-TMD and CTR group were 8.2 ± 5.4 and 6.95 ± 4.3 , respectively, and were significantly higher in the P-TMD than in the CTR group ($p < 0.001$).

Results from the linear regression model showed that GAD-7 was a statistically significant predictor of NF scores ($B = 0.42$; 95 %CI: 0.36–0.49; $p < 0.001$). Next, the binomial logistic regression showed that NF scores significantly predicted P-TMD (OR = 1.146; 95 %CI: 1.11–1.19, $p < 0.001$). When the variable NF was modelled in the mediation analysis testing the direct (GAD-7 \rightarrow P-TMD) and the indirect (GAD-7 \rightarrow NF \rightarrow P-TMD) pathways, GAD-7 was no longer a significant predictor of P-TMD ($B = -0.002$; 95 %CI: -0.037 to -0.034 ; $p = 0.92$), while NF significantly predicted P-TMD ($B = 0.14$; 95 %CI: 0.09–0.18; $p < 0.001$) (Fig. 1). The results of the Sobel's test confirmed that NF scores significantly mediate the relationship between GAD-7 and P-TMD ($Z = 0.058$; 95 %CI: 0.040–0.079, Fig. 1), while the direct effect of GAD-7 on P-TMD was -0.002 . This corresponds to approximately 103 % of the total effect being mediated, confirming that the relationship between anxiety and painful TMD is fully mediated by NF oral behaviors.

Overall, anxiety increases the frequency of NF oral behaviors, and these behaviors, in turn, are linked to painful TMD.

Model fit was evaluated using the statistics available under the GSEM framework: log-likelihood = -2621.4 ; degrees of freedom = 6; Akaike Information Criterion (AIC) = 5254.7; and Bayesian Information Criterion (BIC) = 5282.4

4. Discussion

Pain is a subjective experience shaped by an individual's sociocultural context, as well as their emotional and psychological states and traits (Engel, 1980). Recent research employing network analysis to classify patients with TMD based on biopsychosocial variables has shown that elevated anxiety levels are associated with clinical pain features suggestive of central sensitization (i.e., nociplastic pain) (Asquini et al., 2025). Anxiety also influences the sympathetic nervous system contributing to peripheral hyperexcitability and increased production of pro-inflammatory mediators (Maes et al., 1998; Richards & Bertram, 2000), both of which promote peripheral sensitization. Neuroimaging studies further reveal that brain regions within the cortico-limbic system involved in pain modulation (Vachon-Presseau et al., 2016) are altered in anxiety disorders (Kim & Kim, 2021). Notably, individuals with chronic p-TMD present structural and functional changes in cortical regions implicated in pain perception and pain modulation (Moayedi et al., 2011), along with dysfunction in the fronto-striatal-limbic circuit (Chen et al., 2022). These findings support a strong link between anxiety and p-TMD (Reis et al., 2022), as further

corroborated by our results showing higher GAD-7 scores in participants with p-TMD.

Although the mechanisms underlying p-TMD remain elusive, recent research has increasingly emphasized the central role of stress in the etiology of oral behaviors and their contribution to p-TMD (Câmara-Souza et al., 2023; Ohrbach & Michelotti, 2018). The mediation analysis performed in this cross-sectional retrospective study demonstrates that non-functional oral behaviors mediate the relationship between anxiety and p-TMD. Importantly, this proposed causal pathway is supported by studies showing that interventions aiming at improving mood, such as guided music listening, reduce the electromyographic amplitude of oral behaviors in individuals with chronic p-TMD (Imbriglio et al., 2020), while also alleviating anxiety and pain in those with musculoskeletal pain (Garza-Villarreal et al., 2017). Additionally, behavioral interventions such as habit reversal (i.e., targeting and reducing oral behaviors) combined with relaxation and stress-management techniques have been shown to be effective in managing p-TMD (Aggarwal et al., 2019, 2021; Donnarumma et al., 2022). Notably, this indirect pathway of association is further supported by evidence of a neural circuit connecting the central nucleus (CeA) of the Amygdala—a region critical for mediating anxiety responses—to the trigeminal motor nucleus (Kaya et al., 2023). Optogenetic activation of CeA affects jaw motor control in mice and enables predatory hunting behavior (Han et al., 2017). However, whether this circuit is involved in regulating oral behaviors in response to stress remains unexplored.

While this mediation may represent a unique feature of TMD pathogenesis, it is important to note that anxiety has been associated with the maintenance of guarding behaviors—such as muscle tension and movement avoidance—in other clinical conditions, notably chronic low back pain (Olugbade et al., 2019). Oral behaviors have been proposed as acquired coping strategies to manage stress (Soto-Goñi et al., 2020), and our analysis may suggest that an increased jaw motor activity as a coping mechanism for anxiety could contribute to p-TMD. However, it is also possible that the observed mediation reflects the role of non-functional oral behaviors—such as keeping the jaw in a tense position or clenching the teeth—as a protective strategy to limit jaw movement. This interpretation aligns with the fear-avoidance model of chronic pain (Vlaeyen and Linton, 2000). Importantly, in a previous study using the same research sample, we showed that functional oral behaviors—such as talking and chewing—are reduced in individuals with TMD (Donnarumma et al., 2021). Other research has also reported high levels of functional limitation in TMD (Ohrbach et al., 2008), which supports the hypothesis that the mediation observed may be more closely related to the maintenance of guarding behaviors than to the use of non-functional oral behaviors as acquired stress-coping strategies. However, given the nature of this cross-sectional retrospective study, it cannot be determined whether non-functional oral behaviors in the investigated TMD cohort represent a true anxiety or stress coping response or, alternatively, a protective strategy aiming at minimizing jaw movement to prevent further muscle injury and pain. Prospective studies are needed to clarify the temporality of these associations.

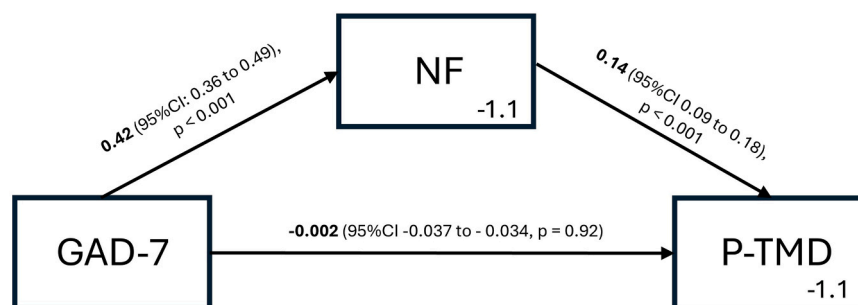


Fig. 1. Non-functional oral behaviors (NF) mediate the relationship between anxiety scores (GAD-7) and painful TMD (P-TMD), which support an indirect pathway of relationship between anxiety and TMD. Coefficients are reported in the diagram.

Importantly, these effects may act synergistically or predominate in one direction or the other depending on individual clinical presentations.

This study has some limitations. While mediation analyses in a cross-sectional study design can illustrate patterns of relationship between variables, a longitudinal design would better support a causal relationship. Indeed, while the observed relationship exists cross-sectionally, it may not necessarily hold with a longitudinal design. Cross-sectional studies lack the ability to account for temporality, meaning they cannot confirm where the putative cause (anxiety) precedes the effect (non-functional oral behaviors and p-TMD). Therefore, longitudinal studies testing this mediation should be performed. Another limitation is that we measured oral behaviors using self-reports (Markiewicz et al., 2006) rather than a more objective methodology, such as ambulatory electromyographic recordings or ecological momentary assessment. However, the oral behavior checklist used in this study has been previously validated against electromyography (Ohrbach et al., 2008) and ecological momentary assessment (Kaplan & Ohrbach, 2016). In addition, our analysis did not account for the potential influence of sleep-related disorders, such as obstructive sleep apnea or sleep bruxism, on the pathways investigated. Finally, although our study demonstrated a mediation effect of oral behaviors on the relationship between anxiety and p-TMD, the magnitude of this effect appears to be small. This suggests that other factors, not considered in the present study, may also contribute to explaining this association.

5. Conclusions

Our study demonstrated that non-functional oral behaviors mediate the relationship between anxiety and p-TMD in a large clinical cohort. While these findings highlight a significant association, the cross-sectional retrospective design precludes conclusions about causality or temporal direction. Longitudinal and experimental studies are needed to clarify whether these behaviors contribute to the onset or maintenance of p-TMD as protective responses, maladaptive stress-related coping strategies, or both. In addition, future studies could further stratify non-painful and subclinical TMD populations to clarify how behavioral and psychological profiles vary along the continuum of TMD severity. Our work lays the foundation for future studies exploring the biopsychosocial mechanisms underlying the association between oral behaviors and p-TMD.

CRedit authorship contribution statement

Ambra Michelotti: Writing – review & editing, Writing – original draft, Supervision, Resources, Methodology, Investigation, Conceptualization. **Rosaria Bucci:** Writing – review & editing, Methodology. **Valeria Donnarumma:** Writing – review & editing, Methodology, Investigation, Data curation, Conceptualization. **Roberto Rongo:** Writing – review & editing, Supervision, Methodology. **Vittorio Simeon:** Writing – review & editing, Software, Methodology, Formal analysis, Data curation. **Iacopo Cioffi:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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