

The Association Between Self-Reported Awake Oral Behaviors and Orofacial Pain Depends on the Belief of Patients That These Behaviors Are Harmful to the Jaw

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Aims: To examine if the existence of an association between self-reported awake oral behaviors and orofacial pain depends on the belief of patients that these behaviors are harmful to the jaw and to investigate if an additional variable (ie, somatic symptoms, depression, and/or anxiety) indirectly affects the association between the causal attribution belief and the report of awake oral behaviors. **Methods:** Prior to the first clinical visit, patients referred to a specialized clinic for complaints of orofacial pain and dysfunction completed a digital questionnaire. Data of 329 patients diagnosed with myalgia according to the Diagnostic Criteria for Temporomandibular Disorders (82.4% women; mean \pm SD age = 41.9 ± 14.7 years) were analyzed. **Results:** Causal attribution belief moderated the association between awake oral behaviors and orofacial pain intensity. In addition, the relationship between causal attribution belief and self-reported oral behaviors was partially mediated by the presence of somatic symptoms (8%), depression (9%), and anxiety (16%). **Conclusion:** Awake oral behaviors were positively associated with orofacial pain, but only under the condition of a strong belief of the patients in causal attribution of these behaviors to the jaw pain complaint. No such association was present in case of a low causal attribution belief. It appeared that, within this patient cohort, the relationship between causal attribution belief and self-reported oral behaviors was (in part) the result of shared psychologic risk factors. *J Oral Facial Pain Headache* 2020;34:273–280. doi: 10.11607/ofph.2629

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Bruxism is defined as a repetitive masticatory muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible during sleep and/or wakefulness.¹ Numerous scientific publications have indicated that bruxism is associated with pain-related temporomandibular disorders (TMD).^{2–5} The applied forces presumably lead to overloading of the masticatory system, with subsequent pain in the muscles of mastication and the temporomandibular joints (TMJ). Surprisingly, this positive association between bruxism and TMD pain complaints is not only observed in studies investigating TMD pain patients who visit specialty clinics, but also among children, scuba divers, musicians, etc.^{6–9} Even popular online sources of medical information advocate that bruxism can lead to TMD pain.¹⁰ Strangely, however, it is difficult to find strong evidence to support the assumption that bruxism-induced overloading of the masticatory system causes TMD pain.^{2–4} Most evidence for the existence of a cause-and-effect relationship between bruxism and TMD pain is derived from epidemiologic surveys in which data are collected at a given point in time.³ Cross-sectional studies preclude the demonstration of a temporal relationship between the two conditions. It should also be kept in mind that pain associated with bruxism is not a compulsory finding because many patients who appear to brux at night have no TMD complaints.⁴ As an alternative hypothesis following the chain of logic from the opposite direction, the presence of complaints in the orofacial area could also direct the self-report of bruxism; that is, a person who

has a nagging pain in the masticatory muscles may be inclined to report more oral behaviors, such as clenching and nail biting, than someone without such complaints in an attempt to find an explanation for the cause of the complaints.¹¹ In other words, recall bias might influence the outcomes of studies that rely on self-report information,¹² as patients with higher levels of orofacial pain might be more inclined to recall the exposure to certain risk factors (eg, oral behaviors) compared to those with lower levels.

When persons are confronted with signs of illness, they generally develop beliefs about the cause of their condition.^{13,14} This type of belief can have several names, such as causal attribution belief, illness belief, and illness attribution.^{15,16} Medical knowledge, whether accurate or not, is integrated with previous experiences of the patients themselves, their relatives, or acquaintances with similar symptoms or diagnoses in order to interpret why the illness has occurred.¹⁷ A fascinating aspect of causal attribution belief is that patients with the same illness can have diverging beliefs about the cause of their condition. The meanings given by a patient about the cause of their condition are influenced by a host of psychosocial and cultural factors.^{18,19} For example, causal explanations for common somatic symptoms are influenced by gender in that women use more psychologic explanations than men.²⁰ Likewise, catastrophizing may give rise to dysfunctional beliefs regarding the cause of illness.²¹ On the other hand, causal illness beliefs can strongly influence the emotional response, particularly if the patient blames him- or herself for the illness.¹⁸ This may result in feelings of helplessness, guilt, and/or depression.^{19,22}

To the best of the authors' knowledge, only one study has examined TMD pain patients' causal attribution beliefs in relation to bruxism.²³ According to that study, most patients who were diagnosed with a pain-related TMD believed in the potentially harmful effects of excessive masticatory muscle activity. The authors also reported that patients who bruxed more also believed more strongly in this relationship.²³ In other words, bruxism may have a different association with TMD pain under conditions of a high belief in causal attribution compared to conditions of low belief. Since the authors of that study did not investigate if the association between self-reported oral behaviors and TMD pain was modified by the belief that these behaviours are harmful to the jaw (ie, causal attribution belief), the first aim of this study was to investigate whether the causal attribution belief moderated the relationship between awake oral behaviors and orofacial pain among patients diagnosed with TMD pain. The focus was on the self-report of awake oral behaviours, and not on sleep bruxism, because poor validity of sleep bruxism self-reports has repeat-

edly been shown.^{11,24} In addition, a recent systematic review of the literature suggested a positive association between awake bruxism and TMD pain, whereas the association with sleep bruxism is still unclear.²

Another noteworthy finding of the study by Van der Meulen et al was that a strong correlation was observed between the strength of the belief in muscle activity as the causation of TMD pain and the amount of self-reported bruxism.²³ Apparently, the "believers" (ie, patients with TMD pain who were fully convinced that awake/sleep clenching and/or grinding was causing their pain) scored significantly higher in the self-reported frequency of these behaviors than the "nonbelievers." So far, little is known about the factors that affect the beliefs of patients with TMD pain regarding whether their oral behaviors can cause their pain complaint, and if these factors also influence the frequency of reporting these behaviors. Since psychologic factors can have an influence on causal attributional beliefs²⁵ as well as on the frequency of self-reported bruxism,²⁶ it can be speculated that the previously reported association between oral behaviors and the belief in these behaviors as the cause of TMD pain was in fact partly due to shared psychologic risk factors. Given this background, the second aim of the present study was to examine if the association between causal attribution belief and the report of awake oral behaviors is (in part) the result of shared risk factors. It was hypothesized that the association between awake oral behaviors and the belief that these behaviors cause TMD pain would be mediated by a third variable (eg, somatic symptoms, depression, and/or anxiety).

Materials and Methods

Study Design and Participants

The study design was a retrospective medical record review study from patients attending the specialty Clinic for Orofacial Pain and Dysfunction of the Academic Centre for Dentistry Amsterdam (ACTA), The Netherlands. Participants in this study were consecutive patients referred to this clinic between September 2014 and January 2017 in whom a diagnosis of myalgia according to the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) was established.²⁷ This study focused on myalgia because myalgia is the pain-related subtype of TMD that is especially believed to be associated with prolonged nonfunctional jaw activities.² Participants were at least 18 years of age. Only data of patients who gave permission for the anonymous use of their information for research purposes were used. This study was considered by the ethical committee of ACTA not to fall under the provisions of the Medical

Research Involving Human Subjects Act and to comply with the ethical research code of conduct at ACTA (ref no. 2017006).

Questionnaire

As part of the usual care, all patients completed a digital diagnostic questionnaire before their initial visit to the clinic. Among others, this questionnaire includes screening tools for TMD pain intensity, pain-related disability, oral behaviors (including bruxism), and psychologic functioning; these tools are part of the DC/TMD Axis II protocol.²⁷ Frequency of oral behaviors was obtained using the Dutch version of the Oral Behaviours Checklist (OBC).²⁸ The OBC is a 21-item scale for identifying and quantifying self-reported frequency of oral behaviors.²⁹ The response options range between 0 ("none of the time") and 4 ("all of the time"). For this study, the sum score of the 19 activities that can be performed during waking hours was used for analysis, where a higher score indicates a higher frequency of awake oral behaviors. Subsequently, patients' beliefs about the causal relation between oral behaviors as mentioned in the OBC and the patient's jaw pain complaint was assessed using the question: "Do you think these behaviours are harmful to your jaws, jaw muscles and/or teeth?" This question is modified from the one formulated by van der Meulen et al in such a way that it inquires about oral behaviors only instead of also asking about other factors, such as stress and occlusion.²³ The response options are 0 ("no"), 1 ("a little"), 2 ("somewhat"), 3 ("much"), and 4 ("very much"). Orofacial pain intensity was assessed by means of three questions from the Graded Chronic Pain Scale as implemented in the DC/TMD²⁷ (orofacial pain: right now, worst pain, and average pain). The average of the three questions, as rated by numeric rating scale (NRS) scores (0 to 10), was calculated and multiplied by 10 in order to give a 0–100 score.

Clinical Examination

At the initial visit, all patients underwent a standardized clinical examination performed according to the DC/TMD protocol.²⁷ The clinical diagnosis 'myalgia' was based on information derived from both the questionnaire and the physical examination as implemented in the DC/TMD Axis I protocol. Myalgia was considered present when at least one of the following DC/TMD pain diagnoses was present: local myalgia, myofascial pain, and/or myofascial pain with referral.²⁷ The physical examinations were performed by one of 10 well-trained dentists from the OPD clinic. Each year, the dentists are calibrated to perform the DC/TMD clinical examination by a dentist previously trained and calibrated in the DC/TMD clinical examination.³⁰

Mediating Variables

The variables for which a mediating effect on the association between a person's causal attribution belief and report of oral behaviors examined were somatic symptoms, depression, and anxiety. Three DC/TMD Axis II instruments were used to obtain a comprehensive assessment of these variables.²⁷ The Patient Health Questionnaire (PHQ)-15, which is a list of 15 somatic symptom clusters that account for more than 90% of all physical complaints, was used to assess somatic symptom severity.³¹ Each symptom is scored from 0 ("not bothered at all") to 2 ("bothered a lot"), and the total score ranges from 0 to 30. To screen for depression, the PHQ-9 score was used.³² The total score is calculated by assigning scores from 0 ("not at all") to 3 ("nearly every day") to the answers on the 9 items (range 0 to 27). Finally, for screening for anxiety, the Generalized Anxiety Disorder (GAD)-7 was used.³³ The total anxiety score (range 0 to 21) is based on the scores of the 7 items (0 = "not at all" to 3 = "nearly every day").

Statistical Methods

Descriptive statistics were used to characterize the patients who were clinically diagnosed with myalgia. In order to investigate if a patient's causal attribution belief influences the association between oral behaviors and orofacial pain, moderation statistics were used. Moderation is a methodology that statistically defines the relationship between two variables as a function of a third variable. First, the bivariate association between the independent variable (awake oral behaviors) and the dependent variable (orofacial pain intensity) was analyzed using a single linear regression model. Subsequently, the independent variable, the potential moderator variable (causal attribution belief), and the interaction term between the potential moderator and independent variable were added simultaneously into a multiple regression model to study their association with the dependent variable. Moderation is considered present in cases where the interaction term in that model is significant, meaning that the relationship between the independent variable and the dependent variable is different at different levels of the moderator variable.^{34,35} In case of a significant interaction effect, post hoc probing of moderation effect was performed by examining the slopes of regression lines at different conditions of the moderator. Aiken and West recommended computing the simple slopes of high and low values of the moderator, which represent the relationship between the independent and dependent variables at those values irrespective of what this relation is at other values of the moderator.³⁶ The steepness of the slopes indicates the strength of the association under the various conditions of the moderator. Moderator value

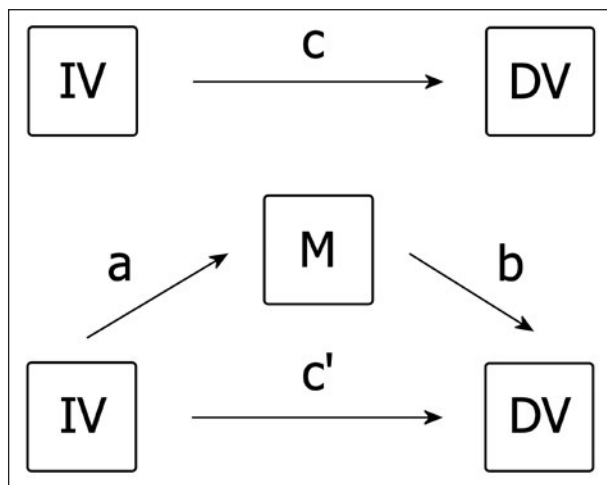


Fig 1 Graphical representation of the relationships between the independent variable (IV), dependent variable (DV), and mediator variable (M). The influence of the IV on the M is represented by path a; the influence of the M on the DV after controlling for IV is represented by path b; the relationship between the IV and the DV is represented by path c; and the relationship between the IV and the DV after adjusting for M is represented by path c'.

Table 1 Median (Interquartile Range) Scores for Awake Oral Behaviors, Causal Attribution Belief, Pain Intensity, and the Three Potential Mediators as Reported by Myalgia Patients (n = 329)

	Range	Median (IQR)
Awake oral behaviors	0–76	24 (18–32)
Causal attribution belief	0–4	1 (1–2)
Pain intensity	0–100	60 (42–73)
Somatic symptoms	0–30	10 (6–13)
Depression	0–27	5 (2–9)
Anxiety	0–21	4 (1–8)

0 ("no") represented the low condition of causal attribution belief, whereas 4 ("very much") was the high condition; the intermediate value 2 ("somewhat") was added to complement this method.

In order to test the second hypothesis, which was to investigate if the association between causal attribution belief and oral behaviors was (in part) the result of shared risk factors, mediation statistics were used. Mediation statistics attempt to determine if the relationship between an independent variable and a dependent variable is better explained via the inclusion of a third hypothetical variable, known as a mediator or mediating variable (Fig 1).^{35,37} The four different pathways included in the mediation model can be estimated by means of a series of regression models and are termed as follows. The association between the independent variable and the mediator (unstandardized regression coefficient) is represented by path a. Path b is the effect of the mediator on

the dependent variable, controlling for the independent variable. The relationship between the independent and dependent variables without adjustment is the total effect (path c). Finally, the c' coefficient in Fig 1 defines the effect of the independent variable on the dependent variable controlling for the mediating variable. A basic assumption of mediation is that path c should get smaller with the addition of a mediator.³⁵ The Sobel test was used to determine the significance of the indirect effect of the independent variable (causal attribution belief) on the dependent variable (self-reported oral behaviours) through the mediators (somatic symptoms, depression, and anxiety). The Sobel test makes use of the regression coefficients of path a and path b and their standard errors³⁸ and was conducted using an online calculator.³⁹ Finally, the percentage of the total effect (path c) accounted for by the indirect effect ($a \times b$) was interpreted as the relative magnitude of the mediation effect (ie, percent mediation: $P_M = a \times b/c$).⁴⁰ All analyses were performed using the IBM SPSS Statistics 26 software package (IBM). Probability levels of less than .05 were considered statistically significant.

Results

During the study period, a clinical examination was performed on 924 eligible patients. Of these patients, a clinical diagnosis of myalgia was established in 329, who were included in the study. Most study participants were female (82.4%), and the mean age was 41.9 years (SD 14.7; range 18 to 78). Patients' characteristics for awake oral behaviors, the patient's belief that these behaviors are harmful to the jaw, pain intensity, and somatic symptoms, depression, and anxiety are summarized in Table 1.

According to the single regression model, the report of awake oral behaviors was positively associated with orofacial pain intensity ($P = .029$, Table 2). Since the interaction term "awake oral behaviors \times causal attribution belief" was significant ($P = .046$), it appeared that the causal attribution belief was a moderator of the association between awake oral behaviors and orofacial pain intensity. Post hoc probing showed that the simple regression slope for the "very much" belief that awake oral behaviors causally attribute to the orofacial pain complaint was positive, whereas the slope was almost horizontal (ie, no association) in case of the causal attribution belief being "no" (Fig 2). According to Pearson correlation coefficient, the strength of the linear relationship was $r = 0.512$ ($P = .001$) for the causal attribution belief condition "very much"; $r = 0.131$ ($P = .238$) for the condition "somewhat"; and $r = 0.013$ ($P = .928$) for the condition "no belief." Finally, the scatter plots

Table 2 Results of Linear Regression Analyses with Orofacial Pain Intensity as the Dependent Variable, Awake Oral Behaviors as the Independent Variable, and Causal Attribution Belief as Moderator in Patients with TMD Pain (n = 329)

Independent variables	Single regression			Multiple regression		
	B	t	P	B	t	P
Awake oral behaviors	0.300	2.191	.029	0.143	0.592	.554
Causal attribution belief				-8.728	-2.890	.004
Awake oral behaviors × causal attribution belief				0.209	2.007	.046

The multiple regression analysis includes the interaction term between the independent variable and the potential moderator. B = unstandardized regression coefficient.

indicate that patients with a high belief in a causal attribution to their pain complaint reported more awake oral behaviors compared to those who did not believe in this attribution (Fig 2).

Table 3 shows the results of the mediation analyses. For each of the potential mediators (somatic symptoms, depression, and anxiety), their effects on the relationship between causal attribution belief (independent variable) and self-reported oral behaviors (dependent variable), as described in Fig 1, were calculated and tested for significance using Sobel test statistics. It appeared that a causal attribution belief was indeed positively associated with the report of oral behaviors (path c). In addition, causal attribution belief was also positively associated with all potential mediators (somatic symptoms, depression, and anxiety; path a), whereas these potential mediators were associated with self-reported oral behaviors, controlling for the independent variable attribution belief (path b). When the effects of somatic symptoms, depression, and anxiety were controlled for, the respective unstandardized regression coefficients between causal attribution belief and self-reported oral behaviors (path c') were reduced. For all potential mediator variables, the Sobel test statistics demonstrated significant mediating effects. In other words, the relationship between causal attribution belief and self-reported oral behaviors was partially explained by somatic symptoms, depression, and anxiety. Somatic symptoms mediated almost 8% of the relationship between causal attribution belief and self-reported oral behaviors, and depression mediated 9%, while the highest proportion of mediation was due to anxiety (almost 16%).

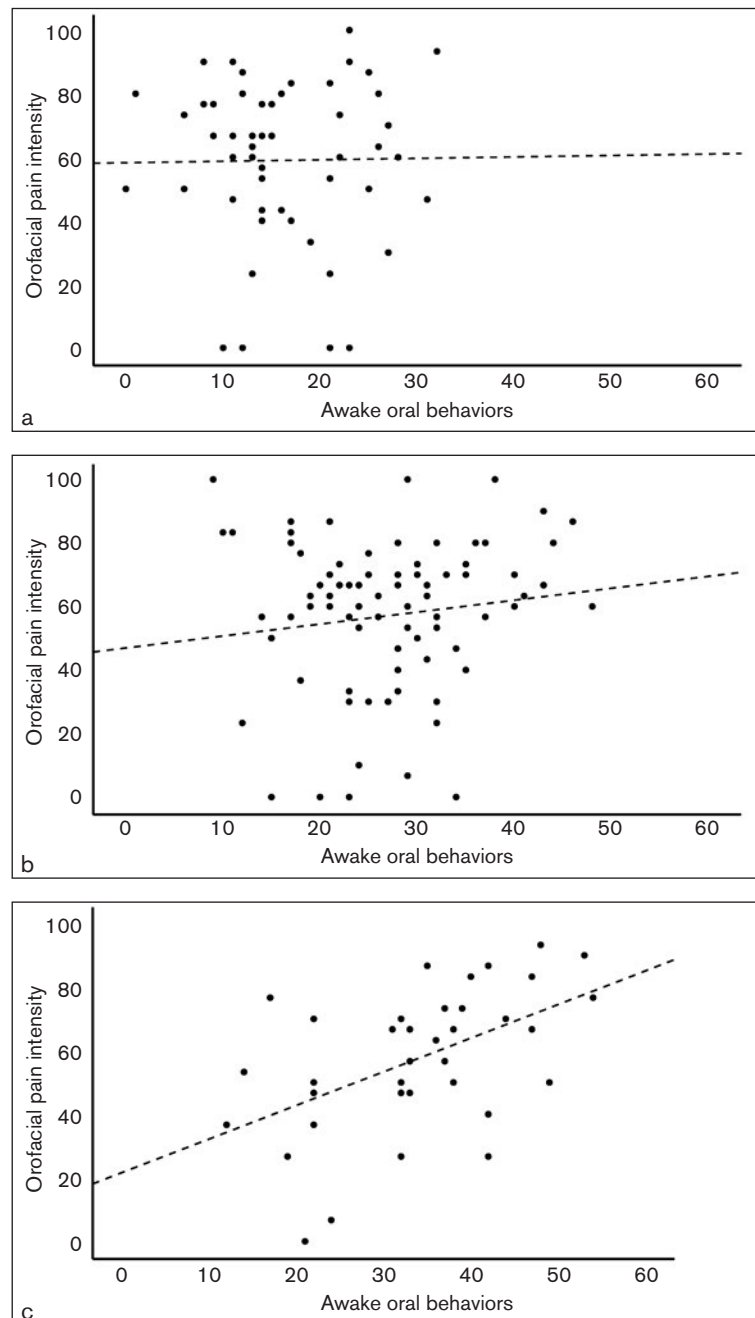


Fig 2 Scatter plots (with best-fitting regression lines) depicting associations between awake oral behaviors and orofacial pain intensity under three conditions of the moderator: **(a)** causal attribution belief is "no" (n = 54); **(b)** causal attribution belief is "somewhat" (n = 83); and **(c)** causal attribution belief is "very much" (n = 36).

Table 3 Results from the Mediation Analyses Exploring the Indirect Effects of Causal Attribution Belief on Self-Reported Awake Oral Behaviors via Potential Mediators

Total effect (path c)	Regression coefficients					
	Path a	Path b	Path c'	Sobel test statistic (SE)	P	% mediated
4.27*						
Mediators						
Somatic symptoms	0.58*	0.57*	3.96*	2.30 (0.14)	.021	7.7
Depression	0.73*	0.52*	3.91*	2.73 (0.14)	.006	9.0
Anxiety	1.20*	0.56*	3.61*	4.05 (0.17)	< .001	15.9

Depicted are the unstandardized regression coefficients for paths a, b, c, and c' that reflect the paths as described in Fig 1.

In addition, the outcomes of the Sobel test statistic, including standard error (SE), and the proportions of mediation are presented.

*Significant at the 5% level ($P < .05$).

Discussion

A commonly held view in the literature and dental practice is that oral behaviors, such as clenching and nail biting, play a causal role in the presence of orofacial pain. Furthermore, it is often believed that there is a dose-response gradient between both conditions so that more muscle activity leads to more overloading and thus to more pain.⁴¹ However, as noted by Manfredini et al, it is very likely that other and more complex relationships exist between the amount of performed masticatory muscle activities and the purported consequences of those activities, such as orofacial pain.⁴² Therefore, an interesting finding of the current study was that self-reported oral behaviors and orofacial pain could have a dose-response association. This association was, however, moderated by the belief that these behaviors are harmful to the jaw (aim 1). Since little is known about the factors that affect the beliefs of patients with TMD pain regarding whether or not their oral behaviors can cause their orofacial pain, it was also investigated if the association between causal attribution belief and self-reported oral behaviors was (in part) the result of shared risk factors (aim 2). It appeared that somatic symptoms, depression, and anxiety each partially explained the relationship between causal attribution belief and these behaviors.

The main finding of the present study is that there was a dose-response gradient between awake oral behaviors and orofacial pain intensity; this association was, however, only present among the group of patients with TMD pain who strongly believed that these behaviors are harmful to the jaw. An explanation for this might be that patients with a strong belief in a causal attribution of awake oral behaviors to their jaw pain complaint are better capable of recalling the exposure to awake oral behaviors compared to those who do not believe in this relationship. This can be considered as an extension of the earlier suggested recall bias concept, which stated that patients who suffer from a disease may be more likely to recall the exposure to certain risk factors compared to those without that disease (ie, controls).¹²

This study confirmed earlier results, namely that there is a correlation between causal attribution belief and frequency of self-reported oral behaviors.²³ This implies that TMD patients with myalgia report more awake oral behaviors in cases of a strong belief that performing these behaviors is harmful to the jaw, whereas those who do not believe in it report less behaviors. As a consequence, this finding seems to undermine the strength and validity of questionnaire-based assessment of oral behaviors because this assessment is based on the assumption that the self-report of oral behaviors is sufficiently accurate to reflect the actual frequency of these behaviors. This underlines the need to search for valid, accurate, and reliable measurement tools that reflect the actual frequencies of oral behaviors.⁴²

The current study provides evidence to support the hypothesis that the relationship between causal attribution belief and self-reported oral behaviors is partially mediated by the presence of somatic symptoms, depression, and anxiety. The strongest mediator appeared to be anxiety (almost 16%), followed by somatic symptoms (almost 8%) and depression (9%). In other words, these psychological factors may affect the beliefs of TMD pain patients regarding whether or not their oral behaviors can cause their orofacial pain. Already in 1993, Joubert reported that the frequency of several oral behaviors is related to anxiety, and that it is the person's qualification of these behaviors as a problem rather than their frequency that is related to higher anxiety and lower self-esteem.⁴⁴ The present finding that the report of frequent oral behaviors was associated with higher anxiety and depression scores makes it very likely that self-report measures of oral behaviors indeed partly depend on psychological determinants of health. This corroborates well with the suggestion that the complex bruxism-psyche relationship could actually drive self-reporting of awake oral behaviors, meaning that self-report of oral behaviors reflects psychological distress rather than actual masticatory muscle activity.²⁴ In addition, both somatosensory amplification (ie, an estimate of bodily hypervigilance) and trait anxiety have been found to be positively associated with the report of oral

behaviors.⁴⁵ As such, the odds of noticing physiologic symptoms may be higher among anxious patients due to an increased internal focus of attention.⁴⁶ This would mean that in patients who are clinically diagnosed with myalgia, the general awareness of occlusal sensations is higher in cases of elevated anxiety scores. Since anxiety appeared to be the strongest mediator of the association between awake oral behaviors and the belief that these behaviors cause orofacial pain, an examination of comorbid anxiety disorders may be an important tool for the clinician to effectively manage these types of patients.

A limitation of the present study involves its design. All participants completed the digital questionnaire prior to their first visit to the Clinic for Orofacial Pain and Dysfunction. Consequently, the questionnaire may thus have functioned not only as a tool to gather information, but may also have made patients more aware of oral behaviors, thereby possibly changing their beliefs about the cause of their TMD pain complaint.²³ Since it is known that patients with the same illness can have diverging beliefs about the cause of their condition, beliefs that are influenced by a host of psychosocial and cultural factors, it might thus well be possible that patients with elevated scores for somatic symptoms, depression, and anxiety become more aware of their oral behaviors just by completing the digital questionnaire. Future research is needed to elucidate the role of such factors in the beliefs of patients with TMD pain regarding whether their oral behaviors can cause their pain complaint. Another point of concern deals with the test statistics employed to consider mediation to be present. In mediation analysis, the most common method to test the effect of an intervening variable is the Sobel test, which comprises the ratio of the effect size and the standard error of the indirect effect. Even though the Sobel test is quite conservative and as such has a low power,⁴⁷ a comparison with other mediation tests revealed comparable results.⁴⁸

Conclusions

Based on the present findings, it is concluded that causal attribution belief moderates the association between awake oral behaviors and orofacial pain intensity among TMD patients clinically diagnosed with myalgia. It appeared that self-reported awake oral behaviors were positively associated with orofacial pain intensity, albeit only under the condition of a high belief that such behaviors can cause the jaw pain complaint—no association between these conditions was present in case of a low belief. In addition, there was a positive correlation between causal attribution belief and frequency of self-reported oral behaviors.

Finally, the relationship between causal attribution belief and self-reported oral behaviors was partially mediated by the presence of somatic symptoms, depression, and anxiety. Anxiety in particular appeared to be an important component of the relationship between causal attribution belief and the report of awake oral behaviors.

Highlights

- The existence of an association between the frequency of self-reported awake oral behaviors and the intensity of orofacial pain depends on the belief of patients that these behaviors are harmful to the jaw.
- Causal attribution belief and frequency of self-reported oral behaviors are correlated, implying that TMD patients with myalgia report more awake oral behaviors in cases of a strong belief that performing these behaviors is harmful to the jaw, and vice versa.
- Somatic symptoms, depression, and anxiety partially explained the relationship between causal attribution belief and self-reported oral behaviors, which may imply that the self-report of oral behaviors reflects psychologic distress rather than actual masticatory muscle activity.

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