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MALOCCLUSIONS, ORTHODONTIC TREATMENT AND OROFACIAL PAIN

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Dedicated to Matija, Prosper and Vedrana

ABSTRACT

Temporomandibular disorders (TMD) are myoarthropathies of the orofacial region, characterized by orofacial pain and dysfunction of the temporomandibular joint (TMJ). The aims of the study were to translate and validate TMD-Pain Screener instrument in Croatia, to assess the what extent to which orofacial pain and TMJ dysfunction are present in patients referred for orthodontic consultation and to identify the predictors of clinically diagnosed temporomandibular disorder and those of its two components (pain disorder and joint disorder).

The validation study included 134 participants (students of Universtiy of Rijeka and patients of University Dental Clinic Rijeka, Croatia) aged 11-62 years (median 23, interquartile range 21-24), 76% females and 82% adults who self-administered TMD-Pain Screener. For the assessment of temporal stability 23 participants completed the questionnaire twice in a two week interval without any interventions; 14 had painful TMD. The orthodontic sample consisted of 352 consecutive subjects who came for orthodontic consultation at the Department of Orthodontics of the University Dental Clinic in Rijeka in 2018. The age range was five to 52 years, with a median of 12 years (interquartile range 10-15), with 52% female and 9% adult subject. Screening for orofacial pain and TMJ dysfunction was undertaken using a TMD-Pain Screener. Clinical examination and diagnostics were performed according to the Diagnostic Criteria for Temporomandibular Disorders protocol. Occlusal characteristics, breathing and swallowing patterns, facial asymmetry, previous orthodontic treatment, self-reported parafunctions and chewing problems, were recorded.

The Croatian version of the TMD-Pain Screener has good ability to detect subjects with painful TMD. Orofacial pain and TMJ dysfunction are not frequent in people referred to orthodontists. Malocclusions and previous orthodontic treatment are not predictors of TMD. The TMD-Pain Screener is a strong predictor of clinically confirmed orofacial pain, identifying up to 6.9-times-higher odds, but it is not a significant predictor of TMJ dysfunction.

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1.0. INTRODUCTION

1.1. Temporomanbibular Disorders

Temporomandibular disorders (TMD) are myoarthropathies of the orofacial region characterized by orofacial pain of nonodontogenic origin and dysfunction of the temporomandibular joint (1). Signs of disorder include primarily the impairment of mandibular kinematics, which can be quantified as a reduced amount of mouth opening (maximum painless, maximum active/unassisted and maximum passive/assisted opening), limited protrusion movement and/or an asymmetric degree of laterotrusive movement. Signs also include the inability to close the mouth and an opening pattern with deviation from a straight line. In addition, the presence of sounds such as clicking and crepitus was recorded. Symptoms reported by the patient are crucial for diagnosis and are often a better indicator of a condition than clinical examination (2).

Symptoms include pain in muscles and joints and headache at rest, with localization and spreading of the pain, as well as changes in pain due to function (induction or reduction). In fact, pain is the main reason why patients seek help, and these patients are treated. The patient reports the characteristics, intensity and duration of pain, as well as initiating and inhibiting factors. Acute TMD is not a big problem; it often has a good prognosis and is well rehabilitated, but chronic pain is a major problem that significantly reduces working ability and quality of life.

A common criterion for acute painful TMD is the presence of pain for at least five days in the last 30 days in masticatory tissues, confirmed by palpation, together with pain in the muscle and/or jaw joint provoked during the examination - whether by palpation or mandibular movement. If the condition lasts more than three to six months, or if the pain persists after healing of injured tissues, it is considered chronic TMD.

Painful TMD is present in 5% of the general adult population aged 18+ years, twice as often in women as in men (6 vs 3%) (3). This estimate is considered to be quite accurate, since more than 30,000 people

were surveyed in the United States, with all age groups from of 18 to 75+ eaqually represented, including Caucasian and South African races. Symptomatology fluctuates throughout the lifespan. A study that followed patients for five years indicated that TMD occurring over a longer period during life, persists in one third of people with the same intensity and shows remittent symptoms in another third, and recurrent symptoms in the remaining third (4). The prevalence of temporomandibular disorders and pain tends to increase in adolescence and up to the age of 40, gradually diminishing thereafter. Therefore, there is a favourable prognosis, since the condition tends to improve (5).

In addition to twice the prevalence of TMD pain in women than in men, women are more sensitive to pain than men (6). This is conditioned by the physiological and psychological characteristics of the sexes, and the sensitivity tends to decrease with age. Racial differences are not great, but Caucasians are the least sensitive, and South Africans the most. However, differences are not conditioned by tissue characteristics or innate sensitivity of the nociceptors but by cognitive, psychological and affective factors (8).

There are two aetiological models of acute TMD. The first defines the symptoms as a consequence of impaired regulation within the central nervous system, i.e., outside the chewing structures (9). Hence, the pain of the masticatory system is a primary manifestation of dysregulation, and the limitation of jaw function and joint problems are only consequences. According to the other model, oral parafunctions or trauma cause masticatory tissue damage, and so the peripheral nociceptor changes are a consequence of damage contributing to pain and function restriction (10, 11). TMD probably starts with peripheral pain, after which peripheral sensitivity becomes a normal part of the protective role of nociceptors. The chronicity occur in the form of neuroplasticity, central sensitization and reorganization of the cortex.

Great progress in understanding the aetiopathogenesis of chronic TMD has resulted from the research project Orofacial Pain: Prospective Evaluation and Risk Assessment (OPPERA), which recruited more than 3,000 subjects without TMD (not experiencing pain) aged 18-44 years, between 2006 and 2013, in four centres in the United States, followed them for an average period of three years and recorded the incidence of TMD. The study also included a cohort of around 200 subjects with painful TMD who were

also followed. The criterion for painful TMD was the presence of pain for at least five days in the last 30 days, confirmed by palpation and provoked by mandibular movement (12).

The incidence of clinically confirmed TMD is 4% per annum in the adult population aged 18-44, but the annual rate of initial symptoms of orofacial pain is higher (19%) (13, 14). The aetiology of chronic painful TMD is complex and includes a range of biopsyhosocial, environmental and genetic factors that contribute to the onset and presence of disorders as predisposing, initiating and perpetuating factors (10).
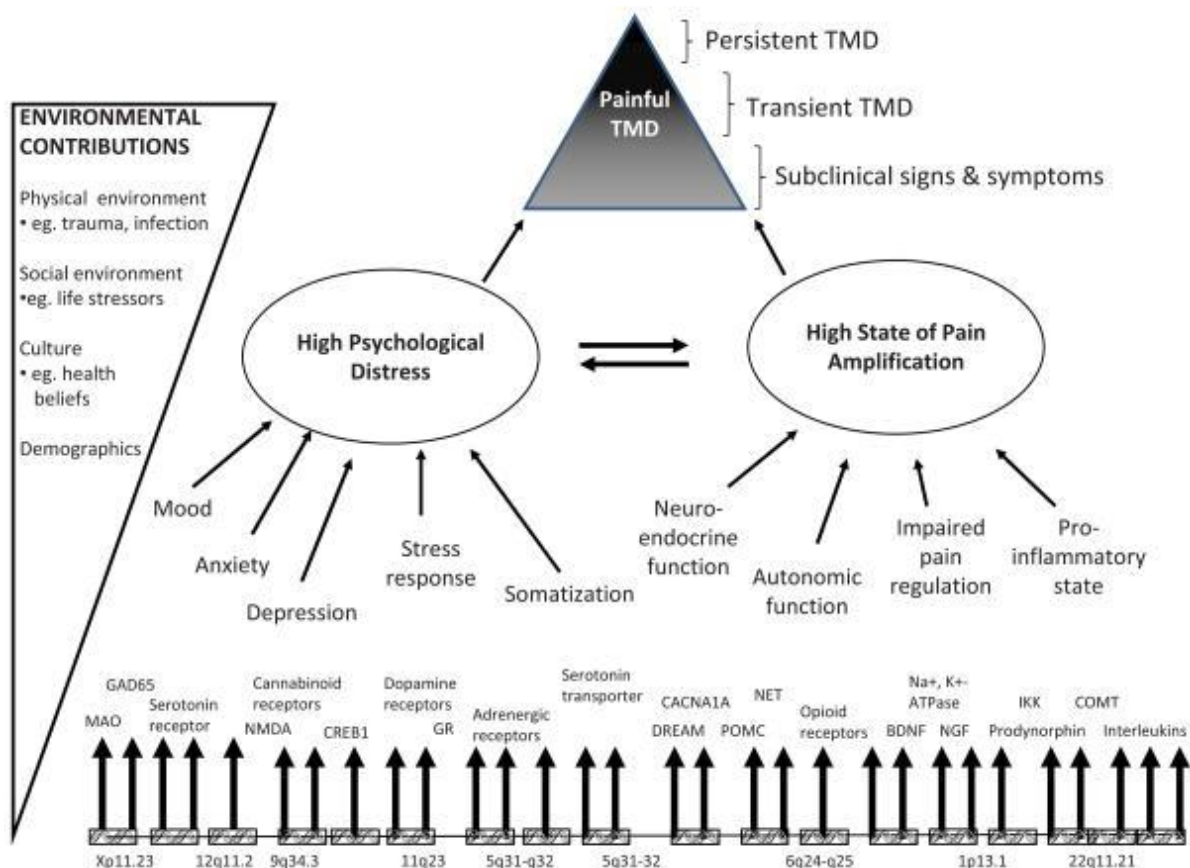

Figure 1. Aetiology of paintful TMD (15)

Two phenotypes are responsible for the onset and persistence of painful TMD - psychological suffering and pain amplification. These may act in synergy (15).

Each of these phenotypes is a combination of specific risk factors. Pain amplification is a condition that causes normal pain sensations to be stronger and more intense than usual, and it includes several specific phenomena, such as excessive sensitivity to pain (hyperalgesia), painful experience of painless stimuli

(allodynia) and excessive excitation of the spinal cord neurons (central sensitization). It is manifested as increased sensitivity during sensory testing and spontaneous pain from deep structures (muscles, joints and internal organs). Pain amplification is affected by impaired pain regulation, neuroendocrine and cardiovascular function, and a pro-inflammatory state. Psychological distress is an unpleasant feeling of emotional pain, psychological discomfort and suffering of a non-physical origin, that interferes with the activities of daily life and impacts on the level of functioning. It is influenced by anxiety, depression, somatization, stress and mood.

Environmental factors (parafunctions, injuries and life stress situations) have a secondary effect on interactions between phenotypes and the risk factors associated with phenotypes, and they also contribute to the onset and persistence of painful TMD. Genetic regulation of biological mechanisms determines the expression of phenotypes and their risk factors, and time is an indispensable factor in the development of chronic pain. The TMD-vulnerable phenotype is therefore generated by the interaction of genetic variations affecting psychological traits and pain sensitivity, and environmental factors such as physical damage and emotional stress (15).

In order to be effective in treating TMD pain, cases should be anatomically classified using aetiological principles. Although TMD is heterogeneous condition composed of a mosaic of complex biopsychosocial phenotypes, it is possible to identify three groups of chronic TMD cases: adaptive cases, pain-sensitive individuals and those with global symptoms (16). Adaptive cases have a localized pathology with low pain susceptibility, and the other two clusters have high sensitivity to pain due to sensation from the central nervous system. People with global symptoms in addition to sensitivity to pain, also have a pronounced dimension of psychological suffering.

Most people with TMD have increased sensitivity to pain alone or pain associated with global symptoms. In addition, they report higher pain intensity, jaw function constraints and other painful comorbidities. The most common comorbidities are irritable bowel syndrome, pelvic pain, chronic headache and chronic pain in the lower back. Healthy people with generalized symptoms, have a 2.8-times higher risk of TMD

development during a three-year follow-up period. Psychological suffering, along with neurosensory regulatory processes, is a very important determinant of TMD.

The presence, frequency and type of headache are important determinants of TMD. Migraines and a mixed type of headache are predictors, but tension headaches are not (17). A headache frequency of two to four per month increases the risk of TMD by 1.6-3.1 times. An increasing number of headaches over time increases the llikelihood of TMD during the five-year follow-up. In people with TMD, the presence of migraine increases the risk by 10 times, and exacerbation of lower hierarchical forms of headaches towards migraine also occurs. The likelihood of progression is increased by 1.9-2.8 times. Therefore, screening, monitoring and adequate treatment of migraines should be implemented as a preventative strategy for reducing the risk of TMD development.

Impaired sleep quality contributes to TMD onset, doubling the risk, to a large extent directly but also to a certain extent mediated by increased psychological stress (18). As poor sleep quality leads to increased stress, leading to painful TMD, sleep hygiene can reduce stress and reduce the risk of TMD development. Obstructive sleep apnoea (OSA) almost doubles the odds ratio (OR) for TMD, increasing it even more for chronic TMD (OR = 3.6). In screening for OSA, the presence of at least two of the following signs is sufficient: loud snoring, daily tiredness, observed sleep apnoea and hypertension (19).

Bruxism is associated with TMD in children and adults, especially night bruxism with myofacial pain, arthralgia and disc displacement in adults (20, 21). Night bruxism could actually be a defence mechanism against obstructive sleep apnoea in some cases. In order to open the airway through the mouth during sleep, a person must move the mandible forward, causing the teeth to grind. In some people, OSA and bruxism appear independently of each other, while bruxism can sometimes induce OSA due to the mucosal oedema induced by the trigemino-cardiac reflex (22-25). Sleeping and waking bruxism should not be considered a sleep disorder or a movement disorder but a parafunctional behaviour of healthy persons characterized by unconscious activity of the masticatory muscles (26). Gastroesophageal reflux could be also associated with TMD through bruxism, in the same way as OSA (27, 28).

Of the many potential factors that could be predictors of the onset of clinically confirmed painful TMD in previously asymptomatic persons, the most significant are self-reported comorbid health conditions, jaw parafunctions, somatization and orofacial symptoms where the pain is not specific (jaw tension, spasm, fatigue, pressure or discomfort) (29-32).

Self-reported symptoms are particularly important, especially those related to organ systems distant from chewing structures, and these are more significant predictors than clinical examination. Clinically detected joint sounds and wear facets are not predictors of TMD. Obviously, the aetiology of TMD is complex. It is influenced by local disorders of chewing structures but also by systemic mechanisms of pain regulation. It is impossible to find a single cause that is sufficient in itself for inducing TMD; rather, chronic TMD is a disorder of several organic systems with overlapping comorbidities. Therefore, it cannot be considered only as a localized orofacial pain condition, and primary prevention of TMD should be oriented towards general health promotion (32).

1.2. Malocclusions and Temporomandibular Disorders

Malocclusion includes a broad range of structural occlusal characteristics that differe from a theoretically ideal occlusion. Although the prefix 'mal' means 'bad' or 'ill', the malocclusion is not a non-physiological condition and treatment is not necessarily needed. Malocclusion is often an occlusal adaptation to skeletal or dento-alveolar discrepancy or enlarged or altered position of soft tissues, that manages to create a functional equlibrium. No clear boundary between acceptable and pathological occlusion has yet been defined (33).

Some static occlusal characteristics have long been associated with dysfunctions of the joints and orofacial pain: unilateral crossbite, skeletal open bite, overjet over 6mm and absence of lateral teeth. However, they have also been related with dynamic characteristics: mediotrusion interference, orthopaedically unstable occlusion with forced bite and discrepancy between retruded contact position and maximum intercuspation (RCP-ICP) (34-36).

However, in the population of TMD patients, the odds for joint clicking are minimally increased in persons with mediotrusion interference and RCP-ICP over 2 mm (OR = 1.6 and 1.9). Occlusion characteristics account for a very small share of clicking prevalence (4.5%) without clinical relevance (37). This is a result of a recent study in a group of 442 subjects aged 25-44, which controlled for the influence of other occlusal characteristics. Furthermore, in a sample of 625 subjects in the same age range with painful disorders and joint disfunctions of the jaw joint, no correlation was found between the characteristics of static and dynamic occlusion and painful disorder (38). Moreover, the prevalence of occlusal characteristics is similar among subjects with painful and painless forms of TMD and is much the same as in the TMD-free population. During a 20-year follow-up of 100 examinees, only the forced crossbite, out of all occlusal characteristics, showed an association with some sign of joint dysfunction (in this case clicking), but the link was weak (39). The correlation in this case was r = 0.31, and in interpreting the power of the

association, the usual criteria are: 0.1-0.3 = small, 0.3-0.5 = moderate, 0.5-0.7 = large and > 0.7 = very large (40, 41).

Malocclusion used to be thought to be related to body posture, and posture related in turn with TMD. It was argued that scoliosis creates a risk of unilateral crossbite and TMD. Studies focused on posturography assessed cases using postural platforms that were not suitable for studying the relation between the masticatory system and body posture, due to large variations in the measured postural variables (42, 43).

Malocclusion cannot be associated with posture, as confirmed by a recent observational study on a cross-sectional sample of children and young adolescents, which demonstrated no correlation between the presence of scoliosis and the more frequently present unilateral crossbite (44-46). In addition, posture is not related to TMD (47). Experimental studies also deny that acute alteration of occlusion could induce changes in posture, and that postural changes could induce orofacial pain and dysfunction (48, 49).

Therefore, there is no scientific evidence of the correlation between occlusion, posture and TMD, and the link is probably missing due to the numerous compensatory mechanisms that exist within the neuromuscular system, balancing the body (50).

The shortcomings of observational research in the field include cross-sectional design and non-evaluation of the strength of correlations (low, moderate or high). Due to the first limitation, a time sequence and a cause-consequence relationship could not be established. Therefore, it was not possible to say whether malocclusion was the cause of TMD or the consequential occlusal adaptation to TMD. There are few longitudinal observational studies with long-term follow-up of function and pain in the orofacial area regarding occlusal characteristics and development and changes of occlusion. Apart from the research design and evaluation of the effect size, another problem of research in the field is the use of non-uniform methodological criteria (51). Some studies were based on the Helkimo index, and part on Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD or DC TMD) established by an international consortium network, while a few used other criteria (2, 52). Studies focusing on signs of

impaired mandibular function sometimes failed to take into account the patient's reported symptoms, and sometimes did not record symptomatology from the past. Thus, intermittent locking with clicking or limited opening without clicking in the past, together with the present finding of normal mandibular kinematics without joint sounds indicates that the patient has a disc displacement without reduction, with functionalization by fibrosing the retrodiscal tissues. Failure to use standardized examination methods led to overestimation of the prevalence of myofascial pain in the orofacial region and arthralgia of the jaw joint. Examination was accompanied by palpation of the temporomandibular joint through the external auditory canal, and too much force was applied during palpation of the joint.

Occasionally, the prevalence of painful disorders has been underestimated due to palpation with too little force or too short a duration, lack of palpation of all key points on the muscle or failure to find trigger points, or due to the absence of a patient's confirmation of a known pain, its spreading, and the modification of the pain by function. These problems have been mitigated by standardization of the criteria by an international consortium of experts focusing on clinical translation of research on orofacial pain and temporomandibular joint dysfunction - now known as the International Network for Orofacial Pain and Related Disorders Methodology (INfORM) (previously known as the International Research - Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) Consortium Network) (1, 2).

Many persons in the geriatric population are completely edentulous, some are partially dentate, and some edentulous persons are not prosthetically rehabilitated; nevertheless, TMD prevalence is very low at that age. A recent systematic review indicates that occlusion or malocclusion is not related to TMD (53).

Only the tooth contact on the balance side during laterotrusion movement is more frequent in TMD subjects, but as studies on this had a cross-sectional design, it cannot be established whether this is a cause of TMD or perhaps only occlusal adaptation to TMD. Since there is no evidence that occlusion plays a role in the onset and development of TMD, it is necessary to abandon the concept of such an association. This does not mean that dentition and occlusion no longer need to be evaluated in TMD

patients. On the contrary, they help in registering the signs that point to TMD, i.e., mandibular dynamics and wear of tooth surfaces.

There is no optimal three-dimensional position of the condyle within the glenoid fossa. Absence of a central position of the condyle is a common characteristic of asymptomatic persons with normal occlusion, as well as asymptomatic persons with malocclusions (54).

Therefore, the condition of the condyle and the disc should be considered as a variation of the normal state. Instead of the mandibular condyle and the glenoid fossa, the condyle and the articular eminence should be considered as two articulating surfaces, since that is where the condyle functions, i.e. condyle is not a ball in a pocket but on a hill (55). Each person has his or her own individual anatomical position of the condyle in relation to the articular eminence. Condyle position has no diagnostic and predictive value. The relationship of the condyle with the fossa and eminence may change due to muscle fatigue, parafunctions, body posture, tongue thrusting and fluid hydration of the articular disc (54).

Flattening of the articulating surfaces should be considered a normal adaptation to an increased load and not as a pathological degenerative change (56). The human body has a great potential for adaptation and functionalization, which is why the mandible can function without an articular disc and also without condyle, as well as without a fossa.

1.3. Orthodontic Treatment and Temporomandibular Disorders

Few high-quality studies have followed changes in signs and symptoms of temporomandibular disorders in persons who were orthodontically treated, compared to untreated persons, using an observational or experimental design. A case-control study that collected and analysed data from 1,818 subjects (185 with chronic TMD pain and 1,633 controls) aged 18-44, indicated that the incidence of chronic painful TMD (arthralgia and myalgia) was greater by 1.4 times in those who were previously orthodontically treated, in comparison to those who were not (57). This has been estimated when the influence of age, sex and race is controlled for, and in the population the ratio ranges between values of 1 and 2 (95% confidence interval). In the interpretation, it should be borne in mind that odds of 1.5 are considered mild or small, while odds of > 3 are considered moderate and odds of > 9 are considered large (58). The aforementioned study showed that an incidence of chronic TMD is highly related to numerous and frequent oral parafunctions (OR = 16.8; 95% CI 8.6-32.9) and moderately to highly related to jaw injury (micro and macro trauma) due to prolonged jaw opening (OR = 8.3; 95% CI 4.5-15.2), frequent yawning (OR = 7.3; 4.2-12.7) and external jaw trauma (OR = 4.2; 95% CI 2.8-6.5) (57).

Previous literature also points out the very poor relationship between orthodontic treatment and symptomatology of TMD. Moreover, even failure to achieve the gnathological concept of ideal occlusion does not necessarily result in the occurrence of TMD (59).

A cohort study of 174 women aged 18-42 years identified an incidence of arthralgia and myalgia of 8.6% in persons previously without TMD, in an average period of three years of follow-up, and found that previous orthodontic treatment did not create a significantly higher risk of TMD (60). After 20 years of follow-up, there was no correlation between the signs of TMD and orthodontic treatment (61). However, the interaction of genes and the environment have been proven. Among subjects with a variant of the gene that encodes the pain response-related enzyme regulating the synaptic level of dopamine (catechol-

O-methyl-transferase), the risk for TMD development was higher in those who had previously been orthodontically treated (60).

Therefore, it could be said that orthodontic treatment does not increase the risk of developing TMD, but it may be a trigger in people who are predisposed to pain. A large cohort study that followed 2,737 people aged 18-44 over a period of about three years, did not detect orthodontic treatment as a likely risk factor for the onset of painful TMD (29).

Systematic reviews and meta-analyses indicate that no type of orthodontic treatment, regardless of the type of appliance, biomechanics or teeth extraction, can prevent the onset of TMD, increase the frequency, cause TMD or exacerbate or cure TMD (62-64). Orthodontics is therefore TMD neutral (65).

A typical dentate patient generally has a well-adapted position of condyles (in a stable musculoskeletal orthopaedic position) that does not need to be analysed. There is no evidence that asymptomatic temporomandibular joint with posteriorly placed condyle creates a risk of disc disorder, and there is no evidence that a centric condylar position means a healthy temporomandibular joint or that a centric position should be achieved to limit the risk in treating TMD patients (54).

There is no scientific evidence that the position should be changed by repositioning the mandible using therapeutic or preventive procedures (55). It makes no sense to manufacture an occlusal splint at the beginning of orthodontic treatment (or before starting orthodontics) to properly position the condyle in the centric relation, because orthodontic treatment lasts two years on average, and all teeth change their position. It cannot be guaranteed that the position of the condyles at the end of the treatment will be in the centric relation.

Since there is no evidence that malocclusion induces the onset of TMD, no interceptive orthodontic treatment can be recommended for the prevention of TMD. Of course, improving the conditions for the development of normal occlusion may be recommended, but not for the prevention of dysfunction of the temporomandibular joint or for orofacial pain.

A Michigan court lawsuit is well known, in which an orthodontist lost the lawsuit against a patient who claimed that orthodontic therapy had caused him TMD (68). If today's information had been available to his attorney, the outcome would have been different.

Not even early orthodontic treatment in mixed dentition, with interceptive orthopaedic appliances in class II and III malocclusion, creates a risk of TMD development (69-71).

Class II malocclusions are very frequent. They are also the most commonly treated malocclusions, but considering their high frequency there is no evidence of higher incidence or prevalence of TMD in these patients (72). Mesial displacement of the condyle during orthodontic treatment of class II malocclusion tends to return to the previous original position after termination of active treatment (73). Although symptomatology improvement is reported, or at least no deterioration occurs during or after class II malocclusion treatment with various appliances and mechanics, the condition is mainly dependent on the initial disc position and its function (74-76). Functional appliances in the treatment of class II malocclusions can have a beneficial effect in patients presenting disc displacement with reduction (with or without intermittent locking) as they can allow the disk to be re-captured (77, 78). This is not the case in patients with a disc displacement without reduction and a limited opening, because in this case they will not allow the disc to be re-captured but could push it even further anteriorly.

Orthognathic surgery may reduce the symptomatology of TMD for most patients who had TMD prior to surgery, but it could also create symptoms for a smaller proportion of the population that was asymptomatic before surgery. Predictors of improvement are not known, but it seems that presence of parafunctional and dysfunctional oral habits before surgery could be predictors of the occurence of symptomatology after surgery (79-83).

Surgical mandibular advancement, mandibular anterior rotation and rigid fixation increase the risk of condylar resorption, but resorption and remodelling are physiological processes, and resorption is not a contraindication for surgery (84, 85). Therefore, there is a somewhat greater risk for patients with vertical growth pattern in class II, due to overload of the temporomandibular joint when the maxillofacial surgeon

rotates condyles is too excessively within the fossa during orthognatic surgery after bilateral mandibular osteotomy, fixing the segments only with bicortical screws without bone plates (86).

It is true that all patients have premature teeth contacts for a large part of orthodontic treatment, induced by moving teeth from the malposition to the correct position or by placing the bite raisers on two teeth to allow for correction of crossbite or scissor bite, reverse overjet or deep bite. However, following these occlusal interferences, no high incidence of temporomandibular joint dysfunction in orthodontic patients has been observed. Furthermore, no more frequent lockinig, reduced opening or protrusion, midline deviation during opening, asymmetry in lateral movement or subluxation has been detected. Indeed, during orthodontic treatment patients sometimes report the onset of clicking, but if it is pain-free and without functional limitations then it is not considered a pathological condition. It cannot be argued that the click in a particular patient would not have appeared even if he had not start orthodontic treatment. It could have been a natural course in a particular case, which coincided by chance with the orthodontic treatment. In addition, if myoarthropathy develops during orthodontic treatment, this does not necessarily have a cause-consequence relationship. As far as the incidence of orofacial pain during orthodontic treatment is concerned, it is actually increased, but it is of odontogenic, rather than non-odontogenic origin. Pain occurs after the application of force and pain a day after is reported by over 90% of people (87). Pain modulation is achieved with nonsteroidal anti-inflammatory drugs and by masticating chewing gum (88). A low-energy laser is also effective to a certain extent in reducing this type of pain (89, 90).

The previously mentioned presence of variation in the enzyme for the regulation of pain, catechol-O-methyl transferase, may also be responsible for some people feeling greater discomfort and pain during orthodontic treatment (91, 92).

For screening for TMD, orthodontic patients are advised to use a very simple, short, self-administered questionnaire before the orthodontic examination (93). The TMD-Pain Screener includes six questions focusing on the pain of orofacial region and the function of the temporomandibular joint. By filling out the questionnaire, the patient becomes aware of activities that might not otherwise have been reported to the

orthodontist because the patient believed they were not important for orthodontic treatment. The patient reports whether he has experienced pain in the temporal area or the jaw, unilaterally or bilaterally, in the last 30 days, and if present, for how long it lasted (appearing occasionally or constantly present). Additionally, the patient reports pain or stiffness in the jaw on waking, and whether some jaw activities change the pain, either diminishing it or worsening it. Activities that are evaluated include chewing hard or tough foods, opening the mouth, protrusion and lateral movements, parafunctional habits such as holding the teeth together, clenching, grinding or chewing gum and daily activities such as talking, yawning or kissing. Summing the responses (the first question receives 0-2 points (a (no pain) =0, b (appearing occasionally) =1, c (constantly present) = 2), while the remaining questions are scored simply as a (no) = 0, b (yes) = 1) produces a value in the range of 0-7 where a cut-off value of ≥ 3 indicates that TMD may be present (93). There is even a shorter version which includes only first three questions (experience of orofacial pain, stiffness/pain in jaw on waking and changes in pain due to chewing hard/tough food). Values exceeding a cut-off of ≥ 2 indicate the presence of TMD.

From this questionnaire, it is apparent that the presence of joint clicking without pain or restriction of function is not considered a serious condition. It is important to screen in order to register conditions that the patient considers as unimportant or not very pronounced, but to which he may start to pay attention only during orthodontic treatment and then start to relate them to the orthodontic procedures. It is necessary to establish a proper diagnosis of the type of TMD. Pain and dysfunction should be eliminated prior to orthodontic treatment, and the patient should be advised regarding the fluctuation of the symptoms and the possibility of their re-emergence during orthodontic treatment (94). Management of such patients may include counselling, cognitive-behavioural therapy, physiotherapy, home massages, pharmacotherapy, and sometimes occlusal splint (95; Figure 2).

There is no doubt that some form of TMD is present in some patients during orthodontic treatment. In this case the active orthodontic mechanics should be temporarily stopped to avoid exacerbating factors, and appliances should be left in passive form. Activating orthodontic appliances applies forces to teeth that

can cause transient discomfort or pain. Fixed and retention appliances and mini-implants are left in the mouth while the use of functional and removable appliances and intermaxillary elastics is temporarily suspended (95; Figure 2). The patient is approached and managed as any other person with TMD. It is diagnosed whether there is a pain disorder and/or joint disorder, and which subtype, and factors that may be related to the occurrence (trauma, stressful events, parafunctions, etc.) are investigated.
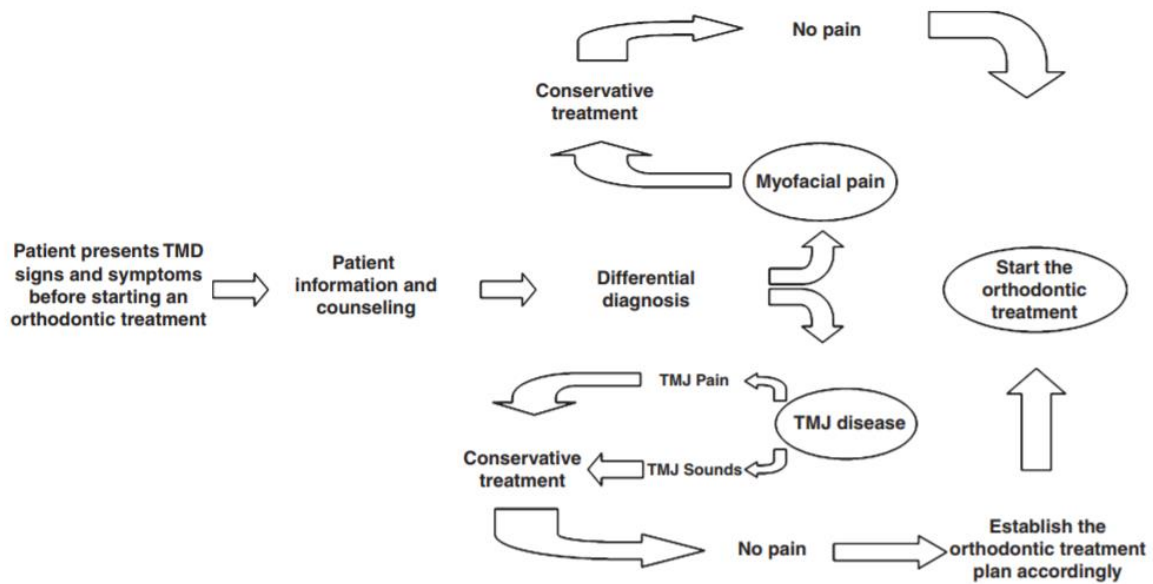
Figure 2. Algorithm for patients presenting TMD before starting an orthodontic treatment (95)
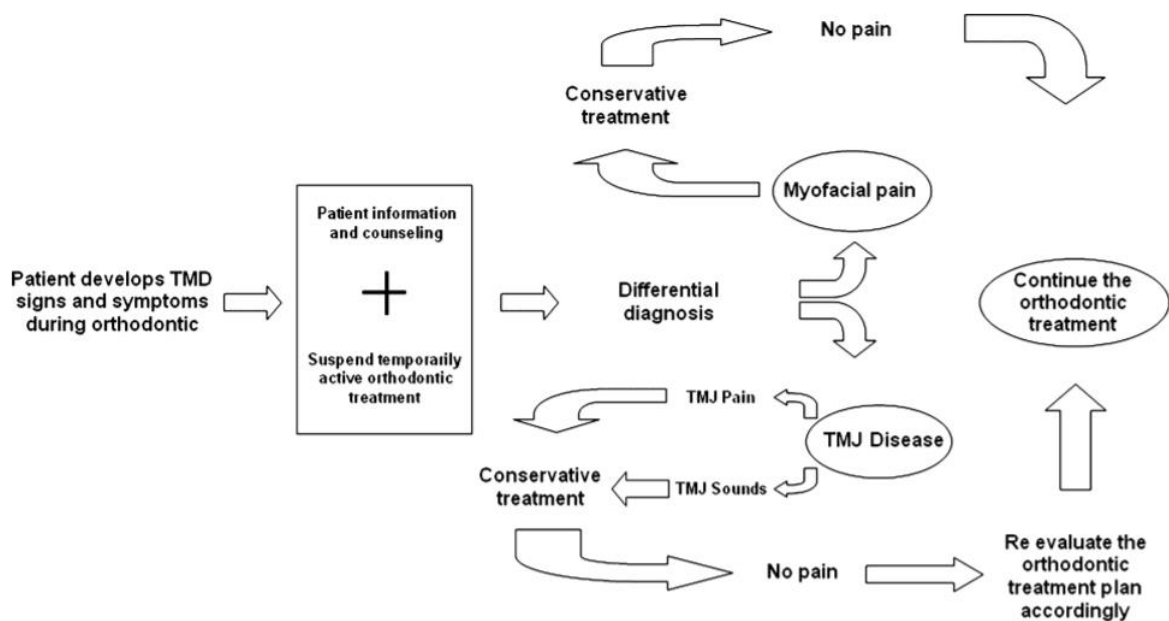
Figure 3. Algorithm for patients developing TMD during orthodontic treatment (94)

It is advisable for the patient to first fill in the DC TMD Axis II instruments, which consist of several structured questionnaires that will alert the patient to his or her own condition and allow him or her to think about related events and report them during clinical examination and orthodontic interview.

The DC TMD Symptom Questionnaire focuses on five key clinical entities: orofacial pain, mouth opening problems, inability to close the mouth, joint noises and headache (2). A patient reports the signs and symptoms he or she has noticed and the activities that modify the condition. After completion, the Symptoms Questionnaire items are checked in front of the patient (and in conversation with him or her) because there is a chance that some of the items were not fully understood by the patient. Any illogicality is checked by direct questions to the patient and clarification. Questions 1, 3 and 4 are, with a very small modifications, contained in the TMD-Pain Screener. Therefore, by summing the answers to these three questions, we get a very similar value to the TMD-Pain Screener score, and thus we are able to determine whether TMD is present. The analysis of the five self-reported components allows us already to have some guidance when confirming signs and symptoms during clinical examination and making the diagnosis, i.e., it allows us to assess whether the patient has a painful condition (arthralgia, myalgia (localized myalgia or myofacsical pain), myofascial pain with referral, headache attributed to TMD), disc displacement with or without reduction or mandibular subluxation.

Table 1. Instruments recommended for use

Domain	Suggested instrument	No of items	Screening before treatment	Comprehensive evaluation
Symptoms	DC TMD Symptom Questionnaire	20	+	+
Pain intensity and pain-related disability	Graded Chronic Pain Scale (GCPS)	8	+	+
Pain locations	Pain drawing	1	+	+
Functional limitations	Jaw Functional Limitation Scale	8 or 20	+ 8 item	+ 20 item
Parafunctions	Oral Behaviours Checklist (OBC)	21	+	+
Distress	Pain Health Questionnaire (PHQ-4)	4	+	
Depression	Pain Health Questionnaire (PHQ-9)	9		+
Somatisation	Pain Health Questionnaire (PHQ-15)	15		+
Anxiety	Generalized Anxiety Disorder (GAD-7)	7		+

The Pain Drawing instrument helps the patient to indicate all locations of the pain by using the drawing of the mouth, head, face and body, and also to indicate the directions of spreading, if any (2). In addition to the drawings of the pain, the patient should be asked questions about the initial symptoms (whether it started after a trauma, a traffic accident, a stressful event, in the morning, after masticating or gum chewing, etc.), pain characteristics (dull, sharp, stabbing, aching, tingling, constant, intermittent, etc.), triggers (moving jaw, cold/heat, etc.) and inhibitors (massage, warmth/cold, humidity, moving the jaw, stillness, medication, etc.). More than three marked painful places point to a serious painful condition.

The Graded Chronic Pain Scale Instrument is used to report the duration of pain (acute or chronic), pain intensity and pain-related disability (96). Pain intensity is assessed as the average value of reported

present pain, worst pain, average pain (on a scale fo zero to 10 for each of the three items), while disability is assessed as the average of scores of daily, work and social activities (also on a scale of zero to 10 for each of the three items). As mentioned above, the pain is characterized as chronic if it is present or repeated over a period of more than three months (almost every day or several times a week), if it lasts longer than one month after healing of acute tissue injury or if it is related to damage that cannot heal (97). The usual criterion for acute painful TMD is at least five days of pain in the last 30 days. Acute pain is a normal sensation indicating a possible injury. Sometimes, patients can manage their chronic pain quite well, but often the pain limits their everyday activities and causes disability. A subject is categorized into one of five possible groups on an ordinal scale: (0) no chronic pain, (1) low-intensity pain without disability, (2) high-intensity pain without disability, (3) moderately limiting pain or (4) with severely limiting pain.

The Jaw Functional Limitation Scale is used for reporting in situations where there are restrictions, whether in mastication, mandibular mobility or communication (98). Even the low limitations regarding verbal and non-verbal communication point to a serious painful state. The instrument is available in longer and shorter form, with 20 and eight items respectively.

The Oral Behaviours Checklist instrument contains two night-time and 19 daytime parafunctions, whose frequency is estimated on a scale of 0 = never to 4 = constant or four to seven nights per week (99). Patients should be consulted about the parafunctions that are present more than once weekly. As a risk factor for TMD, a score of ≥ 25 is associated with a 17-times-higher probability of TMD onset (57).

Regarding psychological traits, psychological suffering can be evaluated by a short Patient Health Questionnaire (PHQ-4) that contains four questions focusing on distress as a combination of depression and anxiety (100). Alternatively, three separate questionnaires can be used within the DC TMD Axis II to assess the level of anxiety, somatization and depression (101-103). Additional psychological features that can help evaluate how the patient will cope with a health condition are hypervigilance (attentional – having increased sensitivity to the symptoms), somatosensory amplification (perceptual - perceiving somatic

sensations as intense, noxious and disturbing), catastrophizing (cognitive - assuming things are worse than they are) and health competence (ability to cope with health conditions and health outcomes) (104-107). Their assessment is based on questionnaires that are not a standard part of the DC TMD protocol.

Table 2. Additional instruments recommended for use

Domain	Suggested instrument	No of items	Screening before treatment	Comprehensive evaluation
Catastrophizing	Pain Catastrophizing Scale (PCS)	13	-	+
Hypervigilance	Brief Hypervigilance Scale (BHS)	5	-	+
Somatosensory amplification	Somatosensory Amplification Scale (SAS)	10	-	+
Health Competence	Perceived Health Competence Scale (PHCS)	8	-	+

Pain perception, including orthodontically induced pain, is influenced by anxiety, catastrophizing, and somatosensory amplification (108-110). Hypervigilance requires special attention, since, together with anxiety, it could be a risk factor for TMD when the therapeutic management includes a modification of the occlusion (33, 95). Such a person is placed on high alert, which includes a high rate of perpetual scanning of the environment to search for signs of threat and a reduced ability to switch attention away from the threatening stimulus. Individuals with bodily hypervigilance also present with occlusal hypervigilance and continuously check their occlusion (110). Parafunction may be a coping response to potential threat when coupled with hypervigilance and somatosensory amplification, and patients with high-frequency parafunctional activity could be more disturbed by occlusal interferences (111-113). Even a minimally invasive alteration of the existing occlusal pattern in subjects who are occlusally hypervigilant can lead to increased activity of the masticatory muscles, which in turn may lead to pain and dysfunction. This explains why some patients are disturbed and do not adapt to occlusal interferences present throughout

a long period duration of orthodontic treatment. Hence, TMD symptomatology that develops in occlusally hypervigilant patients is misdiagnosed as being caused by orthodontically changing the occlusion. Occlusal hypervigilance, besides being attentional, involves a perceptual habit of subjective amplification of a variety of painful but also non-painful sensations (95, 114). Thus, if attention is focused on sensations, their amplification increases, and they become autonomous (115; Figure 3).
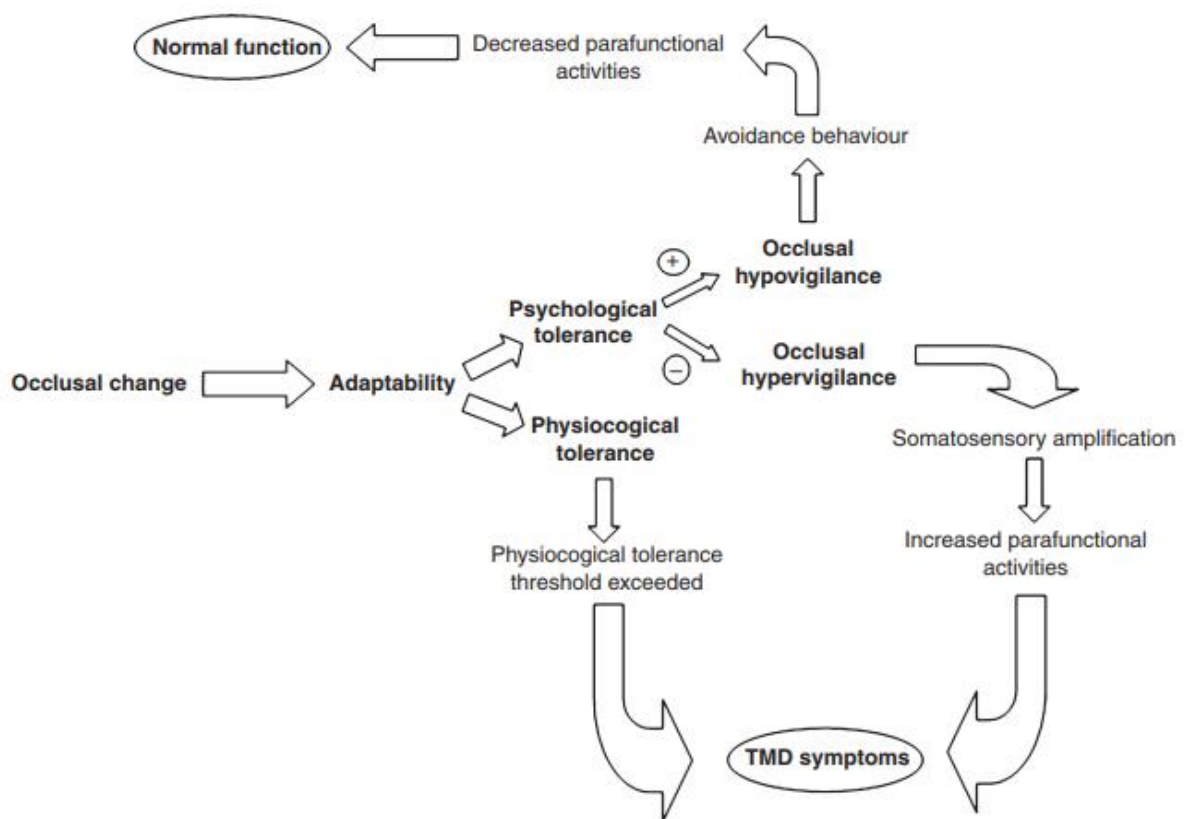
Figure 4. Occlusal hypervigilance theory (95)

Therapeutic options in TMD subjects are focused on reducing pain and improving jaw function, to allow a person to continue with daily activities. The first and most important step after diagnosis is cognitive-behavioural therapy. An international consensus suggests six components of self-management for use in clinical practice: education/counselling about the problem, parafunctional behaviour identification, monitoring and avoidance, jaw exercises, massage, thermal therapy and dietary and nutrition advice that

includes chewing mainly soft food while the painful state persists, with a gradual return to normal food (116). Chronic pain cannot be cured, but it can be managed.

Education includes explaining the aetiology of the disorder, the functioning of the joints and muscles, chronicity and rehabilitation, emphasizing a generally good prognosis. It is important to emphasize the avoidance of overloading the mastication system via the control of parafunctions. Parafunctional behaviours should be avoided, with reinforcement from the clinician for several months. A patient is also advised to keep the muscles relaxed, with the mandible in a neutral position and the teeth not in the occlusion but separated, as if wearing an occlusal splint made of air. This is achieved by pronouncing 'N'. These procedures have a confirmed effectiveness in the management of chronic TMD pain (117).

Sometimes, pharmacological therapy and occlusal splints are also included, when needed (118). Education and physiotherapy seem to be more effective than an occlusal splint for myogenous TMD (119, 120). Even when the use of an occlusal stabilization splint presents a short-term benefit for patients with TMD, the long term effect is equallized by other therapeutic modalities such as physiotherapy, behavioural therapy and counselling (121). Clicking and locking often resolve over time with minimal intervention (122).

Therapy must be conservative and reversible, because occlusal modification can overcome the adaptive ability of the organism and trigger the onset of iatrogenic TMD.

2.0. Aim

The aim of the study was:

1. to translate and validate the TMD-Pain Screener instrument in Croatia,

2 to assess the extent to which orofacial pain and temporomandibular joint dysfunction were present in patients referred for orthodontic consultation,

3. to explore predictors of clinically diagnosed temporomandibular disorder, and of its two components (pain disorder and joint disorder).

We expected the instrument to be valid and reliable in Croatia, with good internal consistency, and that it would have good ability to detect subjects with painful TMD, and temporal stability.

Hypothetical predictors of TMD were type of dentition, malocclusion (crowding, cross bite, forced bite), facial asymmetry, previous orthodontic treatment, age, gender, self-reported parafunctions, chewing problems and self-reported pain and dysfunction.

3.0. Materials and Methods

A Croatian version of the TMD-Pain Screener and DC TMD was produced in forward-backward translation independently by four dentists (two forward and two backward) with experience in temporomandibular disorders and a good knowledge of Croatian and English (2, 93). Translations were reviewed by a panel of five dentists also with a good knowledge of both languages and experience in the fields of validation of questionnaires and temporomandibular disorders. A consensus on the Croatian version was reached. The validation study included 134 participants (student of local university and dental clinic patients) aged 11-62 years (median 23, interquartile range 21-24), 76% females and 82% adults who self-administered the TMD-Pain Screener. Clinical examination and diagnostics were performed according to the DC TMD protocol (2). For the assessment of temporal stability 23 participants completed the questionnaire twice in a two-week interval without any interventions; 14 had painful TMD. The orthodontic sample consisted of 352 consecutive subjects who came for orthodontic consultation at the Department of Orthodontics of the University Dental Clinic Rijeka, Croatia in 2018. The age range was five to 52 years, with a median of 12 years (interquartile range 10-15), with 52% female and 9% adult subjects. Screening for orofacial pain and temporomandibular joint dysfunction was performed using the TMD-Pain Screener instrument evaluating pain, stiffness of the jaw and the modification of pain via jaw activities through six questions. Summing the responses produces a value in the range of 0-7, where a score of ≥ 3 indicates that a person could have a TMD (93). In a shorter 3-items version a score of ≥ 2 indicates TMD (out of range 0-4). Participants completed the questionnaire independently or, for underage participants, with the help of a parent or caregiver. DC TMD was used for clinical examination and diagnostics (2).

The following occlusal characteristics were recorded: type of dentition (deciduous, mixed, permanent), sagittal class by Angle, presence of crowding, crossbite / scissor bite and forced bite. The swallowing pattern (infantile, somatic) and breathing pattern (nasal, oral, combined) was recorded. Parafunctional activities such as nail biting, tonguge-thrusting, clenching and grinding, were noted. The presence of facial

asymmetry was also estimated. Subjects reported whether they had problems during mastication and whether they had been previously orthodontically treated.

Factor analysis and Cronbach alpha were used for assessment of internal consistency of the Croatian version of the TMD-Pain Screener. Discriminant ability was tested by comparing scores between participants with and without TMD using t-test, while temporal stability by intraclass correlation coeficient and Cohen kappa. Sensitivity, specificity, positive and negative predictive values, and likelihood ratio were used to verify the predictive value of the Croatian version of instrument in screening TMD subjects.

The prevalence of orofacial pain and joint dysfunction was estimated with 95% confidence intervals (CI) (123). For comparing the TMD-Pain Screener scores between the occlusal characteristics groups, gender and age, a t-test and an analysis of variance (ANOVA) with the Student-Newman-Keuls post-hoc test, were used. Predictors of TMD were explored by applying Fisher's exact test and logistic regression, and the odds ratios (OR) were calculated with 95% CI. Effect size, as a measure of the difference between groups, was quantified for Fisher's test by means of Cramer's V, for ANOVA via partial η^2 and for the t-test according to formula $r=\sqrt{(t2/(t2+df))}$. For interpretation, the Cohen criteria were used: 0.1-0.3 = small effect size, 0.3-0.5 = medium effect size, 0.5-0.7 = large effect size and >0.7 = very large effect size (40, 41). In the interpretation, OR = 1.5 was considered mild or small, > 3 was considered moderate and > 9 was considered large (58). IBM SPSS 22 statistical software (IBM Corp, Armonk, USA) was used.

4.0. Results

4.1. TMD-Pain Screener

In the validation study, the TMD-Pain Screener score for the 6-item instrument ranged from 0-7 (mean 2.3±2.2) and 46% of subjects reported a score ≥3 indicating that the person could have TMD (95% CI 38-55). In the 3-item instrument, the score ranged 0-4 (mean 1.5±1.3), with 50% of subjects indicative for TMD (scoring ≥2; 95% CI 42-58). Clinical confirmation of TMD was for 73% of subjects, 50% painful, 63% with joint disorder and 40% with painful and joint disorders. Out of TMD subjects, an isolated painful disorder was present in 14%, solitary joint dysfunction in 32% while both joint+painful in 59%. There were significant correlations between items (r=0.308-0.616; p<0.001). Factor analysis demonstrated one-factor structure accounting for 55% of variance. Internal consistency was higher for the 6-item than for the 3-item instrument (Chronbach α 0.831 vs. 0.712). None of the items would increase the alpha coefficient if deleted from the scale. Discriminant validity was better for orofacial pain than joint dysfunction and in the 3-item than in the 6-item instrument (Table 3). For the 6-item instrument sensitivity was 74.6%, specificity 82.1%, positive predictive value 80.7% and negative predictive value 76.4%, while for the 3-item instrument all values were 83.6%. Likelihood ratio was 4.2 indicating that someone with a positive test is 4.2 times more likely to have the disease than someone with a negative test.

In test-retest no significant differences were present between the first and second administration of the instrument. Intraclass correlation coefficients were 0.706 (95% CI 0,424-0.864; p<0.001) and 0.632 (95% CI 0.302-0.826; p=0.001) for score of the six- and three-items instrument, while for dichotomous outcome Cohen Kappa was the same for both forms (0.635; p=0.002).

Table 3. Comparison of TMD-Pain Screener scores between TMD groups

	variable		N	mean±SD	p	r
6-item	clinically diagnosed TMD	no	36	0.6±1.2		
		yes	98	3.0±2.2	<0.001	0.609
	clinically diagnosed	no	67	0.8±1.4		
	orofacial pain	yes	67	3.8±1.9	<0.001	0.693
	clinically diagnosed	no	50	1.1±1.6		
	TMJ dysunction	yes	84	3.1±2.2	<0.001	0.465
3-item	clinically diagnosed TMD	no	36	0.4±0.8		
		yes	98	1.9±1.2	<0.001	0.644
	clinically diagnosed	no	67	0.6±0.9		
	orofacial pain	yes	67	2.5±0.9	<0.001	0.725
	clinically diagnosed	no	50	0.8±1.1		
	TMJ dysunction	yes	84	2.0±1.2	<0.001	0.412

r=effect size for t-test.

The TMD-Pain Screener score ranged from 0-6 (mean 0.6±1.3) and 10% of subjects reported scores ≥ 3, indicating TMD (95% CI 7-14). Orofacial pain was reported by 24% of participants (95% CI 20-29), but only 1% had constant pain (95% CI 0.3-3). Pain modified by function was reported by 21% of participants (95% CI 17-25), and pain modification occurred mainly in one of four functions (interquartile range 1-2). Stiffness was present in 3% of cases (95% CI 1-5). TMD was clinically confirmed in 10% of subjects (95% CI 7-14), and of these, pain disorder was clinically confirmed in 4% (95% CI 3-7) and joint disorder in 7% (95% CI 5-10). Myalgia / myofascial pain was confirmed in 3% of cases (95% CI 2-5), arthralgia in 2% (95% CI 1-4), headache attributed to TMD in 1% (95% CI 0.2-2), disc displacement without reduction and

without intermittent locking in 5% (95% CI 3-7), disc displacement with reduction and intermittent locking in 1% (95% CI 1-3) and subluxation in 1% (95% CI 1-3).

Of the examinees, 2% had deciduous dentition, 47% had mixed dentition and 51% had permanent dentition. Previous orthodontic treatment was reported in 5% of examinees.

The TMD-Pain Screener scores that differed significantly between subjects grouped by clinical, behavioural and socio-demographic characteristics are shown in Table 4. Higher scores were observed for permanent dentition, adults, women, those who had previously been in orthodontic treatment, those with nasal breathing, those who reported that they could not chew well, those with parafunctions of biting pencils, lips, cheeks and/or tongues, and those with clinically confirmed temporomandibular disorders, orofacial pain and joint dysfunction, with small to moderate effect sizes (p <0.05). The weakest effect size was for previous orthodontic treatment. The presence of malocclusion (sagittal, transverse or crowding), forced bite and other parafunctions was not related to the TMD-Pain Screener score.

Table 4. Comparison of TMD-Pain Screener scores between groups of participants classified according to sociodemographic, clinical and behavioural characteristics

variable		N	mean±SD	P	r
age	child/adolescent	321	0.5±1.1		
	adult	31	1.7±2.0	0.001	0.277
gender	male	147	0.4±0.9		
	female	205	0.8±1.4	0.001	0.189
previous orthodontic tx	no	332	0.6±1.2		
	yes	19	1.2±1.7	0.031	0.117
dentition type	deciduous or mixed	174	0.3±0.9		
	permanent	178	0.9±1.5	<0.001	0.224
breathing	oral or combined	98	0.4±0.8		
	nasal	250	0.7±1.4	0.005	0.170
mastication	report no problems	310	0.5±1.1		
	report problems	38	1.4±2.0	0.008	0.225
biting objects/tissues	no	331	0.6±1.2		
	yes	21	1.3±1.3	0.007	0.143
clinically diagnosed TMD	no	317	0.5±1.0		
	yes	35	1.8±2.1	0.001	0.315
clinically diagnosed orofacial pain	no	337	0.5±1.1		
	yes	15	2.7±2.4	0.003	0.355
clinically diagnosed TMJ dysunction	no	327	0.5±1.1		
	yes	25	1.8±2.2	0.010	0.253

r=effect size for t-test.

4.2. Clinically Diagnosed TMD (Pain or/and Joint Disorder)

In univariate analyses, TMD predictors were female sex, adult age, permanent dentition, crowding, facial asymmetry, previous orthodontic treatment, reported problems during mastication, and a TMD-Pain Screener score of ≥3. Deviation from sagittal class I, transversal discrepancies, forced bite or parafunctions were not found to be predictors.

The oddsa of TMD are 3.2 times higher in female subjects (95% CI 1.3-7.5; p = 0.006; V = 0.147), 5.6 times higher for adult ages (95% CI 2.4-13.3; p<0.001; V=0.232), 4.5 times higher for permanent dentition (95% CI 1.9-10.5, p <0.001, V = 0.196), 2.3 times higher for in crowding (95% CI 1.1-4.9; p=0.031; V=0.125), 2.7 times higher for facial asymmetry subjects (95% CI 1.3-5.5; p=0.008; V=0.149), 3.6 times higher in those who had been previously orthodontically treated (95% CI 1.2-10.7, p = 0.031, V = 0.130), 4.9 times higher in subjects with chewing problems (95% CI 2.2-11.0; p <0.001; V = 0.220), and 6.7 times higher for the TMD-Pain Screener scores of ≥ 3 (95% CI 3.0-15.1; p <0.001; V = 0.270). All effect sizes were small to moderate. However, in the multiple logistic regression, when all factors were controlled for, the only significant predictors were difficulties with chewing (OR 2.8; 95% CI 1.1-7.4; p = 0.034) and a TMD-Pain Screener score ≥3 (OR 4.7; 95% CI 1, 8-12.4; p = 0.002).

4.3. Clinically Diagnosed Orofacial Pain

In univariate analyses, predictors of painful TMD were female gender, adult age, reported chewing problems, clinically diagnosed joint dysfunction and TMD-Pain Screener score ≥3. No occlusal characteristics or parafunctional behaviours were found to be predictors. The odds for painful TMD were 4.9 times higher in females than in males (95% CI 1.1-22.1; p = 0.030; V = 0.122), 4.2 times higher for adult age (95% CI 1.2-14; p = 0.034; V = 0.133), 8.5 times higher for chewing problems (95% CI 2.9-25.1; p<0.001; V=0.243), 7.9 times higher for clinically diagnosed joint dysfunction (95% CI 2.5-25.4; p=0.002; V=0.215), and 13.1 times higher for a TMD-Pain Screener score ≥3 (95% CI 4.4-39.0; p <0.001; V = 0.306). All effect sizes were small to moderate. However, in the multiple logistic regression, the only

significant predictors were chewing problems (OR 4.7; 95% CI 1.4-16.0; p = 0.013) and a TMD-Pain Screener score ≥3 (OR 6.9; 95% CI 2.0-23.6; p = 0.002).

4.4. Clinically Diagnosed Joint Dysfunction

Predictors of joint dysfunction in univariate analyses were adult age (OR 6.2; 95% CI 2.4-15.9; p = 0.001; V = 0.226), permanent dentition (OR 4.3; 95% CI 1.6-11.7; p=0.003; V=0.163), previous orthodontic treatment (OR 4.0; 95% CI 1.2-13.0; p=0.037; V=0.130), facial asymmetry (OR 3.4; 95% CI 1.5-7.8, p = 0.004, V = 0.164), reported chewing problems (OR 3.7; 95% CI 1.4-9.5; p = 0.012, V = 0.152), grinding (OR 4.8; 95% CI 1.2-19.1; p = 0.045, V = 0.131), clinically diagnosed painful TMD (OR 7.9; 95% CI 2.5-25.4; p = 0.002; V = 0.215) and TMD-Pain Screener score ≥3 (OR 5.2; 95% CI 2.1-13.1; p = 0.001; V = 0.204). No occlusal characteristics (except permanent dentition) were found to be predictors. However, in the multiple logistic regression all predictors became insignificant.

5.0. Discussion

The present study demonstrates that TMD is not a frequent problem in subjects referred for orthodontic consultation, and malocclusions and previous orthodontic treatment are not predictors of TMD.

Orofacial pain was reported by 24% of subjects, mainly modified by function, but a minority had constant pain or stiffness on waking. In fact only 10% of subjects reported a score ≥ 3 indicating TMD -pain, which is less then in general population of children nine to 11 years in Italy (15%), using the same instrument (72). Self-reported painful TMD in children and young adolescents in the general population ranges from 5-32%, although different screening methods are used (124-129).

TMD was clinically confirmed in 10% of subjects referred for orthodontic consultation, with more joint disorders than pain disorders, while the prevalence of TMD -symptoms using a broad range of methodologies was up to 80% in children and adolescents from different populations (130-136). TMD appears to reach its peak in young adults between 20 and 40 years of age (5). TMD, especially painful TMD, can impact on the individual's psychosocial functioning, daily activities and overall quality of life (137-139).

Occlusal characteristics and malocclusions were not related to self-reported TMD pain in our study, but several variables were identified in the general population of children in Italy in recent study, namely unilateral and bilateral crossbite and open bite, producing odds ratios of 2.3-4.5 (72). Nevertheless, these are the results of univariate analyses where other variables were not simultaneously controlled for. Another recent study implied that unstable occlusion, especially the amount of lateral deviation in RCP-ICP slide, as well as negative overjet, were related to painful TMD (140). Nevertheless, the cross-sectional design does not imply a causal relationship, and there is no evidence that dental occlusion plays a role in the pathophysiology of TMD (53).

Oral parafunctions are frequent in children and adolescents; gum chewing is the most prevalent oral parafunction followed by biting tissues, nails and objects while holding teeth in contact, grinding, clenching

and jaw play are not as prevalent (141). Persistence in these activities might have detrimental effects on the orofacial structures, disrupting the functional balance within the orofacial system, which can induce TMD or worsen TMD which is already present TMD (29). Our study and also others confirmed parafunctions as factors related to TMD -pain (72, 126, 141). Clenching is related to myofascial pain, while jaw play with disc displacement with reduction was also implicated, but with low odds ratios (141). Not all oral behaviours contribute equally to TMD, and among waking activities, several seem to be more influential: grinding, clenching, pressing, touching or holding teeth together during waking hours, biting, chewing or playing with tongue, cheeks or lips, holding objects between the teeth or biting objects such as hair, pipes, pencils, pens and fingers, together with gum chewing (99, 142, 143). Frequency appears to be better predictor than the number of parafunctions, with a high frequency having 2.3-times-higher odds for TMD pain than a low frequency (72). Avoidance of parafunctional behaviour is effective in management of TMD pain (117). However, parafunctions were not predictors of clinically confirmed TMJ dysfunction or pain in our research; they were only related to self-reported pain. Probably children, who composed the majority of our sample, are not able to accurately express the presence / absence and characteristics of their orofacial pain.

Female gender is a known factor related to TMD -pain, in children, adolescents and adults, as outlined in present and previous research (3, 6, 126, 141, 144-147). Again, in multiple logistic regression, female gender did not predict clinically diagnosed TMD, dysfunction or pain, which could be explained by the small number of participants with those conditions in the present study.

The weakest effect size was that of previous orthodontic treatment on self-reported TMD -pain, but not on clinically diagnosed painful disorders. This was related to TMJ dysfunction only in univariate, not in multivariate analyses. The association between orthodontic treatment and TMD appears to be small or non-existent (57, 59, 65).

Excellent internal reliability is reported for the short and long versions TMD-Pain Screener instrument, with high sensitivity for correct classification of the presence or absence of TMD and high specificity in

the correct identification of people with unpainful TMD (93). However, the TMD-Pain Screener seems to lack diagnostic accuracy for differentiating pain of non-odontogenic origin from odontogenic pain without adjunctive clinical examination. It has low specificity (148). Nevertheless, its sensitivity is acceptable, i.e., it is able to identify subjects who have a painful condition. Its high negative predictive value implies that when the screening is negative, one can be reasonably sure that TMD is not present. Overall, it is a useful screening instrument when odontogenic aetiology for pain can be excluded on clinical and radiographic grounds. The Croatian version of the instrument met the sensitivity of ≥ 0.70 necessary to be declared valid, but the specificity wis lower than suggested ≥ 0.95 (149). Therefore, 75% of those with painful TMD will be correctly indentified as positive by the Croatian six-item instrument, and 84% with four-item instrument, which further implies that 16-25% of cases with painful TMD will not be correctly classified. Accordingly, considering specificity, 82% without a condition will be correctly indentified in longer form and 84% in shorter form, and 16-18% of negative cases will be false positive. A test with high sensitivity is useful for ruling out disease, attempting to avoid false negative findings, which makes it appropriate for screening. Tests with high specificity are better in detecting disease and are appropriate when a decision has to be made concerning therapy. Clinical assessment by using DC TMD protocol is able to reach target sensitivity and specificity for painful TMD conditions, but not for majority of joint disfunctions (150). Temporal stability of the instrument is moderate or substantial (151).

The TMD-Pain Screener has been used not only in adults, but also in adolescents and children, although it has not yet been validated in children (72, 110, 152-154). It was observed during investigation that younger children do not fully understand questions from the TMD-Pain Screener, and some of them tend to answer no to the first two questions (presence of pain in jaw or temple area and stiffness of jaw on waking) but yes to activities that changed the pain, namely, clenching, chewing gum or chewing hard food. Furthermore, sometimes, parents needed explanation. Nevertheless, the present study found that the TMD-Pain Screener score was a strong predictor of clinically confirmed orofacial pain, although not of joint dysfunction.

This study has several limitations. First of all, the sample size is low and the age range quite broad. In addition, the majority of participants were children or young adolescents. Since there was a low prevalence of self-reported TMD -pain and clinically confirmed painful conditions and TMJ dysfunctions, the majority of significant predictors from univariate analyses became insignifican in multiple regression. No distinction was made between waking and sleeping oral parafunctions and the frequency of parafunctions was not recorded. Due to cross-sectional design, cause-effect relationships could not be established.

6.0. Conclusion

The Croatian version of the TMD-Pain Screener has good ability to detect subjects with painful TMD. Orofacial pain and temporomandibular joint dysfunction are not frequent in people referred to orthodontists. Malocclusions and previous orthodontic treatment are not predictors of TMD. The instrument is a strong predictor of clinically confirmed orofacial pain in subjects reffered for orthodontic consultation, with a high score indicating 6.9-times-higher odds, but it is not a significant predictor of joint dysfunction.

7.0. Reference

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Nationality	Croat
Date of birth	4TH FEBRUARY 1974

WORK EXPERIENCE

• Dates (from – to)	**SINCE AUGUST 1ST 2009**
• Name and address of employer	University of Rijeka, Faculty of Medicine & Dental Clinic at Clical Hospital Centre Rijeka
• Type of business or sector	Science, teaching, health sector
• Occupation or position held	Associate Professor, Orthodontist, Head of Department of Orthodontics
• Main activities and responsibilities	Teaching Orthodontics and Public Health Dentistry, providing orthodontic treatment
• Dates (from – to)	**2002-2009**
• Name and address of employer	Public Health Center Vukovar
• Type of business or sector	Health sector
• Occupation or position held	General dentist, orthodontic resident, orthodontist
• Main activities and responsibilities	Team head, Dental unit head
• Dates (from – to)	**1998-2002**
• Name and address of employer	Public Health Center Senj, Dental Office Kuzmic Crikvenicar
• Type of business or sector	Health sector
• Occupation or position held	General dentist
• Main activities and responsibilities	Intern, Team head

EDUCATION AND TRAINING

• Dates (from – to)	2004-2008
• Name and type of organization providing education and training	Clinical Hospital Centre Zagreb - Dental Clinic
• Principal subjects/occupational skills covered	Orthodontics, dentofacial orthopedics, facial growth and development
• Title of qualification awarded	Specialist in orthodontics
• Level in national classification	Spec.

(if appropriate)	
• Dates (from – to)	2005
• Name and type of organization providing education and training	School of Dental Medicine University of Zagreb
• Principal subjects/occupational skills covered	Public Health Dentistry and Oral Epidemiology
• Title of qualification awarded	PhD
• Level in national classification (if appropriate)	Dr. sc.
• Dates (from – to)	1998-2000
• Name and type of organization providing education and training	School of Dental Medicine University of Zagreb
• Principal subjects/occupational skills covered	Periodontology and Oral Epidemiology
• Title of qualification awarded	Master of science
• Level in national classification (if appropriate)	Mr. sc.
• Dates (from – to)	1992-1998
• Name and type of organization providing education and training	School of Dental Medicine University of Zagreb
• Principal subjects/occupational skills covered	Dental Medicine
• Title of qualification awarded	Doctor of Dental Medicine
• Level in national classification (if appropriate)	Dr. med. dent.
• Dates (from – to)	1998
• Name and type of organization providing education and training	Faculty of Political Sciences University of Zagreb
• Principal subjects/occupational skills covered	Journalism, Communications, Political Sciences
• Title of qualification awarded	Master of journalism
• Level in national classification (if appropriate)	Mag. nov.
• Dates (from – to)	2006, 2007, 2009, 2010
• Name and type of organization providing education and training	University Computational Centre Zagreb, E-academy University of Rijeka
• Principal subjects/occupational skills covered	Statistical methods – descriptive, graphical, univariate analysis of variance and correlations, multivariate analyses
• Title of qualification awarded	
• Level in national classification (if appropriate)	

MOTHER TONGUE

CROATIAN

OTHER LANGUAGES

ENGLISH, ITALIAN, GERMAN

	English	Italian	German
• Reading skills	C1	B2	A2
• Writing skills	B2	B1	A2
• Verbal skills	B2	B1	A2

COMUNICATION SKILS acquired during studying journalism, and working as a journalist in daily newspapers Novi list, Vecernji list and Jutarnji list during the period of 2001-4. Assistant editor in Croatian Journal of Dental Medicine 2010-2017

Coordination and administration of people, projects and budgets as a head of Dental Unit of Public Health Centre Vukovar in 2008/9 and head of Department of Orthodontics at University of Rijeka, Faculty of Medicine since 2014

Principle investigator at three scientific projects:
- Immunological and regenerative implications of corrosion of dental materials in children and adolescents (IP-2014-09-7500; duration 2015-2019) – funding Croatian Science Foundation
- Determinants of effectiveness of treatment of altered orofacial functions and appearance (uniri-biomed-18-22; since 2019) – funding University of Rijeka
- Predictors of success of orthodontic treatment in children and adolescents (13.06.2.1.53; duration 2013-2018) – funding University of Rijeka

Actively participated in three scientific projects at University of Rijeka and Zagreb:
- Bioactive dental materials – modulation of the active matrix for improvement of the clinical efficiency and reduction of the DNA damage (18.07.2.2.03.; duration 2018-2019; principal investigator Visnja Katic) - funding University of Rijeka
- New diagnostic methods in orthodontics and biocompatibility of appliances (065-0650444-0436; duration 2007-2012, principal investigator Professor Mladen Slaj) - funding Ministry of Science of the Republic of Croatia
- Systemic aspects of periodontal disease (065-0650444-0415; duration 2007-2009, principal investigator Professor Darije Plancak) - funding Ministry of Science of the Republic of Croatia
- Epidemiology of periodontal diseases and caries in Croatia (065102; duration 2002-2006, principal investigator Professor Darije Plancak) - funding Ministry of Science of the Republic of Croatia
- Reorgansation and modernization of specialist network in City of Zagreb based on evaluation of orthodontic treatment need of schoolchildren, and epidemiological survey, and quality assessment (duration 2005-2008, principal investigator Professor Mladen Slaj) - funding Zagreb Municipality

Course teacher Oral epidemiology at Master and PhD program at School of Dental Medicine University of Zagreb since 2007

	Course head Public Health Dentistry, Principles of Dentistry, Orthodontics, Antropometry and cephalometry, and Dental photography for graduate program at School of Medicine Universtiy of Rijeka since 2008.
	Course head Assessment of orthodontic treatment needs at PhD program at School of Dental Medicine University of Zagreb since 2009
TECHNICAL SKILLS AND COMPETENCIES *With computers, specific kinds of equipment, machinery, etc.*	Work on computers – Word, Excel, Acces, Power Point, SPSS, Statistica – skils gained at Faculty of Political Sciences, University Computational Centre and e-academy at University of Rijeka
ARTISTIC SKILLS AND COMPETENCIES *Music, writing, design, etc.*	
OTHER SKILLS AND COMPETENCIES *Competences not mentioned above.*	
DRIVING LICENCE(S)	B category

ADDITIONAL INFORMATION

MARRIED TO VEDRANA TUDOR, MD, 2 CHILDREN – PROSPER, BORN 2007 AND MATIJA, 2009.

PUBLICATIONS: CO-AUTHOR OF 78 PUBLICATIONS - 49 PAPERS IN JOURNALS INDEXED IN CC, 10 IN SCIE

MENTORING: 5 PHD THESIS AND 8 GRADUATE THESIS

REVIEWER IN JOURNALS: EUR J ORTHOD, ANGLE ORTHOD, ORTHOD CRAIOFAC RES, QUAL LIFE RES, HEAD FACE MED, J PUBLIC HEALTH DENT, PLOS ONE, INT J DENT HYG, J APPL ORAL SCI, BIOMED RES INT, MED SCI MONIT, BMC MUSCULOSKELET DISORD, ANN MED HEALTH SCI RES, J AFFECT DISORD, ACTA MED ACAD, SAUDI DENT J, AUSTRALAS MED J, COLL ANTROPOL, ADV MED SCI, INT J ADOLESC MED HEALTH, ACTA STOMATOLO CROAT, SOUTH EUR J ORTHOD DETOFAC RES, JMED RES, INT RES J PURE APPL CHEM, STOM EDU J, J DENT APPLIC, J INT MED RES

RESEARCH INTERESTS: PERCEPTION OF DENTOFACIAL ESTHETICS, ORTHODONTIC TREATMENT AND QUALITY OF LIFE, ORAL CORROSION AND BIOCOMPATIBILITY, ORAL EPIDEMIOLOGY AND PUBLIC HEALTH

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ANNEXES   Bibliography: (Articles in journals indexed in CC, SCIE and SSCI)

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3. **Spalj S**, Plancak D, Jurić H, Pavelić B, Bosnjak A. Reasons for extraction of permanent teeth in urban and rural populations of Croatia. Coll Antropol. 2004;28:833-9. **(**Q3; CC; **IF 0.373**)

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5. **Spalj S**, Mestrovic S, Lapter Varga M, Slaj M. Skeletal components of class III malocclusions and compensation mechanisms. J Oral Rehabil. 2008;35:629-37. **(**Q2; CC; **IF 1.356**)

6. **Spalj S**, Plancak D, Bozic D, Kasaj A, Willershausen B, Jelusic D. Periodontal conditions and oral hygiene in rural population of post-war Vukovar region, Croatia in correlation to stress. Eur J Med Res. 2008;13:100-6. **IF 1.040**

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13. Varga S, **Spalj S**, Lapter Varga M, Anic Milosevic S, Mestrovic S, Slaj M. Maximum voluntary molar bite force in subjects with normal occlusion. Eur J Orthod. 2011;33:427-33. (Q1; CC; **IF 0.893**)

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51. Vuletic L, **Spalj S**, Peros K, Jakovac H, Ostroski Anic A, Vodanovic M. Assessing the influence of the English language on professional vocabulary of Croatian dental students by analyzing their word choice for translation of medical/dental terms. Terminology. 2017:23:2. (Q2; CC; **IF 0.619**)

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ANEXES

Anex 1. Croatian version of TMD-Pain Screener

TMP-PROBIR BOLI

1. U posljednjih 30 dana, koliko dugo je trajala bilo kakva bol u području čeljusti ili sljepoočnica na jednoj ili obje strane?

a.– Bez boli

b.– Bol dolazi i prolazi

c.– Bol je stalno prisutna

2. U posljednjih 30 dana, jeste li imali bolnu ili zakočenu čeljust nakon buđenja?

a. - Ne

b. - Da

3. U posljednjih 30 dana, jesu li sljedeće aktivnosti promijenile bol (odnosno, smanjile ju ili pogoršale) u području čeljusti ili sljepoočnica na jednoj ili obje strane?

A.– Žvakanje tvrde ili žilave hrane

a. - Ne

b. - Da

B.– Otvaranje usta ili pomicanje čeljusti naprijed ili u stranu

a - Ne

b - Da

C. - Čeljusne navike, kao npr. držanje zubi spojenima, stiskanje, škrgutanje ili žvakanje žvakače

a. - Ne

b - Da

D.– Ostale aktivnosti čeljusti, kao npr. govorenje, ljubljenje ili zijevanje

a. - Ne

b. – Da

UKUPNI ZBROJ =_____

DK- Upitnik o simptomima

BOL

	ne	da

1. Jeste li ikada imali bol u čeljusti, sljepoočnici, uhu ili ispred uha na jednoj ili obje strane?
Ako ste odgovorili NE, preskočite na pitanje 5

2. Prije koliko godina ili mjeseci su Vam počeli bolovi u čeljusti, sljepoočnici, uhu ili ispred uha?   ____godina_____mjeseci

3. U posljednjih 30 dana, što od navedenog najbolje opisuje bol u Vašoj čeljusti, sljepoočnici, uhu ili ispred uha na jednoj ili obje strane?
Izaberite JEDAN odgovor.
Ako ste odgovorili BEZ BOLA na pitanje 3, preskočite na pitanje 5

☐ **bez bola**

☐ **bol dođe i prođe**

☐ **bol uvijek prisutna**

4. U posljednjih 30 dana, je li koja od sljedećih aktivnosti promijenila bilo koju bolnost (pogoršala ili poboljšala) čeljusti, sljepoočnice, uha, ispred uha na jednoj ili obje strane?

	ne	da
A. Žvakanje tvrde ili žilave hrane		
B. Otvaranje usta, pomicanje čeljusti naprijed ili u stranu		
C. Navike poput držanja zubi spojenima, stiskanja/škripanja zubima ili žvakanja žvakaće gume		
D. Druge aktivnosti čeljusti kao pričanje, ljubljenje ili zijevanje		

GLAVOBOLJA

	ne	da

5. U posljednjih 30 dana jeste li imali glavobolje koje su uključivale područja sljepoočnica?
Ako ste odgovorili NE na pitanje 5, preskočite na pitanje 8

6. Prije koliko godina ili mjeseci su Vam počele glavobolje u području sljepoočnice?   ____godina_____mjeseci

7. U posljednjih 30 dana, je li koja od sljedećih aktivnosti promijenila glabovolju (pogoršala ili poboljšala) u području sljepoočnice na jednoj ili obje strane?

	ne	da
A. Žvakanje tvrde ili žilave hrane		
B. Otvaranje usta, pomicanje čeljusti naprijed ili u stranu		
C. Navike poput držanja zubi spojenima, stiskanja/škripanja zubima ili žvakanja žvakaće gume		
D. Druge aktivnosti čeljusti kao pričanje, ljubljenje ili zijevanje		

ZVUKOVI U ZGLOBU

	ne	da	D	L	ne zna
8. U posljednjih 30 dana jeste li imali zvukove u zglobu pri pomaku ili korištenju čeljusti?					

KOČENJE ČELJUSTI PRI OTVARANJU

9. Je li Vam <u>ikada</u> čeljust zapela ili se zakočila, čak i na trenutak, da niste mogli U POTPUNOSTI <u>otvoriti</u> usta
Ako ste odgovorili NE na pitanje 9, preskočite na pitanje 11

10. Je li Vam čeljust zapela ili se zakočila tako ozbiljno da Vam ograniči otvaranje usta i onemogući Vas u jelu?

11. U posljednjih 30 dana je li Vam se čeljusti toliko zakočila da niste mogli otvoriti U POTPUNOSTI, čak i na trenutak, a onda otkočila da ste mogli otvoriti U POTPUNOSTI?
Ako ste odgovorili NE na pitanje 9, preskočite na pitanje 13

12. Je li Vaša čeljust trenutno zakočena ili ograničena tako da se <u>ne može otvoriti</u> DO KRAJA?

KOČENJE ČELJUSTI PRI ZATVARANJU

13. U posljednjih 30 dana, kada bi otvorili jako usta, je li Vam čeljusti zapela ili se zakočila čak i na trenutak tako da <u>niste mogli zatvoriti</u> usta iz tog položaja?
Ako ste odgovorili NE na pitanje 13 onda ste završili s odgovorima na ovoj stranici.

14. U posljednjih 30 dana, kada bi Vam čeljust zapela ili se zakočila pri otvorenim ustima, jeste li morali napraviti nešto da bi zatvorili usta, uključujući odmaranje, micanje, pritiskanje ili neki manevar sa čeljusti?

Anex 3. Croatian version of DC TMP Axis I – clinical examination form

DK TMP obrazac za pregled

Datum ispunjavanja (dd-mm-gggg)

| | | – | | | – | | | | |

pacijent_____liječnik _____

1a. Lokalizacija boli: posljednjih 30 dana (označite sve što je točno)

BOL DESNE STRANE	**BOL LIJEVE STRANE**
O bez boli O temporalis O drugi mišići O nežvačne	O bez boli O temporalis O drugi mišići O nežvačne
O maseter O TMZ strukture	O maseter O TMZ strukture

1b. Lokalizacija glavobolje: posljednjih 30 dana (označite sve što je točno)

O bez glavobolje   O temporalno područje   O ostalo   O bez glavobolje   O temporalno područje   O ostalo

2. Incizalni odnosi   referentni zub   O 11 O 21 O drugo

pregriz (OJ)   O ako je negativan [|] mm   prijeklop (OB) O ako je negativan [|] mm   pomak sredine   D L N/P O O O [|] mm

3. Obrazac otvaranja (označite sve što je točno)   neispravljena devijacija

O po ravnoj liniji   O ispravljena devijacija   O desno   O lijevo

4. Kretnje otvaranja

A. bezbolno otvaranje [|] mm

B. maks. neasistirano otvaranje [|] mm

DESNA STRANA	bol	poznata bol	poznata glavobolja	LIJEVA STRANA	bol	poznata bol	poznata glavobolja
temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maseter	Ⓝ Ⓓ	Ⓝ Ⓓ		maseter	Ⓝ Ⓓ	Ⓝ Ⓓ	
TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ		TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ	
ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ		ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ	
nežvačne strukture	Ⓝ Ⓓ	Ⓝ Ⓓ		nežvačne strukture	Ⓝ Ⓓ	Ⓝ Ⓓ	

C. maks. asistirano otvaranje [|] mm

D. prekinuto? Ⓝ Ⓓ

	bol	poznata bol	poznata glavobolja		bol	poznata bol	poznata glavobolja
temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maseter	Ⓝ Ⓓ	Ⓝ Ⓓ		maseter	Ⓝ Ⓓ	Ⓝ Ⓓ	
TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ		TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ	
ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ		ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ	
nežvačne strukture	Ⓝ Ⓓ	Ⓝ Ⓓ		nežvačne strukture	Ⓝ Ⓓ	Ⓝ Ⓓ	

5. Lateralne krenje i protruzija

A. desna lateralna [|] mm

DESNA STRANA	bol	poznata bol	poznata glavobolja	LIJEVA STRANA	bol	poznata bol	poznata glavobolja
temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maseter	Ⓝ Ⓓ	Ⓝ Ⓓ		maseter	Ⓝ Ⓓ	Ⓝ Ⓓ	
TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ		TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ	
ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ		ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ	
nežvač. strukture	Ⓝ Ⓓ	Ⓝ Ⓓ		nežvač. strukture	Ⓝ Ⓓ	Ⓝ Ⓓ	

B. lijeva lateralna [|] mm

	bol	poznata bol	poznata glavobolja		bol	poznata bol	poznata glavobolja
temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maseter	Ⓝ Ⓓ	Ⓝ Ⓓ		maseter	Ⓝ Ⓓ	Ⓝ Ⓓ	
TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ		TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ	
ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ		ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ	
nežvač. strukture	Ⓝ Ⓓ	Ⓝ Ⓓ		nežvač. strukture	Ⓝ Ⓓ	Ⓝ Ⓓ	

C. protruzija [|] mm

O ako je negativno

	bol	poznata bol	poznata glavobolja		bol	poznata bol	poznata glavobolja
temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maseter	Ⓝ Ⓓ	Ⓝ Ⓓ		maseter	Ⓝ Ⓓ	Ⓝ Ⓓ	
TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ		TMZ	Ⓝ Ⓓ	Ⓝ Ⓓ	
ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ		ostali žv. mišići	Ⓝ Ⓓ	Ⓝ Ⓓ	
nežvač. strukture	Ⓝ Ⓓ	Ⓝ Ⓓ		nežvač. strukture	Ⓝ Ⓓ	Ⓝ Ⓓ	

6. Zvukovi TMZ tijekom otvaranja i zatvaranja

DESNI TMZ						LIJEVI TMZ				
	ispitivač		pacijent	bol sa škljocajem	poznata bol		ispitivač		pacijent	bol sa škljocajem	poznata bol
	otvaranje	zatvaranje					otvaranje	zatvaranje			

	ispitivač otvaranje	ispitivač zatvaranje	pacijent	bol sa škljocajem	poznata bol		ispitivač otvaranje	ispitivač zatvaranje	pacijent	bol sa škljocajem	poznata bol
škljocaj	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ [Ⓓ] →	Ⓝ Ⓓ	Ⓝ Ⓓ	škljocaj	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ [Ⓓ] →	Ⓝ Ⓓ	Ⓝ Ⓓ
krepitacija	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ			krepitacija	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ		

7. Zvukovi TMZ tijekom protruzije i lateralnih kretnji

DESNI TMZ				LIJEVI TMZ			
	ispitivač	pacijent	bol sa škljocajem	poznata bol		ispitivač	pacijent	bol sa škljocajem	poznata bol

	ispitivač	pacijent	bol sa škljocajem	poznata bol		ispitivač	pacijent	bol sa škljocajem	poznata bol
škljocaj	Ⓝ Ⓓ	Ⓝ [Ⓓ] →	Ⓝ Ⓓ	Ⓝ Ⓓ	škljocaj	Ⓝ Ⓓ	Ⓝ [Ⓓ] →	Ⓝ Ⓓ	Ⓝ Ⓓ
krepitacija	Ⓝ Ⓓ	Ⓝ Ⓓ			krepitacija	Ⓝ Ⓓ	Ⓝ Ⓓ		

8. Zakočenje zgloba

DESNI TMZ		redukcija		LIJEVI TMZ		redukcija	
	zakočenje	pacijent	ispitivač		zakočenje	pacijent	ispitivač
tijekom otvaranja	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	tijekom otvaranja	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maksimalno otvaranje	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	maksimalno otvaranje	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ

9. Bol mišića i zgloba na palpaciju

DESNA STRANA (1 kg)	bol	poznata bol	poznata glavobolja	prenesena bol	LIJEVA STRANA (1 kg)	bol	poznata bol	poznata glavobolja	prenesena bol
temporalis (stražnji)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis (stražnji)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
temporalis (srednji)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis (srednji)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
temporalis (prednji)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	temporalis (prednji)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
maseter (polazište)	Ⓝ Ⓓ	Ⓝ Ⓓ		Ⓝ Ⓓ	maseter (polazište)	Ⓝ Ⓓ	Ⓝ Ⓓ		Ⓝ Ⓓ
maseter (srednji dio)	Ⓝ Ⓓ	Ⓝ Ⓓ		Ⓝ Ⓓ	maseter (srednji dio)	Ⓝ Ⓓ	Ⓝ Ⓓ		Ⓝ Ⓓ
maseter (hvatište)	Ⓝ Ⓓ	Ⓝ Ⓓ		Ⓝ Ⓓ	maseter (hvatište)	Ⓝ Ⓓ	Ⓝ Ⓓ		Ⓝ Ⓓ

TMZ	bol	poznata bol	prenesena bol		bol	poznata bol	prenesena bol
lateralni pol (0,5 kg)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	lateralni pol (0,5 kg)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
oko lateralnog pola (1 kg)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	oko lateralnog pola (1 kg)	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ

10. Mišićna bol na palpaciju u dodatnim područjima

DESNA STRANA (0,5 kg)	bol	poznata bol	prenesena bol	LIJEVA STRANA (0,5 kg)	bol	poznata bol	prenesena bol
stražnja mandibularna regija	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	stražnja mandibularna regija	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
submandibularna regija	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	submandibularna regija	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
područje lateralnog pterigoida	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	područje lateralnog pterigoida	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ
tetiva temporalisa	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ	tetiva temporalisa	Ⓝ Ⓓ	Ⓝ Ⓓ	Ⓝ Ⓓ

11. Dijagnoze

bolni poremećaji	poremećaji desnog zgloba	poremećaji lijevog zgloba
○ nijedna	○ nijedna	○ nijedna
○ mialgija	pomak diska (izaberi jedan)	pomak diska (izaberi jedan)
○ prenesena miofascijalna bol	○ ...s redukcijom	○ ...s redukcijom
	○ ...s redukcijom, s povremenim kočenjem	○ ...s redukcijom, s povremenim kočenjem
○ artralgija desno	○ ... bez redukcije, s ograničenim otvaranjem	○ ... bez redukcije, s ograničenim otvaranjem
○ artralgija lijevo	○ ... bez redukcije, bez ograničenog otvaranja	○ ... bez redukcije, bez ograničenog otvaranja
	○ degenerativna bolest zgloba	○ degenerativna bolest zgloba
○ glavobolja koja se pripisuje TMP-u	○ subluksacija	○ subluksacija

12. Komentari