A literature review on the causal relationship between Occlusal Factors (OF) and Temporomandibular Disorders (TMD) V: effect of changes in occlusal factors achieved with orthodontic treatment

Una revisión de la literatura sobre la relación causal entre los factores oclusales (FO) y los desórdenes temporomandibulares (DTM) V: efecto de los cambios en los factores oclusales conseguidos con el tratamiento de ortodoncia

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ABSTRACT

Introduction*: this is the fifth article in this series, which aims to present a literature review regarding the causal relationship between occlusal factors (OF) and temporomandibular disorders (TMD).* ***Methods:*** *This article presents and analyzes epidemiological reports (cross-sectional and analytical) in which the relationship between orthodontic treatment (OT) and TMD was studied.* ***Results:*** *most of the reports (70%) did not support any association between OT and TMD development.* ***Conclusion:*** *even though the general tendency was the lack of association between OT and TMDs throughout different reports, perhaps in the future, an improvement in the research design will allow obtaining more concrete results that can help us reach a solid interpretation about the relationship between OT and TMDs. Other factors, such as genetics, may play a role as confounding variables and should be studied and considered in the etiology, prevention, or management of TMDs. Apart from the possible relationship between OT and TMDs, an important clinical recommendation is to carry out the appropriate TMD screening in patients who will receive an OT.*

Keywords: *occlusion, temporomandibular disorders, etiology, occlusal factors, temporomandibular joint, orthodontic treatment.*

RESUMEN

Introducción: *este es el quinto de una serie de artículos que tienen como propósito presentar una revisión de la literatura sobre la relación causal entre los factores oclusales (FO) y los desórdenes temporomandibulares (DTM).* ***Métodos:*** *en este artículo se presentaron y analizaron los estudios epidemiológicos (transversales y analíticos) en los que se investigó la posible relación entre el tratamiento de ortodoncia (TO) y los DTM.* ***Resultados:*** *la mayoría de los reportes (70%) no soportaron al TO como un factor asociado al desarrollo de los DTM.* ***Conclusión:*** *a pesar de que la tendencia general fue la de presentarse una ausencia de asociación entre el TO y los DTM en los diferentes reportes; quizás en el futuro, un mejoramiento de los diseños de las investigaciones permita obtener resultados más claros que ayuden a hacer una sólida interpretación acerca de la relación existente entre el TO y los DTM. Adicionalmente, otros factores; como los genéticos, pueden ser variables de confusión y deberían ser investigados y considerados dentro de la prevención, etiología y manejo de los DTM. Mas allá de que exista una asociación entre el TO y los DTM, es importante destacar como recomendación clínica, la ejecución de un apropiado tamizaje de DTM en los pacientes que van a recibir un TO.*

Palabras clave: *oclusión, desórdenes temporomandibulares, etiología, factores oclusales, articulación temporomandibular, tratamiento de ortodoncia.*

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INTRODUCTION

This is the fifth article in a series that aims to review the validity of different existing epidemiological studies about the relationship between TMDs and OFs to weigh the current scientific evidence that supports a possible causal relationship between these two factors. The first two articles analyzed descriptive and analytical epidemiological observational studies.[1,2] The third article presented and analyzed experimental studies in which artificial occlusal interference (OI) was used to evaluate changes in oral physiology and the presence of characteristics associated with TMDs.[3] The fourth article analyzed studies in which TMD signs and symptoms (TMDSS) were evaluated in relation to the use of occlusal adjustment by occlusal carving.[4] Similarly, because orthodontic treatment (OT) can modify or impact dental occlusion (or occlusal factors), it has been considered an etiological, preventive, or therapeutic factor of TMDs. Historically, the possible relationship between OT and TMDs has been a clinical event recognized long ago by the dentistry profession (especially by orthodontists). However, in the first eight decades of the last century, the existing publications about the relationship between OT and TMDs were mostly personal opinions and case reports completely lacking controlled studies.[5] This shows that although TMDSS have been long identified as a clinical problem in orthodontic patients, it has become of particular interest in the last three decades. This interest may have been partially motivated (especially in the United States) by the results of a lawsuit brought against an orthodontist by one of his patients who developed TMDs during OT.[6] The patient received a large sum of money as compensation because it was considered at the time that the OT caused the TMDSS displayed by the patient. This situation would most likely not occur today since the dentistry profession has done more and better research, and the conclusions of different literature reviews do not support OT as an etiologic, preventive, or therapeutic factor of TMDs.[7-10] However, despite many years of research, not every publication or sector of the dentistry profession dismisses the relationship between OT and TMDs. Therefore, this continues to be a controversial point worth further analyzing from different perspectives.[11-15] This fifth article will present and analyze important methodological aspects of the studies in which the effect of changes in occlusal factors achieved with OT and the presence of TMDs have been evaluated.

METHODS

The literature review was performed using different sources of information:

1. The standard medical information Medline database, specifically using the MedlineOVID library (from 1966 to 2008). The abstracts of the articles in English and whose titles suggested the study of the relationship between OT and TMD were reviewed. In order to narrow the search, OT and orthodontics were used as keywords and cross-referenced with the relevant terms under the TMD heading (temporomandibular joint (TMJ), temporomandibular joint disorders).

2. The bibliography of the articles initially found in the MedlineOVID database search.

3. The bibliography of different books on the TMD and occlusion domain.

4. The bibliography of several literature reviews on the subject matter from the MedlineOVID database.

The validity of the studies was evaluated following the parameters recommended by Mohl.[16] These parameters were thoroughly described in previous articles 1 and 2 and are shown in Table 1.

Table 1. Parameters for assessing the validity of scientific studies

1. Definition of the gold standard.
2. Establishment of an acceptable system of diagnostic classification.
3. Use of clearly defined measures.
4. Intra- and inter-examinators acceptable reliability.
5. Use of suitable samples
6. Using matched groups
7. Random assignment of patients and subjects to study groups.
8. Data collection by "blind" examiners.
9. Study replicability
10. Consideration of alternative hypotheses

Source: by the authors

RESULTS

Among the 56 studies found, 22 were descriptive of transversal type in which subjects with a history of OT and without it were compared[17-38] (table 2), 8 case-control studies[39-46] (table 3), [25] longitudinal studies[47-71] and a randomized clinical study[72] (table 4). The results showed a discrepancy between the reports, a large group (30%) associated OT with TMDs, while 39 of these studies (70%) showed no association between OT and TMDs. When analyzing the research studies as a whole, it is observed that out of the 17 investigations reporting an association between OT and TMDs, 10 (3 descriptive and 7 longitudinal studies) reported improvement of signs and symptoms in patients with OT history (therefore, OT was presented as a therapeutic or preventive factor for TMD) [18, 19, 26, 54, 57, 58, 62-64, 67]. In turn, 7 (3 descriptive, 3 case-control, and 1 longitudinal study) reported the opposite, concluding that there was a more significant number of signs and symptoms in patients with an OT history, therefore presenting OT as a risk or etiologic factor for TMDs. [24, 35, 36, 39, 44, 45, 70]. On the other hand, when analyzing research studies according to their design, it is observed that out of the 22 publications shown in table 2 —comparing individuals with OT history versus individuals with no treatment— 16 of these reported the lack of any association between having received OT and the presence of TMDSS. Out of the remaining [6, 3] reported improvement in patients (considering it as a preventive or therapeutic factor)[18, 19, 26], and 3 reported greater presence (worsening)[24, 35, 36] of certain TMDSS in patients who had received OT (considering it as a risk or etiological factor). The 8 case-control studies presented in Table 3 — comparing patients with TMD versus asymptomatic individuals— included only 3 studies reporting a positive association with a greater presence of OT history in TMD patients.

[39, 44, 45] The 26 longitudinal studies (a single randomized clinical study) shown in the table include 7 studies reporting improvement or lower presence of TMDSS in patients who received OT (considering it as a preventive or therapeutic factor) [54, 58, 59, 62, 63, 64, 67] and include only 1 study showing OT as a risk or etiologic factor for TMDs.[70] The remaining 18 reported lack of association.

Analysis of the parameters guaranteeing the validity of the reviewed studies:

1. Definition of the gold standard. In the descriptive (cross-sectional) and longitudinal studies, TMDs were defined by the presence or absence of one or more generally isolated signs or symptoms, which were identified in patients through questionnaires, interviews, and physical examination. Only a few of these studies attempted to make an analysis based on the presence or absence of the diagnosis of TMD or TMD subgroups.[35, 62] In the case-control studies, patients were recruited from university hospitals specializing in TMDs and only 4 research studies focused on a specific muscle or joint diagnosis of TMD. [40-42, 44]

2. Diagnostic classification system. Only a few reviewed studies attempted to use a systematic diagnostic classification that would allow a differential diagnosis between the different TMDs. The clinical research criteria for TMDs proposed by Dworkin (1992)[73] —whose reliability and validity have been studied—[74-77] were used in some of the studies,[35, 42, 44, 45, 62, 70] while in others, general criteria were used for the diagnosis of TMD39, [43, 46] or very specific diagnoses such as disc displacement confirmed with magnetic resonance imaging (MRI).[40, 41]

3. Use of clearly defined measures. In general terms, the criteria for identifying TMD signs were described similarly throughout the studies; however, the techniques used during the physical examination to establish its presence were different. For example, for the assessment of articular noises (AN), some used stethoscopes, others used TMJ palpation, and others considered the patients' reports. Some studies made use of indexes whose reliability has been researched.[35,42,44,45,62] However, most of them were limited to using methods considered "standard" in each report and whose reliability has not been reported. In the studies in which questionnaires were used as an evaluation method, only one reported having considered its reliability.[32]

4. Intra- and inter-examinators acceptable reliability. Only some studies clarified the number of examiners used, being usually one or two. Most studies did not report this number. No reliability measurement was reported in studies where only one examiner was used.[40, 41, 44, 45, 59, 66] While in other studies [29, 30, 31, 33, 35, 46, 5254, 57, 62, 70] where two or more examiners were used, some reported calibration, but concrete values of examiners reliability were revealed only in two studies.[63, 70-72]

5. Use of suitable samples. In most studies, participants were selected from university clinics specializing in orthodontics. These samples were chosen by convenience, and few studies reported any statistical calculation to determine the size of these samples, and the use of randomization was limited.[42, 72] In addition, the severity of TMD cases was not determined by factors such as intensity, duration, or frequency of signs and symptoms of the subjects. Thus, it can be observed that the

samples do not lead to obtaining an adequate representativeness that would allow the generalization of the results obtained in these reports.

6. Using matched control groups. A good number of the studies reported having considered possible confounding variables that were included in the analysis of the results or having used subject matching.[17, 21, 23, 31, 32, 37, 42, 44, 45, 48, 55, 57, 63, 64, 68, 72] Some occlusal, social (occupation, education level, economic situation, marital status), parafunctional (reported bruxism) or emotional (anxiety levels, depression) variables, among others, were considered in the studies. However, age and sex were the most commonly controlled factors.

7. Random assignment of patients and subjects to study groups. A limited number of studies used randomization as a mechanism for choosing or assigning participants.[42, 72]

8. Data collection by "blind" examiners. The vast majority of studies did not report considering data collection by "blind" examiners, and only a few did some sort of control of this bias variable for examiners. In order to control this aspect, at the time of clinical evaluation, examiners were prevented from knowing previous information obtained from questionnaires or interviews.[25, 36, 46, 57, 62, 63]

9. Study replicability. The lack of replicability between studies was evident. However, most of the descriptive studies reported no association between orthodontic treatment and the development of TMDSS. Only a few articles reported that having a history of OT was a predisposing factor for TMD development.[24, 35, 36] On the contrary, some reported a lower prevalence of TMDSS in patients who had received OT.[18, 19, 26] Among the case-control studies, the results were slightly more balanced; three reported an association between the history of OT [39, 44, 45] and TMD, and five reported no association.[40-43] Most longitudinal studies reported no association between OT and the development of TMDSS. None of these studies reported OT as a risk factor for the development of signs or symptoms of TMD. On the contrary, longitudinal studies reported lower TMDSS prevalence in patients who had received OT (presenting OT as a preventive or therapeutic factor of TMDSS).[54, 58, 59, 62-64,67] The only randomized clinical study reported no association.[72]

9. Consideration of alternative hypotheses. The lack of replicability and consistency of conclusions between studies leads to the creation of certain alternative hypotheses. If one made a more specific analysis regarding the type of patient or patient conditions (joint or muscle), could it be clarified when the OT would behave as a preventive, therapeutic or etiological factor for TMDs? In addition, because epidemiology shows that TMDs are more frequent in women[78] and if OT is considered preventive or therapeutic, alternatively, one could ask: is there a greater need for OT in women than in men? If, on the other hand, one considered the OT to be etiological, the alternative question would be: is there a higher prevalence of OT history in women than in men? These are some of the hypotheses that could alternatively arise from the results of the review of those studies.

Table 2. Cross-sectional descriptive studies comparing treated and untreated cases with orthodontics

Main author and year	Sample (♀ / ♂)	Average age (range)	TMD evaluation method	Appliances	Extrac. vs. No extrac. (Number of participants)	Occlusal examination	Controlled confounding variables	Follow-up time	General conclusion (description of results)
Sadowsk&Begole, 1980	75 (46/29) private practice patients.	NR (25-55).	Pain, TMJ AN, parafunctional habits (quest., interv., examination)	Upper and lower fixed arch wire	NR	NC working, non-working or protrusive interferences. Displacement in centric was higher in the controls	Age, sex, occupation, education level, family economic situation	10-35 years after OT	No association (OR: 0,43 for history of pain; OR: 0,83 for the presence of noises in TMJ. OR: 0,5 of the patient's displacement in centric center vs. controls)
	75 (47/28) Untreated malocclusions	NR (25-55).							
Larsson & Ronner- man, 1981	23 (12/11) private practice patients	25 (24-28)	Helkimo Index (Di, Ai, Oi) (interv., examination)	Activator or arch wire with and without extractions	NC (17/6).	Oi NC	NR	10 years after OT	Improvement in patients
Janson & Hasund, 1981	Patients from a university		Helkimo Index (Di, Ai) (interv., examination)	Fixed arch wire, headgear, and activator	NC	NR	NR	5 years after OT	Improvement in patients (OR: 0,3 for patient pain history vs. controls. OR: 1,8 for patient pain history with and without extractions)
	30 (15/15) Class I. Division 1 with extractions	22 (18-36)							
	30 (15/15) Class I. Division 1 with extractions	19 (14-27)							
	30 (18/12) untreated subjects	23 (18-36)							
Helm & col., 1984	83 (NR) with OT history vs. 758 (NR) untreated subjects	NR (28-34)	TMD symptoms, headaches, and parafunctional habits	NR	NR	NR	NR	NR	No association
Sadowsk & Polson, 1984	96 (64/47) private practice patients	87,7 (NR)	Pain, TMJ AN, functional occlusion	Fixed	NC (first group: 28/68. Second group 39/67)	NC Working, non-working or protrusive interferences. Displacement in centric was higher in the controls	Age, sex, occupation, education level, family economic situation	10 years after OT	No association (OR: 0,63 and OR: 1,01 for patient pain history vs. controls for the first and second group, respectively)
	111 (64/47) patients from a university.	29,3 (NR)							
	103 (68/35) untreated subjects	37,7 (NR)							
	111 (62/39) untreated subjects	32,9 (NR)							
Dahl y col.,	51 (28/23) with OT history vs.	19	Helkimo Index (Di, Ai, Oi) (interv., examination)	NR	NR	NC Oi	NR	5 years after OT	No association
	47 (19/28) untreated subjects	19							
Smith & Freer 1989	87 (60/27) with OT history vs.	21,1 (NR)	Sensitivity, TMJ AN, occlusion, Helkimo index (Di, Ai, Oi) (interv., examination)	Fixed	NC (26/61)	NC Working, non-working or protrusive interferences. Displacement in centric, type of occlusion, stability	Matched by the buccal segments relationship, overjet, overbite after treatment	52 months after OT	Non-association (soft snap in the TMJ was occurred twice as much in the orthodontic group, and lateral displacement in centric was close to being statistically significant)
	28 (16/12) untreated subjects	19,7 (NR)							
Loft & col.,	219 (44/175) with OT history vs. 349 (50/299) untreated patients	NR (20-43) of the whole group.	Temporomandibular, facial, and head pain (quest.)	NR	NR	NR	NR	NR	Worsening of patients with a partial association displayed only in F (OR: 3 for patient pain history vs. controls)

Nielsen & col., 1990	295 (NR) with OT history vs. 411 (NR) untreated subjects	NR (14-16) of the whole group.	Mandibular movements, TMJ AN, muscle and TMJ tenderness, dental occlusion (examination)	Fixed and removable	NC (145/150)	NR	NR	NR	No association (OR: 1,2 for patient dysfunction history vs. controls. OR: 1,2 for patient dysfunction history with and without extractions).
Keb & col., 1991	54 (NR) with OT history vs. 52 (NR) untreated dentistry students	NR (20-30) of the whole group.	Mandibular movements, TMJ noises, muscle and TMJ tenderness, dental occlusion. Di (examination)	Fixed and removable	NR	Correlational Working, non-working or protrusive interferences. Displacement in centric, type of occlusion, occlusal stability	NR	"Many years" after OT	Improvement in patients (better results of orthodontic patients in the occlusal and instrumental clinical evaluation).
Artun & col., 1992	29 treated Class II malocclusion patients. Division 1 with extractions vs.	16,9 (11-25).	History or presence of TMJ noises TMJ tenderness and TMJ tomography	Fixed and removable	NC (29/34).	NC Displacement in centric	NR	From 0,1 to 3 years after OT	No association
	34 patients with Class I malocclusion without extractions	16,6 (13,1- 24,9)							
Wadhwa & col., 1993	31 (28/3) (NR) patients with history of malocclusions with OT history vs.		Di, Ai (interv., examination)	Fixed	NC (27/4)	NC Angle's class, crowding or spacing, crossbite, overjet, and overbite	NR	From 6 months to 6,5 years after OT	No association
	41 (26/15) with untreated malocclusions vs.	19,58 (15-24) 16,9 (13-5)							
	30 (15/15) with normal occlusion	20,97 (14-25)							
Luppanapornlarp & Jhonson, 1993	62 (36/26) Angle's Class II treated patients with and without extractions	NR	Craniomandibular index (CMI) and cephalometry (examination)	Fixed	NC (33/29).	NR	NR	An average of 15,3 years (10,8-22,5) after OT	No association
Beattie & col., 1994	63 (31/32) Angle's Class II treated patients with and without extractions	NR	CMI (examination)	Fixed	NC (33/30)	NR	NR	An average of 14,5 years (±3,4) after OT	No association
Lagerstorm & col., 1998	260 (137/123) with OT history vs.	19	Di (quest., examination)	Fixed and removable	NR	NC Displacement in centric and non-working contacts	Age, examiners calibration	NR	No association
	121 (62/59) untreated subjects	20							
Macfalane & col., 2003	427 (137/123) patients with OT history vs. 1960 untreated subjects	NR (18-65).	Orofacial pain report (quest.)	NR	NR	NR	Age, sex, reliability of the questionnaire	NR	No association (OR: 1,2 for patient dysfunction history vs. controls)
Conti & col., 2003	200 (120/80) Angle's Class I and II adolescents treated with and without OT	12 (9-14).	Ai (quest., examination)	Fixed and removable	NR	NC Open bite, crossbite, displacement in centric and lateral, and anterior guidance	NR	NR	No association

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Arat & col., 2003	32 (18/14) Class III malocclusion subjects treated with chin strap	18,4 (13,9-22,5)	TMD signs and symptoms	Removable	NR	NR	NR	Average post-OT retention 5,6 (2-11 years) and average OT time 1,8 years	No association (orthodontic treatment is not a risk or preventive factor for TMD)
	32 (14/20) Class III malocclusion subjects without treatment	15,5 (12,5-31,1)							
	53 (29/24) dentistry students.	19,2 (18-12,4)							
Huddlestn Slater & col., 2007	2153 (NR) subjects, out of which 410 had OT history (1964 had not received any OT)	Older than 4 years old	AN in the TMJ for diagnosis of disc displacement with reduction and hypermobility.	NR	NR	Positive correlation Overjet and overbite	NR	NR	Worsening of patients with an 1,57 OR for the presence of disc displacement with reduction and hypermobility (using TMJ noises for patient diagnosis) in patients with a history of orthodontia vs. untreated patients
Godo& col., 2007	35 (NR) with OT history vs. 309 (NR) untreated subjects	NR (16-18)	TMD signs and symptoms Ai (quest., examination)	NR	NR	NC Type of Angle's malocclusion, open bite, crossbite, tooth crowding or spacing	NR	NR	Worsening of patients with an 3,08 OR for self-report of TMD symptoms in orthodontic vs. untreated patients
Akhter & col., 2008	2374 (642/1732) subjects, out of which 1947 had OT history (427 had not received any treatment)	18,7 (±1,1)	TMD symptoms (quest.)	NR	NR	NR	Age, sex, emotional stress, and parafunctional habits	NR	No association (orthodontic treatment is not a risk factor for TMD)
Rey & col., 2008	25 (16/9) Angle's Class III patients.	16,7 (12-24) for all groups.	TMD signs and symptoms Ai (examination)	Removable	NR	NR	NR	2-3 years after starting treatment	No association (prevalence of TMD symptoms is similar between groups)
	25 (13/12) Angle's Class I patients with previous OT history. 25 (14/11) subjects with no OT history								
	25 (14/11) subjects with no OT history								

Orthodontic treatment (OT); Helkimo anamnestic index (Ai); Helkimo dysfunction index (Di); Helkimo occlusal index (Oi); questionnaire (quest.); interview (interv.); no correlation (NC); not reported (NR); female (F); disc displacement without reduction (DDwoR); magnetic resonance imaging (MRI); electromyography (EMG); temporomandibular disorders (TMD); temporomandibular joint (TMJ); articular noises (AN); odds ratio (OR).

Table 3. Case-control studies

Main author and year	Sample (♀ / ♂)	Average age (range)	TMD evaluation method	Appliances	Extrac. vs. No extrac. (number of participants)	Occlusal examination	Controlled confounding variables	OT tracking time	General conclusion (description of results)
Pulinger et al., 1988	152 (102/50) university patients	NR (18-35)	TMD (quest.)	NR	NR	NR	NR	NR	Worsening of patients with an OR: 1,6 for orthodontic treatment history in F patients with TMD
	131 (192/139) oral hygiene and dentistry students	NR (18-35)							
Katzberg et al., 1996	102 (90/12) university patients	29,9 (10-66)	Disc displacement in MRI-diagnosed TMJ (quest., examination)	NR	NR	NR	NR	NR	No association (OR: 1)
	76 (39/37) asymptomatic volunteers	28,3 (19-49)							
Tallents et al., 1996	236 (197/24) university patients	NR	Disc displacement in MRI-diagnosed TMJ (quest., examination)	NR	NR	NR	NR	NR	No association (OR: 0,83)
	82 (42/40) asymptomatic volunteers								
Huang et al., 2002	274 (229/45) patients of an insurance plan	40 (22-82)	Patients with myofascial pain, arthralgia, and a combination of both (myofascial pain and arthralgia) (quest., interv., examination)	NR	NR	NR	Age, sex, economic status, education level, marital status	NR	No association (OR: 0,98)
	195 (109/86) symptomatic and asymptomatic subjects from the general population	40 (18-74)							
Macfarlane et al., 2001	152 (102/50) university patients	18-65	NR	NR	NR	NR	Age and sex	NR	No association (OR: 0,88)
	131 (192/139) dentistry students								
Velly et al., 2002	59 (43/16) university patients	Approximately 35 (18-60). For both groups	Disc displacement in the TMJ (quest., examination)	NR	NR	NR	Age, sex, economic status, education level, bruxism report, trauma, marital status, depression, and anxiety	NR	Worsening of patients with an OR: 1,6 for orthodontic treatment history in TMD patients
	100 (64/36) dental patients								
Velly et al., 2002	162 (119/43) university patients	Approximately 35 (18-60). For both groups	TMD (quest., examination)	NR	NR	NR	Age, sex, economic status, education level, bruxism report, trauma, marital status, depression, and anxiety	NR	Worsening of patients with an OR: 3,62 for orthodontic treatment history in a TMD patients subgroup
	100 (64/36) dental patients								
Mohlin et al., 2004	62 (41/21) general population subjects with severe symptoms	30 for both groups	DTM symptoms (quest. interv., examination)	Fixed and removable	NC (NR)	NC Angle's class, crowding, crossbite, overjet, overbite, non-working interferences, type of occlusion	Occlusal condition, psychosocial condition. Reliability of examiners.	11 years	No association (OR: 1,02 for orthodontic treatment history in TMD patient)
	72 (44/28) asymptomatic subjects								

Orthodontic treatment (OT); Helkimo anamnestic index (Ai); Helkimo dysfunction index (Di); Helkimo occlusal index (Oi); questionnaire (quest.); interview (interv.); no correlation (NC); not reported (NR); female (F); disc displacement without reduction (DDwoR); magnetic resonance imaging (MRI); electromyography (EMG); temporomandibular disorders (TMD); temporomandibular joint (TMJ); articular noises (AN); odds ratio (OR).

Table 4. Longitudinal studies

Main author and year	Sample (♀ / ♂)	Average age (range)	TMD evaluation method	Appliances	Extrac. vs. No extrac. (number of participants)	Association with occlusal factors (occlusal factors)	Controlled confounding variables	OT tracking time	General conclusion (description of results)
Pancherz, 1985	23 (NR) division 1 Class II patients with OT history. 20 (NR) Class II individuals. Division 1 without OT history	NR	TMJ noises, and muscle and TM sensitivity EMG (examination)	Functional	NR	NC Angle's class, displacement in centric, overjet, overbite, occlusal stability	NR	1 year after finishing OT	No association
Dibbets & Ven derWeele, 1987	172 (95/77) different types of malocclusions	12,5 (8-15 for 85% of the sample)	Objective and subjective presence of TMJ noises (interv., examination)	Fixed and functional	NR	NR	Matched by age	10 years after starting OT	No association (orthodontic treatment does not cause TMD development)
Sadowsky et al., 1991	160 (92/68) various types of Angle's malocclusions	14,5 (9-41)	Objective and subjective AN presence in TMJ, limitation of normal movements, muscle, and TMJ sensitivity (interv., examination)	Fixed	NC (87/68)	NR	NR	Right after finishing OT	No association (orthodontic treatment, with or without extractions, does not cause the development of AN in the TMJ)
Dibbets & van der Weele, 1991	172 (95/77) different types of malocclusions	12,5 (8-15 for 85% of the sample)	Pain, TMJ noises, limitation of jaw movements (interv., examination)	Fixed and functional	NC (114/58)	NR	NR	15 years after starting OT	No association (orthodontic treatment, with or without extractions, does not cause the development of AN in the TMJ)
Dibbets & van der Weele, 1992	172 (95/77) different types of malocclusions	12,5 (8-15 for 85% of the sample)	Pain, TMJ noises, limitation of jaw movements (interv., examination)	Fixed and functional	NC (114/58)	NR	NR	20 years after starting OT	No association (orthodontic treatment, with or without extractions, does not cause the development of noises in the TMJ)
Kremenak, 1992	63 (44/21) university patients with OT history, with and without extractions	19,5 (16-25)	Di	Fixed	NC (26/39)	NR	Examiner calibration	< 3,5 years after starting OT	No association (no significant clinical differences between patients with or without extractions during orthodontic treatment)
Kremenak, 1992	109 (69/40) university patients with OT history, with and without extractions	19,7 (16-25)	Di	Fixed	NC (76/33)	NR	Examiner calibration	From 0 to 6 years after starting treatment	No association (orthodontic treatment, with or without extractions, does not cause the development of noises in the TMJ)
Egermark & Thilander, 1992	100 OT patients 193 without treatment history	NR (7-15)	Di (quest., examination)	Fixed and functional	NR	NC (Angle's class, lateral bite force, non-working interference, displacement in centric)	Examiner calibration	10 years after treatment	Improvement in patients (subjects with a history of orthodontic treatment had a lower prevalence of subjective symptoms)
Hirata et al., 1992	102 (59/43) OT patients 41 (20/21) patients without treatment history	16,2 (NR) 16,2 (NR)	Subjective symptoms, mandibular movements, TMJ AN (quest., examination)	Fixed	NR	NC (midline, overjet, overbite)	Matched by age	1-2 years after starting treatment	No association (orthodontic treatment does not increase or decrease the risk of developing TMD)
Rendell et al., 1992	462 (NR) OT patients from a university	NR (10-35). 90% < 18. 10% > 18	Ai, Di (quest., examination)	NR	NR	NR	NR	18 months during active treatment	No association (orthodontic treatment is independent of TMD development)
O'Reilly et all., 1993	60 (30/30) OT patients 60 (35/25) subjects without OT history	15,3 (14,3-16,1) NR	Mandibular movements, TMJ AN, muscle sensitivity (quest., examination)	Fixed	CN 60/0	NC Angle's class, overjet, and overbite	Age, examiner calibration	Before, during, and right after treatment is finished	No association (no significant clinical differences between patients with or without extractions during orthodontic treatment) (orthodontic treatment with tooth extraction does not cause TMD development)

Egermark & Ronnerman, 1995	50 (27/23) consecutive patients from an orthodontic clinic. 135 (NR) subjects without OT history.	12,9 (7,8-16,8). 15 (NR)	Di (quest., examination)	Fixed	NC (35/15)	NC (Angle's class, lateral bite force, non-working interference, displacement in centric)	NR	Before, during, and right after treatment is finished	Improvement in patients (subjects with a history of orthodontic treatment had a lower prevalence of subjective symptoms)
Olson & Lindqvist, 1995	210 (116/94) pre-OT subjects	12,8 (7,1- 20,4) at the beginning of the study	Muscle palpation, Di (quest., examination)	Fixed		NC (Angle's class, non-working interference, displacement in centric)	NR	Before and after completion of post-retention treatment (18 months, 9-63 months)	Improvement in patients (orthodontic treatment may prevent or cure TMD development)
Pille et al., 1997	148 (80/68) OT patients 573 (274/299) subjects without OT history	12 for both groups at the beginning of the study	TMD symptoms (quest. interv., examination)	Fixed and removable	NC (NR)	NC Angle's class, crowding, crossbite, overjet, overbite, non-working interferences, type of occlusion	NR	7 years	No association (differences between subjects with or without a history of orthodontic treatment showed minor differences)
Ngan et al., 1997	10 patients with OT Class III from a university clinic	12,8 (7,1- 20,4) at the beginning of the study	Muscle palpation, EMG (examination)	Functional	NR	NR	NR	Before, during, and right after treatment is finished (< 2 months after treatment ends)	No association (orthodontic treatment does not cause the development of muscle sensitivity or EMG changes)
Henrikson et al., 1999	65 (F) patients with Angle's class II OT		TMD signs and symptoms (quest. interv., examination)	Fixed	NC (35/30)	NC(displacement in centric, protrusive, working and non-working interference)	Examiner calibration	Before, during (1 and 2 years), and right after treatment is finished	Improvement in patients (patients showed a lower prevalence of some subjective signs and symptoms during and after orthodontic treatment)
Henrikson et al., 2000	65 (M) patients with Angle class II with OT, 58 (M) untreated Angle's class II patients. 60 (M) with untreated normal occlusions	12,8 (11-15) 12,9 (11-15) 12,7 (11-15) at the beginning of the study	TMD symptoms (quest. interv., examination)	Fixed	NC (35/30)	NC(displacement in centric, crowding, protrusive, working and non-working interference)	Matched by age, examiner calibration	Before and two years after starting treatment	Improvement in patients (patients showed a lower prevalence of muscle-origin signs after orthodontic treatment)
Henrikson & Nilner, 2000	65 (M) patients with Angle class II with OT, 58 (M) untreated Angle's class II patients. 60 (M) with untreated normal occlusions	12,8 (11-15) 12,9 (11-15) 12,7 (11-15 at the beginning of the study)	TMD symptoms and diagnosis and headaches, need for TMD (quest., interv., examination)	Fixed	NC (35/30)	NC(displacement in centric, crowding, protrusive, working and non-working interference)	Matched by age, examiner calibration	Before and two years after starting treatment	Improvement in patients (patients reported fewer TMD symptoms and headaches after orthodontic treatment. There was no difference in the diagnosis or need for TMD therapy)
Imai et al., 2000	18 (14/4) with OT/occlusal plate 27 (24/3) with OT/no occlusal plate 13 (9/4) with occlusal plate only	18,6 (NR) 18,2 (NR) 17,9 (NR)	TMD signs (TMJ pain and noises, pain and restriction of movement) (quest., examination)	Fixed	NR	NC (open bite, crossbite, crowding, maxillary protrusion)	NR	Before and after plate therapy. Right after and one year after orthodontic treatment	No association (orthodontic treatment does not cause recurrence or exacerbation of TMD symptoms)
Tullberg et al., 2001	22 (14/8) patients with early OT and 22 (12/10) patients with late posterior crossbite OT	21 (±1,5)	TMD signs and symptoms (quest., examination)	Fixed	NR	NC(displacement in centric, protrusive, working and non-working interference)	NR	14-18 years after treatment	No association (early orthodontic treatment, even if it fails and needs to be continued later, does not result in an increased future risk of developing TMD symptoms and signs)
Henrikson & Nilner, 2003	65 (F) patients with Angle's Class II OT	12,8 (11-15)	TMD symptoms and diagnosis and	Fixed	NC (35/30)	NC(displacement in centric,		Before and three years after	Improvement in patients (orthodontic treatment, with or

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	58 (M) patients with untreated Angle's Class II	12,9 (11-15)	headaches, need for TMD (quest., interv., examination)			crowding, protrusive, working and non-working interference)	Matched by age, examiner calibration	starting treatment	without extractions, does not increase prevalence or worsen pre-existing TMD signs or symptoms)
	60 (M) with untreated normal occlusions	12,7 (11-15) at the beginning of the study							
Egermark et al., 2003	102 OT patients 192 subjects without treatment history	NR (7-15) across the group	Di (quest., examination)	Fixed and functional	NR	NC (Angle's class, lateral bite force, non-working interference, displacement in centric)	Examiner calibration	10 years after treatment	No association (orthodontic treatment does not increase or decrease the risk of developing TMD. Lateral forced bite due to displacement in centric and unilateral crossbite could be important in certain individuals)
Egermark et al., 2005	40 (27/23) consecutive patients from an orthodontic clinic.	12,9 (7,8-16,8)	Di (quest., examination)	Fixed	NC (35/15)	NC (Angle's class, bite force, non-working interference, displacement in centric)	NR	17 (15-18) years after finishing OT	No association (OT does not increase the risk of developing TMD)
	86 (NR) subjects without OT history.	15 (NR) at the beginning of the study							
Slade et al., 2008	186 (M) subjects without OT or TMD history	NR (18-34).	TMD signs and symptoms (quest. interv., examination)	NR	NR	NR	Examiner calibration and blood test to evaluate genetic markers predisposing pain development	3 years	Worsening of patients with association (OR: 3,03) to the genotype determining sensitive to pain production
Macfarlane et al., 2009	337 (191/146) OT patients (150) and without it (187)	NR (30-31).	TMD and Di signs and symptoms (quest. interv., examination)	Fixed and functional	NR	Malocclusion (isolated presence of certain occlusal factors)	Examiner calibration, age, sex, social class, and psychological aspects.	19 years	No association (OR: 1,0; orthodontic treatment does not increase or decrease the risk of developing TMD)
Randomized clinical trials									
Keeling, 2005	60 (25/38) patients received a bionator	9,8 (NR)	TMD signs (examination)	Functional	NR	NC Molar class, overjet, and overbite	Age, sex, mandibular plane angle, severity of Class II molar discrepancy, calibrated and "blind" examiners	Two years (approximately) after starting treatment	No association (OT does not increase the risk of TMD development, at least, in the short term after finishing treatment. Failure to achieve a Class I molar ratio puts symptom-free children at increased risk of developing muscle pain during palpation)
	71 (30/41) patients received craniomaxillary appliance	9,93 (NR)							
	60 (20/40) observation group	9,71 (NR)							

Orthodontic treatment (OT); Helkimo anamnestic index (Ai); Helkimo dysfunction index (Di); Helkimo occlusal index (Oi); questionnaire (quest.); interview (interv.); no correlation (NC); not reported (NR); female (F); disc displacement without reduction (DDwoR); magnetic resonance imaging (MRI); electromyography (EMG); temporomandibular disorders (TMD); temporomandibular joint (TMJ); articular noises (AN); odds ratio (OR).

DISCUSSION

Different literature reviews [7-10, 79] have concluded that OT should not be considered an etiological, preventive, or therapeutic factor for TMDs. However, this article, based on the different studies that have explored this relationship (Tables 2, 3, and 4), shows that the association of OT with TMDs is not clear and still controversial. An overall assessment of the studies presented in this article shows that slightly more sophisticated reports using control groups are only beginning to be seen in the last three decades, and out of these, 70% found no association between OT and TMDs. However, there is also a significant number (30%) of publications that reported opposite conclusions to the above and did show an association between OT and TMDs. Noticeably, most studies (70%) do not support an idea that for a long time was the general belief in the dentistry profession: that OT was closely related to TMDs.[80,81] This discrepancy in the results and conclusions throughout different reports is perhaps due to the divergence and inconsistency of the methodologies used among these research studies (poorly defined and differently done measurements, reliability of the examiners, etc.); which in turn directly jeopardizes the strength of factors ensuring the validity of the results. Although not as in much detail as the one presented in this article, other literature reviews have discussed some of these methodological aspects.[7-10, 79, 82-84]

An important methodological aspect is how TMDs were defined. Except for case-control studies (Table 3), in most of the reported research studies, TMDs are defined in terms of signs and symptoms, which are generally presented in isolation and do not necessarily represent a disease state with the clinical characteristics of patients diagnosed with TMD. As it is already known, prevalence studies have shown that TMDSS are relatively frequent in the general population. It is estimated that 65% of the general population presents at least one sign in the TMJ (abnormality in mandibular movements, clicks, crepitation, and muscular or joint tenderness to palpation), and 35% presents at least one symptom (limitation of mandibular opening, muscular or joint pain).[85] In a few studies, a clinical diagnosis of TMD was used, and among these, few used systems having some reliability and validity reported.[35, 40-46, 62, 65] Some of these studies [35, 42, 44, 45, 62] used the evaluation and diagnostic criteria for TMD research published by Dworkin (1992)[73-77] , which were made to standardize different TMD researches, in order to facilitate the comparison between studies. However, even following these diagnostic criteria, there were critical differences between research studies. In the report by Huddleston et al.,[35] isolated AN without the presence of joint pain were used as a clinical criterion for diagnosing disc displacement with TMJ reduction and hypermobility. At the same time, Velly et al.[44], included pain as part of the diagnostic criteria for the same condition. Other researchers used magnetic resonance imaging (MRI)[40, 41] for the most accurate diagnosis of internal TMJ disorders. In contrast, others created their own subgroups of TMDs based on statistical calculations of patient characteristics.[45] Similarly, it is known that despite the relative TMDSS frequency in the general population, these fluctuate over time, and it is considered that only 7 to 10% could have problems severe enough to require treatment. [84] Henrikson and Nilner[63, 68] reported that although subjects were diagnosed with TMDs, only a small percentage presented a moderate severity condition requiring treatment. They also found a high fluctuation of signs and symptoms among patients. In

fact, despite Henrikson and Nilner (2000)[63] establishing a treatment need of 13%, only 3% of patients sought to be treated. This fact highlights the "overdiagnosis" of TMD in which cases with low levels of severity with subclinical signs and symptoms are considered TMD patients. Thus, the "overdiagnosis" of the studied condition would contaminate any associations found in research. These inconsistencies in the diagnosis are also related to the fact that the criteria for identifying TMDSS were not completely similar between the different studies, nor were the techniques used during the physical examination. The information regarding the presence of TMD symptoms was collected using several methods, such as interviews or questionnaires (answered by mail or in person by the patient). Regarding the signs, muscle tenderness on palpation was determined with different techniques and muscles depending on the type of protocol used. Some authors used stethoscopes to assess AN, while others used audition or palpation of the TMJ. Sometimes, the joint sounds reported by the subjects participating in the study were also considered. The fact that AN were a diagnostic criterion in most studies was interesting. However, this isolated sign or symptom is not a good TMD indicator.[86, 87] In fact, in some research studies, due to the high prevalence of AN in the general population, they were considered normal, and AN were allowed to be present in the control or "asymptomatic" group.[44,45] Regarding the assessment of dental occlusion, static and dynamic OFs were considered in almost half (52%) of the reports. Although inconsistently, certain reports showed some association between OFs and OT. For example, a minor displacement in centric in patients with a history of OT was reported by some authors [17, 26, 31] while others reported otherwise.[23, 47, 64] In many of the research studies, Helkimo[88] index was used to establish the presence of TMDSS and determine their severity. However, as mentioned in the previous articles of this review,[1, 2] this index tends to overestimate the TMD prevalence when used in the general population. Applying this index to the general population, Helkimo[88] reported that only 12% of the population was free of signs and symptoms, while 47% had at least one severe symptom of TMD. As mentioned above, the percentage of individuals seeking treatment is much lower (7-10%); therefore, this index overestimates the presence of TMD, and a possible association with OT cannot be considered entirely valid.

The sample sizes varied among the reports and were mostly not randomly drawn, which makes it evident that the representativeness, the power to generalize or extrapolate the results, is limited.[42, 70] Huang et al.[42] used randomization to choose the control group; however, in this study, they reported dissimilar groups with significant differences in sex and educational level of the participating subjects. In addition, the severity levels of TMDs or of signs or symptoms in the participants were not considered, let alone including important factors related to severity such as intensity, frequency, duration, or chronicity of the cases. When it came to establishing the severity of TDM cases, the Helkimo[88] index was used; as mentioned above, the diagnostic validity of this index is doubtful. Control of confounding variables or bias was attempted in several of the research studies through subject matching, randomization, and the use of "blind" examiners.[17, 21, 23, 31, 32, 37, 42-45, 48, 52-55, 57, 62-64, 67, 68, 70-72] Subject matching or other statistical strategies were used in an attempt to control possible confounding variables; but only rarely was interference of any variable in the results of the investigations observed. This was the case with the anxiety variable, which, when considered, maintained and strengthened the positive relationship between OT and TMDs.[44] Some researchers

tried to keep the examiners "blind", preventing the examiner from accessing the results of the anamnesis, the occlusal examination, or the history of OT at the time of the examination of the TMDSS. This is perfectly valid and understandable when there are different groups of examiners in the study for each section of the subject's data. However, it is rather doubtful that this can happen when it is not made entirely clear whether the examiner also handled other aspects of the patient characteristics information. Despite not having the information available at the time of the physical examination, it should be taken into account that examiners may remember specific information about the subject they are examining, which may influence their clinical judgment, resulting in what is commonly called memory bias. Additionally, although examiner training and calibration were reported in some cases, intra- and inter-examiner reliability levels were reported in a limited number of studies.[63, 70, 72] The possible lack of reliability of the clinical evaluation, coupled with the fluctuation inherent to TMDSS, makes the reliability and validity of the measurements made in the different studies questionable. The validity of studies is compromised when the reliability of measurements and examiners is inadequate. Likewise, most of the variables included in the occlusal, orthodontic, or TMD evaluation were mostly qualitative and not quantitative. In other words, and to illustrate the case, the presence of balancing (non-working) contacts or interferences in the occlusal examination was evaluated as absent or present without quantifying other variables that could be confounding, such as the force and the time of duration in which they occur. Perhaps these variables that are generally not taken into account could help to explain, in addition to the adaptive capacity of individuals, why some studies show that in certain subjects there is a relationship between certain OFs and TMDs, while in other individuals there is not. Regarding the orthodontic assessment, many researchers only classified the subjects depending on the type of malocclusion (Angle I, II, III). However, no report showed the use of a severity index to look for any difference in the type of orthodontic patient under study. Likewise, as mentioned before, the TMD severity was classified in some studies, but very few studies also took into account the classification of TMDs in terms of chronicity and associated characteristics.[44]

Lastly, TMDs are more frequent in females than in males. Therefore, if there is any causal or etiological correlation between OT and TMDs, one possible alternative hypothesis could be expecting females to receive OT more often than males. Similarly, if OT were considered preventive or therapeutic, it would be expected that women would have a greater need for OT than men. None of these hypotheses can be supported by the existing epidemiological reports since the need for OT is similar in both sexes. Therefore, this suggests that other hypotheses or explanations should be considered.[89-91]

It is possible that the inconsistency or lack of partial replicability between the results and conclusions of the different research studies is directly related to the methodological problems that have been discussed throughout this article. Therefore, the validity of the research hypothesis or clinical premise will always be compromised as long as there is no evidence of replicability of the conclusions and presence of methodological differences in the studies. However, other aspects could be highlighted and analyzed. Interestingly, only one of the longitudinal studies —which are the highest ranked in the hierarchy of scientific evidence— presented OT as a risk or etiological factor, while

seven of them showed OT as a preventive or therapeutic factor for TMDs. OT was presented as a risk or etiological factor for TMDs, mainly in the cross-sectional studies or case-control studies (which are ranked lower in the hierarchy of scientific evidence). These data would suggest a trend that OT could improve TMDSS. From a biomechanical point of view, considering TMDSS improvement in patients after receiving OT could make some positive clinical sense and be explained because some malocclusions (crossbite) that have been considered as risk factors for TMDSS development2, [92] are corrected or its severity its permanently decreased during OT. Likewise, none of the objectives of an adequate OT is to permanently increase or create this type of malocclusion. Other occlusal factors (working, non-working interference), although weakly associated with TMDs, generally occur or are created temporarily during OT. However, the creation of these OFs is also not included in the objectives of a finalized OT. Therefore, one could speculate that OT —in patients with certain malocclusions (e.g., crossbites)— could often be therapeutic or preventive, and in very rare cases could become a risk factor, since the principle of a good OT is to reduce the presence of these malocclusions (e.g., balancing interferences) and not to create them. Although it is known that OFs represent a slight risk in TMDSS development, these occlusal interferences are decreased in patients receiving OT.[62-64] On the other hand, it is very interesting to note that the only longitudinal study in which OT was reported as a TMD risk factor was the one in which genetic aspects of predisposition to pain development were considered.[70] In this study, Slade et al.[70] considered the variants of the gene encoding catechol-O-methyltransferase (COMT) enzyme, which is involved in pain regulation in the central nervous system. The enzyme activity is proportionally related to pain regulation; a low-activity COMT is present in patients more likely to develop pain (pain-sensitive haplotype), whereas a higher activity COMT is present in patients less likely to develop pain (pain-resistant haplotype). These researchers 70 showed that orthodontic patients who developed TMDSS after three years of follow-up were only those with the presence of the pain-sensitive haplotype. Although the general results of the epidemiological reports analyzed in this article indicate that there is no tendency to favor the association of OT (either as an etiological, preventive or therapeutic factor) with TMDs, this is a point of utmost importance since it is possibly these other factors (genetic) that may be participating as confounding variables and therefore helping to produce confusing results that interfere in the establishment of the real association between OT and TMDs. [93-95] These factors should be considered to find the actual role that OT may play as a preventive or therapeutic mean and an etiological factor of TMDs. Additionally, from a clinical point of view, this suggests that in the near future, using genetic evaluation, haplotypes should be established for the modulation of patients' pain. This process would allow establishing the risk of developing pain symptoms or TMDSS in the masticatory system, not only with OT but with any type of treatment in which it is necessary to produce occlusal changes.

CONCLUSIONS

Although different literature reviews have concluded that OT should not be considered an etiological, preventive, or therapeutic factor for TMDs, this article shows that the role of OT in TMDs is not

entirely clear. The scientific literature related to TMDs is undoubtedly controversial, and the evaluation of epidemiological reports in this area is not easy to analyze and interpret. Although the general trend (70% of the studies) was to show a lack of association between TMDs and OT (either as an etiological, preventive, or therapeutic factor) in the different reports, a considerable number of research studies (30%) associated OT (as an improvement or worsening factor) with TMDs. Although there is a more significant trend (more than twice as many studies) to show no correlation between OT and TMDs, this relationship should not be considered completely null. On the contrary, the door should be left open for more and better research to understand if there is any association.

Possibly other factors, such as genetic ones, may be confounding variables and should be considered in the prevention, etiology or management of TMDs. It is very likely that the improvement of research designs will lead to more concrete results that will help to make a clearer interpretation and conclusion about the possible relationship between OT and TMDs. However, beyond the fact that there might be an association between OT and TMDs, as a clinical recommendation, it is important to insist on the execution of appropriate screening for TMDSS in patients who will receive an OT. This will ensure the recognition of subclinical signs and symptoms before the onset of OT. If these are not recognized beforehand, they could later appear simultaneously to OT, and a causal relationship between the two might be mistakenly established. Additionally, in the near future, this screening could also be done genetically, hence establishing the haplotypes for the modulation of pain in patients. This step would allow knowing the risk of developing pain symptoms or TMDSS in the masticatory system, not only with OT but with any type of treatment in which it is necessary to produce occlusal changes.

CONFLICT OF INTEREST

The authors state that they have no conflict of interest.

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