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Multifactorial etiology of *Torus mandibularis*: study of twins

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SUMMARY

Objective. The aim of this study is to investigate the multifactorial etiology of mandibular tori analyzing the influence of genetics, occlusal overload, various clinical variables and their interactions.

Methods. Overall, plaster casts of 162 twins (81 twin pairs) were analyzed for the presence or absence of mandibular tori. Atypical wear facets on canine tips or incisors were recorded to diagnose bruxism. Angle Class, any kind of anterior open bite and positive, negative or flat curve of Wilson were recorded. Zygosity determination was carried out using a DNA test.

Results. Mandibular tori were found in 56.8% of the cases. In 93.6% of all monozygotic twin pairs both individuals had or did not have mandibular tori ($\kappa=0.96\pm0.04$; $p<0.001$), compared to 79.4% concordance of mandibular tori in dizygotic co-twins ($\kappa=0.7\pm0.12$; $p<0.001$). Prevalence of mandibular tori was significantly higher in the group of bruxers (67.5%) compared to non-bruxers (31.3%) ($p<0.001$). Significant association between mandibular tori and negative or flat curve of Wilson in the maxillary second premolars and first molars was found (OR=2.55, 95% CI (1.19-5.46), p=0.016). In all monozygotic bruxers, 97.1% showed concordance of mandibular tori presence in both co-twins compared to 78.9% dizygotic bruxers, and this difference is statistically significant (p=0.007).

Conclusion. Our results suggest that the mandibular tori are of a multifactorial origin. Mandibular tori seem to have genetic predisposition, and may be associated with teeth grinding as well as with negative or flat CW in region of maxillary second premolar and first molar.

Key words: *torus mandibularis*, twins, zygosity, genetic factor, bruxism.

INTRODUCTION

Torus mandibularis (TM) is a common oral bony outgrowth formed by compact bone with small amount of trabecular bone and fibrofatty marrow (1, 2). It is mostly found bilaterally in the lingual surface of the mandible, in the region of canines or premolars (2). Records in the incidence of TM vary inconsistently from 0.54% to 64.4% depending on the ethnic group, race, or the investigated sample (3). These bony protuberances are non pathological and usually do not produce any symptoms, therefore, the cases of surgical removal are rare (4, 5).

Various possible causes were discussed to explain the etiology of TM, but the accepted model for the formation of these bony protuberances is still under question. Historically, the dominant focus is on genetics, and the heredity of TM has been analyzed using familial (6, 7), regional studies (8, 9), or comparing ethnic groups (10-12).

However, the heredity does not explain all the cases of TM. As stated by Eggen (13), genetic determination of TM was estimated to be 30%, whereas 70% of the causes could be explained by influence of occlusal overload and other clinical variables. In the studies analyzing TM etiology, occlusal overload is

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mostly described as bruxism (14-17) or heavy food consumption (8, 18). In most of the studies association between TM and occlusal overload was found.

Other clinical variables related with the occlusal characteristics, malocclusion and oromaxillofacial function (e. g. Angle class, open bite, buccal overjet, curve of Spee etc.) were hypothesized as possibly having a role in creating TM (19), but the studies are limited. Significant links between TM and Eichner Index, occlusal support at the premolar and molar areas, occlusal force and presence of TM were found (14). Moreover, number of teeth and adequately developed jaws were also positively related with the incidence of TM (20). In the literature, significant relation between TM occurrence and temporomandibular disorders was reported as well (15, 17).

These controversial findings of multifactorial nature give much space for the discussions about the origins of TM. The key to explain multifactorial etiology of TM could be functional matrix hypothesis (21). It is based on Wolff's law, which state that loading force prompts remodeling and strengthening of the bone (22, 23). According to functional matrix hypothesis, compressive stresses may lead to buckling of the mandible in the mental foramen region, which has a reduced bone volume. Osteogenic periosteum in these regions is stretched and this tension leads to new bone formation in the form of tori (19). This functional matrix hypothesis considers heredity as well, since children inherit jaw form from parents (19). However, this multifactorial hypothesis of TM occurrence still lacks evidence.

Therefore, the primary purpose of this research is to investigate the multifactorial etiology of TM, studying the influence of genetics, occlusal overload and other clinical variables taking twins as an investigation sample. Explanation of the potential causes for TM formation may give valuable knowledge about the biomechanical mechanisms improving bone quality (24, 25).

MATERIAL AND METHODS

A total 162 pairs of maxillary and mandibular plaster casts of individuals (81 pairs of twins) were examined. The plaster casts were collected using the database of Orthodontic Department and Scientific Twin Center at the Lithuanian University of Health Science. Plaster models were selected from nationwide population-based database randomly.

We used the criteria for inclusion, which were described as follows: a) subjects have to be with permanent dentition; b) no orthodontic treatment has to be performed; and c) zygosity has to be confirmed by DNA analysis. The sample consists of 47 monozygotic and 34 dizygotic twin pairs. There were 100 females and 62 males in the sample. The age of the subjects ranged from 12 to 51 years (mean age of 20.3±0.9 y.).

Zygosity determination was carried out using a DNA test. The polymerase chain reaction set AmpFℓSTR® Identifiler® (Applied biosystems, USA) was used to amplify short tandem repeats and 15 specific DNA markers (D8S1179, D21S11, D7S820, CSF1PO, D3S1358, TH01, D13S317, D16S539, D2S1338, D19S433, vWA, TROX, D18S51, D5S818, FGA) and the Amel fragment of the amelogenin gene were used for comparison of genetic profiles. The zygosity determination using this molecular genetic technique reaches 99.9% accuracy.

Plaster casts were chosen as a reliable method to diagnose TM, bruxism, and various occlusal variables (26, 27). Two calibrated observers (periodontologist and general dentist) evaluated the data manually analyzing the plaster casts and looking for TM. The nodular bone protuberance on the lingual surface of the mandible in the region of canines or premolars was considered as TM. Bruxism was diagnosed by recording the atypical wear facets on incisors and canine tips or non-functional surfaces. Eccentric wear facets, arising only during extreme extrusive mandible movements, were taken as an evidence for teeth grinding (2, 28-32).

Occlusal variables such as Angle Class, any kind of anterior open bite in habitual occlusion and positive, negative or flat curve of Wilson (CW) on the maxillary premolars and molars were recorded. To identify Angle Class, relationship between first molars was recorded. In a case of Angle Class II subdivision or Angle Class III subdivision, when molar relationship was asymmetrical and was Class I on the one side and Class II or III on the other, the cases were considered as Angle Class II or Angle Class III (33). CW was considered flat when the tips of vestibular and palatal cusps were at the same level on the both sides of the maxillary premolars or molars. Patients with negative CW exhibited longer vestibular cusps compared to palatal cusps, and on the contrary for the patients with positive CW.

For calibration purposes two examiners were trained for better reliability diagnosing TM, bruxism and occlusal variables. After training, the examiners evaluated 10 plaster casts, not belonging to the study sample, twice. Recorded Kappa (κ) index for inter-rater agreement was >0.8, and intraclass correlation coefficient (ICC) was >0.8 for all parameters.

The obtained data were analyzed using the IBM SPSS Statistics 22 (SPSS, Inc, Chicago, IL). The interdependence of qualitative evidence was evaluated by Chi-square (χ^2) criteria. Risk estimates were calculated

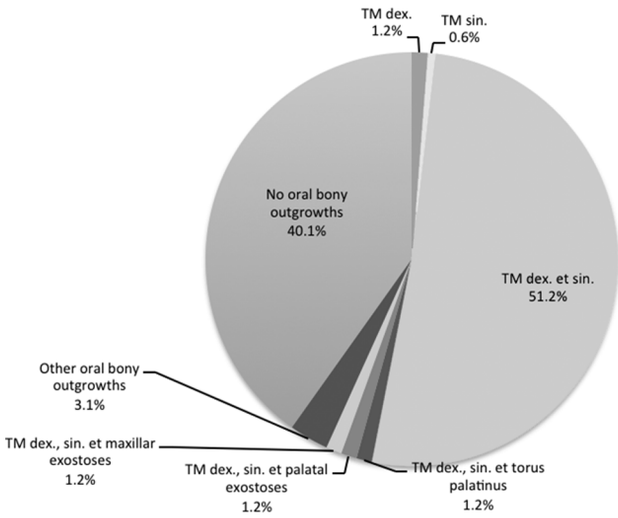

Fig. Distribution of TM

as unadjusted odds ratios (ORs) with 95% confidence intervals (CIs) using logistic regression analyses, and the *P* value was set at 0.05. Strength of agreement between the measurements was evaluated using *Kappa* (κ) coefficient. κ values ≤0.2 were considered as poor agreement; 0.21–0.40 fair; 0.41–0.60 moderate; 0.61–0.80 good; and 0.81–1.00 very good (34).

Ethical approval for the research was obtained from Kaunas Regional Biomedical Research Ethics Committee.

RESULTS

Overall, 56.8% of the subjects had TM and it was the most frequent type of bony outgrowth in the sample. Predominantly, subjects had TM bilaterally or with another oral bony outgrowth, and the cases with unpaired TM are rear (Figure).

Higher incidence of TM was diagnosed for females and for the group over 18 years, but no statistically significant differences were found (Table 1).

Table 1. Prevalence of TM associated with different variables

	TM prevalence		Difference p-value (Chi-squared test)
	n	%	
Gender			
Females	60	60.0	p=0.3
Males	32	51.6	
Age			
=<18	43	52.4	p=0.25
>18	49	61.3	
Bruxism			
Bruxers	77	67.5	**p<0.001**
Non-bruxers	15	31.3	
Angle Class			
Angle Class I	21	58.3	p=0.326
Angle Class II Division 1	24	70.6	
Angle Class II Division 2	29	55.8	
Angle Class III	18	45.0	
CW			
Negative or flat CW at first premolar	88	56.4	p=0.7
Negative or flat CW at second premolar	77	61.6	**p=0.02**
Negative or flat CW at first molar	35	64.8	p=0.15
Negative or flat CW at second molar	5	83.3	p=0.18
Negative or flat CW at second premolar and first molar	78	61.9	**p=0.01**
Anterior open bite			
No open bite	73	56.6	χ^2 =3.117; df=3; p=0.44
Open bite between incisors	11	57.9	
Open bite between canine	8	66.7	
Open bite between premolars	0	0	

In bold – p<0.05. Abbreviations: TM, torus mandibularis; CW, curve of Wilson; χ^2 , chi square test; df, degree of freedom

In the investigated sample, 70.4% of the subjects were identified as bruxers. Prevalence of TM was significantly higher in this group compared to non-bruxers (Table 1).

Analyzing influence of the various occlusal variables on TM occurrence, significant association between negative and flat CW in the maxillary second premolars and first molars was found (OR=2.55, 95% CI (1.19-5.46), p=0.016) (Table 2).

The influence of genetics on the etiology of TM was evaluated calculating concordance values for the presence or absence of TM between first and second twin (co-twin) in MZ and DZ pairs. 93.6% of all MZ and 79.4% of DZ co-twins showed concordance in the occurrence of investigated bony outgrowths, i.e. both individuals in the pair had or did not have TM. High κ values (0.96±0.04) show very good strength of agreement between the measurements in MZ co-twins, and good agreement (0.7±0.12) in DZ co-twins. This difference is statistically significant (p<0.001) (Table 3).

The multifactorial etiology of TM was evaluated grouping the sample into four groups: MZ bruxers, MZ non-bruxers, DZ bruxers and DZ non-bruxers. In our study, 97.1% of all MZ bruxers showed concordance of TM presence in both co-twins compared to 78.9% of all DZ bruxers (p=0.007). Significant difference between

MZ non-bruxers (91.7%) and DZ non-bruxers (80.0%) was found as well ($p<0.001$).

Multiple logistic regressions analysis did not show significant increase in TM occurrence for the bruxers with negative or flat CW in region of maxillary second premolar and first molar ($p=0.089$).

DISCUSSION

Prevalence

This report demonstrates that TM is dominant oral bony outgrowth and has a high incidence (56.8%) in the investigated sample of Lithuanian twins. In the worldwide studies, prevalence of TM has a wide variety in rates – between 0.54% to 64.4% (3). Higher prevalence of TM in our study may be partially explained by method of data collection. Plaster cast analysis leads to more precise data collection and possibility to calibrate the examiners, to recalculate and compare the cases seeking for maximum accuracy. After training and calibration procedure inter-rater agreement (weighted Kappa) and ICC between researchers diagnosing TM were 0.82 and 0.97 respectively.

Genetics

TM dominates in Japanese, Spanish, Ghanaian populations (14,35,36). On the other hand, German, Norwegian, Croatian, Thai, Malaysian populations were reported to have torus palatinus more commonly (11, 16, 27, 37). Consequently, researchers suggest genetics as responsible factor in the etiology of TM and other bony protuberances. However, until now the influence of genetics was mostly analyzed using regional, ethnicity research, or familial studies (6-8,10-12). The present study gives a new perspective since it takes the sample of twins as an object to verify the influence of genetic factor on the occurrence of TM for the first time. MZ twins are genetically identical and are expressing more similar traits than DZ twins. If genes determine the trait, agreement between traits (Kappa) in both individuals is close to 1.0 in MZ twin pair and near to 0.5 in DZ twins (38) (0.96 and 0.7 respectively in our research). The results of our study prove influence of genetic factor in TM etiology.

Data on TM prevalence in the groups of females and males (60.6% and 50.8% respectively, but no statistically significant differences) do not confirm the X chromosome-linked heritability of TM and support some previous reports (14, 39). Moreover, our study did not show statistically significant differences of TM prevalence according to age. These findings differ from other reports showing gradual growth of oral bony outgrowths, which is greater in second or third decade of life (12, 36). Thus, our results may be influenced by the young mean age (20.3±0.9 y) of the sample, which dominates in the database of Scientific Twin Center. Seeking to evaluate the age dependency on TM prevalence more epidemiological studies including various age groups should be implemented.

Bruxism

In our study the prevalence of TM is significantly correlated with teeth grinding, and these findings are consistent with other studies (13, 14, 16). The diagnostics of bruxism is controversial and various methods are known (2). In our study bruxism was diagnosed

Table 2. Association between TM and different variables

Variables	OR*	95% CI	P value
Age (>18 years)	1.43	0.77-2.68	0.258
Gender (women)	1.41	0.74-2.66	0.296
Bruxism	4.58	2.22-9.46	**<0.001**
Angle Class II Division 1**	1.71	0.64-4.62	0.287
Angle Class II Division 2**	0.9	0.38-2.13	0.811
Angle Class III**	0.58	0.24-1.45	0.247
Anterior open bite	1.04	0.48-2.26	0.919
Negative or flat CW at first premolar***	0.65	0.12-3.64	0.621
Negative or flat CW at second premolar***	2.35	1.11-4.98	**0.025**
Negative or flat CW at first molar***	1.65	0.84-3.24	0.146
Negative or flat CW at second molar***	3.97	0.45-34.74	0.213
Negative or flat CW at second premolar and first molar***	2.55	1.19-5.46	**0.016**

* Unadjusted odds ratio of logistic regression.
** Compared to Angle Class I.
*** Compared to positive CW.
In bold – $p<0.05$. Abbreviations: OR, unadjusted odds ratio; CI, confidence intervals; CW, curve of Wilson.

Table 3. Concordance values of TM in twin pairs

	Concordance values of TM		Discordance values of TM		κ value
	First and second twin with TM, or first and second twin without TM		First twin with TM and second twin without TM, or first twin without TM and second twin with TM		
	n	%	n	%	
MZ twins	44	93.6	3	6.4	0.96±0.04
DZ twins	27	79.4	7	20.6	0.7±0.12

Abbreviations: TM, torus mandibularis; MZ, monozygotic; DZ, dizygotic.

analyzing plaster casts, because atypical bruxism facets on plaster casts show a sum of all diurnal and nocturnal parafunctional activities retrospectively in all life periods (26). For this reason, plaster casts analysis is more reliable and objective assessment than self-report of the grinding activity (29). However, calibration of examiners and standard methods for assessment of bruxism has extremely important role in this regard (28). After training and calibration for standard bruxism assessment inter-rater agreement (Kappa) and ICC between two examiners were 0.87 and 0.87 respectively.

Obviously, there are some limitations in this regard. Tooth wear may not be evident in all cases of bruxism and depends on the type of bruxism. Atypical, eccentric wear facets are usual for teeth grinding. On the other hand, teeth clenching and gnashing may be difficult to recognize evaluating plaster casts. In fact, our study identifies bruxism only as parafunctional teeth grinding. Therefore, extended studies analyzing influence of teeth clenching and gnashing on the etiology of TM are necessary. Furthermore, teeth grinding is more common for young people (2) and our study sample average age was 20.3±0.9 years.

Other clinical variables

Some authors hypothesized that formation of TM may be related to negative teeth inclination or deep buccal overjet in Angle II Class. On the other hand, when direction of the bite force vector is changed (e.g. in Angle Class III) TM may not be prominent (19). Nevertheless, our study does not confirm TM dependency on Angle Class. Moreover, our results show that anterior open bite in habitual occlusion does not decrease TM incidence rate.

However, significant association between negative or flat CW in the maxillary premolars and first molars was found. The degree of curvature of the CW controls the inclination of occlusal guidance and occlusal contact point orientation (40). In case of negative or flat CW inclination, the occlusal guiding path becomes steeper, and it often leads to group function and determines greater occlusal load on teeth (40, 41). In addition, maxillary premolars and molars with the negative CW usually have negative torque and dominant occlusal contact in static occlusion on vestibular slope of vestibular cusps of mandibular premolars (A contact) (42). This finding supports functional matrix hypothesis (19, 21) as shear load and lingual buckling of the mandibular teeth initiates TM formation.

Multifactority

Scholars agree that the potential causes of TM are not limited to only one factor (13-15, 43). High concordance values for the presence or absence of TM between first and second twin in MZ bruxers pair indicate multifactorial etiology of TM. Our results suggest that TM formation could be associated with parafunctional teeth grinding, but genes may play predisposition role in TM etiology.

Higher, but statistically insignificant TM occurrence rates for bruxers with negative or flat CW in the region of maxillary second premolar and first molar may be explained by multicollinearity. In this case, two separately significant variables are highly correlated, and are insignificant conducting multiple logistic regression analysis (44). In our study high multicollinearity between negative or flat CW and bruxism was proved by *Spearman's rho* correlation coefficient ($r=0.2$, $p=0.009$), and significant correlation between the same variables was confirmed by logistic regression analysis (OR=2.742, 95% CI (1,27-5.92), $p=0.01$). On the other hand, as it was discussed before, negative or flat curvature of CW may provoke lingual buckling of the mandibular teeth in static occlusion or normal group function, and this occlusal overload is not related to parafunctional teeth grinding.

The formation of TM seems to be a compensatory and protective bone reaction to occlusal overload in the most vulnerable mandible area. This study leads us to a discussion about tori removal and their possible use for autogenous bone grafting for dental implantation (45, 46). Moreover, some authors report recurrent growth of TM following their removal due to repetitive bruxism (26).

Apparently, gene effects on the morphologic level are pleiotropic (13). Moreover, bruxism becomes almost habitual function for contemporary patients (47). Therefore, the search for other clinical and occlusal variables may play an important role analyzing and understanding TM etiology. Further clinical investigations, retrospective computer tomography studies or finite-element analysis evaluating correlation of TM and other clinical and occlusal variables may give valuable knowledge for implant angulation, fabrication of teeth or implant supported posterior restorations, and occlusal adjustment.

CONCLUSION

Our results suggest that the etiology of TM is multifactorial. TM seems to have genetic predisposition, and may be associated with teeth grinding as well as with negative or flat CW in region of maxillary second premolar and first molar.

STATEMENT OF CONFLICTS OF INTEREST

The authors state no conflict of interest.

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