Correlated characteristics of the jaws: association between torus mandibularis and marginal alveolar bone height

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The factor marginal alveolar bone height and torus mandibularis (TM) were studied in 2 groups of dentate patients more than 20 years of age, altogether 571 individuals. Subgroups possessing TM had the higher prevalence of unimpaired bone height as compared with those without the trait ($P < 0.001$). Different environmental factors considered, such as food habits, exposure to infection, oral hygiene habits, bruxism, access to professional dental care, and habits as to seeking dental treatment, did not seem to account for or to have any influence on the observed phenotypic correlation between TM and more favorable bone height. It was therefore suggested that TM and marginal bone seem to be influenced by common pleiotropic genes. It is hypothesized that future disclosure of the loci that regulate the capacity to develop TM at the same time may throw some light on the genetic contribution and mechanisms that tend to make the marginal alveolar bone more resistant to destructive agents. □ *Anatomy; osteology; periodontology*

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Periodontal diseases involve a series of symptoms such as gingival color and texture alterations and bleeding on probing to the base of the pocket. The decisive threat to the tooth, however, is destruction of the periodontal ligament fibers and the bone into which they are inserted. It is now recognized that the susceptibility to such destruction is individually variable and partly under genetic control: some persons are at relatively high risk of being dentally handicapped through loss of teeth, whereas others have a greater capacity to maintain the integrity of the tooth-supporting tissues when exposed to similar adverse environmental influences (1). There is growing evidence that individuals with significant periodontal destruction represent a subfraction of the adult population that is similar in many parts of the world, 7–15% (2–5). Furthermore, a recent study indicated that the proportion of individuals at risk did not seem to have changed during the past 3–4 decades (6). Therefore, the detection of the genetic mechanisms that contribute to make the alveolar bone resistant to destructive agents seems to be an important objective for study.

Most of the genes that have so far been identified by the techniques developed during recent years are responsible for simple Mendelian conditions (7). However, almost every common 'character' or disease that varies in size or degree is multifactorial in etiology—that is, influenced by gene differences at many loci plus environmental factors (8). Recombinant DNA technology is not immediately so useful for disclosing the gene loci involved in such conditions as it is for characters transmitted by single genes (9). Various methods for investigating the role of host genotype in the variation of susceptibility to periodontal breakdown have been used, such as family studies, studies of an association between the disease and a known genetic marker, periodontal pathology in inherited disorders with more widespread effect, and animal models (1).

Even torus mandibularis (TM), a bony outgrowth on the lingual surface of the mandible, has been found to be partly determined by heredity (10). On the morphologic level gene effects are almost without exception 'pleiotropic' (11); that is, they have more than one phenotypic outcome and thus

tend to produce correlated characters. Correlation, therefore, is of interest as far as the characters are influenced by the same genes (12). Eggen & Natvig (13) observed some association between the presence of TM and more adequately developed arches. The purpose of the present study was to investigate the alveolar bone height among individuals with and without TM.

Materials and methods

The two study groups consisted of dentate individuals more than 20 years old who on their own initiative reported for routine dental treatment at a private practice (6, 10, 14). One group consisted of 245 patients treated for bruxism, and a comparison group was composed of 326 consecutive patients. All participants were indigenous to a narrow district in the Gudbrandsdal Valley defined as Lillehammer and the surrounding rural municipalities. The sex and age patterns of the groups did not differ significantly (14).

Estimates of clinical and morphologic variables may differ from one population to another. During the 1960s and 1970s Norway changed from being an emigration country to an immigration country (15). However, besides casual internal migration no regular immigration of foreign ethnic groups into Gudbrandsdal had been reported at the time of the investigation (16). The study groups were therefore believed to carry similar genetic predispositions to the variables studied, TM and alveolar bone height. None of the patients had subjective problems related to these variables, and none was seeking treatment for such problems. Consequently, there seemed to be no particular reason to believe that the present groups were systematically different from the individuals of corresponding blocks in the resident population with regard to the conditions discussed.

Torus mandibularis

In the bruxism group TM was measured on study models and classified into three size categories as previously described (10, 14). In the comparison group the character was assessed clinically by visual inspection and digital palpation and classified on the basis of the experience from the study model measurements. Small thickenings recognized by palpation, but not discernible by sight, were regarded as normal lingual outline (no torus) in both groups. Comparison of 72 double clinical determinations made at intervals of more than 7 days and of 74 assessments made both on study models and clinically showed no statistically significant difference (14). Most tori were of the small category; large tori made up only 6–7% of observed tori in both groups. During the analysis TM therefore was considered either present or absent. There was no significant difference in prevalence between men and women, and no increase with age in either of the groups. TM was, however, more frequent in the bruxism group than in the comparison group ($P < 0.001$) (14).

Marginal alveolar bone height

In the bruxism group the interdental bone level, estimated by the linear distance between the cementoenamel junction (CEJ) and the adjacent bone support, was examined on full mouth periapical radiographs. Approximate parallelism was obtained by the insertion of cotton rolls. Two basically different measures have been used to quantify this distance: absolute values (in millimeters) and relative values—that is, percentage of the root length or tooth length (17). It was recognized that absolute measurements would have required a filmholder for standardizing the angle and distance variables, to minimize the variation in geometric distortion of the image (18, 19). However, no convenient device was at my disposal at the time of the investigation, so a relative assessment procedure, modified from the method used by Stahl et al. (20), was applied. Bone level within the coronal quarter of the root length was considered intact or compatible with a reasonably good state among adults and recorded as normal; bone level situated within the second quarter of the root was considered moderate bone loss; and bone level in the apical half as

severe bone loss. Most bone/root relations determined the diagnosis of each entire set as either normal, moderate bone loss, or severe bone loss.

In the comparison group the bottom of the clinical pocket was estimated mesially and distally on each tooth with a graded probe at the first visit, and all patients had an overall clinical diagnosis as either normal, moderate periodontal destruction, or severe destruction. The degree of gingival inflammation was not scored. Optimal attachment level, mainly observed among individuals aged 20–29 years, were recorded as normal without being radiographed. In 107 cases of doubt as to classification the patients were radiographed in addition to the clinical diagnosis. Analysis of the radiographic series was performed on a horizontal viewbox without knowledge of the clinical estimate and was repeated at intervals of 1–6 months without knowledge of the first assessment.

Tests of the data showed no significant difference between the first and the second radiographic assessment (6) and no significant difference between the clinical and radiographic diagnosis 'normal'. There was, however, some discrepancy between the radiographic and clinical diagnosis 'severe destruction'. The clinical estimates were then adjusted in accordance with the radiographic assessment, thus relating the diagnosis to bone height also in the comparison group.

Individuals showing severe bone loss were few (Table 1). During the statistical analysis the individual bone level state was therefore classified either as 'normal', defined as bone level within the coronal quarter of the root length on most of the teeth, or as 'bone loss present', defined as bone level situated apically of the coronal quarter of the root on most of the teeth. The chi-square test was used to test differences in prevalence of bone level classes, accepting a probability level of $P \leqslant 0.05$ as statistically significant.

Results

There was no significant difference between men and women as to bone level status in either of the groups; the sexes therefore were combined. The prevalence of bone loss present appeared to be higher among individuals with bruxism than among those of the comparison group (Table 1), both among younger adults ($P < 0.01$) and older individuals ($P < 0.01$), enabling combination of age classes but making it necessary to analyze the groups separately. Each of the groups was then divided into two subgroups in accordance with the presence or absence of TM, and bone level status among individuals with and without the trait was examined.

Individuals with TM had a higher prevalence of the diagnosis normal alveolar bone height than those without the trait at the same level of significance ($P < 0.001$) in both patient groups (Table 2).

Discussion

The validity of the method

The coronal level of the tooth-supporting tissues is usually situated at some distance from the CEJ (5), but there is no generally accepted standard against which to estimate

Table 1. Prevalence of bone level categories by age

	Comparison group				Bruxism group			
Age, years	*n*	Normal, %	Moderate bone loss, %	Severe bone loss, %	*n*	Normal, %	Moderate bone loss, %	Severe bone loss, %
20–39	205	90.7	7.3	2.0	136	80.1	16.1	3.7
40–70	121	68.6	24.0	7.4	109	50.5	38.5	11.0

Table 2. Prevalence of normal bone level among individuals with and without torus mandibularis, sexes and age classes combined

	Comparison group			Bruxism group		
	n	Percentage with normal bone level	*P* value	*n*	Percentage with normal bone level	*P* value
Individuals with torus mandibularis	102	93.1	<0.001	121	85.1	<0.001
Individuals without torus mandibularis	224	77.7		124	49.2	

whether the bone height is intact or impaired or, rather, 'normal' versus 'diseased' in terms of bone loss. Different epidemiologic studies have used different criteria, and direct comparison of observations from one investigation to another may be difficult. There are, however, two Swedish studies with which the present method to a certain extent may be compared, both investigating strict random samples (2, 21). In the study by Salonen et al. (21) the bone level was considered to be intact when 80% or more of the root was supported by bone. The present method implied that bone covering 75% or more of the root was considered as optimum bone level among adults and recorded as 'normal'. Hugoson & Jordan (2) also used the classification 'normal' but without any specific definition. Table 3 gives an outline of corresponding age groups in the three studies. It appears that in the age group 20–39 years the frequency of optimal bone level was virtually identical, both in the two Swedish samples and in the present comparison group. This seems to sustain the validity of the present observations. Consequently, approximately 9% of the individuals of the three samples seemed to have impaired bone levels before the age of 40, in accordance with the criteria used. This estimate is in agreement with the estimates for periodontal high-risk groups in many populations, 7–15% (2–5).

Severe periodontal destruction was rare or absent under the age of 40 in these studies, although the criteria used by Hugoson & Jordan (2) did not lend themselves to direct comparison. However, the prevalence of advanced bone loss between 40 and 70 years of age in the present comparison group (Table 3) did not seem incompatible with that of Salonen et al. (21). Furthermore, both prevalences are within the boundaries of worldwide estimates for periodontal high-risk groups. Thus the present study seemed to show similar population patterns of susceptibility to periodontal destruction as have previously been described when using more detailed methods of analysis.

Bone height and torus mandibularis

Phenotypic correlation between characters in adult individuals may be the result of either common genetic control or of common environment influence, or a combination of both (12). Dietary habits have been shown to be important environmental factors affecting oral tissues (16, 22). A nationwide survey of food habits in Norway (23) concurrently with the present investigation showed differences between geographic areas and occupational groups. Residence, however, seemed to have a greater influence than economy, implying that dietary habits were likely to be similarly distributed among individuals living in the same area. Since all participants of the study were indigenous to a narrow district, they were assumed to subsist on similar types of food items on the average. Additional environmental factors expected to be of importance for the periodontal health were exposure to infection, oral hygiene habits, access to professional dental care, and habits as to seeking dental treatment. The patient groups were considered to have similar probability of being influenced by these variables too, and this circumstance seemed to be

Table 3. Distribution of marginal alveolar bone heights adapted from published data

Age, years	Bone level state	Ref.	Criteria	Unit	*n*	Prevalence (%)
20–39	Optimum	(2)	No reduction of 'normal' alveolar bone height	Individual	200	90.5
20–39	Optimum	(21)	Alveolar bone height/root length ratio ≥80%	Site	17,553	91.5
20–39	Optimum	*	Alveolar bone height/root length ratio ≥75%	Individual	205	90.7
40–69	Advanced bone loss	(2)	Not comparable			
40–69	Advanced bone loss	(21)	Alveolar bone height/root length ratio ≤59%	Site	13,178	10.3
40~70	Advanced bone loss	*	Alveolar bone height/root length ratio <50%	Individual	121	7.4

* Present study, comparison group (Table 1).

independent of the presence or absence of TM. Consequently, neither of the above non-genetic factors seemed to account for the observed difference in marginal bone height between persons with and without TM (Table 2).

The only known environmental variable making the study groups systematically different was clenching and grinding habits. The bruxism group was considered to be uniform, whereas the comparison group was mixed (10). Muscular forces during bruxism exceed the forces of mastication and may lead to periodontal trauma (24). The etiology of the habit has been subject to controversy, but a close relationship between psychic stress and bruxism has been observed by most students of the condition (24, 25).

The role of traumatic occlusion in the etiology of periodontal destruction is highly controversial (24, 26, 27). In the present patient material bone loss appeared to be more frequent in the bruxism group than in the comparison group (Table 1). It is noteworthy, however, that not even the environmental factor bruxism seemed to have any influence on the observed connection between alveolar bone level and TM, since the analysis showed the same relationship in both patient groups (Table 2). Self-consistency of estimates obtained among different groups is an important indication of the validity of the observations (28). It therefore seemed plausible that the observed phenotypic correlation between TM and more favorable marginal bone state to a noticeable extent may be a result of common genetic control.

So far, little is known about the genes that control development (12). It has been suggested, however, that as the mapping of the human genome progresses, the genes contributing to the more common morphologic characters, which are usually of multifactorial origin, will also be discovered and characterized (7).

With the development of the functional matrix principle it became reasonably certain that bone does not regulate its own growth. The genetic and epigenetic determinants are in the functional soft tissue matrix, not within the cells forming the bone. The control of growth and resorption remodeling is essentially a system of feedback pathways, informational interchanges, and reciprocal responses between the soft tissues and the bone (29, 30).

Different approaches to the detection of periodontal high-risk groups and individuals, such as laboratory markers from peripheral blood, saliva, gingival crevicular fluid and subgingival bacterial plaque, have been proposed (4). It has been suggested that an 'antiantibody' first described by Waaler (31), today known as the rheumatoid factor, may contribute to chronic periodontal inflammation and loss of bone and that patients with rheumatoid arthritis may be regarded as a risk group (32). On the basis of the present observations it is hypothesized that future disclosure of the kind of loci that

regulates the capacity of TM formation at the same time might provide some indication of the genetic mechanisms that contribute to make the alveolar bone more resistant to destructive agents.

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