Eggen S, Natvig B, and Gåsemyr J. Variation in torus palatinus prevalence in Norway.

Abstract. The variation of torus palatinus (TP) prevalence was investigated among altogether 2010 dental patients over 10 yr of age indigenous to two different regions in Norway, the Lofoten Islands in North Norway, and the Gudbrandsdalen Valley, an inland area in the south-eastern part of the country. The two groups were presumed to belong to the same Caucasian stock, but were extremes with regard to the fish-to-meat ratio of the diet, being 3:1 in Lofoten and 1:3 in Gudbrandsdalen, representing a soft type of diet, and a tough type requiring greater muscular forces during mastication respectively. The study revealed that 1) the prevalence of TP increased among women compared to men (P < 0.001), 2) TP seemed likely to arise from a multifactorial liability, with part of the genetic factors residing on the X chromosome, 3) the prevalence of TP appeared to increase among individuals native to Lofoten, consuming the softer food, compared to those living in Gudbrandsdalen (P < 0.05), an increase being hypothesized to have some connection with nutrient substances present in saltwater fish, possibly $\Omega 3$ polyunsaturated fatty acids and vitamin D, 4) TP seemed to be a dynamic phenomenon capable of growth and subject to resorption remodelling, 5) torus palatinus and torus mandibularis appeared to exhibit different patterns of occurrence when related to sex, masticatory stress and age of onset, indicating that the two types of tori do not directly seem to be biologically equivalent characters wholly sharing a common morphogenetic background.

Torus palatinus (TP), a non-pathologic bony overgrowth in the mid-palatine region first described in 1814 by Fox (1), is the most common of the "tori" of the human jaw skeleton, but it has been the subject of as much controversy as has torus mandibularis (TM) (2). However, the long-established polarization of views about whether the causes of variation are either environmental or genetic has outlived any dialectic purpose, since virtually any trait is the result of the combined action of genetic and environmental factors (3,4).

Most reports on the occurrence of TP are from American, Mongoloid and Eskimo populations, with fewer papers from European communities. The present study groups were constituted by altogether 2010 dental patients indigenous to two different geographical regions in Norway, the Lofoten Islands and the Gudbrandsdalen Valley. The purpose was to examine the variation of TP in relation to sex, age, residence and diet; and, in an appendix, to compare the phenotypic behavior of TP and TM.

STUDY POPULATIONS AND METHODS

Consecutive patients reporting for dental treatment in two private clinics were examined during the second half of 1954 and the first half of 1955 (Lofoten), and during the second half of 1955 and the first half of 1956 (Gudbrandsdalen). Lofoten, a fishing district in Nordland County of North Norway, is made up by a row of islands piercing the Norwegian Sea at 68° latitude. The North Atlantic Drift carries warm water to the shores, and westernly winds blowing inland across the water provide a more favourable climate than is commonly found at that latitude. Gudbrandsdalen was defined as the town Lillehammer and the neighbouring rural municipalities, representing an inland district in the southeastern part of the country in the County of Oppland, situated at 61° latitude (Fig. 1). Recent residents and persons under 10 yr of age were not included in the study. A series of clinical variables was recorded in the dental chair by one examiner (S.E.) on a specially prepared form.

TORUS PALATINUS (TP)

Shape

The existence of a TP was ascertained by visual inspection and palpation, and the shape was classified as either flat, spindle, or irregular (nodular, lobular or 3-bladed). The flat and spindle types were responsible for nearly 98% of observed tori. During the analysis no distinction was made as to the form of TP.

Size

The maximum elevation was applied as the determining criterion for the size of TP, usually agreeing with the length and width (5). No actual measurements were performed during the assessments, but on the basis of previous training on stone models obtained from accurate impressions the size was estimated according to 3 categories: 1) small, torus elevation less than 2 mm, 2) medium, elevation between 2 and 4 mm, and 3) large, elevation 4 mm or more.

Altogether, 725 patients were found to possess a TP, of which only 4 persons had large torus (0.5% of affected), and 62 (8.5%) had medium torus. The majority of affected, 659 (91%) had small tori. Because of the low frequency of the large and medium expressions TP was regarded as either present or absent during the statistical analysis.

Persons with TP were hardly aware of its presence. It was of no worry and none was seeking treatment for the condition. It therefore seemed to be no particular reason to believe that the patients within a certain sex-place-age block were systematically different from the individuals in the corresponding block in the resident population with regard to the presence of TP.

STATISTICS

The material was divided into 20 sex-place-age blocks. Let X_{ijk} denote the number of persons of sex i from place j and within age class k with TP out of the total number n_{ijk}

of persons within this block. Here i = 1 (i = 2) corresponds to men (women), j = 1 (j = 2) to Gudbrandsdalen (Lofoten) whereas k = 1, 2, 3, 4, 5 correspond to the age classes 10-19, 20-29, 30-39, 40-49 and 50~70 respectively. Furthermore, let p_{ijk} be the probability of having TP for a person of sex i from place j and within age class k. We assume X_{ijk} to be binomially distributed (n_{ijk}, p_{ijk}) for all 20 blocks.

To analyse the effect of sex, place, and age on the prevalence of TP we used logistic regression (6). The models were fitted using the statistical package GLIM (7).

More specifically, we used a model which relates p_{ijk} to the factors via the logit function:

$$logit(p_{ijk}) = log(p_{ijk}/(1 - p_{ijk})) = \mu + \alpha_i + \beta_j + \gamma_k$$
$$i = 1, 2; \ j = 1, 2; \ k = 1, 2, 3, 4, 5$$

The analysis revealed that there was no significant difference between the three age classes 20-29, 30-39, 40-49 (P > 0.25). Hence we divided the material into just three new age classes k = 1, 2, 3 corresponding to 10-19, 20-49, 50 \sim 70 (Table 1). We then used the logit function

$$logit(p_{ijk}) = \mu + \alpha_i + \beta_j + \gamma_k, i = 1, 2; j = 1, 2; k = 1, 2, 3$$

Omission of any of the addends α_i, β_j , and γ_k gave a statistically significant poorer fit in the goodness of fit statistics. There were no significant interaction effects between the factors above.

RESULTS

The prevalence of torus palatinus was significantly greater among women than among men, the χ^2 -statistic with 1 degree of freedom being 34.63 (P < 0.001). Furthermore, it was greater in Lofoten than in Gudbrandsdalen on a level of significance P < 0.05. The prevalence increased among persons in the age class 20-49 compared to the one below and decreased again in the age class 50~70 (P < 0.001).

DISCUSSION

Prevalence of torus palatinus and sex

The higher prevalence of TP among women than among men in the present study groups (P < 0.001) is in accordance with previous observations in different living populations (8-15) as shown in Table 2. It appears that the women/men ratio does not seem to be influenced by whether the prevalences are high or low. The ratio turned out to be 1.7:1 on the average, ranging between 1.3:1 and 2.3:1. Consequently, the data from the present and previous studies indicated that TP seems to be sex-linked.

Suppose that TP is determined by genes at a single locus on the X chromosome. Then there are two alternatives: X-linked recessive or X-linked dominant transmission. A characteristic for X-linked recessive inheritance of a trait is that it typically occurs in males only (3); consequently this pattern must be ruled out. Thoma (16) suggested an X-linked dominant pattern for TP. A feature of X-linked dominants is that they are approximately twice as common in women as in men (3). This condition does not seem incompatible with the empirical data given in Table 2. However, the chief characteristic of X-linked dominant inheritance is that an affected male transmits the gene (and the trait) to all of his daughters and to none of his sons (3); but pedigree charts of family studies on TP show that this feature is not met (9,17). From this it follows that TP cannot be explained by the X-linked dominant single-gene model either.

It is suggested that TP, like TM (18), can be explained in terms of an underlying continuity in the population, "liability", with a threshold value above which individuals will be affected (4). This hypothesis is in accordance with the opinion of Haugen (8). Liability is defined as having a certain genetic attribute, most often polygenic, which in practice always is accompanied by environmental effects on individual variation (19). The combination of genetic and environmental factors makes the system "multifactorial" (20). It is suggested that at least part of the genetic determinants of the liability to TP resides on the X chromosome. This conclusion is similar to the hypothesis proposed by Kari & Alvesalo (9).

Age Change

In the present patients aged 10 yr and over the prevalence of TP increased from the second to the third decade of life in both sexes and both localities at which time it reached a plateau through 20-49 yr of age, again followed by a decrease in the age class $50\sim70$ yr (P < 0.001). This pattern of variation with age is similar to those appearing in previous studies of living populations (8, 9, 13-15), indicating that TP, like TM (21), seems to be a dynamic character, capable of growth and subject to resorption remodelling.

Prevalence and residence

By definition, the difference in prevalence of a threshold trait from one population to another may be due to different gene pool or different environmental influences, or a combination of both. In a discussion of the ethnical descent of the two present patient populations it was concluded that they seemed to be of the same Caucasian stock (21). The strain of people of Lapp and Kven (Finnish) origin, being more or less predominant on some districts of the northernmost counties, was presumed to be negligible in Lofoten (22,23), and absent in Gudbrandsdalen (21). During the 60s and 70s Norway changed from being an emigration country to an immigration country (24). The present investigation was, however, performed prior to this new era. Beside casual internal migration no regular immigration of foreign ethnic groups was found to have been reported in either of the two localities at the time of the examinations, making the two patient groups likely to carry the same genetic predisposition to TP on the average. The higher prevalence of TP in Lofoten than in Gudbrandsdalen (P < 0.05) was therefore presumed to be attributable to non-genetic factors.

Nevertheless, the prevalence estimates for TP were high in both groups; admittedly, somewhat lower than those among residents of the Island of Hailuoto, Finland (9), but being of the same order of magnitude as those observed among U.S. citizens of European, mainly British and German extraction (11) (Table 2). In comparison, the figures given by Haugen (8) obtained among Norwegians living in the Oslo area may seem strikingly low (Table 2). A remark may be added to the problem pertaining to the comparison of prevalence

estimates for TP from one study to another. There may be uncertainty as to the demarcation between the initial grades of the condition and its absence, and it is possible that the method applied by Haugen may have been more exclusive than ours. In addition, Haugen (8) suggested that there may be a decreasing tendency of torus formation in the Norwegian population. Confessedly, there is a span of time of approximately 30 years between our collection of data and that of Haugen. However, the difference is so great that it seems to reveal a real disparity between the provinces and the capital as regards the prevalence of TP. Actually, the prevalences observed in the Oslo area are more or less compatible with those found in Rostock, Germany (10) and in London, United Kingdom (11) (Table 2).

Prevalence and diet

It has repeatedly been suggested that an important factor influencing the prevalence of the tori is diet (11, 12, 25-27). The most popular theory on causation of the variation of TP is that its presence is triggered by pressure toward the median palatine region from mastication of tough food, and that the thickening of the palatine vault on each side of the median palatine sutur acts as a buttress to resist the pressure (25).

In a previous study (21) the present patient groups were described as being extremes with regard to the fish-to-meat ratio of the diet, nearly 3:1 in Lofoten and less than 1:3 in Gudbrandsdalen (28). The large amount of fresh fish consumed in Lofoten rendered the diet a rather soft type, whereas the diet in Gudbrandsdalen was characterized as a tough type requiring greater muscular forces during mastication because of the great proportion of meat. Much of the meat selected for hot meals in Lofoten consisted of mutton, whereas a large part of that consumed in Gudbrandsdalen consisted of pork, including cured pork served uncooked (21).

The present observation did not exclude masticatory function as a factor contributing to the development and maintenance of the presence of a TP; but the "hard chewing" theory suggested by Hooton (25) did not seem capable of explaining the higher prevalence of TP among the group native to Lofoten (P < 0.05) consuming the softer food. It therefore seemed that the explanation should be searched for among nutrients with impact upon the

very development of bone tissue.

As to the proportional contribution of most nutrients from foodstuffs other than fish and meat the calculations made by Øgrim & Homb (28) revealed marked similarities among different groups of the people. Consequently, the only clear dietary inequality between the two regions seemed to be the much greater consumption of saltwater fish in Lofoten.

The biochemistry of skeletal growth is complex, involving a chain of regulatory links. The program for bone morphogenesis does not reside in the cells forming the bone, but in adjacent tissues with which they interact, bone cell differentiation and bone development being initiated by the growth factors (cytokines) bone morphogenetic proteins (BMP) (29). Most of the MBP proteins so far identified have been found to be members of the transforming growth factor β (TGF β) family of growth factors (30,31).

To control the complex production of proteins the cells are equipped with hormonlike signal substances, autacoids (32). The base substance for the production of all autacoids is the essential polyunsaturated Ω 6 fatty acid linoleic acid, the main component of vegetable fat (33). (The term essential fatty acids (EFA) implies that these important acids cannot be synthesized de novo in the human organism, but must be present in the diet.) During recent years it has been established that an adequate production of the various signal substances seems to be regulated by the ratio of the Ω 6/ Ω 3 fatty acids (33). Ω 3's are also important for growth and developmental processes (33). This class of fatty acids is derived from the essential α -linolenic acid, and is the main component of fat from fish and other marine animals.

There are still facts unknown concerning the actual mechanisms. However, since the two classes of EFA, Ω 6 and Ω 3, constitute a significant part of practically all cell membranes (33) it does not seem unreasonable to hypothesize that they may be involved even in the production of BMP's and in the development and growth of bony characters such as TP. Fish has a rich supply of the Ω 3 eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the Ω 6/ Ω 3 ratio of codfish being 0.11:1 (33). Mutton has an acceptable ratio 4.8:1, whereas bacon has an unfavourably high ratio, 12.7:1 (33).

Furthermore, it is established that vitamin D participates in the intestinal absorption of calcium, that it participates in the skeletal metabolism, and that its main natural source is fish liver. On the basis of the present observations it is hypothesized that the higher prevalence of TP in Lofoten compared with Gudbrandsdalen (P < 0.05) could have some connection with essential substances present in saltwater fish, possibly vitamin D and $\Omega 3$ polyunsaturated fatty acids.

APPENDIX

Torus palatinus versus torus mandibularis

Interest has been focused on the question whether TP and TM are manifestations of the same morphogenetic phenomenon or represent separate biological units; the findings are, however, controversial (8). In the following some comments are given on the pattern of occurrence of the two characters, when related to some defined variables.

Sex

In a previous analysis of the present patient groups TM appeared to be more frequent in men than in women (21). This is in accordance with observations in a couple of other studies of living populations (8,34). However, some authors have found the reverse (14,35), whereas the majority of investigators did not observe any significant difference in prevalence of TM between men and women (13, 15, 36-38). Consequently, in contrast to TP (Table 2), the sexual pattern of occurrence of TM is variable, and does not indicate any X-linkage of the trait. (Virtually, no common human anomaly is Y-linked (3).)

Masticatory stress

In the previous study (21) the prevalence of TM was found to increase among individuals in Gudbrandsdalen compared to those in Lofoten, an increase mainly being presumed to be ascribable to the mastication of the tougher type of food in Gudbrandsdalen. The present analysis of the variation of TP in the same groups revealed an opposite pattern:

individuals native to Lofoten, consuming the softer food, exhibited the higher prevalence. This indicated that the developmental process of TP to a lesser degree seemed to be sensitive to masticatory stress than TM, implying that other factors, possibly nutrient substances present in saltwater fish, may have come into play during the development of TP.

Age of onset of the tori

According to the literature TP does not infrequently appear in the first decade of life, and may even be present at birth (5, 9, 10, 12-15). Once more the phenotypic pattern of occurrence of TP seems to be dissimilar to that for TM, which character typically begins to manifest itself only at the beginning of puberty onward (2, 26). In conclusion, the above conditions indicated that TP and TM do not directly seem to be biologically equivalent characters wholly sharing a common morphogenetic background.

Inheritance

Even the "heritability" of liability to threshold traits, that is, the proportion of the total phenotypic variance ascribable to genes, has been found to be influenced by the time of onset of the traits. Whereas heritability is high for conditions that are present at birth such as cleft lip and cleft palate, 70-90%, it may drop to 30-40% for traits developing later in life (20,39,40), because of more opportunity for environmental factors to influence liability (20). The heritability of liability to TM has been estimated at approximately 30% (18). Since TP has an earlier onset, sometimes even prenatal, the heritability might be expected to turn out somewhat higher than for TM. So far this problem has not been studied. Further familial investigations seem desirable.

REFERENCES

- 1. Fox J. Natural history and diseases of teeth. London, 1814; 135.
- 2. Ossenberg NS. Mandibular torus. A synthesis of new and previously reported data and a discussion of its cause. In: Cybulski JS, ed. Contribution to physical anthro-

- pology 1978-1980. Ottawa: National Museum of Canada, 1981; 1-52.
- Thompson J, Thompson MW. Genetics in medicine. 3rd ed. Philadelphia: W.B. Saunders Co, 1980; 3, 69-76.
- 4. Falconer DS. Introduction to quantitative genetics. 2nd edn. London: Longman, 1985; 314.
- 5. Woo J-K. Torus palatinus. Am J Phys Anthrop 1950; 8: 81-111.
- Freeman DH Jr. Applied categorial data analysis. New York: Marcel Dekker, 1987;
 chapter 7.
- 7. The generalized linear interaction modelling (GLIM) system. Oxford: Numerical algorithms group (NAG), 1986; Release 3.77. Manual.
- 8. Haugen LK. Palatine and mandibular tori. A morphologic study in the current Norwegian population. Acta Odontol Scand 1992; 50: 65-77.
- Kari M, Alvesalo L. A dental field investigation in Hailuoto. VI. Torus palatinus: incidence and some viewpoints concerning its inheritance. Proc Finn Dent Soc 1973; 69: 120-7.
- 10. Körner O. Der Torus palatinus. Zeitschr Hals Nasen Ohrenheilk 1910; 61: 24-9.
- 11. King DR, Moore GE. An analysis of torus palatinus in a transatlantic study. J Oral Med 1976; 31: 44-6.
- 12. Miller SC, Roth H. Torus palatinus: a statistical study. J Am Dent Assoc 1940; 27: 1950-7.
- Kolas S, Halperin V, Jefferis K, Huddleston S, Robinson HBG. The occurrence of torus palatinus and torus mandibularis in 2478 dental patients. Oral Surg 1953; 6: 1134-41.
- 14. Austin JE, Radford GH, Banks SD. Palatal and mandibular tori in the Negro. N Y State Dent J 1965; 31: 187-91.
- Schaumann BF, Peagler FD, Gorlin RJ. Minor craniofacial anomalies among a Negro population. Oral Surg 1970; 29: 566-75.
- Gorlin RJ, Goldman HM. Thoma's oral pathology. St. Louis. C.V. Mosby Co, 1970, Volume one; 45.
- 17. Suzuki M, Sakai T. A familial study of torus palatinus and torus mandibularis. Am

- J Phys Anthrop 1960; 18: 263-72.
- Eggen S. Torus mandibularis: an estimation of the degree of genetic determination.
 Acta Odont Scand 1989; 47: 409-15.
- Bodmer MF, Cavalli-Sforza LL. Genetics, evolution, and man. San Fransisco: W.H. Freeman and Co, 1976; 487-96.
- 20. Fraser FC. The multifactorial/threshold concept uses and misuses. Teratology 1976; 14: 267-80.
- 21. Eggen S, Natvig B. Variation in torus mandibularis prevalence in Norway. A statistical analysis using logistic regression. Community Dent Oral Epidemiol 1991; 19: 32-5.
- Qvigstad J. Den kvænske indvandring til Nord-Norge. Tromsø Museums årshefter
 1920; 43: No. 1; 46.
- 23. Qvigstad J. Sjøfinnene i Nordland. Tromsø Museums årshefter 1929; 51: No. 1; 35-40.
- 24. Haagensen E, Kvisler L, Birkeland TG. Innvandrere gjester eller bofaste? Oslo: Gyldendal Norsk Forlag 1990; 21.
- 25. Hooton EA. On certain Eskimoid characters in Icelandic skulls. Am J Phys Anthrop 1918; 1: 53-76.
- 26. Hrdlicka A. Mandibular and maxillary hyperostoses. Am J Phys Anthrop 1940; 27: 1-67.
- 27. Mayhall JT. The effect of culture change upon the Eskimo dentition. Arctic Anthropol 1970; 7: 117-21.
- Øgrim ME; Homb E. Kostvaner og næringstilførsel hos grupper av norske familier.
 Oslo: Universitetsforlaget 1960; 1-173.
- Urist MR, DeLange RJ, Finerman AM. Bone cell differentiation and growth factors.
 Science 1983; 220: 680-6.
- Wozney JM, Rosen V, Celeste AJ, Mitsock LM, Whitters MJ, Kriz RW, Hewick RM, Wang EA. Novel regulators of bone formation: molecular clones and activities. Science 1988; 242: 1528-34.
- 31. Centrella M, McCarthy TL, Canalis E. Skeletal tissue and transforming growth

- factor β . FASEB J 1988; 2: 3066-73.
- 32. Kinsella JE. Food lipids and fatty acids: Importance in food quality, nutrition, and health. Food Technology 1988; October: 124-43.
- 33. Skjervold H. Lifestyle diseases and the human diet. How should the new discoveries influence future food production. N-1432 Ås: Department of animal science, Agricultural University of Norway 1992; 1-48.
- 34. Alvesalo L, Kari M. A dental field investigation in Hailuoto. V. Torus mandibularis: incidence and some viewpoints connected with inheritance. Proc Finn Dent Soc 1972; 68: 307-14.
- Johnson CC, Gorlin RJ, Anderson VE. Torus mandibularis: A genetic study. Am
 J Hum Gen 1965; 17: 433-9.
- 36. Levesque RP. Torus mandibularis a clinical survey. Georgetown Dent J 1965; 31: 12-5.
- 37. Muller TP, Mayhall JT. Analysis of contingency table data on torus mandibularis using a log linear model. Am J Phys Anthrop 1971; 34: 149-54.
- 38. King DR, King AC. Incidence of tori in three population groups. J Oral Med 1981; 36: 21-3.
- 39. Falconer DS. The inheritance of liability to certain diseases, estimated from the incidence among relatives. Ann Hum Genet 1965; 29: 51-76.
- 40. Falconer DS. The inheritance of liability to diseases with variable age of onset, with particular reference to diabetes mellitus. Ann Hum Genet 1967; 31: 1-20.

Table 1. 2010 patients by residence, sex, and age, with prevalence rates for torus palatinus (TP).

Lofoten

Age, yr	$oldsymbol{n}$	With TP %	Women n	With TP %	
10-19	97	30.9	138	36.9	
20-49	371	35.0	441	46.0	
50~70	86	24.4	48	37.5	
Total	554(47%)	32.7	627(53%)	43.4	

${\bf Gudbrandsdalen}$

Age,	Men	With TP	Women	With TP	
yr	$m{n}$	%	$m{n}$	%	
10-19	109	15.6	122	28.7	
20-49	198	29.8	280	45.0	
50~70	55	18.2	65	38.5	
Total	362(44%)	23.7	467(56%)	39.8	

Table 2. Prevalence of torus palatinus (TP) in living populations, altogether 15156 individuals, by residence and sex, age classes combined. Dental patients if not otherwise indicated.

	Men		Women			
		Percent		Percent	Women/Me	n .
Population	n	with TP	n	with TP	quotient	References
Norway						
Lofoten	554	32.7	627	43.4	1.3	Present study
Gudbrandsdalen	362	23.7	467	39.8	1.7	Present study
Oslo area	2217	6.7	2783	11.2	1.7	(8)
Finland						
Island of Hailuoto (Karlö). Field stu	dy 328	37.8	339	56.9	1.5	(9)
Germany						
Rostock	584	7.5	525	15.8	2.1	(10)
United Kingdom						
London	100	12.0	100	15.0	1.3	(11)
United States						
Kentucky. Mainly of British						
and German extraction	100	25	100	42.0	1.7	(11)
New York	533	16.3	507	32.3	2.0	(12)
Ohio. Mainly Whites	1193	14.7	1272	26.7	1.8	(13)
Tennessee. Negro population	696	11.6	813	26.3	2.3	(14)
Washington D.C. Negro population	446	12.3	510	25.9	2.1	(15)
Total	7113	14.3	8043	24.2	1.7	

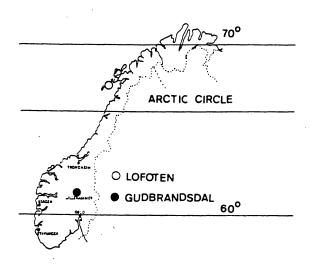


Fig. 1 Domicile of the study populations.