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ARTICLE / *ARTICLE*

Oral exostoses: An assessment of two hundred years of research

Les exostoses orales : bilan de deux siècles de recherches

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Abstract Oral exostoses, also called *tori*, are considered as non-metrical anatomical variants of the human skull. Although oral exostoses have been extensively studied in various materials and populations since they were first described, they are still a poorly understood anatomical feature. They vary in size and shape and their prevalence fluctuates according to the population, although no obvious population-based selection can be put forward. Their relationship with age and sex is still in debate and their etiology remains unknown. Despite this, they are often included in trait lists for assessments of distances between populations. Furthermore, there is a wide range of study protocols that may also account for the heterogeneity of observations. A detailed review of the literature is proposed here to assess current knowledge on oral exostoses and highlight new possibilities for their study.

Keywords Oral exostoses · *Torus mandibularis* · *torus palatinus* · Population variability · Incidence

Résumé Les exostoses orales, aussi dénommées *tori*, sont considérées comme des variations anatomiques non métriques du crâne humain. Bien qu'elles aient été largement étudiées depuis leur première description, sur différents matériels et différentes populations, elles apparaissent toujours comme une caractéristique anatomique mal comprise. Elles sont de forme et taille variables et leur prévalence fluctue entre populations, bien qu'aucune sélection populationnelle claire ne puisse être affirmée. Le lien au sexe et à l'âge est toujours débattu et leur étiologie reste à ce jour inconnue. Pourtant, les exostoses orales sont souvent utilisées en tant que caractères discrets crâniens dans l'étude des distances populationnelles. De plus, le large éventail de protocoles d'étude peut aussi expliquer l'hétérogénéité des observations. Ainsi, nous proposons une revue détaillée de la littérature pour faire le bilan des connaissances actuelles sur les exostoses orales et suggérer quelques nouvelles approches pour leur étude.

Mots clés Exostoses orales · *Torus mandibularis* · *Torus palatinus* · Variabilité populationnelle · Incidence

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Introduction

Oral exostoses, also known as *tori*, have been extensively studied for more than a century, in physical anthropology and in dentistry as well as other medical fields, but they remain a puzzling anatomical feature. Although their existence has been recognized since the beginning of the nineteenth century, the mandibular exostosis [1] was first described in 1884 and the palatine exostosis [2] in 1889. Since then, they have been the subject of numerous publications. The first studies focused on their anatomical description. Subsequent research mostly attempted to assess population variation and clarify etiological processes to answer one core question: are oral exostoses non-metrical cranial variations that could be used to assess distances between populations? However, their biological morphological significance is still not fully understood. They have not yet been identified in non-human primates, although a slight thickening on the lingual aspect of the mandible, resembling a mandibular exostosis, can be detected in the largest species such as gorillas [1], and a palatine torus in chimpanzee skulls [2]. In humans, they appear sporadically in the Palaeolithic. Two cases have been reported in Asian *Homo erectus* from Zhoukoudian (China) [3]. Some bony protuberances have also been identified on the *Homo heidelbergensis* mandible from Mauer (Germany) and in

Neanderthals, in particular on the Guattari 1 skull (Monte Circeo, Italy) [4] and on mandibles from Ehringsdorf (Germany), Spy and la Naulette (Belgium) [1]. Vallois [5] reports one possible exostosis in the Upper Palaeolithic modern human skull from Chancelade (France). In modern populations, their frequency and expressivity vary widely according to ethnic affiliation. They are considered as discrete anatomical variants of the jaws [6,7]. However, close examination of the abundant literature shows that the relationship with age and sex remains unclear, no population-based selection can be put forward with certainty and no definitive conclusion can be drawn as to etiology [8-14]. The latter observations are fundamental since cranial non-metric variants, including oral *tori*, have sometimes been included in trait lists to quantify distances between populations [15-19], on the assumption that their variation is representative of quantitative genetic differences. This hypothesis implies that non-metric variables do not vary significantly with sex, age and environment [20], which is yet to be proven for oral exostoses. Moreover, the disparities observed between studies could also reflect the extreme variability of study protocols [21]. This methodological aspect, although critical, is often ignored in the few reviews of the literature [8-14]. In order to use this particular variable in anthropological studies, we need a clearer understanding of its factors of influence and causation. Two hundred years after they were first described, what can be said about oral exostoses regarding their definition, clinical aspects, population-based variation, relationship with age and sex, possible etiologies and methodological considerations?

Definition

The general term of exostoses describes different types of benign, localized, bony protuberances, composed of both compact and cancellous bone arising from the local cortical bone [12]. When identified on jaws, they are often called *tori*, from the Latin word *torus* meaning "protuberance" or "outgrowth" [8-10], and consist of an enlargement of the bone tissue in different parts of the upper and lower jaws [6,7]. They all share the same histological structure: dense cortical bone and a limited amount of cancellous bone, coated with a thin and poorly vascularised mucosa [8-14]. They seem to be continuous with the surrounding bone, which explains the complexity of surgery in the event of removal. Similarly, they cannot be differentiated from the surrounding bone on X-rays or CT-scans [22-26].

Their discovery is usually asymptomatic and occurs incidentally during a routine clinical or anthropological examination. In rare instances, they prompt medical consultations: cancerophobia, significant growth causing ulcerations of the mucosa [27] or spontaneous exposure of the bone tissue below [28], alteration of the mobility of the tongue [22,29,30] or mastication [31], difficult endotracheal intubation [32,33] and hampering of prosthetic rehabilitation in edentulous patients [29]. The emergence of oral exostoses has also been observed following free gingival grafts [34,35], distraction osteogenesis procedures [36], orthodontic implant placement [37] and traumatizing blows [38], and has been recently associated with obstructive sleep apnoea disorder [39]. Surgical removal is prescribed for extremely large exostoses in cases of prosthetic rehabilitation, correction of bone defects of the jaws [40,41], management of sleep apnoea disorders [42] or maximization of periodontal surgery outcomes [43].

Various types of exostoses have been described according to their anatomical location (Fig. 1) :

- Palatine exostosis (PE), also referred to as palatine torus: a bony protuberance located on the median palatine suture;
- Mandibular exostosis (ME), also referred to as mandibular torus: a bony protuberance arising from the lingual aspect of the mandibular alveolar process and located between the mylohyoid line and the alveolar margin;
- Maxillary exostosis (MxE), also referred to as maxillary torus: a bony protuberance arising from the lingual aspect of the maxillary alveolar process, mostly in the molar area;
- Buccal maxillary and mandibular exostosis (BMxE and BMdE): a bony protuberance arising from the buccal aspect of the maxillary alveolar process, mostly in the molar area.

Most authors agree on these definitions [8-14]. Nevertheless, according to some researchers, a maxillary torus, for instance, refers to either irregular bony nodules of varying sizes or a mound-like thickening of the alveolar process at the buccal *and* the lingual sides [6,44]. Differences in definition might lead to differences in observed prevalence.

Oral exostoses *sensu lato* vary in shape, size and frequency according to population origin. Different types of exostoses can sometimes be observed in the same individual. However, although they display a similar layout, it is still unclear whether they are related to each other. The very denomination of *torus* maintains a sort of grey area implying that some of them are indeed linked [1,21], although they may also represent different biological units. Therefore, we chose to use the name "oral exostoses", a term that describes their nature but remains neutral.

Population variability

Both the prevalence and expressivity of oral exostoses vary widely between populations. Although no definite association with population has been established, their frequency is often

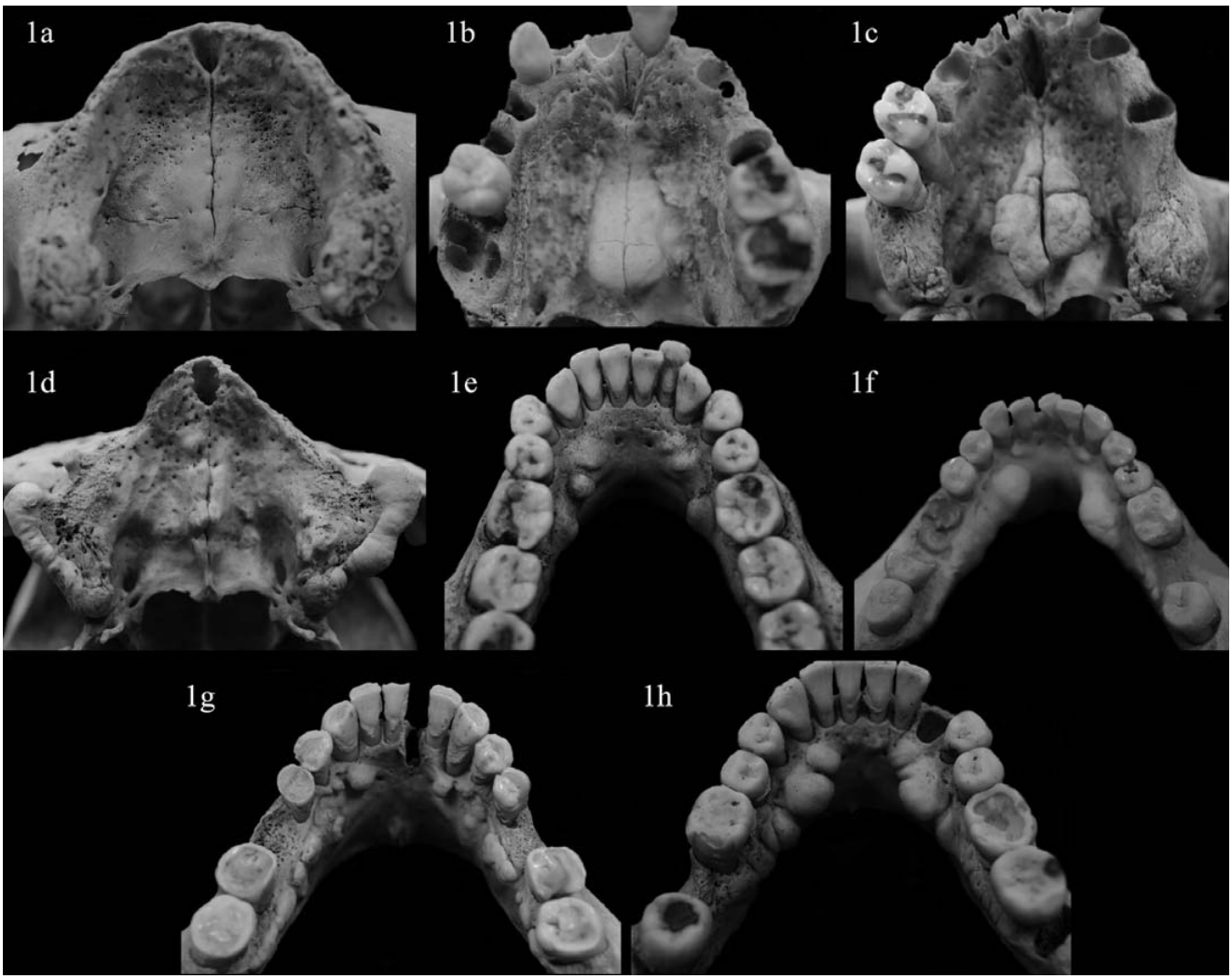

Fig. 1 Examples of palatine and mandibular exostoses. Photographs 1a to 1d show the different degrees of expression of palatine exostoses (PE). 1a shows a rather flat and slight PE. 1b is a moderate-sized PE. 1c displays a nodular, very pronounced palatine exostosis. On 1d, the PE is ridge-shaped and quite small but large bilateral buccal maxillary exostoses are clearly visible. Photographs 1e to 1h illustrate mandibular exostoses (ME). 1e shows a small, bilateral, rather discontinuous ME, while 1f shows a medium-sized, bilateral and continuous ME. 1g is a discontinuous ME. 1h is a nodular, continuous and pronounced expression of ME. Note that ME are often bilateral but not strictly symmetrical. All the individuals are Thais from the Chiang Mai anatomical collection. 1a is a 57 year-old female, 1b a 71 year old female, 1c a 46 year-old female and 1d a 66 year-old male. 1e is a 49 year-old female, 1f a 68 year-old female, 1g a 84 year-old male and 1h a 70 year-old female / *Exemples d'exostoses palatines et mandibulaires. Les photographies 1a à 1d montrent différents degrés d'expression de l'exostose palatine (EP). 1a montre une EP plutôt petite et plate. 1b est une EP de taille modérée. 1c montre une EP de grande taille et nodulaire. Sur 1d, l'EP est petite et en forme de crête mais de larges exostoses vestibulaires maxillaires sont clairement visibles. Les photographies 1e à 1h illustrent l'exostose mandibulaire (EM). 1e montre une EM de petite taille, plutôt discontinue, tandis que 1f est une EM continue, bilatérale et de taille modérée. 1g est une EM discontinue. 1h est une forme très marquée, continue et nodulaire d'EM. Remarquez que les EM sont le plus souvent bilatérales mais pas strictement symétriques. Tous les individus sont thaïlandais et proviennent de la collection anatomique de Chiang Mai. 1a est une femme de 57 ans, 1b une femme de 71 ans, 1c une femme de 46 ans, 1d un homme de 66 ans, 1e une femme de 59 ans, 1f une femme de 68 ans, 1g un homme de 84 ans et 1h une femme de 70 ans*.

higher in populations of Asian origin [45-63] and lower in people with an African background [2,64-70]. Arctic and sub-Arctic populations display the highest prevalences of oral exostoses [71-79], to the point where it has been considered an "eskimoid character" [71]. European populations are mostly intermediate while South American populations display low frequencies of oral exostoses [80-103]. In addition, there are considerable divergences in oral exostoses not only between populations, but also between groups with an apparently close genetic background [1,8-14,21] (Table 1).

Reported prevalences also vary according to the type of exostosis. The palatine exostosis seems to be the most frequent type in most populations [8-14,45,49,51,74,90, 101-103] but the reverse has been reported as well [47,65,103]. Buccal exostoses appear to be the rarest of all, with reported frequencies of 2 to 3% [21,83,90,99], but prevalences higher than 20% have also been found [47,51,94]. Data on the concurrence of exostoses is scarce and mainly deals with the association of palatine and mandibular exostoses, which are the most frequent [8,10,12,21]. This specific association may be addressed because of the common denomination of *tori* [1]. The overall rarity of concurrence of oral exostoses may also explain the lack of studies: the prevalence of concurrence ranges from 0.7 to 39.5% [47,99]. The concurrence of all types of exostoses in the same individual is extremely rare. However, the probability of observing a mandibular exostosis is twice as high if the individual already possesses a palatine exostosis [10,21,45,48]. Moreover, individuals with both palatine and mandibular exostoses are more likely to display other types of oral exostoses, which suggests that all types of oral exostoses may be linked. But these features can also arise together in some populations with no influence on each other or any correlation whatsoever [1,2].

Relationship with sex and age

Most authors acknowledge a relationship with sex, such as a predominance of palatine exostoses in females and a predominance of the mandibular exostoses in males [8-14], although the difference is not always significant [21,45-47,53,61, 97,98,102]. The reverse is described as well [70,103]. Buccal and maxillary exostoses seem more recurrent in males [48,103,105-107], although sexual dimorphism is not always at a significant level [102].

Data on the sexual dimorphism of concurrent exostoses is sparse. Haugen [21] reported a more frequent, although not significant, association of palatine and mandibular torus in females (2.34%) than in males (2,07%). Al-Bayati et al. [90] and Bruce et al. [65] also show no significant influence of sex on the concurrence of palatine and mandibular exostoses.

Sexual dimorphism also affects the expressivity of oral exostoses. For instance, a palatine exostosis is commonly more developed in females, while mandibular exostoses are larger in males [8-14]. Moreover, the sexual dimorphism of oral exostoses may begin at a fairly young age, since it has been identified in children and adolescents [71,108], though not always significantly so [72].

A correlation between the presence and development of oral exostoses and age is often reported, but depends on both the type of exostosis and the population. However, studies are not easily comparable since the age ranges are not standardized [21].

The onset of oral exostosis formation is far from clear. Woo [2] reports the presence of palatine exostoses in foetuses, newborns and some children. Many more studies have identified oral exostoses (mostly palatine and mandibular) in children [71,77,79,83,94,101,108-110]. However, because the presence of both palatine and mandibular exostoses is exceptional before the age of 5 and rare before 10 years of age, the onset of their formation is thought to begin between 10 and 20 years of age. Hrdlička [1] has even stated that mandibular exostoses form around the age of 12, which coincides with the eruption of the second permanent molars and would tend to support the hypothesis that occlusion has a major influence. Nevertheless, it is worth noting that most studies about oral exostoses in children lack illustrations, except in Woo [2], who provided an image of a palatine exostosis identified in a foetus of approximately 34 weeks. In contrast, the age of emergence of maxillary and buccal exostoses remains unknown, although they seem to form later than palatine and mandibular exostoses.

The development of exostoses with increasing age is also debated. Woo [2] posits that oral exostoses (particularly the palatine exostosis) start to form before birth and grow until the age of 20. As the skeleton reaches maturity, oral exostoses cannot become any larger. Conversely, other studies support the hypothesis of continuous growth with increasing age [111-113]. However, although the reverse relationship with increasing age is sometimes noted [57], the frequency and expressivity of oral exostoses both tend to increase with age up to a peak and then decrease [8-14] or level off [45], but the precise age of that peak is still under discussion. Various frequency peaks have been observed: at around 30 [114-116], up to 40 [72], 50 [70,103] or 60 years of age [21,51,53]. However, it must be borne in mind that age classes are often expressed differently in each study. For instance, Haugen [21] expresses age in twenty-year intervals, while Halffman et al. [76] only refer to two age classes (under and over 35 years). The distribution of age classes usually depends on the type of material studied and on statistical considerations. For example, Haugen observed a large sample of living individuals, while Halffman et al. [76] examined archaeological assemblages.

Table 1 Reported prevalences of oral exostoses on different materials and populations / *Prevalences des exostoses orales sur différents matériels et populations.*

Geographical area	Population	Type of material	N	FPE	FME		FMxE	FBMxE	FBMdE	FPE+ME	Fcooc	Fexost	References
Arctic and sub-Arctic populations	Iceland	skulls	148	67.9	-		-	-	-	-	-	-	[71]
	Eskimo	skulls	31	87.1	-		-	-	-	-	-	-	
	Chukchi	skulls	1	100	-		-	-	-	-	-	-	
	Eskimo	skulls	366	65.47	-		-	-	-	-	-	-	[2]
	Aleut (children)	living	108	-	35		-	-	-	-	-	-	[72]
	Iceland (Children)	living	763	33.3	-		-	-	-	-	-	-	[73]
			213	14.6	-		-	-	-	-	-	-	
	Norway	living	326	-	31.3		-	-	-	-	-	-	[74]
			245	-	49.4		-	-	-	-	-	-	
	Norway	living	5000	9.22	7.23		-	-	-	2.2	-	-	[21]
	Norway	living	1181	38.55	12.7		-	-	-	-	-	-	[85]
			829	32.81	27.5		-	-	-	-	-	-	
	Finland	living	Women X0	-	20.8		-	-	-	-	-	-	[86]
			Women	-	25.4		-	-	-	-	-	-	
			Men	-	43.5		-	-	-	-	-	-	
	Northern Europe (Neolithic-Medieval)	skulls	333	57.4	-		-	-	-	-	-	-	[15]
	Inuit	skulls	12	50	-		-	-	-	-	-	-	[93]
	Iceland (Viking)	skulls	48	-	50		-	-	-	-	-	-	[75]
Asia	Mongolia	skulls	163	47	-		-	-	-	-	-	-	[2]
	Thailand	living	947	23.1	9.2		-	-	-	-	-	-	[45]
	Vietnam	living	550	6	3		-	-	-	-	-	-	[46]
	Thailand	living	609	61.7	29.9		-	-	-	-	-	-	[47]
	Thailand	living	960	43.54	10.9		-	-	-	28.12	-	-	[48]
	Korea	skulls	160	18.75	-		-	-	-	-	-	-	[52]
	Thailand	living	1200	58.1	31.9		-	-	-	-	-	-	[49]
	China (Neolithic)	skulls	66	-	19.7		-	48	-	-	38.7	-	[50]
	Thailand	living	1520	60.5	32.2		-	-	-	23.2	-	-	[51]
	Japan	living	224	-	76.6	70.3	-	-	-	-	-	-	[53]
	Japan (elderly)	living	664	17	29.7		-	-	-	7.7	-	-	[54,56]
	Malaysia	living	65	50.8	4.6		-	-	-	-	-	-	[55]
	Korea	CT-scans	726	-	24.1		-	-	-	-	-	-	[57]
	Thailand	living (dialysis)	91	41.7	-		-	-	-	-	-	-	[58]
	North Malaysia	living	1532	12	2.8		-	-	-	2.3	-	12.5	[60]
Europe and North America	South California (Indians)	skulls	46	4.3	-		-	-	-	-	-	-	[71]
	Italia	skulls	30	3.3	-		-	-	-	-	-	-	
	USA (African origin)	skulls	873	43.3									[2]
	USA (Caucasian origin)	skulls	667	44.82	-		-	-	-	-	-	-	

(Suite page suivante)

Table 1 *(suite)*

Geographical area	Population	Type of material	N	FPE	FME	FMxE	FBMxE	FBMdE	FPE+ME	Fcooc	Fexost	References
	American Indian	skulls	175	54.74	-	-	-	-	-	-	-	
	USA	living	2478	20.9	7.75	-	-	-	-	3.03	-	[80]
	Yugoslavia	skulls	400	45	-	-	-	-	-	-	-	[82]
	Germany	living	1317	13.5	5.2	-	-	-	-	-	-	[45]
	USA	x-rays		-	16.9	-	-	-	-	-	-	[89]
	USA (African origin)	skulls	52	-	57.7		34.6		-			[68]
	Spain	living	278	44.3	54.7	50.7			-	-	-	[44]
	Crakow	skulls	-	62.2	-	-	-		-	-	-	[91]
	Southern Europe (historical-modern)	skulls	147	7.5	-	-	-		-	-	-	[15]
	Canary Island (400-900 AD)	skulls	130	23.8	-	-	-	-	-	-	-	
	Alsaciens 1885-1908	skulls	72	34.7	-	-	-	-	-	-	-	[93]
	Lorrains 1885-1908	skulls	16	6.25	-	-	-	-	-	-	-	
	Badois 1885-1908	skulls	12	16.5	-	-	-	-	-	-	-	
	Alsace	skulls	50	30	-	-	-	-	-	-	-	
	Croatia	living	1679	42.9	12.6	-	-	-	-	-	-	[100]
	Croatia (elderly)	living	251	48.2	33.46	-	-	-	-	-	-	[29]
Western Asia	Israel	living	1002	21	-	-	-	-	-	-	-	[87]
	Israel (families)	living	168	38.7	-	-	-	-	-	-	-	[88]
	Western Asia (Bronze Age-Modern)	skulls	95	2.1	-	-	-	-	-	-	-	[15]
	Turkey	living	253	20.9	-	-	-	-	-	-	-	[92]
	Tunisia	skulls	48	0	-	-	-	-	-	-	-	[93]
	Ancient Egypt	skulls	15	0	-	-	-	-	-	-	-	
	Turkey (children)	living	1943	-	30.9	-	-	-	-	-	-	[94]
	Jordan	living	338	29.8	42.6	-	-	-	27.7	-	-	[95]
	Turkey	living	2660	4.1	-	-	-	-	-	-	-	[96]
	Saudi Arabia	living	2552	1.3	2	-	-	-	-	-	-	[98]
	Irak	living	932	5.3	7.2	-	-	-	0.5	-	-	[99]

(Suite page suivante)

Table 1 *(suite)*

Geographical area	Population	Type of material	N	FPE	FME	FMxE	FBMxE	FBMdE	FPE+ME	Fcooc	Fexost	References
Africa	South Africa (Prehistoric Bushmen)	skulls	28	32	32	-	-	-	66.7	-	-	[64]
	South Africa (Historic Bushmen)		50	6	24	-	-	-	-	-	-	
	Niger	living	2506	2	3.2	-	-	-	-	-	2.86	[67]
	Jamaica	living	958	-	-	-	-	-	-	-	6.6	[66]
			23	-	4.30	-	-	-	-	-	-	
	Ghana	living	926	3.9	12.1	-	-	-	2.9	-	14.6	[65]
	North West Africa (Late Paleolithic-20th century)	skulls	167	4.2	-	-	-	-	-	-	-	[15]
	East Africa (Late Paleolithic-20th century)	skulls	327	6.1	-	-	-	-	-	-	-	
	Sub-Saharan Africa (19th -20th century)	skulls	777	1.8	-	-	-	-	-	-	-	
	Tunisia	skulls	48	0	-	-	-	-	-	-	-	[93]
	Ancient Egypt	skulls	15	0	-	-	-	-	-	-	-	
	Niger	living	1392	3.7	2.5	-	-	-	-	-	-	[70]
	South Africa (African origin)	skulls	246	-	24.4	-	-	-	-	-	-	[69]
South America	Chile	living	1966	0.37	1/1966	-	-	-	-	-	-	[81]
	Brazil (Indians)	living	200	10	0.5	-	-	-	1	-	-	[83]
	Peru (Pre-Columbian)	skulls	1000	0	8.5			-	-	-	-	[84]
	Old Peruvians	skulls	67	10.8	-	-	-	-	-	-	-	[93]
	Brazil	living	1006	1	2.1	-	-	-	-	-	-	[97]
Multiethnic	Multiethnic	living	667	6.6	14.3	-	-	-	2.8	-	12.3	[90]
	Multiethnic	skulls	723	10.5	-	-	-	-	-	-	-	[93]

Peaks may also differ according to the type of exostoses in the same sample. For instance, Apinshashmit et al. [49] observe that the prevalence of palatine exostoses is similar in each age class while the prevalence of mandibular exostoses increases until the age of 59. Moreover, both palatine and mandibular *tori* tend to enlarge with age. However, with a very similar Thai sample and following the same study protocol, Jainkittivong et al. [51] observed a peak frequency of palatine torus between 20 and 29 years of age.

The peak frequency for other oral exostoses seems to be observed at around 50 to 60 years of age, although there are few studies [10,48,50,105-107]. No data on the relationship between age and the concurrence of oral exostoses were found.

Age and sex have been related to oral exostoses to varying degrees. The existence of sexual dimorphism is interesting because it would support the hypothesis of a genetic component in the etiology of oral exostoses. In contrast, a relationship with age points to an environmental influence. Despite various observations, one rule seems to prevail: the prevalence and expressivity of oral exostoses both increase gradually from adolescence and then decrease after the third or fourth decades of life. Therefore, the growth of oral exostoses seems to occur when teeth are most likely to be present and osseous stimulation is maximal. With increasing age, as tooth loss increases and muscular strength decreases, bone stimulation lessens, resulting in remodelling. This phenomenon is well described for the development of the alveolar bone and could affect oral exostoses in the same way [50].

If proven, the influence of age could rule out the possibility of using oral exostoses to assess distances between populations. Moreover, other factors could also play a part in the appearance and development of this feature.

The etiology of oral exostoses: an endless debate?

Many possible causes have been put forward for the presence of oral exostoses since their discovery and are summarized in Table 2. Etiologies such as madness, cancer, rickets, scurvy, syphilis, criminality or even the regularity of sexual activity [1,2,8-14] have now mostly been abandoned. Chemical irritation of the oral mucosa has also been postulated as a causative factor [117]: the mixture of food and saliva, when in contact with the mucosa for a long time, would cause localized inflammation that could spread to the bone surface, resulting in an overproduction of bone. Hooton [71] suggested that oral exostosis may be caused by sutural activity. The palatine exostosis is indeed located along the median palatine suture, which may overproduce bone tissue with mechanical stimulation. But none of the other types of exostoses are located along or even near a bone suture and, if sutural activity was the sole etiology of oral exostoses, then both the maximum frequency and size could never exceed the closing age of the suture (i.e. 20 years of age), which is not what is usually noticed. Other options therefore have to be considered.

The relationship with systematic diseases and medication has also been investigated. Thus, a positive correlation between the size of palatine exostoses and bone density has been demonstrated in postmenopausal European-American women, with or without hormone replacement therapy [118,119]. This correlation might be related to the polymorphism of the gene coding for the LRP5 protein or some other gene yet to be discovered. Hosoi et al. [120] have further established that women with palatine exostoses display higher radial and femoral bone density, while women with mandibular exostoses have higher bone density only at the femoral neck. Maxillary exostoses seem to be unrelated to bone density. Such observations suggest that bone density and oral exostoses could share a common mechanism, but the final effect of that mechanism may vary according to the location of bone deposit, possibly because of occlusal stress [120]. More data on the association between oral exostoses and bone density are needed to determine whether oral exostoses could be a bone-forming characteristic.

A connection between the presence of primary hyperparathyroidism (HPT, a systemic disease resulting in hypocalcaemia and altering bone remodelling), and a high prevalence of palatine exostosis has been reported [121]. This seems surprising since primary HPT is more commonly associated with bone loss. However, recent studies show that primary HPT leads to a loss of cortical bone mass but also to the preservation, or even an increase, of cancellous bone. Additionally, the anabolic effect of parathormone is supposedly higher at bone sites subjected to mechanical stress: the parathormone would raise the response of osteoblasts to mechanical stimulus. This anabolic response requires the bone tissue to possess enough trabeculation and may be hampered by low bone mass despite mechanical deposition [122]. Given the correlation with HPT, a palatine exostosis might be an expansion of cancellous bone at the expense of the cortical layer in response to the high blood levels of parathormone and mechanical stimulation. But oral exostoses mainly consist of compact bone and this theory seems at odds with the observation of oral exostoses in postmenopausal women, whose low bone mass would counteract the anabolic effect of parathormone. Additionally, Rai et al. [123] failed to identify any correlation between oral exostoses and primary HPT in their sample.

Oral exostoses have also been linked to end-stage renal disease treated with peritoneal dialysis [58], multiple dermatomas [124], osteosclerosis [125], hereditary multiple exostoses [126], allergy to penicillin or anti-hypertensive medication [127] and chronic phenytoin therapy [128]. However, it is unclear whether these pathologies affect or share a

Table 2 Summary of the main etiologies proposed in the literature. The table divides the main etiologies proposed for oral exostoses by major types of causes. For each of these, the theoretical principles, main arguments and limitations are briefly described / *Résumé des étiologies principales rapportées dans la littérature. Le tableau sépare les principes étiologies évoquées pour les exostoses orales par grands types causals. Pour chacun d'entre eux, les principes théorique, principaux arguments et limites sont brièvement décrits.*

Proposed etiology		Theory	Argument	Limitation/counter-argument	References
Epistemological Etiology		Possible relationship with scurvy, rickets, cancer, syphylis, madness, criminality, the regularity of sexual activity	The joint presence of oral exostoses and those instances	The absence of scientific proof	[1,2,8-12]
The chemical irritation of the oral mucosa		The mucous inflammation spreads to the underlying bone and leads to bone deposit	The frequent ulceration of the mucosa covering the exostoses Analogy with the auditory torus	A long contact between the blend of food and saliva and the oral membrane is required The absence of scientific proof	[117]
Sutural activity		The possible persistence or reactivation of sutural activity leading to bone deposit	The location of the palatine exostosis along the midline palatal suture	The other types of oral exostoses are not near any suture The emergence of oral exostoses in adults after the closing of sutures	[71]
Continuous growth with age		Age as the major influence	Case reports showing the continuous enlargement of oral exostoses with increasing age	No prospective studies on large samples to test the theory	[111-113]
Systemic disorders	**Bone density in post-menopausal women**	The polymorphism of the gene coding for the LRP 5 protein or some other genes, jointly with functional loading	Women with oral exostoses display higher bone density	More studies required	[118-120]
	Primary Hyperparathyroidism	The anabolic effect of PTH on bone sites undergoing mechanical loading	The presence of oral exostoses in patients with primary HPT	More studies required to attest the correlation One study failed to find the same correlation	[121-123]
	End-stage renal disease	Oral exostoses may be a consequence of renal osteodystrophy	The increased size and prevalence of palatine exostosis in patients with renal disease	Only one study	[58]
	Multiple dermatomas, Osteosclerosis, hereditary multiple exostoses	A common genetic basis	The observation of oral exostoses in patients with those pathologies	More proof required	[124-126]

(Suite page suivante)

Table 2 *(suite)*

Proposed etiology		Theory	Argument	Limitation/counter-argument	References
Medications	**Allergy to penicillin**	None	The observation of the presence of oral exostoses in that instance	Only one study	[127]
	Anti-hypertensive medication	None	The observation of the presence of oral exostoses in that instance	Only one study	[127]
	Phenytoin therapy	The anabolic effect of that drug on bone cells	The emergence of oral exostoses following the beginning of the treatment	Only one case report	[128]
Genetic factors		The existence of a genetic control for both the presence and the expressivity of oral exostoses	The existence of a sexual dimorphism: a participation of the sex chromosomes?	The sexual dimorphism is not always reported or is not always significant	[2,21,45,7-1,86,104,1-29-137]
			The observation of oral exostoses in children:	Genes involved unknown	
			Population variation	Every mode of inheritance reported	
			Heritability observed in family studies	Different mode of inheritance reported for a same kind of exostoses	
			Varying frequencies and expressivities in two populations of different genetic background living in the same environment	The genetic component may account for only 30% of the total variance of oral exostoses	
			The relationship between frequency and development: a reflection of the link between penetrance and expressivity?		
Environmental factors	**The "food" theory**	Relationship with sufficient nutritional intake, food consistency and high content of vitamin D and polyunsaturated fatty acids (omega 3 and 6).	Higher prevalences and expressivity of oral exostoses in arctic and subarctic populations	The reverse is also observed	[1,6,7,15,5-0,85]
			High prevalences and expressivity in populations eating a lot of marine food		
	Functional loading	Oral exostoses as a consequence of heavy or excessive occlusal loading and acting as a buttress to strengthen the jaws	Positive correlations with bruxism, bite force, pronounced dental wear, number of present teeth and TDM	The reverse is also observed. The correlation with stress proxies may vary with the type of exostosis	[1,45,47,4-8,54,56,68-,70,127]

(Suite page suivante)

Table 2 *(suite)*

Proposed etiology	Theory	Argument	Limitation/counter-argument	References
Oral health status	The inflammatory processes in case of periodontal disease may spread and lead to bone deposition The possible alteration of chewing habits in cases of painful teeth	Positive correlations between tooth decay or periodontal disease and oral exostoses	More studies required	[50,152]
Quasi-continuous model	Oral exostoses as threshold characters, resulting in a complex interplay of both genetic and environmental factors	The best-fit model at present	The hypothesis has not been demonstrated *per se*	[50,154-157,161]

common genetic basis with oral exostoses or if they behave as an epigenetic influence. The correlation might also be coincidental or unrelated to the causative process.

More importantly, the existence of sexual dimorphism points to a genetic component while the relationship with age suggests the involvement of environmental factors. Therefore, research has divided into two opposing scientific camps: hereditary versus environmental.

The genetic component

Several facts support a genetic basis for oral exostoses. First, these seem to arise in childhood and could be part of the overall body growth pattern, which is assumed to be genetically determined [2,71,108]. Secondly, the existence of sexual dimorphism points to a genetic control mediated by the sex chromosomes [86,129]. Moreover, two populations of different genetic origins living in the same environment do not display the same frequency and expressivity of oral exostoses [8-14]. More broadly, population variation itself indicates a genetic component [45]. Furthermore, the often reported relationship between the prevalence and size of oral exostoses may reflect a link between penetrance and expressivity [21]. Finally, family studies demonstrate the inheritability of oral exostoses [98,104,130-136], although almost every pattern of inheritance has been observed: autosomal dominant [130-132], autosomal recessive [133] or dominant X-related [134]. Moreover, different patterns of inheritance have been described for the same type of oral exostosis. For instance, the inheritance of mandibular exostoses is autosomal dominant for Johnson et al. [104] but autosomal recessive for Alvesalo and Kari [135]. Such observations should be considered with caution, however, as a family shares not only a common genetic background but also the same environment and cultural habits, which may also play a role in the appearance of oral exostoses.

The genes controlling for the presence and expressivity of oral exostoses are yet to be discovered. The determinants regulating the initiation, growth, differentiation and limits of the processes of facial growth are intricate polygenic systems, possibly governed by major mutant genes [136]. The emergence and growth of oral exostoses could be regulated by the same genes. This theory does not rule out the potential action of genes related to sex chromosomes. The influence of sex chromosomes is supported by the existence of sexual dimorphism and also by the observation of oral exostoses in women diagnosed with Turner syndrome (45, X0) and Klinefelter syndrome (46, XXY). Alvesalo hypothesizes that mandibular exostoses originate from periosteal bone deposition, which depends on cell proliferation and secretion, which in turn can be regulated by sex chromosomes. On the assumption that sex chromosomes have a genetic

pleiotropic effect, the influence of the X and Y chromosomes could express itself at different levels of skeletal development, be it the maturation of the skeleton, statural growth or the presence and development of mandibular exostoses. It can also be postulated that the same could occur for other types of oral exostoses as well [86-129].

Nevertheless, the genetic theory alone fails to account for all the data observed. Eggen and Natvig [137] even demonstrate that the genetic component for mandibular exostoses is no greater than 30%. Therefore, other factors might play a part in the origin of oral exostoses.

Environmental factors

The environmental influence corresponds to two intricate factors: masticatory stress and diet.

Dietary quantity (sufficient nutritional intake) and quality (nutritional composition and food consistency) are both known to influence bone growth and remodelling. The possible influence of diet is fairly well illustrated by Arctic and sub-Arctic populations. These have a high prevalence of oral exostoses and share a similar and specific diet because of climatic conditions, comprising raw or dry meat and sea mammals and few cereals, vegetables and dairy products [76]. On the one hand, such hard, tough food would be very demanding on the masticatory system and require strong jaws. Oral exostoses would then consolidate the jaw bones, like a kind of buttress [1]. On the other hand, while a low intake of dairy products could lead to deficiencies in calcium and vitamin D, both of which are essential to bone growth and development, daily consumption of fish and other seafood ensures a large intake of polyunsaturated fatty acids (omega 3 and 6) and vitamin D, which promote bone development [15,76,85]. The high prevalence of oral exostoses in Arctic and sub-Arctic populations could then result at once from the toughness of food and its content in bone promoting nutrients. In contrast, the reduction of both prevalence and expressivity of oral exostoses in contemporary populations may result from the increasingly tender food consumed [50] and/or from changes in food preferences. For instance, the lower intake of raw fish in the current Japanese population could explain the decrease in oral exostoses [56].

However, the "food" theory cannot account for every observation. Other populations, for example from Cappadocia in Turkey, display a high prevalence of exostoses although their diet is not particularly tough and low in marine food [94]. On the other hand, African populations or Aborigines test their jaws severely, in particular by using them as a tool, as do the Inuits, but the prevalence and development of oral exostoses is low [64]. Moreover, the general idea of a reduced masticatory function in modern populations could be more apparent than real, since large contemporary populations tend to keep their natural teeth into old age [21], and the prevalence of edentulism is decreasing steadily thanks to preventive action [138]. Finally, the human diet may vary according to the period of life (childhood, old age…), access to various food supplies, culinary preparations, religious beliefs, etc., all of which are difficult to assess precisely and, most of all, need to be considered together with masticatory function.

Severe or excessive masticatory demands could stimulate bone production. Indeed, alveolar bone deposition occurs in cases of bruxism, through the continuous eruption of teeth, to compensate for dental wear and maintain the vertical occlusal dimension [139]. Bone deposition can also be observed in edentulous patients when they are fitted with a full removable prosthesis stabilized on implants, thanks to the increase in functional loading [140,141]. Finally, the occurrence of subpontic bone proliferation under a fixed prosthetic denture (FPD) could also illustrate the relationship between occlusal stress and bone deposition. When the FPD is put in place, the orientation and amount of occlusal loading change and may stimulate osteogenesis, according to Wolff's law, thus producing a bony protuberance under the pontic [142-144]. Oral exostoses may act in a similar way. However, Yamashita [145] did not observe any difference in *in vitro* strain patterns of mandible loading before and after fitting an FPD, at least as regards its overall direction.

Theoretically, heavy occlusal loading (bruxism, occlusal trauma, tough food, use of the jaws as a tool, etc.) contributes to both the emergence and development of oral exostoses "*much as a vigorous weightlifting program produces a Schwarzenegger-like physique*" [68]. These would then act as buttresses strengthening the jaws. In this case, the configuration of the internal bone structure would follow the direction of the mechanical forces exerted on the bone [145]. Woo [2] observed that the pressure lamellae of palatine exostoses have an antero-posterior direction. If the mechanical loading exerted on the palatine exostosis were vertical, the lamellae would display a medio-lateral orientation.

Several observations could support the influence of functional stress. First of all, bruxism has been positively correlated with the presence and development of oral exostoses [85], as well as pronounced dental wear [45,48,50,53,69-71,83,103] or strength of bite force [54,56]. Moreover, the presence of oral exostoses has been linked to the number of teeth present [70,147], since their prevalence decreases in the elderly, supposedly because of the increase of edentulism with age, which hampers the transmission of mechanical loading, hence stimulation, to the jaw bones [73,81,85,103,137]. Finally, the presence of oral exostoses has been correlated with temporomandibular disorder (TMD) [47,50,127,148]. Pechenkina et al. [50] noted that the chance of observing oral exostoses is twice the normal rate on the upper jaw and 4.5 times higher on the mandible when TMD is also present. TMD often occurs in cases of heavy to excessive occlusal

loading. As the masticatory apparatus undergoes heavy occlusal loading, dental wear increases. In turn, the orientation of the occlusal plane changes and the load tends to shift from a posterior to an anterior position, resulting in the alteration of the temporomandibular joint (TMJ) [148,149].

The position of tooth apices might explain why oral exostoses arise in specific locations [150]. Teeth would transfer occlusal loading through their roots to the surrounding alveolar bone. Once these forces reach the tooth apex, the pressure would be transmitted in the opposite direction to the position of the apex.

However, some studies could not demonstrate a positive correlation between occlusal stress proxies, such as dental wear or TMJ alterations, and the presence of oral exostoses. Yoshinaka et al. [54] found no connection between palatine exostoses and bruxism, TMD, occlusal forces or the Eichner Index[1].

However, in the same sample, mandibular exostoses appear to be related to the Eichner index and to nocturnal bruxism [56]. Sellevold [151] observed no correlation between oral exostoses and attrition or gonial eversion in Inuits and Norse. Moreover, if such local environmental factors affect both the maxillary and the mandible, and given that any exostosis strengthens the jaw bones, then the influence of masticatory stress *sensu lato* should produce a strong correlation between the various types of oral exostoses, which is not the case in many populations [21,82,115]. Finally, the theory of the transmission of forces in the opposite direction to the tooth apex fails to explain how oral exostoses might occur simultaneously on the lingual and buccal aspects of the alveolar bone. In addition, this theory does not take into account the extreme variability of the root system. For instance, upper molars have three roots, oriented in three different directions: where are the occlusal forces supposed to go?

Finally, few studies have investigated the correlation between oral health and oral exostoses, although poor oral health might influence dietary habits and alter the masticatory function. A positive correlation with periodontal disease has been suggested by Glickman and Smulow [151]: local inflammation processes resulting from periodontal disease may promote bone formation, hence the occurrence of oral exostoses. Pechenkina et al. [50] report a positive correlation between the presence of buccal maxillary exostoses and calculus deposit and periodontal disease, while buccal mandibular exostoses show no correlation with these factors. But how could periodontal disease explain the appearance of palatine exostoses, which are located at a distance from the teeth and from any inflammation of the alveolar bone? Pechenkina et al. [50] also observed a positive correlation between the presence of buccal maxillary exostoses and tooth decay, but then again, the buccal mandibular exostoses do not share a similar association and the amount of tooth decay in their sample is very small.

However, it could be interesting to check for the influence of oral health status. Indeed, deep tooth decay and/or pronounced periodontal disease, being painful, may disturb chewing and thus result in a change in both dietary habits and masticatory function. This may, in turn, lead to a redistribution of oral forces, just as much as dental wear.

To conclude, it is apparent that environmental factors do play a role in the genesis of oral exostoses, although the proportion of their participation in the causative process is still unknown and may vary according to the type of exostosis and the population. Moreover, the various aspects of environmental influence have mostly been investigated separately, although they probably interact with each other and are related to age and sex as well. TMD, in particular, is a multifactor pathology, known to be influenced by stress, posture, hypermobility, systemic disease, age and sex [153]. Therefore, it could be interesting to consider these environmental factors, including data on diet, as a matrix, taking into account their interrelationships as well as their effect on oral exostoses. In any case, environmental factors alone cannot account for all the observations. A more comprehensive, dynamic etiological theory then emerges.

Towards a compromise: the threshold theory

Oral exostoses can be considered as a multifactor process: they result from a complex interplay of both genetic and environmental factors [8-14]. The Functional Matrix Hypothesis (FMH) [154-157] fits this concept. According to the FMH, the developmental origin of any cranial skeletal unit, its changes in size, shape or location and even bone abnormalities, are mainly secondary, compensatory or adaptive responses to functional demands. Genes alone cannot account for every phenotype and, in Moss's view, the genomic hypothesis reduces morphogenesis to the sole reading of DNA. The FMH particularly stresses the importance of mechanical loading in bone development, regulation and maintenance. Indeed, mechanical loading can be transmitted directly to osteoblasts and even DNA, through mechanotransduction [154] and thanks to the organization of bone tissue as an interconnected cellular network [155]. Bone cells then respond to mechanical forces by commanding either bone deposition, bone resorption or even both. Such mechanisms are also thought to contribute to bone homeostasis and ageing [158-160]. Singh [161] calls on the FMH to

[1]The Eichner Index is used to assess occlusal dysfunction by counting the number of tooth pairs in occlusion. Three classes are defined:
A: from a minimum of one tooth contact between antagonist pairs in the premolar/molar area to a maximum of four supporting zones;
B: one to three supporting zones or tooth contact only in the anterior area;
C: no supporting zones.

explain the etiology of oral exostoses: the mechanical forces applied strongly over a short duration (i.e. <50 Kg and < 1s) during mastication are mainly absorbed by the periodontal fluids, but this process results partly in the bending of the collagen and a piezo-electric effect in the alveolar bone. Singh [158] suggests that jaw bones undergo a similar deformation process during parafunctional activities, such as bruxism. The ensuing stresses and stretching of the osteogenic periosteum eventually lead to bone deposition in the form of site-specific oral exostoses. For instance, repeated compressive stress may cause buckling of the maxilla along the midline. The osteogenic periosteum of the palatal vault (i.e. the palatal suture) would then be stretched intermittently. This tension would promote new localized bone formation along the midline, as this is the epicentre of force distribution, and thereby trigger the emergence of an exostosis. But if the vector of force distribution changes direction, the exostosis would then appear elsewhere. To sum up, bone deposition occurs in the focal areas of force dissemination thanks to mechanotransduction signals. However, this theory cannot account for the concurrence of oral exostoses.

On the assumption of genetic and environmental interaction, oral exostoses can be viewed as quasi continuous traits: the environmental factors have to reach a threshold before the predisposing genetic factors can express themselves [8-14]. The population differences then reflect the position of the environmental threshold. Moreover, quasi continuous characters tend to cluster in families but, unlike single-trait genes, they do not fit any model of dominance or recessiveness. This would account for the results of family studies. Nevertheless, the question of a common origin for every type of oral exostosis is still unanswered. Since all oral exostoses share similar features, the quasi continuous model of inheritance could apply to all of them, although this is yet to be proven [49]. Questions remain nevertheless. The genes involved are yet to be identified, as well as the precise combination and the relative proportions of both genetic and environmental factors in the emergence of oral exostoses.

The threshold theory is certainly the causative model that best fits the data, but it implies that oral exostoses may be influenced by environmental factors in addition to age and sex, which compromises the use of the feature as a population marker. Unfortunately, the literature on oral exostoses is not easily analyzed because of the heterogeneity of study protocols. The differences observed may well echo these discrepancies. The most common assessment protocols for oral exostoses are summarized in Table 3.

Methodological disparities: a major influence?

First of all, data collected from dry skulls should be considered separately from data gathered from living individuals [21,76], since frequencies estimated from cranial samples are generally higher than those based on living individuals. This is probably due in part to the difficulty of identifying small exostoses when they are obscured by the mucosal lining. Moreover, the unqualified term of "trace" to account for very small exostoses may also explain the considerable differences in prevalence in ethnically and genetically close material. It has been suggested that the "trace" concept should be dropped to limit biases [21]. Furthermore, many essential factors such as sample size, sex ratio or age range are more easily controlled for in living samples. Archaeological series, on the other hand, may be small and sometimes poorly preserved. The age at death of adults is determined in broad intervals, so that the age ranges used are wider and cannot be compared with hospital samples [162,163]. Ethnic representativeness can be difficult in both cases. As for osteo-archaeological assemblages, ethnic backgrounds can be inferred from historical contexts, dating and close examination of graves and individuals (dental anatomical variations in particular), all of their possible shortcomings being well known. On the other hand, ethnic origin or homogeneity in current populations could be distorted by migration flows, although such problems can be avoided with a thorough preliminary questionnaire on family history [21]. Finally, selection bias remains an issue in both samples.

Most importantly, as shown in Table 3, there is no consensus on how to classify oral exostoses. The most common evaluation process uses a "simple" presence/absence dichotomy. This method limits inter-observer error [73] but authors do not always explicitly report what they have used as a presence threshold and may disagree about the definition of that starting point. Researchers often classify a trait as present only when it reaches a certain arbitrary size [21,45], but the breakpoint corresponding to the threshold varies a great deal between studies. Once the feature is determined as present, ranked scales are used to characterize the degree of expression. Ranked scales include different presence categories ranging from slight to pronounced. Ordinal scales are inherently problematical because of their subjectivity. Moreover, the graduations from absence of development to very marked are in themselves gradual, so that a clear-cut definition between each graduation is virtually impossible and the distance between ranks is unknown but rarely equal. Therefore, some rely on metric measurements to refine their grading system, thus transforming it into an scale of intervals where the distance between each category is known. This approach can be more precise and allows the use of parametric statistics with no significant uncertainties. But the limits of each class differ between studies, often to account for trait expression in the population studied. As a result, comparisons between populations become haphazard. Finally, some of these grading scales take the "trace" score into account, but this very small

Table 3 Examples of methodologies used to assess oral exostoses.Table 3 shows some examples of methodologies used in previous studies of oral exostoses. PE stands for Palatine Exostosis, ME for Mandibular Exostosis, MxE for Maxillary exostoses, BE for buccal exostoses. Note the diversity of study protocols, which may play a part in the diverging prevalences observed in table 1 / *Exemples de méthodologies utilisées pour étudier les exostoses orales. Le tableau 3 expose quelques exemples de méthodologies employées dans les études des exostoses orales. PE représente l'exostose palatine, ME l'exostose mandibulaire, MxE l'exostose maxillaire, et BE les exostoses vestibulaires. Noter la diversité des protocoles, qui peuvent participer à la divergence des prévalences observées dans le tableau 1.*.

Material	Type of exostoses	Age ranges	Determination method	Presence threshold	Size assessment method	Form assessment method	Metric measurements	Reference
Skulls	ME PE	Decanal age classes	Visual	?	From metric measurements, creation of a 3 point scale (L*l*h): Small: <15*<10*<3 mm Medium: 15-25*10-15*3-5 mm Large: >25*>15*>5 mm	3 point scale: Mound: wide protuberance Ridge: narrow protuberance Lump: irregular protuberance	Yes: maximum length, width and height	[2]
Living individuals	PE ME	Decanal age classes	Visual		ME: 4 point scale such as: bilateral simple bilateral multiple, unilateral simple, unilateral multiple	PE: flat, spindle, nodular, lobular	No	[80]
Living individuals	PE, ME	-	Visual and palpation	Palpable	Presence/absence only	-	-	[83]
	ME		Visual	?	4 point scale: low, moderate, marked, extreme	6-point scale: from no lobuli (0) to more than 4 lobuli (5)	No	[151]
Living individuals (children)	PE	Exact age	Visual and palpation	Palpable	4 point scale: absent, palpable, visible, large	-	No	[85]
Living individuals	PE, ME	-	Visual and palpation		3 point scale based on height estimation: Small: <3 mm high Medium: 3-6 mm high Large: >6 mm high	-	Yes	[45]
Living individuals	PE ME	3 age classes: [16-34], [35-65], [66+]	Visual and palpation	?	Three-point scale: small, medium, large.	PE: 3 classes: ridge-shaped, nodular, lobular ME: 4 classes: Bilateral solitary, bilateral multiple, unilateral solitary, unilateral multiple	No	[21]

(Suite page suivante)

Table 3 *(suite)*

Material	Type of exostoses	Age ranges	Determination method	Presence threshold	Size assessment method	Form assessment method	Metric measurements	Reference
Skulls	PE	2 age classes: [18-35], [35+]	Visual	Above trace	Absent Trace: elevation of 1 to 2 mm Moderate: elevation of 2 to 5 mm Marked: more than 5 mm high Very marked: more than 10 mm high and 10 to 20 mm wide + Metric measurements	-	Yes: maximum length, width and height	[76]
Skulls	PE	?	Visual	?	Position in relation to the incisive foramen, the transverse palatine suture and the posterior nasal spine (4 classes)	-	No	[52]
Skulls	BE, ME	-	Visual	Slight?	3 point ordinal scale: Slight: intermittent ridges Moderate: continuous ridge at least 2 cm long Severe: >0.5 cm thick (BE), >1 cm thick (ME)	-	-	[50]
Living individuals	PE, ME, MxE		Visual and palpation	Visible?	Mx E:2 classes: Weak: affects either the buccal or the palatine aspect Strong: affects both buccal and palatine aspect PE and ME: 2 classes: visible, palpable	ME and MxE: 2 classes: simple or continuous PE: two classes: lobular or non-lobular	No	[44]
Skulls	ME	-	Visual	?	2 classes: Solitary: limited to 1 or 2 teeth Elongated: more than 2 teeth	2 classes: round, multiple (more than one lobuli)	No	[69]
Living individuals	PE ME MxE	Decanal age classes	Visual + palpation	?	PE: [45] ME: [80]	PE: Flat, spindle, nodular, lobular ME: number of lobuli	Yes: maximum height	[47,48,5-1]
Skulls	PE	?	Visual + palpation	?	From metric measurements, classification of Woo, 1950 [2]	Woo, 1950	Yes: maximum length, width and height	[91]

(Suite page suivante)

Table 3 *(suite)*

Material	Type of exostoses	Age ranges	Determination method	Presence threshold	Size assessment method	Form assessment method	Metric measurements	Reference
Living individuals	PE	?	Visual and palpation	Visual and palpable ?	Presence/absence only	-	No	[92]
Living individuals	ME PE	Decanal age classes	Visual + palpation	?	3 point scale based on height: Small: 1-2 cm high Medium: 2-3 cm high Large: > 3 cm high	Solitary or multiple	?	[70]
Skulls	PE	-	Visual	?	From metric measurements, creation of a 3 point scales (L*l*h): Small: <15*<10*<2 mm Medium: 15-25*10-15*2-4 mm Large: >25*>15*>4 mm	-	Yes: maximum length and maximum width. Visual estimation of height.	[93]
Skulls	PE	-	Visual	Moderate	Absent Trace: elevation of 1 to 2 mm Moderate: elevation of 2 to 5 mm Marked: more than 5 mm high Very marked: more than 10 mm high and 10 to 20 mm wide	-	Yes: maximum height	[15]
Living individuals	ME	Decanal age classes	Visual and palpation under illumination	Class 1 = palpable	Product of class by length (expressed in number of teeth) 4 point scale: 0 = not palpable, not visible 1: only palpable 2: partly perceptible and palpable 3: clearly visible	_	No	[53]
Living individuals	ME	[<50], [50-59],[60-69], [70-79], [>80]	Visual + palpation	Visual and palpable?	[45]	-	Yes: maximum height	[29]
Skulls	PE ME	[<35], [36+]	Visual	?	PE: [2] ME: [80]	-	No	[75]

expression of a trait can only be detected on skeletal material and could be misdiagnosed. Theoretically, metric methods should be more efficient and reliable and have been put to the test [2,76,90,93,103]. However, many authors advise against them [21,53], on the grounds that they are laborious, time consuming and difficult to perform, since oral exostoses, as non-metric traits, vary in size and shape and have no fixed landmarks. However, metrics circumvent one of the major issues associated with the ranking of oral exostoses, which is that they produce two key dimensions, height and width, in any possible combination [76]. Besides, despite the absence of landmarks, oral exostoses typically differ from the surrounding bone in colour and sometimes in porosity. Under the quasi continuous hypothesis, only maximum height, width and length can be measured to account for the maximum expression potential and still take into account the fact that the expression of oral exostoses is gradual.

Concerning shape, again different grading systems have been used but scientific criteria to characterize shape as a measurable variable are lacking [21]. Regarding mandibular exostoses, for instance, the method of Kolas et al. [80], one of the most commonly used, combines both shape and symmetry (see table 2).

We also point out that etiological factors have been studied separately. However, the human body is the product of complicated and multiple interactions. The same is likely to apply to oral exostoses, which may well be the sum of different factors, both genetic and environmental, with each of these factors, such as age and tooth wear etc., also interacting. Research could focus on a broader etiological pattern and try to identify the amount of participation of each factor in the emergence of oral exostoses. Complex mathematical models are required to develop a matrix of influence factors and determine which of the genetic or the environmental components play the greater role. This way, we would know whether oral exostoses can be used as a population indicator or as a masticatory, behavioural, etc. indicator in anthropological studies.

Conclusions and perspectives

More than a century after they were first described, oral exostoses are still quite a mystery and offer many possibilities for research. Many contradictory observations have been reported and various theories proposed, but few have really been tested. First of all, a consensual definition of oral exostoses must be clearly stated and unanimously accepted. Secondly, two core issues should be addressed: are the various types of oral exostoses related to one another or different biological units, and what type of character are they? It is often implied that oral exostoses are a particular set of traits, but, although they do seem to share some characteristics, they may well be different entities. These two possibilities should be tested equally, since the question of filiation between each type of exostosis may account for the wide divergences reported in the literature. More data on the concurrence of exostoses has to be collected. On the assumption that oral exostoses are not related, some types could be population markers, while others, being more influenced by environmental factors, could be stress indicators. Moreover, it is also often implied in the most recent studies that oral exostoses are threshold characters. The quasi-continuous model indeed seems to best fit the data for now, but it is often chosen in retrospect without ever being tested *per se*. Ordinal scales are the most frequently used to assess both the presence and development of oral exostoses but such coding systems artificially create an illusion of discrete variation, hence possibly distorting our view of the character.

Therefore, new standards for scoring oral exostoses have to be considered and put to the test, following the example of the work of Scott and Turner [164] with dental anatomical variations. Ideally, the scoring method should be applicable to both skeletal remains and living individuals to limit divergences between observations. The method itself should be both simple and exhaustive, while trying not to mix information. This could prove to be a formidable task, since oral exostoses express themselves in several dimensions. The tremendous advances in radiological imagery could prove useful to replace ordinal ranking systems, as the development of oral exostoses could be assessed in three dimension from CT-scans. Such a method may also provide additional information on the constitution of oral exostoses, especially in cases where the expansion of cancellous bone at the expense of the cortical layer is assumed. Such standardization is absolutely necessary to yield comparable data between studies.

Aside from methodological standpoints, the relative contributions of the genetic and environmental components should be determined more precisely. As for the genetic component, larger and more recent family studies are required. Also, the genes involved in the emergence and development of oral exostoses are yet to be identified. Do the sex chromosomes play a part and, if so, in what way? Assessments of the genetic contribution may allow oral exostoses to be used as a family marker, which could prove useful for the study of archaeological assemblages. Regarding the environmental factors, more comprehensive studies, encompassing the full range of possible influences, would be better. Agreement on the type of occlusal stress proxies is essential. Some suggestions deserve to be examined in depth, especially the relationships with bone density, primary hyperparathyroidism and any other disorders that may affect the bone tissue, and the correlation with oral health status. Finite element analysis could help identify localized strain patterns in jaw bones with oral exostoses: are they indeed sites that focus functional loading? Prospective studies in various populations would be ideal to pinpoint the onset of oral exostoses and

verify the continuous growth hypothesis. More importantly, we believe that all these factors – genetic and environmental – should be studied together, taking into account interactions between them as well as their influence on oral exostoses, in a complex matrix. To achieve this end, new mathematical tools need to be considered. Such tools could also prove useful for studies of other biological variations.

Finally, even though we have not addressed this particular issue in this paper, it could be interesting to review fossil specimens. Why are oral exostoses more frequent in anatomically modern humans, despite their tendency to be less robust?

For now, we believe that the status of research on oral exostoses does not allow them to be used to assess distances between populations. To improve knowledge on this anatomical feature so that it can finally be put to good use, these methodological and theoretical questions have to be resolved. Such a task would benefit from interdisciplinary exchanges involving anthropologists, medical professionals, mathematicians and geneticists. Much still needs to be done if we are to write a whole new story a hundred years from now.

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