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ORIGINAL ARTICLE

Atrophy of the residual alveolar ridge following tooth loss in an historical population

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OBJECTIVES: To study the natural aetiopathology of jaw atrophy after tooth loss, unaltered by prosthetic procedures, an historical population without modern dental treatment was examined.

METHODS: Based on the hypothesis that there are predictable changes in shape during jaw-atrophy, frequency and degree of atrophy as well as clinical aspects of bone quality and resorption were determined in the skeletal remains of 263 individuals. The potential association between age and frequency/severity of atrophy was analysed.

RESULTS: Atrophy in at least one jaw segment was present in 45.2% of the analysed jaw specimens. The residual ridge underwent a series of changes in shape and height following the pattern of resorption described for modern populations. The severity of these alterations was associated with the age of the individual and the region within the jaw. Atrophy was frequently related to structural degradation of the covering cortical layer.

CONCLUSIONS: These findings prove that atrophy of the jaw evidently does occur, displaying similar patterns of resorption in a population without modern prosthetics, where the negative effect of ill-fitting dentures is excluded. The basic information about alterations of shape and the cortical layer covering the residual crest might help to provide a deeper insight into aetiopathological mechanisms of this common oral disease.

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Keywords: jaw atrophy; alveolar ridge resorption; tooth loss; residual ridge; denture adverse effects; dental implants; pathoaetiology

Introduction

As people grow progressively older in modern societies, health problems associated with advanced age increase dramatically. These include, among others, oral diseases that finally may lead to tooth loss and necessitate prosthetic treatment for the patient to obtain the ability to chew and eat as normally as possible.

Tooth loss may have a variety of causes such as periodontal disease, caries and trauma leading to pulpitis. Whatever the reason might be, the effect of tooth loss is always the same: The physiological masticatory forces applied via the roots of teeth to the cancellous alveolar bone no longer persist (Devlin and Ferguson, 1991; Ulm *et al*, 1997). According to Wolff's Law (Wolff, 1892) and the Mechanostat Model (Frost, 2003), disuse and a loss of mechanical stimulation is followed by the reduction of bone mass. This effect was originally demonstrated for limb bones. Whether a lack of mechanical strain has the same impact on the alveolar process of the jaw and other skull bones remains to be studied in detail. Moreover, the influence of other factors such as regionally different gene expression (long bones vs. cranial vault) resulting in site-specific osteoblast behaviour, osteoclast activity and matrix composition has been discussed in this context recently (Rawlinson *et al*, 2009). However, loss of teeth leads invariably to atrophy of the residual alveolar ridge being irreversible, chronic, progressive and cumulative (Atwood, 1971).

The rate of atrophy varies greatly between different individuals (Bras, 1990) and even within one and the same person at different times or in different regions within the jaw (Atwood, 1971, 1973). However, atrophy is the greatest during the first year after tooth loss; the reduction of the residual ridge is a life-long process (Sennerby *et al*, 1988), but the rate of bone loss does decrease (Denissen *et al*, 1993).

Atrophy of the jaw bone can be slowed down, but unfortunately not eliminated, with the help of an appropriate treatment using prostheses, balancing the loads to the underlying bone or preferably using implant-supported overdentures (Kordatzis *et al*, 2003) or endosseous implants (Jokstad *et al*, 2002a,b; Bodic *et al*, 2005).

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Yet, the influence of fixed or removable dentures is an issue of controversial discussion (Devlin and Ferguson, 1991; Carlsson, 2004). As complete dentures do not load the alveolus in the same way as the original teeth do, non-physiological pressure is applied to the bone surface of the affected jaw, which might be a reason for increased resorption. This is particularly plausible for ill-fitting dentures that cause occlusal disharmonies and thus might enhance alveolar bone loss (Devlin and Ferguson, 1991). Hence, the quality of the denture as a holistic system is crucial to prevent local overloading of the underlying bone (Xie *et al*, 1997a).

However, reduction in the height of the residual ridge is also observed in patients without denture treatment (Devlin and Ferguson, 1991). Consequently, atrophy of the alveolar bone is not attributable to prosthetic factors alone (Atwood, 1979).

Anatomic conditions of the jaws, systemic factors such as gender and age, hormonal balance, local inflammations and masticatory habits are supposed to act as co-factors in the development of residual ridge resorption after tooth loss (Atwood, 1971; Gruber *et al*, 1993; Kingsmill, 1999).

To gain information about the pathology of atrophy of the jawbone after tooth loss, unaltered by the influence of prosthetic procedures, a population without modern dental treatment seems appropriate to serve as a model. For this reason, the skeletal remains of a central European population unearthed from a mediaeval cemetery of the 7th-8th century A.D. was used for this study. As dental treatment was not or only rarely practised in mediaeval populations (Watt *et al*, 1997; Vodanovic′ *et al*, 2005), these skeletal remains are very useful for analysing the pure effect of oral diseases, such as jaw atrophy of the residual ridge.

The purpose of this study is to characterise the patterns of jaw atrophy after tooth loss in an historical population without modern dentistry using an adapted classification system according to Atwood (Atwood, 1963) and Cawood and Howell (Cawood and Howell, 1988). The frequency and severity of atrophy as well as four clinical aspects of bone quality and resorption are examined with respect to age within different segments of the jaw.

Understanding the natural history of this disease could help to improve our knowledge about atrophy of the jaw, the final result of tooth loss, and is a prerequisite for the development of new treatment strategies. This study also provides additional new information for the interpretation of living conditions and lifestyles of historical populations (Alt, 1987; Vodanovic′ *et al*, 2005; Meller *et al*, 2009).

Material and methods

Avarian skeletal remains

A large mediaeval cemetery with skeletal remains of an Avarian population was unearthed during archaeological excavations in the 11th district of Vienna, Austria. This Avarian cemetery of the 7th–8th century A.D. consisted of 705 graves. In total, the skeletal remains of 755 individuals were collected.

A total of 263 jaw specimens of this sample met the inclusion criteria of (i) completion of dental development, i.e. presence of permanent dentition and (ii) a sufficient state of preservation of the jaw allowing the evaluation of the features described hereinafter.

Estimation of age at death and determination of sex was based on osteological and dental development criteria, [according to the recommendations of Sarospatak (Ferembach *et al*, 1979)] and had previously been performed by Großschmidt (1990). Age at death was determined according to the degree of cranial suture synostosis, surface characteristics of the symphysis of the pubic bone and of the auricular surface of the ilium and the degree of tooth abrasion (White and Folkens, 1999). The degenerative processes evaluated in this study were not used to establish an age estimate, and both factors are therefore independent of one another.

According to these data, the study sample was divided into three age groups of young (21–40 years), middle-aged (41–60 years) and old (61–80 years) individuals. Demographic data concerning age and sex distribution of the analysed population are presented in Table 1.

Methods

Three investigators (WRL, ST, KMR), experienced in the analysis of degenerative alterations of the jaw, discussed and interpreted the variation and expression of signs of atrophy in the sample. The application of the classification systems employed in this study was standardised and calibrated until a consensus was reached.

The practical evaluations and classifications, however, were performed solely by one investigator (WRL) to avoid interobserver error. While undertaking these evaluations, the investigator was blinded to age and sex of the individuals. The following features were macroscopically diagnosed under bright light.

Classification of atrophy stages. Based on the classification system of Nakamato (1968), six separate segments of the jaw were defined as depicted in Figure 1. Each segment of the 263 jaw specimens was assigned to one of the six stages of atrophy described by Atwood (1963) and Cawood and Howell (1988). (See Figure 2)

Stage 1 describes the physiological state of preextraction in which the tooth is still in the alveolar socket or the tooth is lost postmortem.

Table 1 Age and sex distribution of the analysed historical sample consisting of 263 individuals

	Female	*Male*	*Total (both genders)*
Age group	n *(%)*	n *(%)*	n *(%)*
20–40 years	70 (57.9)	69 (48.6)	139 (52.9)
41–60 years	22 (18.2)	58 (40.8)	80 (30.4)
61–80 years	29 (24.0)	15 (10.6)	44 (16.7)
All age groups	121 (46.0)	142 (54.0)	263 (100)

Values are given in absolute numbers and percentages (%).

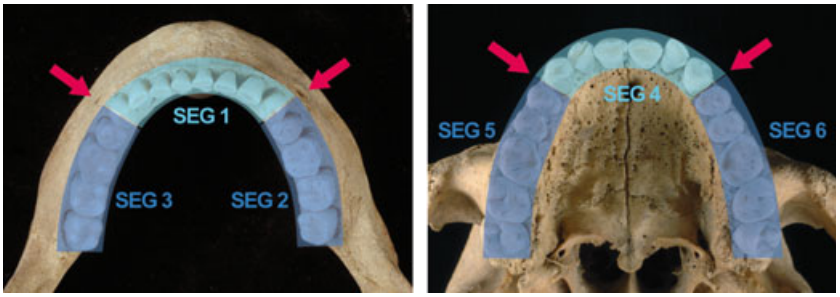

Figure 1 Segments of the jaw according to the classification system of Nakamato (1968). Mandible: SEG 1 (anterior), SEG 2/SEG 3 (posterior, right/left). Red arrowheads indicate mental foramina, separating anterior from posterior segments; Maxilla: SEG 4 (anterior): between the two canines, SEG 5/SEG 6 (posterior, right/left): between the 1st premolar and the tuberosity at the distal end of the alveolar arch. Red arrowheads indicate borderline between canines and first premolars

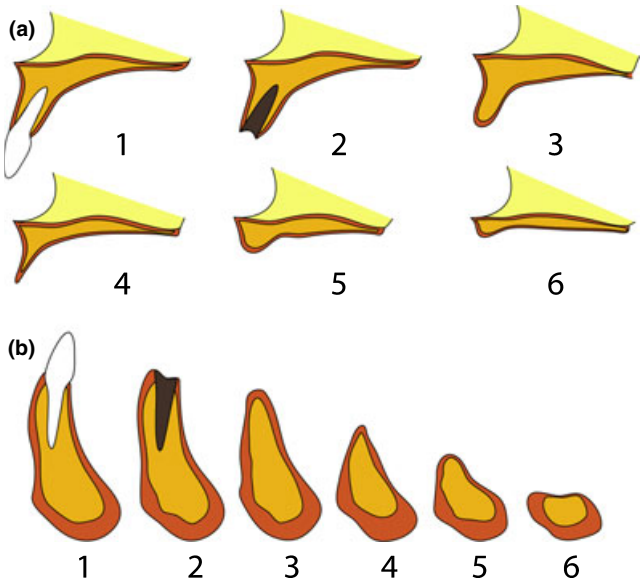

Figure 2 Classification system of six atrophy stages in the maxilla (**a**) and the mandible (**b**) according to Atwood (1963) and Cawood and Howell (1988). Atrophy stage 1: preextraction, stage 2: postextraction, stage 3: high well-rounded ridge, stage 4: knife-edge shaped ridge, stage 5: low well-rounded ridge, stage 6: depressed bone level

Stage 2 is assigned to tooth loss immediately before death. There are slight osseous reactions of new bone formation within the alveolus. The alveolus is still in a good condition and the edges might be sharpened.

In stage 3, the alveolus is completely refilled with newly formed bone. The shape of the original alveolus is no longer identifiable and the top of the alveolar process finally becomes well-rounded due to first signs of resorption. However, there is no notable reduction in height.

In stage 4, the shape of the alveolar crest alters into a thin and sharp knife-edge; the body of the jaw is still adequate in height and width.

Stage 5: Further resorption leads to a low well-rounded ridge, which is flat but already reduced in height and width; the alveolar process is lost.

Stage 6: Continuing excessive atrophy of the residual crest results in a depressed bone level, where even the basal bone shows signs of reduction.

Exposed trabecular bone at the top of the residual ridge. Atwood (1971) and Pietrokovski (1975) stated that in many individuals the cortical layer cannot close over the former alveolus and adjacent areas sufficiently due to degenerative and resorptive alterations of the alveolar crest. Accordingly, the residual crest exhibits trabecular bone, extending over the whole length of an edentulous ridge. (See Figure 3a)

Stage I describes the situation after tooth loss in which the alveolus is totally closed and the crest completely covered by cortical bone.

In stage II, the cortical layer of the alveolar crest is not closed, but discontinuous so that an area of exposed trabecular bone with a width of up to 2 mm is identifiable.

In stage III, the cortical layer of the crest is interrupted by even larger areas of exposed trabecular bone of more than 2 mm in width.

Bony defects in the region of the former alveolus. Due to poor or incomplete repair of the alveolus after tooth loss, bony defects such as trabecular spots and macroscopic perforations (Solar *et al*, 1998) might be observed on the crest of the residual ridge (Nakamato, 1968; Pietrokovski *et al*, 2007). (See Figure 3b)

As opposed to exposed trabecular bone, the occurrence of this feature is thought to be restricted to the

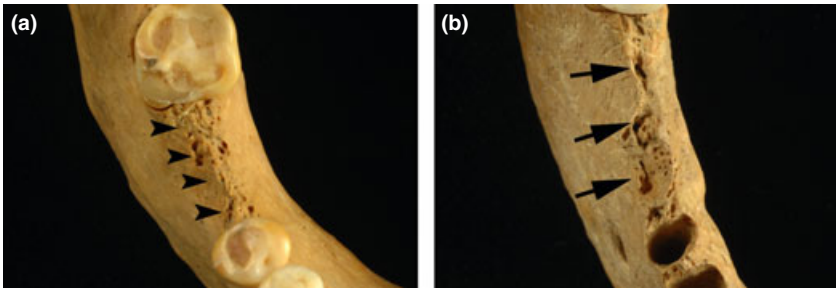

Figure 3 Photographs of two mandibular (right) posterior segments illustrating the differences between the features exposed trabecular bone and bony defects. (**a**) Area of exposed trabecular bone at the crest of the residual ridge (small black arrowheads). (**b**) Bony defects at the crest of the residual ridge (black arrowheads)

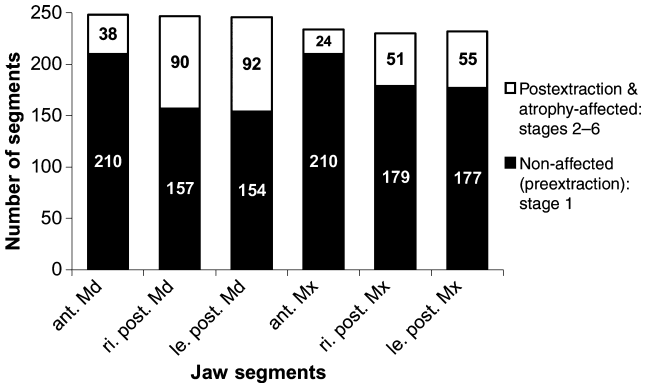

Figure 4 Prevalence of atrophy (stages 2 to 6) and of the non-affected state before tooth loss (stage 1) in anterior and posterior segments of the mandible and the maxilla. Jaw segments: le. = left, ri. = right, ant. = anterior, post. = posterior, Md. = mandible, Mx. = maxilla

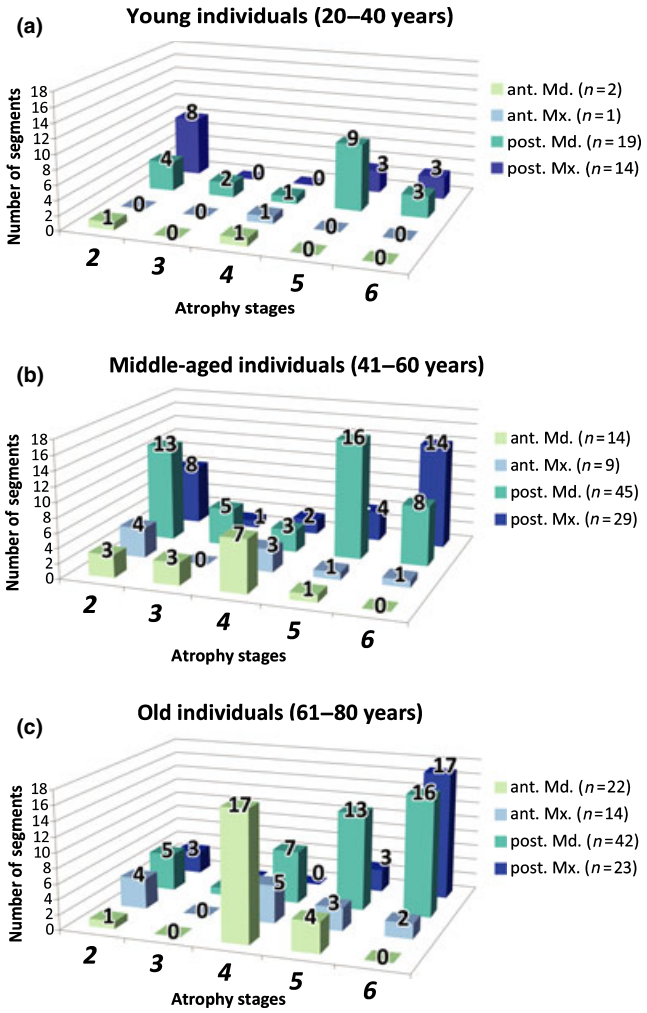

Figure 5 Frequency distribution of the atrophy stages 2 to 6 (postextraction to depressed bone level) in the jaw segments of young (**a**), middle-aged (**b**) and old individuals (**c**). Due to the low frequency of atrophy in young individuals, data were presented as absolute values instead of percentages. By this means, the total increase in affected segments is evident. Atrophy stages: 2 = postextraction, 3 = high well-rounded, 4 = knife-edge, 5 = low well-rounded, 6 = depressed. Jaw segments: ant. = anterior, post. = posterior, Md. = mandible, Mx. = maxilla

area of the former alveolus (Neufeld, 1958; Lammie, 1960). However, Nakamato (1968) doubted this limited extension of such defects as he could not detect a relation between the area of former alveolar sockets and the location of defects. To scrutinise this discrepancy and to check whether a discrimination of exposed trabecular bone and defects is expedient and useful, both features are separately assessed in this study.

In stage I, no defects are present. Stage II characterises a residual alveolar crest with small defects. The cortical layer encircles little trabecular inclusions of e.g. flame-like or channel-like shape.

In stage III, the observed irregularities at the cortical layer are increased in size.

Concave depression of the alveolar crest. In consequence of the loss of several adjacent teeth, the bone tissue in this edentulous space often breaks down. The wound area in this edentulous part of the jaw is too large, so that bone is not able to regenerate up to the height of the former alveolar crest resulting in a depressed level of the residual ridge. Towards the adjacent tooth (or teeth, if existent), the bone level maintains the original height of the alveolar process. So-called concave depressions can be observed at the distal end of the dentate arch (Pietrokovski, 1975) as well as in an edentulous space between remaining teeth.

A segment is classified as stage I if there is no concavity observed.

Stage II describes an edentulous ridge portion with a concave depression looking like a recess in the residual ridge.

Exposed mandibular canal. Excessive resorption of the mandibular residual ridge can lead to a reduction in the alveolar crest towards the mandibular canal (Ulm *et al*, 1989). In the final stage of resorption, the canal containing the mandibular nerve and blood vessels might even lie directly on the surface, solely covered with gingiva in the living individual. In this case, the canal is opened and its borders are well-defined.

In stage I, the mandibular canal is still completely covered with bone. In stage II, the mandibular canal is exposed.

Statistical Analysis

The frequency distribution of ante-mortem tooth loss and the degree of subsequent atrophy and its concomitant features were determined for all segments of the upper and the lower jaw. In case of absence or poor preservation of a segment within an otherwise well-preserved jaw, only segments that could be evaluated accurately were included.

To facilitate evaluation of potential trends between anterior and posterior segments, only the worse affected of both posterior segments was selected for analysis. This pooling of data was based on a high correlation of the analysed parameters between the right and the left posterior segments.

The main purpose of the study is to determine at which frequency the different stages of atrophy are

present after tooth loss has occurred. Thus, segments without atrophy were specified as 'non-affected' and excluded from further calculations. By this means, data about different stages of a certain feature are presented as a percentage of the affected jaw segments only and also as absolute values.

Descriptive analysis, two-sided Fisher's exact tests and cross tabulations were used to assess potential interrelationships between the occurrence and degree of the features.

Results

Frequency and severity of analysed features

Atrophy. Atrophy in at least one segment was present in 120 of the 263 analysed specimens.

The frequency of occurrence of jaw atrophy (including the stage of recent, intravital tooth loss) in this population is presented in Figure 4 for the anterior and posterior segments of the mandible and the maxilla.

Three general trends concerning frequency and distribution of atrophy of the jaw are apparent:

1) Frequency and also severity of atrophy were the lowest in the youngest age group (20–40 years) and increased significantly with advancing age. The occurrence of atrophy is highly correlated with age in all segments of the jaw ($P \leq 0.001$).
2) The mandible (Md.) tended to be worse affected than the maxilla (Mx.), independent of the position of the segment and the age group.
3) The posterior segments (post.) of both the mandible and the maxilla exhibited an increased frequency and also more severe stages of atrophy than the anterior segments (ant.) in all age groups.

Differences in the distribution of the five stages of atrophy, from postextraction state to a depressed level, in the analysed segments of all age groups are shown in Figure 5. Percentages of atrophy stages were calculated from affected segments only (atrophy stages 2 to 6), hereinafter referred to as 100%.

In the youngest age group (20–40 years), atrophy was negligibly rare in the anterior segments. In the posterior segments, atrophy was slightly more frequent. In the older age groups (41–60 years and 61–80 years), a shift from the lower to the higher, more severe atrophy stages was identified. Atrophy stage 4, representing a residual ridge shaped like a knife-edge, was most frequent in anterior segments, particularly of the mandible. By contrast, in posterior segments, atrophy stage 5, characterised by a reduced height of the ridge with a well-rounded crest, and stage 6, a depressed ridge level, were most frequent. Particularly in posterior segments of the mandible, atrophy showed two peaks, one at stage 2 (postextraction) and one at stage 5 (low well-rounded), whereas the stages in between were very rare.

In brief, the knife-edge shape was the predominant stage of atrophy in anterior segments. Stage 5, the low well-rounded ridge, and stage 6, the depressed ridge, played a less prominent role in anterior segments than in posterior segments, where atrophy appeared to attain more severe stages more rapidly with increasing age. Mandibulary posterior segments of old individuals seemed to be the most affected of all.

Fisher's exact tests showed a statistically significant relationship between age group and atrophy stage in anterior segments of the mandible (ant. Md: $P = 0.049$) and posterior segments of the maxilla (post. Mx: $P = 0.034$). In the other segments, no significant correlation was detected (post. Md: $P = 0.095$; ant. Mx: $P = 0.974$).

Exposed trabecular bone at the top of the residual ridge

This feature was more frequent in the mandible than in the maxilla. Posterior segments were more severely and more often affected than anterior segments. Yet, frequency and extent of exposed trabecular bone increased only up to the middle-aged group and decreased again in the oldest age group. (See Table 2)

As a result of the low rate of atrophy in anterior segments of the youngest age group (20–40 years), the feature of exposed trabecular bone at the crest of the residual ridge was also very rare. In posterior segments, the rate of affection was higher with stage III (area of exposed trabecular bone ≥2 mm in width) being the predominant stage in young individuals. In the middle-aged group (41–60 years), the prevalence of a discontinuous cortical layer increased in all segments, posterior segments again being worse affected by stage III. In old individuals (61–80 years), the frequency of stage III decreased compared with the middle-aged group. In contrast, stage I, characterised by a closed cortical layer at the crest, became more frequent and even obtained its peak frequency in this age group.

A significant correlation between age group and the occurrence of different stages of exposed trabecular bone was identified for the posterior segments of the mandible (post. Md: $P = 0.011$) and the maxilla (post. Mx: $P = 0.010$), but not for anterior segments of either jaw (ant. Md: $P = 0.375$; ant. Mx: $P = 1.000$).

Bony defects at the crest of the residual ridge

Bony defects occurred with a higher frequency in the mandible than in the maxilla. Posterior segments were more frequently affected than anterior segments. Similarly to atrophy, frequency increased with advanced age and reached a plateau in the middle-aged (41–60 years) and old group (61–80 years). Indeed, also the frequency of stage I (properly healed alveolus covered by a continuous cortical layer) was increased in old individuals. (See Table 2)

In detail, bony defects in the region of the former alveolus were very rare in anterior segments of young adults (20–40 years), which is the result of low affection by atrophy. In posterior segments, the frequency of poorly healed alveoli was higher, showing a pronounced occurrence of small defects (stage II) in the posterior segment of mandible. The middle-aged group showed a notable increase in stage I in anterior segments and of stage III (large trabecular spots at the crest) in posterior segments. In old individuals, stage III was mainly identified in posterior segments of the maxilla. In the

other segments, the frequency of stage III decreased in favour of an increase in stage I, when compared with the middle-aged group.

Bony defects did not show a statistically significant correlation with age in any jaw segment (ant. Md: $P = 0.699$, post. Md: $P = 0.120$; ant. Mx: $P = 0.312$, post. Mx: $P = 0.218$).

Concave depression of the alveolar crest

With advanced age, the frequency of concave depressions increased. In the mandible, concave depressions occurred more frequently than in the maxilla. Posterior segments were affected with a higher frequency than anterior segments. (See Table 3)

In anterior segments of young adults (20–40 years), concave depressions were almost non-existent. In the middle-aged group (41–60 years), this feature occurred with a frequency of 14.3% in the anterior mandible and 22.2% in the anterior maxilla. In the oldest age group (61–8 years), the anterior segments of the lower and the upper jaws were conversely affected with an increased frequency of 31.8% in mandibular segments and a decrease to 14.3% in maxillary segments.

In posterior segments, the frequency of concave depressions was increased compared with anterior segments in young and middle-aged individuals; the latter representing the most affected age group. Posterior segments of old individuals (61–8 years) showed a decline in frequency.

This tendency was not pronounced enough to show a significant relationship between the occurrence of concave depressions and age in any segment (ant. Md: $P = 0.544$, post. Md: $P = 0.542$; ant. Mx: $P = 0.300$, post. Mx: $P = 0.431$).

Exposed mandibular canal

An exposed mandibular canal (stage II) was a very rare feature. With increasing age, its frequency rose slightly. In the middle-aged group (41–60 years), 3 (6.7%) and in old individuals 7 (16.7%) posterior segments were affected by such signs of excessive resorption.

Correlation of atrophy and concomitant features

Exposed trabecular bone & atrophy. In both the anterior and the posterior segments of the mandible, a statistically significant correlation between atrophy classes and the extent of exposed trabecular bone was identified (ant. Md: $P = 0.004$; post. Md: $P < 0.001$).

The size of exposed trabecular bone areas seemed to decrease with the progress of atrophy in the mandible. (See Table 4)

A continuous cortical layer covering the former alveolus without exposed trabecular bone at the surface (stage I) was the predominant feature in anterior mandibular segments classified as atrophy class 4 (knife-edge ridge). In posterior segments, this feature increased with the progress of atrophy and reached the highest frequency in depressed residual ridge (atrophy stage 6).

Exposed trabecular bone of up to 2 mm in width (stage II) was the most common in posterior segments classified as atrophy stage 5 and 6. Exposed trabecular bone of more than 2 mm in widths (stage III) was

Table 2 Frequency distribution of the features exposed trabecular bone and bony defects stages I to III in the jaw segments of young (20–40 years), middle-aged (41–60 years) and old individuals (61–80 years)

	Mandible		*Maxilla*			*Mandible*		*Maxilla*	
	anterior	*posterior*	*anterior*	*posterior*		*anterior*	*posterior*	*anterior*	*posterior*
Trab. bone	n *(%)*	n *(%)*	n *(%)*	n *(%)*	*Defects*	n *(%)*	n *(%)*	n *(%)*	n *(%)*
20–40 years									
I	0	5 (26.3)	0	4 (28.6)	I	1 (33.3)	3 (15.8)	0	7 (50)
II	1 (50)	4 (21.1)	1 (50)	0	II	2 (66.7)	13 (68.4)	1 (100)	3 (21.4)
III	1 (50)	10 (52.6)	1 (50)	10 (71.4)	III	0	3 (15.8)	0	4 (28.6)
Total	2 (100)	19 (100)	2 (100)	14 (100)	Total	3 (100)	19 (100)	1 (100)	14 (100)
41–60 years									
I	3 (21.4)	10 (22.2)	2 (22.2)	1 (3.4)	I	7 (50)	11 (24.4)	6 (66.7)	11 (37.9)
II	7 (50)	8 (17.8)	2 (22.2)	1 (3.4)	II	7 (50)	17 (37.8)	3 (33.3)	4 (13.8)
III	4 (28.6)	27 (60)	5 (55.6)	27 (33.1)	III	0	17 (37.8)	0	14 (48.3)
Total	14 (100)	45 (100)	9 (100)	29 (100)	Total	14 (100)	45 (100)	9 (100)	29 (100)
61–80 years									
I	7 (31.8)	23 (54.8)	4 (30.8)	6 (25)	I	9 (40.9)	15 (35.7)	11 (78.6)	4 (17.4)
II	13 (59.1)	8 (19)	3 (23.1)	4 (16.7)	II	10 (45.5)	16 (38.1)	3 (21.4)	6 (26.1)
III	2 (9.1)	11 (26.2)	6 (46.2)	14 (58.3)	III	3 (13.6)	11 (26.2)	0	13 (56.5)
Total	22 (100.0)	42 (100)	13 (100)	24 (100)	Total	22 (100.0)	42 (100)	14 (100)	23 (100)

Absolute numbers refer to the number of affected jaw segments. Percentages are given in parentheses.
Trabecular bone: Stage I = closed cortical layer; stage II = exposed trabecular bone ≤2 mm in width; stage III = exposed trabecular bone ≥2 mm in width.
Defects: Stage I = no defects; stage II = small defects; stage III = large defects.

Table 3 Frequency distribution of the features concave depression stages I and II in the jaw segments of young (20–40 years), middle-aged (41–60 years) and old individuals (61–80 years)

	Mandible		*Maxilla*	
	Anterior	*Posterior*	*Anterior*	*Posterior*
Concavity	n *(%)*	n *(%)*	n *(%)*	n *(%)*
20–40 years				
I	2 (100)	9 (47.4)	0	8 (57.1)
II	0	10 (52.6)	1 (100)	6 (42.9)
Total	2 (100)	19 (100)	1 (100)	14 (100)
41–60 years				
I	12 (85.7)	15 (33.3)	7 (77.8)	11 (37.9)
II	2 (14.3)	30 (66.7)	2 (22.2)	18 (62.1)
Total	14 (100)	45 (100)	9 (100)	29 (100)
61–80 years				
I	15 (68.2)	15 (35.7)	12 (85.7)	12 (52.2)
II	7 (31.8)	27 (64.3)	2 (14.3)	11 (47.8)
Total	22 (100.0)	42 (100)	14 (100)	23 (100)

Values are given in absolute numbers and percentages (%).
Stage I = no concave depression; stage II = concave depression existent.

most frequently observed in segments classified as atrophy stage 2 (postextraction). At this atrophy stage, tooth loss did not date back very long; the alveolus already showed first signs of regeneration, but the cortical layer had not closed completely over the trabecular area.

In the maxilla, there was no significant relationship between the dimensions of atrophy and exposed trabecular bone (ant. Mx: $P = 0.310$; post. Mx: $P = 0.655$).

Bony defects at the crest and atrophy. A statistically significant correlation between atrophy stages and the occurrence of bony defects at the crest of the residual ridge was found in anterior segments of the maxilla (ant. Mx: $P = 0.046$) and the posterior segments of both jaws (post. Md: $P < 0.001$, post. Mx: $P < 0.001$).

No defects were most frequently found in postextraction segments (atrophy stage 2) with recent tooth loss where the alveolus is not refilled with bone, and thus has no covering cortical layer. In segments with first signs of atrophy (high well-rounded ridge, stage 3), cortical layers lacking defects were non-existent.

In highly atrophic segments with a low well-rounded (stage 5) or a depressed ridge shape (stage 6), size of bony defects was increased compared with earlier stages of atrophy. Thus, the occurrence of perforations of the cortical layer with trabecular spots at the area of the former alveolus increased with the progress of atrophy. Moreover, cortical layers without defects increased slightly with higher atrophy stages.

Concave depression & atrophy. The presence of concave depressions of the crest of the residual ridge and the extent of atrophy were significantly correlated in all segments of the jaw (ant. Md: $P = 0.022$; post. Md: $P < 0.001$; ant. Mx: $P = 0.043$; post. Mx: $P < 0.001$).

Discussion

Atrophy of the jawbone following tooth loss is a very common oral disease particularly in elderly people. Affected patients suffer not only from alterations of

Table 4 Cross tabulation demonstrating the interrelationship between atrophy stages and the occurrence of different degrees of exposed trabecular bone at the crest of the residual ridge

	Mandible anterior							*Mandible posterior*					
	Atrophy stages							*Atrophy stages*					
	2	*3*	*4*	*5*	*6*	*Total*		*2*	*3*	*4*	*5*	*6*	*Total*
Trabecular bone													
I	1 (10)	0	9 (90)	0	0	10 (100)	I	3 (7.9)	1 (2.6)	5 (13.2)	10 (26.3)	19 (50)	38 (100)
II	0	3 (14.3)	13 (61.9)	5 (23.8)	0	21 (100)	II	1 (5)	1 (5)	3 (15)	10 (50)	5 (25)	20 (100)
III	4 (57.1)	0	3 (42.9)	0	0	7 (100)	III	18 (37.5)	6 (12.5)	3 (6.3)	18 (37.5)	3 (6.3)	48 (100)
Total	5 (13.2)	3 (7.9)	25 (65.8)	5 (13.2)	0	38 (100)	Total	22 (20.8)	8 (7.5)	11 (10.4)	38 (35.8)	27 (25.5)	106 (100)

	Maxilla anterior							*Maxilla posterior*					
	Atrophy stages							*Atrophy stages*					
	2	*3*	*4*	*5*	*6*	*Total*		*2*	*3*	*4*	*5*	*6*	*Total*
Trabecular bone													
I	1 (16.7)	3 (50.0)	1 (16.7)	1 (16.7)	0	6 (100)	I	4 (36.4)	0	0	1 (9.1)	6 (54.5)	11 (100)
II	0	3 (50.0)	2 (33.3)	1 (16.7)	0	6 (100)	II	0	0	0	0	5 (100)	5 (100)
III	6 (54.5)	3 (27.3)	1 (9.1)	1 (9.1)	0	11 (100)	III	15 (30)	1 (2)	2 (4)	9 (18)	23 (46)	50 (100)
Total	7 (30.4)	9 (39.1)	4 (17.4)	3 (13)	0	23 (100)	Total	19 (28.8)	1 (1.5)	2 (3)	10 (15.2)	34 (51.5)	66 (100)

Absolute numbers refer to the number of affected jaw segments. Percentages are given in parentheses.
Atrophy stages: 2 = postextraction; 3 = high well-rounded; 4 = knife-edge; 5 = low well-rounded; 6 = depressed.
Trabecular bone: Stage I = closed cortical layer; stage II = exposed trabecular bone ≤2 mm in width; stage III = exposed trabecular bone ≥2 mm in width.

the anatomic structure of their jaw and loss of function but also from the changes of the facial shape and psychological problems (Atwood, 1971), such as a reduced quality of life (Jokstad *et al*, 2002a,b), feelings of shame, insecurity and decreased attractiveness (Trulsson *et al*, 2002). Thus, the major tasks of prosthetic or implant-supported treatment are, on the one hand, to restore the function including mastication (Şahin *et al*, 2002) and speech, and on the other hand, to preserve the appearance and well-being of the patient (Jokstad *et al*, 2002a,b; Sutton *et al*, 2004; Heydecke *et al*, 2005; Fenlon and Sherriff, 2008; Pan *et al*, 2008).

Despite these undoubtedly positive results of prosthetic treatment, there are diverging opinions as to whether there are severe side effects or not. Several studies have shown that in patients wearing dentures the reduction in the residual ridge was worse than in patients without dentures (Thoma, 1959; Campbell, 1960; Carlsson *et al*, 1969). As an explanatory concept, *pressure atrophy* is particularly plausible for ill-fitting dentures (Carlsson *et al*, 1969; Xie *et al*, 1997a). By contrast, atrophy was also described in patients without prosthetic treatment (Thoma, 1959; Devlin and Ferguson, 1991) supporting the concept of *disuse atrophy*.

Atwood (Atwood, 1971, 1973) assumed that three major groups of co-factors seem to influence the series of events from wound healing of the affected alveolus to resorption of the alveolar process. These are (i) anatomic structure of the jaw, the (ii) biology of the individual (age, gender, hormonal balance etc.) and (iii) mechanical factors such as force applied to the alveolar bone and the influence of prosthetic procedures.

Anatomy and biology of the patient are predetermined. Hence, one factor left to influence the inevitable progress of atrophy is prosthetics. Clinicians can select from a wide variety of prosthetic and surgical treatment options including traditional removable prosthesis, implant-supported dentures or a combination of both. However, the outcome of the chosen therapy is hardly predictable (Carlsson *et al*, 1969). For that reason, and to find the best individual solution for every patient, it is necessary to increase our knowledge about the preserving or destructive effect of prosthetic procedures. Due to the complex pathoaetiology of atrophy (Carlsson, 2004), the true, unbiased influence of prosthetic treatment is difficult to isolate from the broad spectrum of co-factors.

Consequently, there is a growing demand for a purpose-made study design, someway comparable to a knockout experiment in mice. In a knockout study, the effect of a specific gene of unknown function is analysed by deactivating the gene. Thus, differences from the normal condition can be examined and the probable function of the gene can be inferred. In this study, the influence of prosthetic treatment was 'knocked out' by analysing an historical population that did not practice any kind of modern dentistry. By this means, the natural course of disease could be studied excluding the mechanical factor of prosthetics.

Our results proved that atrophy of the jaw after tooth loss definitely does occur in a population without prosthetic treatment, which is in strong agreement with the concept of a multifactorial disease (Atwood, 1971). Furthermore, we could show that even without prosthetics, the residual ridge underwent similar changes in height and shape as described by Atwood (1963) and Cawood and Howell (1988). As expected, old individuals were more frequently and worse affected than younger ones. This seems self-evident as tooth loss usually dates back further in old people's lives. Hence, atrophic processes had less time to proceed in individuals who died at an earlier age. In addition, with increasing age, the loss of several teeth accumulates – a situation that might promote the development of advanced atrophy stages. The prevalence of postextraction segments increased in middle-aged individuals, possibly indicating that this age group is very susceptible to tooth loss.

Notable differences in rate and degree of atrophy were observed between the mandible and the maxilla as well as between anterior and posterior segments in all age groups, which might be the result of differences in anatomy. Posterior segments showed a higher frequency and degree of atrophy than anterior segments, which is in concordance with the fact that molars tend to be lost at an earlier age than anterior teeth. Hence, atrophy might develop in the posterior part of the jaw first and progress further (Ulm *et al*, 2009). Interestingly, in maxillary posterior segments, the residual ridge seemed to skip atrophy stages 3 to 5 resulting rather directly in a depressed bone level (stage 6) in both older age groups. Accordingly, stages 3 to 5 seem to be shorter processes, so that the jaw does not remain long in these stages. By contrast, in the mandible, the transition of stage 5 to stage 6 (depressed) took place with some time lag in the oldest group. This might indicate that in the initial phases of atrophy, mandible offers more resistance to vertical resorption, probably due to the greater thickness of its cortical bone, than the maxilla. However, subsequently the mandible was more frequently and worse affected than the maxilla.

Several studies on denture-wearing patients have attributed the higher atrophy rate in the lower jaw to the smaller surface and the unfavourable shape of the mandibular ridge on which mechanical loads are applied to in edentulous patients (Tallgren, 1972; Tallgren *et al*, 1980; Bodic *et al*, 2005). By contrast, the maxilla is thought to resist loads of dentures more successfully, because of the larger supporting surface of the hard plate (Tallgren, 1972). In the study, prosthetics cannot be held responsible for the higher degree of resorption of the lower jaw. That leads us back again to other factors that might be involved. It seems plausible that forces through chewing and swallowing are applied to the residual ridge also in non-denture wearers. Alterations in frequency, intensity and direction of such forces might lead to an imbalance of the neuromuscular stability (Budtz-Jörgensen, 1996; Kingsmill, 1999) and thus to an impaired activation of osteoblasts and osteoclasts, probably resulting in a discrepancy of

resorption (Atwood, 1962). As recently shown by Rawlinson *et al* (2009) also differences in gene expression of developmentally and/or functionally distinct skeletal sites play a role in their response to mechanical stimuli. The described differences in gene expression profiles between the skull vault and limb bones were attributed to cell (osteoblast) populations of distinct embryonic lineages. This developmental 'positional identity' of bone cells has been shown to determine cell activity and matrix characteristics probably leading to differences in their susceptibility to osteoporosis and adaptation to mechanical strain.

In anterior segments of either jaw, a knife-edge shape of the residual ridge was predominant. This stage 4 is particularly problematic for implantology as the width of the crest is insufficient for the insertion of endosseous implants. Heights of 6 mm and 10 mm as well as widths of 6 mm and 5 mm are regarded as minimum dimensions for successful implantation in the maxilla and the mandible respectively (Neukam and Kloss, 2001). Moreover, well-rounded ridges with reduced height and ridges with a depressed bone level (stage 5 and 6), which are predominant in posterior segments, do not meet the standard criteria for implantation and require pretreatment – complex surgical procedures, such as the removal of the thin bone structure (Nishimura and Atwood, 1994) or/and alveolar ridge augmentation by bone graft techniques (Ulm *et al*, 1995; Coulthard *et al*, 2002; Bodic *et al*, 2005). Even in the most recent systematic reviews on the subject (Esposito *et al*, 2006; Aghaloo and Moy, 2007; Rocchietta *et al*, 2008), there are no indications which frequencies of cases that necessitate bone augmentation procedures a surgeon has to expect. According to the results of this study, implantologists might figure that up to 75% of elderly patients require some kind of surgical pretreatment (depending on the region of the jaw). A comparison with modern populations is hardly possible as data on prevalence are very rare, and information about the sample is mostly lacking. Denissen *et al* (1993) analysed a sample of 61 middle-aged patients (40–65 years of age) treated with prostheses. A total of 51.6% of these dried edentulous mandibles showed a knife-edge shape (atrophy stage 4) compareable to a prevalence of 50% in ant. Md. segments of middle-aged individuals of this study. Al-Faleh (2009) found a similar prevalence of 46.5% in an inhomogeneous sample of 30 17–67 year-old patients. Pietrokovski *et al* (2007) studied a sample of 123 human edentulous dried skulls of unknown age, gender and treatment and described 38% of these maxillary and mandibulary ridges to be knife-edged.

Another aspect of clinical relevance is the bone quality of the cortical layer covering the crest of the residual ridge. In fact, a large amount of continuous cortical bone provides good primary stability of the implant and hence decreases the risk of implant failure (Rabel *et al*, 2007). Alterations in cortical layer, such as areas of exposed trabecular bone and osseous defects might have negative implications for the insertion of endosseous implants (Pietrokovski *et al*, 2007). A closed cortical layer offers more resistance to the bur used than does a crest with macroscopic openings and exposed trabeculae. Thus, there is a risk of overdosing the force at the transition from the outer surface to the trabecular compartment when drilling a hole for the implant (Pietrokovski, 1975).

According to our observations, the differentiation based on the localisation at the crest (Neufeld, 1958; Lammie, 1960; Atwood, 1971; Pietrokovski, 1975) was not unequivocal, so that in many cases, trabecular spots were classified as both exposed trabecular bone and bony defects.

Yet, the results demonstrated that exposed trabecular bone and defects at the crest do occur in absence of prosthetic treatment. Moreover, an exposed mandibular canal was detected in a few individuals as a result of excessive atrophy. In the analysed population, the prevalence of an exposed mandibular canal was 6.7% for 41–60 year-old individuals and 16.7% for individuals over 61 years of age. Xie *et al* (1997b) reported an even higher prevalence of 27% in a sample of 128 76–86 year-old patients. The exact localisation of the mandibular canal is essential to know for an implantologist when planning and inserting endosseous implants in the atrophic mandible (Ulm *et al*, 1993; Lindh *et al*, 1995), as an injury would implicate neurosensory damage or even loss of sensation (Ulm *et al*, 1993).

In the present study, some limitations were encountered. As only individuals with a sufficient state of preservation were included, the sample might be biased to some extent (Meinl *et al*, 2009). Due to the lack of information about the time point of tooth loss and hence of the duration being (partly) edentulous, no conclusions about the rate of atrophy (i.e. the amount of bone being resorbed per time unit) could be drawn. The observed features within the jawbone mirror only the conditions at time of death. The role of osteoporosis in the development and progression of jaw atrophy is still controversially discussed (Hildebolt, 1997; Bollen *et al*, 2004; Felton, 2009; Yüzügüllü *et al*, 2009). However, due to the large sample size of 263 jaw specimens, it was economically not possible to study systemic bone disorders, such as osteopenia and osteoporosis by means of x-rays or DEXA. Several studies reported that osteoporosis might promote ridge resorption (Bays and Weinstein, 1982; Kribbs *et al*, 1983; Hirai *et al*, 1993) and tooth loss (Nicopoulou-Karayianni *et al*, 2009) while others failed to demonstrate a correlation (Klemetti *et al*, 1993a,b; Bollen *et al*, 2004). Devlin and Horner (2007) state that age-related bone loss of the jaw resulting in cortical porosity and a reduced BMD and BMC (Hildebolt, 1997) has to be clearly distinguished from external alterations of the mandibular alveolar process due to tooth loss. Moreover, a recent review of Slagter *et al* (2008) reported no more than a minimal evidence for a relation between osteoporosis and jaw atrophy. Future studies evaluating radiographic data of atrophic jaws would be of great value to shed light onto this issue. So far, most published studies dealing with atrophy after tooth loss did not meet modern scientific standards, mainly being written in a descriptive style, and lacking information on the sample. Moreover, they

had been limited to the assessment of single features of this complex oral disease. Here we present a study analysing concomitant features described in literature and questioning/challenging the consistency and applicability of the classification system. The strength of this study lies in the analysis of the natural history of atrophy in a large sample of a mediaeval population, excluding the influence of prosthetic treatment.

Results demonstrated that atrophy evidently did occur in the absence of prosthetics and followed the pattern of resorption described for modern populations (Atwood, 1963; Cawood and Howell, 1988). Concomitant features, i.e. exposed trabecular bone, osseous defects, concave depression and exposed mandibular nerve, were present and often correlated with the occurrence of advanced atrophy stages. A differentiation of exposed trabecular bone and bony defects at the crest of the ridge appears not to be useful as they were hardly distinguishable and their developmental mechanisms might overlap to some extent. Yet, their presence might be of clinical relevance for the planning and realisation of dental implants and prostheses.

Above all, our findings present basic information about the pathology of jaw atrophy and provide a deeper insight into the structural characteristics of this common oral disease. Further research in this field is required to study the effect and relative weights of other factors involved in this multifactorial process.

Conclusions

1. Atrophy of the jawbone following tooth loss is a multifactorial disease that is not attributable to prosthetics alone, as its occurrence is observed in a population without modern prosthetic treatment.
2. The residual ridge undergoes a series of changes in shape and height following the pattern of resorption described for modern populations. The severity of these alterations is associated with the age of the individual and the region within the jaw.
3. Atrophy is frequently related to structural alterations of the covering cortical layer, which all together might have clinical relevance for planning and realisation of dental implants and prostheses.

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Conflict of interest

None declared.

Authors Contributions

KMR was the primary author of the manuscript, was involved in the development of standardised and calibrated evaluation criteria, analysed and interpreted the results, reviewed literature and submitted the manuscript. CDH performed the statistical analyses and was involved in the interpretation of the results. WRL participated in study design, establishment of methods and performed practical evaluation and classification, being blinded to individuals' age and gender. CU and GW supervised the progress of the project, reviewed the manuscript critically and helped to finalise the manuscript. GW also provided funding for the study. ST designed and planned the study and monitored the progress of the project. He was also involved in the development of standardised and calibrated of evaluation criteria and in data interpretation and helped to prepare the study.

All authors have contributed to, seen and approved the manuscript.

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