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# Cross-regional assessment of dental pathologies: Evidence for predicting oral health and intra-vitam stress among Ghanaian ancestors

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**KEYWORDS**
Dental pathologies;
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Dietetics;
Oral health

**Summary**
*Background and aim.* — WHO's definition of oral health extends beyond the state of the teeth and dental pathologies. However, dental pathologies form a significant component of oral health and impact a person's quality of life. Thus, dental pathologies can elucidate intra-vitam oral health and dietetics of ancient or contemporary populations. This paper aimed at predicting the oral health and intra-vitam stress of past inhabitants across five regions in Ghana from dental pathologies.
*Method.* — The study employed the mixed research approach involving standard anthropological observational scoring systems to examine five dental pathologies: caries, calculus, abscess, enamel hypoplasia, and cementum hyperplasia. The scores were translated into absent (0)/present (1) pathology categories and the frequency was calculated in an Excel sheet.

*Abbreviations:* Ab, Abscess; AMP, Anterior medial palatine; CEJ, Cemento enamel junction; CER, Caries expression rate; Cl, Calculus; Cr, Caries; DPs, Dental pathologies; Eh, Enamel hypoplasia; Hc, Hypercementosis; IN, Incisive; LEH, Linear enamel hypoplasia; MNI, Minimum number of individuals; PMP, Posterior medial palatine; TP, Transverse palatine; WHO, World Health Organization; XRF, X-ray fluorescence.

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*Results and discussion.* — Dental pathologies constituted 60% of the total pathologies recorded on human remains, with calculus being dominant (35%), followed by caries (30%), enamel hypoplasia (21%), abscess (9%), and hypercementosis (7%). In general, the individuals from Sekondi presented the highest percentage of dental pathologies, i.e., 69% caries, 66% calculus, 50% abscess, 44% enamel hypoplasia, and 5% hypercementosis. Begho and Yapei individuals had equal percentages of abscesses, enamel hypoplasia, and calculus, while hypercementosis was relatively low in all cases. More than one individual combined multiple dental pathologies, while 6 had none. Grounded on the high caries and calculus expression rates and their direct influence on other dental pathologies, the individual's oral health could have been compromised by dietary patterns, developmental factors, functional stress on the teeth, and inadequate oral hygiene.
*Conclusion.* — The assessment of the dental pathologies of these Ghanaian ancestors offered valuable insights into dietetics, oral health, and some intra-vitam developmental and functional stress on the teeth.


## Introduction

One particular intra-vitam concern is oral health which has an index on the quality of life of individuals/populations [1]. For clinicians and bioarchaeologists, dental pathologies are direct pointers for the oral health of ancient and recent populations. Assessing oral infections and dental pathologies can shed light on a spectrum of customs, diseases, and dietary composition to aid the reconstruction of individual and group dietetics [2] and health. For instance, caries and calculus data have been used to infer population dietetics as indirect reconstructions of carbohydrate intake among populations [3]. According to WHO [1], orofacial pain, oral infections, and dental pathologies impact masticatory processes, speaking, and smiling, as well as an individual's psychosocial well-being. The examination (including sufficient documentation) of the dental pathologies of ancient individuals/populations is as important as that of contemporary groups because it provides a comparative platform for the evolutionary evaluation of causes and severities to complement the understanding of dental pathologies and oral health over time.

In this paper, we attempt to predict, by examining dental pathologies, the intra-vitam oral health of 21 Ghanaian ancestors across five sites and provide a glimpse into their nutritional and occupational stress. To illuminate the oral health and stress of the past Ghanaian populations, the objectives were to determine the expression rate of each pathology (calculus, caries, abscess, enamel hypoplasia, and hypercementosis) per site and individual and to discuss the characteristics and causes and effects of the respective pathologies. Calculus, for instance, preserves micro-remains of food, oral microbiomes, and non-dietary fiber, whose analysis sheds light on the health and dietary patterns of individuals and populations [4—7]. Additionally, caries expose bacterial activity and oral hygiene of individuals. Moreover, abscess, the periapical offshoot of pulpitis, results from caries, dental infections, and extreme dental wear [8] (which compounds the spread of bacteria in the alveoli). Lastly, enamel hypoplasia reflects individuals' developmental and nutritional stress, while hypercementosis relates more to occupational and masticatory stress on teeth.

## Materials

An MNI (minimum number of individuals) of 21 individuals was subjected to the study. They were excavated by different researchers in the early and mid-20th century from sites in the Western (Sekondi), Bono (Begho), Savannah (Ntereso & Yapei), and Northeast (Yikpabongo) regions of Ghana. The remains have since been stored (in wooden boxes) in the Museum of Archaeology at the Department of Archaeology and Heritage Studies, University of Ghana [9]. From Sekondi; 4 56'2.40'' N-1 42'49.32'' W (Fig. 1), the individuals (< 25% & 25%—75% complete) were excavated from an area near Fort Orange in 1954 associated with ostrich shell beads, greenstone celts, and some stone beads. The remains have not been dated. In the Bono region; 7 51'00'' N 2 29'00'' W (Fig. 1), the Begho individuals (25%—75% & > 75% complete) were excavated in 1970 in the Brong Quarter of the site. Some of the individuals were associated with potsherds and animal (dog) bones. Although the remains have not been dated, site chronology places them between the 10th—19th centuries. In the Savannah region are Ntereso; 9 7' 46'' N 1 12' 17'' W and Yapei; 9 9' 4'' N 1 8' 59'' W (Fig. 1). The context of the individuals (25%—75%, and 25%—75% & > 75% complete, respectively) is unavailable but probably excavated in 1960 and 1970, respectively. While one Yapei individual dated to the mid-18th and early 19th century BC, the Ntereso remains have not been dated. The Yikpabongo remains were excavated in 2007, although not dated, site chronology places it between the 6th—12th century BC.

The Sekondi individuals had completely isolated teeth (partially damaged crowns and roots). From Begho, the middle adult male BG'21-F 73.116 (70 B1) had complete arches, and the middle adult male (?) BG'21-E 73.115 (Bg. 70, E) had all teeth but 24, 26, 31, 41, and 36, 45 & 46

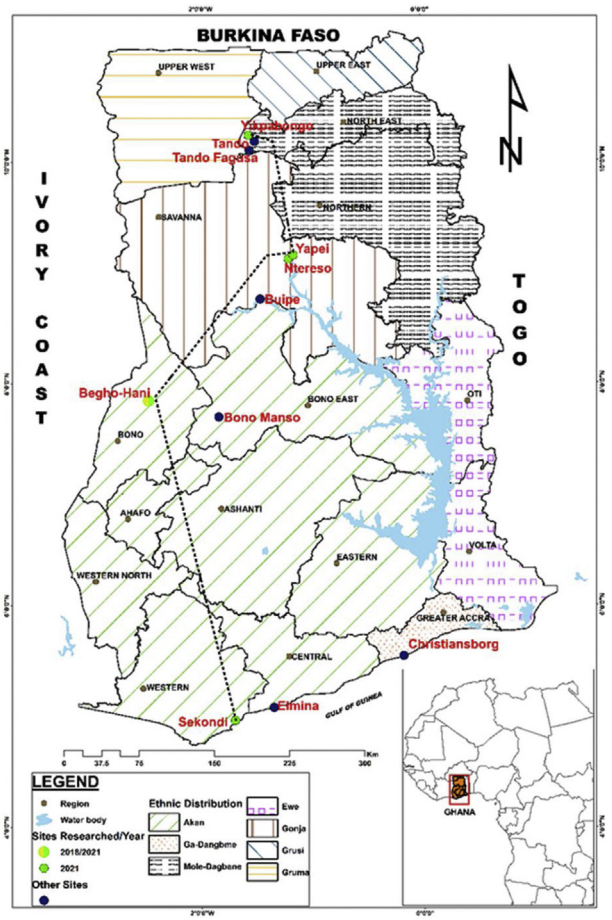


**Figure 1.** Map of Ghana.

(crowns destroyed by caries). The teeth of the other individuals were isolated with damaged roots (some) impossible to side or situate into the upper or lower arches. The Yikpabongo calvarium was accompanied by two extensively damaged isolated teeth (impossible to assess pathologies). Also, the teeth of some individuals within the assemblage were unavailable. Skeletal collections including neonate, baby, & child of < 25% complete were exempted.

## Methods

Combining the mixed research method (quantitative and qualitative) with bioanthropological research standards or scoring systems, the dental pathologies: caries, calculus, abscess, and hypercementosis were assessed through non-metric observation of the maxillary and mandibular teeth. Caries were observed on the occlusal and interproximal surfaces, and at the root & cemento-enamel junction of the teeth. The absence, presence, and extent of the pathologies were coded according to Buikstra & Ubelaker [10]. The presence and quantity/accumulation degree (small, medium, and large) of calculus were assessed on teeth in occlusion and isolated at the buccal, lingual, and interproximal surfaces and coded according to Buikstra &Ubelaker [10]. Abscesses (fenestrations or drainage channels in the alveolar at the root apex) were observed on the buccal and lingual surfaces of the maxillae and mandibles, laterally and bilaterally, according to Buikstra & Ubelaker [10]. We acknowledge, however, that visual identification of abscesses based on the presence and absence of perforations may limit the prevalence assessment of the lesion among populations [11] because of the possibility of non-external fistulas. Lastly, dental enamel hypoplasia was recorded based on the number of hypoplastic lines and pits on the teeth [12], and hypercementosis was noted based on the physical observation of the appearance of teeth roots for abnormal thickness and opacity.

## Results

All the results are displayed in Table 1 and Figs. 2—5. Dental pathologies constituted 60% of the total pathologies recorded on the individuals through the bioanthropological assessment. Of this percentage, calculus was dominant, accounting for 35% in 15/21 individuals, followed by caries which constituted 30% in 13/21 individuals. Caries expression rate (CER) in the skeletal assemblage of all sites, but Yikpabongo was 18 on the occlusal surface, 7 on the interproximal region, 11 at the cemento-enamel junction, three at the root level, and three cases of teeth partially destroyed by caries. Caries were expressed more in molars and premolars, and calculus was on either the buccal or the labial teeth or both. The rest of the pathologies followed as enamel hypoplasia, abscess, and hypercementosis in that order of prevalence. In general, the Sekondi individuals recorded the highest percentage of dental pathologies, i.e., 69% caries, 66% calculus, 50% abscess, 44% enamel hypoplasia, and 5% hypercementosis. Begho and Yapei individuals had equal percentages of abscesses, enamel hypoplasia, and calculus, while hypercementosis was relatively low in all cases. Six of the 21 individuals had none of the dental pathologies, four combined caries and calculus, three combined caries, enamel hypoplasia, and calculus. Two individuals had both enamel hypoplasia and calculus, while six combined up to four dental pathologies on multiple levels of the teeth.

The ratio of males to females with two or multiple types of dental pathologies, including caries and calculus, was 4:6. However, the sex of some of the individuals was determined using only the skull (designated as male?/female?). As a result of the vast percentage of undeterminable cases, we could not conclude the sex prevalence of dental pathologies. The ratio of individuals with two or more dental lesions whose sex could not be determined and undeterminable individuals with no pathologies was 5:6. Due to the irregularities in age distribution, a correlation between the age categories and dental pathologies could not be established discretely. Nevertheless, cumulatively, all adults in the study sample except teeth lost antemortem and postmortem had two or more different dental lesions, the adolescent in the group had minimal calculus and two hypoplastic lines on tooth 12, while the sub-adults presented no pathologies.

## Discussion

Caries result from prolonged bacterial activity in the mouth and the consequent demineralization or degradation of the enamel, dentine, and roots of the teeth [2]. WHO, in 2017,

estimated caries as globally prevalent [1], and Hillson notes caries as a ''pervasive and important disease'' [3]. Some individuals had multi-level caries and a combination of calculus, abscess, and enamel hypoplasia (Fig. 4). The CER is high among populations with increased consumption of carbohydrate-rich foods [13,14], sugar-rich foods, and a high intake of fruits (including dates), honey, and starchy foods/cereals [15]. This custom is not unusual across the

**Table 1** Osteobiographies and details of the dental pathologies presented by the individuals.

| | |
|---|---|
| 1. | |
| Site & ID | Sekondi SK'21- A 73.80 |
| MNI | 1 |
| Age | Middle adult<br>25—29 (IN + PMP); or above? |
| Sex | Female? |
| Dental pathologies | Calculus: minimal (stage 1) on 27, medium (stage 2) on 17, 21, and large (stage 3) on 26. On isolated un-sided molars: stages 1 & 2<br>Caries: at the interproximal surface of 21 and developing root caries on one of the isolated molars<br>Abscess: none<br>Enamel hypoplasia: none<br>Hypercementosis: none |
| 2. | |
| Site & ID | Sekondi SK'21-C 73.88 |
| MNI | 1 |
| Age | Middle-old adult (based on available dentition and extensive wear) |
| Sex | Undeterminable |
| Dental pathologies | Calculus: minimal (stage 1) on 16, 23, 33, 36, 42, 45 & 47, medium (stage 2) on 13, 24, 37 & 48, and large (stage 3) on 17 & 27<br>Caries: on the occlusal surface of 14, 15 &16, and at the interproximal surface of 47<br>Abscess: none<br>Enamel hypoplasia: 1 hypoplastic line on 37, 2 hypoplastic lines on 13, 23<br>Hypercementosis: general in available teeth |
| 3. | |
| Site & ID | Sekondi SK'21- D 73.81 |
| MNI | 2 (two right halves of the temporal bone) |
| Age | Young-old adult (two different wear categories for different dentitions: it is uncertain which individual the teeth belong to) |
| Sex | Female? (for both individuals based on two gracile right-sided mastoids) |
| Dental pathologies | Calculus: minimal (stage 1) on 16 & 47 to medium (stage 2) on 48, unobservable for the rest (teeth were absent)<br>Caries: at the cemento-enamel junction of 46<br>Abscess: none<br>Enamel hypoplasia: none<br>Hypercementosis: none |
| 4. | |
| Site & ID | Sekondi SK'21-E 73.82 |
| MNI | 1 |
| Age | Adolescent |
| Sex | Undeterminable |
| Dental pathologies | Calculus: minimal (stage 1) on 42; Absent on the labial and buccal surfaces but unobservable on the occlusal and interproximal surfaces due to soil<br>Caries: absent on the labial and buccal surfaces and unobservable on the occlusal and interproximal surfaces<br>Abscess: absent on the labial and buccal surfaces and unobservable on the lingual surfaces due to soil endo-cranial<br>Enamel hypoplasia: 2 hypoplastic lines on 12<br>Hypercementosis: none |
| 5. | |
| Site & ID | Sekondi SK'21-F 73.94 |
| MNI | 1 |
| Age | Adult |
| Sex | Male? |

Table 1 (*Continued*)

| | |
|---|---|
| Dental pathologies | Calculus: minimal (stage 1) on 15, 25, 37 to medium (stage 2) on 16 and 33<br>Caries: at the cemento-enamel junction of 25<br>Abscess: on the buccal surface of 35<br>Enamel hypoplasia: none<br>Hypercementosis: none |
| 6. | |
| Site & ID | Sekondi SK'21-G 73.83 |
| MNI | 2 (from different mandible pieces in size, color, and morphology) |
| Age | 1st individual — middle adult? (based on permanent dentition available and significant wear, code 8 for PM1 & PM2. However, code 2 for M3)<br>2nd individual — adult? |
| Sex | Female? & undeterminable for the 2nd |
| Dental pathologies | Calculus: minimal (stage 1) on 16, 17, 22 & 27 and medium (stage 2) on 25<br>Caries: on the occlusal surface of teeth 25 and 36<br>Abscess: none<br>Enamel hypoplasia: none<br>Hypercementosis: none |
| 7. | |
| Site & ID | Sekondi SK'21-H 73.84 |
| MNI | 1 |
| Age | Young adult (significant dental wear)<br>25-29 (IN + PMP); or above? |
| Sex | Female? |
| Dental pathologies | Calculus: minimal (stage 1) on possible 16, 17, possible 37, 38, 47 & 48. Medium (stage 2) on probable 18, 26 & 46 and large (stage 3) on the surface of probable 27 (the surface is covered by what looks like calculus)<br>Caries: on occlusal surfaces of 16, 27 (the surface is partly covered by what looks like calculus), and 36<br>Abscess: none<br>Enamel hypoplasia: 1 hypoplastic line on 36 but absent on available teeth<br>Hypercementosis: present on available teeth roots |
| 8. | |
| Site & ID | Sekondi SK'21-K 73.85 |
| MNI | 1 |
| Age | Adult |
| Sex | Undeterminable |
| Dental pathologies | Calculus: none<br>Caries: none<br>Abscess: none<br>Enamel hypoplasia: none<br>Hypercementosis: none |
| 9. | |
| Site & ID | Sekondi SK'21-L 73.86 |
| MNI | 1 |
| Age | Middle adult (based on permanent dentition and extensive wear observed) |
| Sex | Female? |
| Dental pathologies | Calculus: minimal (stage 1) on 14,15, 17,18, 22, 27, 34, 35—38, 42 & 47. Medium (stage 2) on 13, 21, 41, 46, and 48. Large (stage 3) on 11<br>Caries: at the cemento-enamel junction of 13, mirror caries at the cemento-enamel junction of 25 & 26 (revealed by CT scan), and on the occlusal surface of 42<br>Abscess: none observed<br>Enamel hypoplasia: 2 hypoplastic lines on 13 & 41(although the surface is partially concealed by calculus)<br>Hypercementosis: none |
| 10. | |
| Site & ID | Sekondi SK'21- M 73.87 |

Table 1 (*Continued*)

| | |
|---|---|
| MNI | 1 |
| Age | Middle-old adult? (based on significant crown loss of available teeth) |
| Sex | Undeterminable |
| Dental pathologies | Calculus: minimal (stage 1) on 14, medium (stage 2) on 25, unobservable for the rest (teeth too damaged to assess)<br>Caries: at the occlusal and interproximal surface of 15 and 24, respectively<br>Abscess: none<br>Enamel hypoplasia: none (most teeth had crowns too damaged to assess)<br>Hypercementosis: none |
| 11. | |
| Site & ID | Sekondi SK'21-73.89 (Pit 1 Extension) |
| MNI | 2<br>From differently sized and colored (black and light brown) bone fragments, especially two right pieces of mandibles |
| Age | Adults |
| Sex | Male? & Female? |
| Dental pathologies | Calculus: minimal (stage 1) on 27, 28, and 46. Medium (stage 2) on 11 (for the male?)<br>Caries: on the occlusal surface of 46 (for the male?)<br>Abscess: none observed<br>Enamel hypoplasia: none observed<br>Hypercementosis: none |
| 12. | |
| Site & ID | Begho BG'21-F 73.116 (70 B1) |
| MNI | 2 |
| Age | Middle adult 40—44 (based on auricular surface morphology)<br>45.2 (based on only vault composite scores) |
| Sex | Male |
| Dental pathologies | Calculus: minimal (stage 1) on 11, 15, 16, 18, 21, 23, 27, 32, 45, 46, 47 & 48. Medium (stage 2) on 13, 14, 25, 26, 28, 31, 33, 34, 35, 41, 42, 43 & 44. Large (stage 3) on 24 & 36<br>Caries: none<br>Abscess: none observed<br>Enamel hypoplasia: 2 hypoplastic lines on 18, 33, 34, 43, and one hypoplastic line on 16 & 36 (although calculus is concealing a significant part of the crown surface)<br>Hypercementosis: none |
| 13. | |
| Site & ID | Begho BG'21-E 73.115 (Bg. 70, E) |
| MNI | 1 |
| Age | Adult |
| Sex | Male? |
| Dental pathologies | Calculus: minimal (stage 1) on 18, 17, 15, 14, 21, 22, 23, 25, 27, 32, 31& 47. Medium (stage 2) on 13, 16, 28, 38, 37, 35, 34, 33, 42, 43, 44 & 48<br>Caries on the occlusal, interproximal and buccal surfaces at 16, 15, 14, 11, 22, 37, 36, 45, 46 & 47<br>Abscess: labial and buccal perforations at 12 and 26, respectively (Fig. 6). There were also lingual perforations at 45 & 35 with buccal drainage channels at the level of the mandibular foramen. This, coupled with the infection at the level of the teeth, is the cause of the porosity in the mandibular region<br>Enamel hypoplasia: one hypoplastic line on 33 and two hypoplastic lines on 32 (signs of stress or malnutrition during life)<br>Hypercementosis: none |
| 14. | |
| Site & ID | Begho BG'21-A 73.118 (Bg. 70, A) |
| MNI | 1 |
| Age | 6 years |
| Sex | Undeterminable |
| Dental pathologies | None |
| 15. | |
| Site & ID | Ntereso NT'18-73.90 |

Table 1 (*Continued*)

| | |
|---|---|
| MNI | 1 |
| Age | Adult |
| Sex | Undeterminable |
| Dental pathologies | Calculus: unobservable<br>Caries: unobservable on teeth in occlusion but absent on isolated teeth<br>Abscess: none<br>Enamel hypoplasia: none<br>Hypercementosis: none |
| 16. | |
| Site & ID | Ntereso NT'18-73.91 |
| MNI | 1 |
| Age | Adult |
| Sex | Undeterminable |
| Dental pathologies | Calculus: minimal (stage 1) on possible 35, 43, and un-sided incisors and premolars. Medium (stage 2) on possible 33, 42, 45, 47, and 48. Large (stage 3) on possible 31, 41<br>Caries: on the occlusal surface of 43 & 48 at the cemento-enamel junction of probable upper premolar, at the cemento-enamel junction of 47 of the interproximal aspect, and at the cemento-enamel junction of 23 & 33<br>Abscess: none observed<br>Enamel hypoplasia: two hypoplastic lines on an un-sided upper premolar<br>Hypercementosis: none |
| 17. | |
| Site & ID | Yapei YP'18-73.106 |
| MNI | 1 |
| Age | 34.7—41.1 based on the vault and lateral-anterior sutural composite scores<br>40—44 based on the auricular surface morphology |
| Sex | Female |
| Dental pathologies | Calculus: minimal (stage 1) on 43 & 44<br>Caries: on the occlusal surface and at the cemento-enamel junction of 17<br>Abscess: none observed<br>Enamel hypoplasia: two hypoplastic lines on 43. Indicative of intra-vitam stress or malnutrition<br>Hypercementosis: none |
| 18. | |
| Site & ID | Yapei YP'18-73.105 |
| MNI | 1/2 |
| Age | Significant wear on teeth in occlusion and alveoli resorption of 16-18 suggests middle-old adult<br>25—29 based on the assessment of (IN + PMP) and 25—40 based on (IN + PMP + TP), suggesting young-middle adult<br>40—44 According to auricular surface morphology |
| Sex | Female (based on the presence of the peri-auricular sulcus) |
| Dental pathologies | Calculus: minimal (stage 1) on 13 & 14. Medium (stage 2) on 15; and isolated M1 &M2: M1 = 1 and M2 = 2<br>Caries = mirror caries at the cemento-enamel junction of 14 & 15, and on an isolated un-sided M1 is a non-carious pit or pulp exposure on the lingual surface of the crown. The alveolar margin is extensively resorbed<br>Abscess: at the buccal surface of 14<br>Enamel hypoplasia: one hypoplastic line on 14 & 15 and 2 hypoplastic lines on 13. Indicative of intra-vitam stress or malnutrition<br>Hypercementosis: present on available teeth |
| 19. | |
| Site & ID | Yapei ID: foetus |
| MNI | 2/2 |
| Age | 36 weeks |
| Sex | Undeterminable |
| Dental pathologies | None |
| 20. | |
| Site & ID | Yapei YP'18-73.102 |

Table 1 (*Continued*)

| | |
|---|---|
| MNI | 1 |
| Age | Adult |
| Sex | Undeterminable |
| Dental pathologies | Unobservable |
| 21. | |
| Site & ID | Yikpabongo YK'21, Unit 1/C3-4 |
| MNI | 1 |
| Age | Adult? |
| Sex | Undeterminable |
| Dental pathologies | Unobservable |

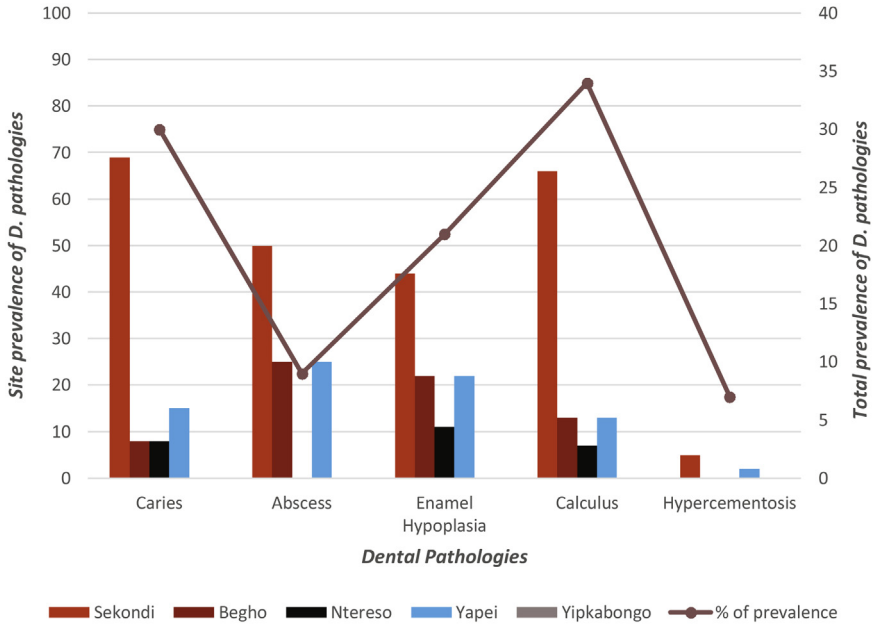


**Figure 2.** Site prevalence of dental pathologies combined with the total prevalence of each dental pathology.

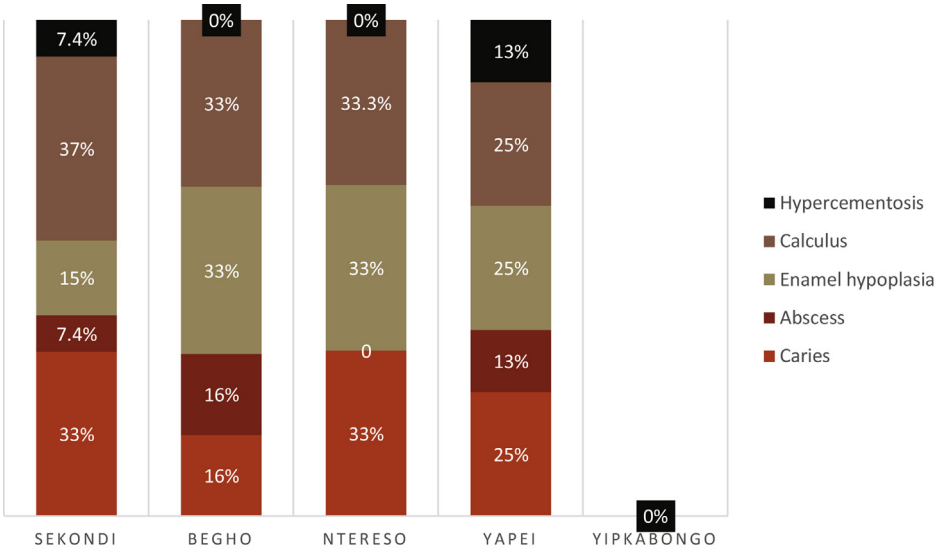


**Figure 3.** The contribution of each dental pathology represented in each site.

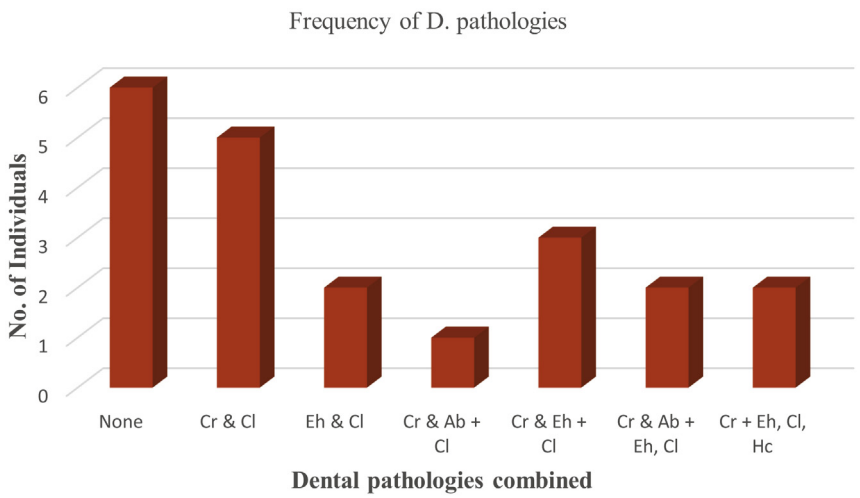


**Figure 4.** The number of individuals with no or combining multiple types (2+) of dental pathologies among 21 individuals.

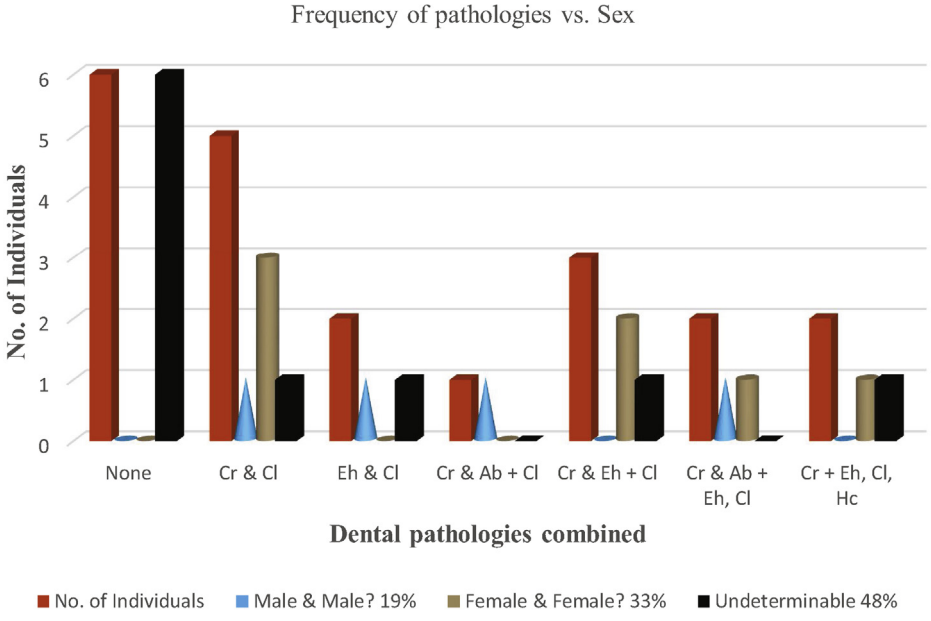


**Figure 5.** Frequency of dental pathologies juxtaposed by the sex of the individuals in the assemblage.

regions in Ghana. Note that both caries and calculus are actively linked to high carbohydrate intake [16]. Extensive caries at the interproximal region and CEJ result from plaque build-up of/from non-abrasive diets at the respective areas [17], whereas less occlusal surface caries represent a relatively coarse diet [18].

In our study, occlusal surface caries were of minimal and extensive expressions. However, the former was more than the latter. The diet composition of most Ghanaian foods is less coarse across all the regions, made from processed grains/cereals like wheat, rice, maize, millet, and tuber crops like cassava, yam, and plantain. Some dishes are also prepared from fermented carbohydrate-rich cereals and crops. This dietary orientation is not any different from paleo-diets, with reference to the $C_4$ plant protein orientation of a mid-18$^{th}$ – early 19$^{th}$ -century Yapei individual from a $^{13}C$ isotopic value of −10.5‰. Additionally, by extension and via the trajectory of trade with a possible blend of $C_3$ plants including, rice, wheat, and barley, paleo-diets of the Ghanaian region may not have changed much. According to Dodd [19], cereals like rice, maize, oats, and wheat are cariogenic foods, with wheat being the topmost. Moreover, carious lesions are equated to the continued consumption of fermentable carbohydrates [13,20]. This likely explains the significant presence of interproximal and CEJ caries. Some studies have attributed high carious lesions in females to the increased peroxidase enzyme and other oral enzyme activities with the elevation in oestrogen levels during pregnancy and menstruation [21–23]: notably, the late gestation phases [24] and before ovulation [25]. These enzymes impact bacterial cells in the mouth, leading to a decrease in oral pH, thus, lower than the critical pH of enamel −5.5 [26]. This extremely acidic environment contributes to enamel erosion and poor oral health.

Furthermore, caries intensify under conditions of induced hydroxyapatite demineralization. For instance, unsaturated

solutions (with pH below the 5.5 critical pH of enamel) such as gastric juice (< 2.0) [26] and fruits (juices) with pH less than 3 [27]. Dawes [27] adds that the extent of enamel demineralization is influenced by individual differences in the concentrations of calcium, phosphate, and hydroxyl ions in oral solutions like saliva and plaque fluids. Given this, these solutions surrounding the teeth tend to be unsaturated and capable of dissolving the enamel. Although saliva and plaque fluids are in themselves not threats to the teeth [3,26,27], extensive calculus accumulation can increase the oral microbiome and decrease the pH of these oral solutions. The low oral pH can then instigate demineralization. Therefore, it was not surprising that most of the individuals with medium to large calculus accumulation also had carious lesions on the same teeth or elsewhere in the mouth. For these reasons, sex dichotomy in caries expression can be attributed to differential levels of carbohydrate and acidic foods intake, estrogen levels, oral pH, and the avulsion or evulsion of carious teeth [14]. Caries predispose individuals to abscesses and hypercementosis (cementum hyperplasia). For instance, bilateral caries was identified in the mandible of an adult male from Begho BG'21-73.115 (Bg. 70, E) at the level of 36 and 46 and mirror caries between 45 and 46. The infection involves complete enamel and dentine demineralization and root destruction at the initial stages. This individual also presented bilateral abscess and resulting porosity in the mandible at the level of caries. The expression of the former directly links to the latter.

Dental calculus results from the mineralization of built-up plaques at the gingiva/sub-gingival level and interproximal regions of maxillary and mandibular teeth. Calculus is an active ''storehouse'' for bacteria, fungi, compounds, minerals, as well as food and non-dietary debris [4,7,28—30]. Consequently, their extensive accumulation around the teeth of these individuals permits the prediction of dietary patterns and oral health/hygiene. According to Lieverse [31], dental plaques mineralize into calculus as early as two weeks if not removed. The long-term decay of the debris releases a pungent smell or odor while decreasing the pH of the oral solution surrounding the teeth. Dental calculus, like caries, is a high correlate of the consumption of carbohydrate-rich foods. According to Lillie and Richards [32], high carbohydrate and low protein diets increase plaque accumulation without proper oral hygiene. However, Keenleyside [16] and Šlaus et al. [33] correlate high protein and low carbohydrate intake to increased calculus. It appears that both protein and carbohydrate play critical roles in caries and calculus manifestations (Figs. 6—7).

The $^{13}C$ and $^{15}N$ isotopic values of a Yapei middle adult were −10.5‰ and 8.8‰ indicative of a high dietary protein orientation to $C_4$ plants like maize, groundnut, beans, and animal protein from fish (riverine resources) and terrestrial animals such as sheep, cattle, and chicken. This $C_4$ plant characterization is not exclusive to Yapei; it cuts across the regions with a high dependence rate on typical carbohydrate-rich food crops dangling across the country via the trade chain. Since Yapei and Ntereso are geographically proximate with similar environmental conditions and subsistence patterns, it is anticipated that the Ntereso individuals may have similar $\delta\ ^{13}C$ and $\delta\ ^{15}N$ values. The Sekondi individuals, however, would have presented much higher $\delta\ ^{13}C$ and $\delta\ ^{15}n$ values for their dependence on marine resources from


**Figure 6.** Maxilla of an adult (male?), Begho -BG'21-73.115 (Bg. 70, E), displaying complete deterioration of teeth 24 & 26 by caries, leading to subsequent damage of the alveoli and abscess at 26. Also, note the abscess at 12.


**Figure 7.** Mandible of an adult (male?), Begho -BG'21-73.115 (Bg. 70, E), showing bilateral caries at 36 and 46 and mirror caries between 45 and 46. The infections translated into abscesses: single lingual and buccal perforation at 35 and double buccal drainage channels at 45 (at the level of the mental foramen). The porosity at the respective regions results from the two DPs.

the sea and food crops from the suburbs that rushed into the community. Comparatively, the individuals from Begho and Yikpabongo may have had analogous δ $^{13}$C and δ $^{15}n$ values, and any variance be associated with regional differential adaptations, individual preferences, and intermittent scarcity of food resources. Furthermore, Posnansky (2004, pp. 39–40), noted that Begho inhabitants had a protein-rich diet; an individual animal (domesticated) protein intake of at least 250 g per week, likely to increase in seasons of abundant game resources. The $^{13}$C and $^{15}$N isotope revelation of the dietary pattern of the Yapei individual enabled the reconstruction of the dietary orientation of the individuals from the Savannah region to $C_4$ plants in addition to riverine and terrestrial animal protein. By inference, the orientation of the individuals from the Western, Bono, and Northeast regions may be a combination of $C_3$, $C_4$ plants, marine, riverine, and lacustrine resources plus terrestrial animal protein.

Oral accounts agree with this dietary orientation, in that carbohydrate-rich foods (cereals and tuber crops) and fish, both riverine and lacustrine, are major components of Ghanaian diets cross-regionally. For instance, the banana, a carbohydrate-rich fruit [34] is cultivated on a large scale in Ghana, although not indigenous to the sub-region. Due to its high carbohydrate content, increased consumption can result in massive plaque build-up in the absence of proper oral hygiene. Moreover, Barbara Teßmann's examination of tartar from 110 19th-century Musila skulls revealed that the build-up resulted from the extensive consumption of bananas and meat (local conference presentation). Sugarcane is also not an exception across the Ghanaian regions. In addition to high sugar and carbohydrate intake and poor oral hygiene, Lieverse [31] notes that the composition of drinking water is another source of calculus formation from which a tone of data (dietetics and health) can be gleaned. For example, through X-ray fluorescence analysis of calculus from mandibular and maxillary teeth, two Begho individuals were found unexposed to industrial pollutants like lead, arsenic, antimony, or mercury [35]. As a reservoir of non-dietary debris such as (cotton) fiber [30], wood and other work-related (mineral) debris [36], trapped vegetal fiber, and quartz crystals [4], occupational or extra-masticatory stress on the teeth from the use of teeth as a 3rd hand for industrial activities can be inferred from dental calculus. Extensive calculus accumulation increases vulnerability to periostitis/periapical lesions [37] from the increased bacterial activity around the teeth and in the mouth.

Periapical lesions or abscesses; offshoots of pulpitis and the subsequent spread of bacteria to the apical region [8] constituted 9% of the dental pathologies recorded. Abscess results from pathological conditions such as extensive caries, sub-gingival infections/inflammation, dental injury, and extreme dental wear [8,14]. Most of the individuals that presented single or bilateral cases of abscess had medium to extensive caries on the occlusal surface or the interproximal region, severe dental attrition, and enamel hypoplasia or combined all, in addition to other dental pathologies. Particularly, a middle adult female from Yapei- YP'18-73.105 presented mirror caries at the cemento-enamel junction (with root extension) of 14 & 15 and extreme calculus accumulation (removed for XRF analysis at the time of taking the photograph). The individual also had an abscess at the buccal surface of 14, one hypoplastic line on 14 & 15, significant wear, and two hypoplastic lines on 13, in addition to about 50% of hypercementosis of the roots of the teeth (Fig. 8). This individual must have experienced ample intra-vitam stress. The functional strain on the teeth could be ascribed to severe dental wear (by mastication and non-masticatory means) and the exposure and infection of the pulp via caries as closely linked variables [8,14].


**Figure 8.** Mid-18th – early 19th Century middle adult female from Yapei (YP'18-73.105) with mirror caries at CEJ and root of 14 and 15. Additionally, an abscess at the buccal surface of 14, one hypoplastic line on 14 & 15, and two hypoplastic lines on 13. There is also about 60% hypercementosis of the roots of 13 & 14. Lateral view.

Enamel hypoplasia or linear enamel hypoplasia (LEH) is an antiphon in pits and lines to developmental and environmental stress interrupting ameloblastic activity in enamel formation [12]. A comparative analysis of dental hypoplasia can infer dietetics and pathological stress among populations [38,39]. Enamel hypoplasia is one of the childhood stress markers that persist into adulthood [40] because teeth do not remodel after complete formation, apart from mechanical alterations by mutilations (chipping and filing [8]), caries, and attrition from masticatory and non-masticatory activities. Moreover, because hypoplastic lines or pits represent a stress-informed deficiency in enamel development, it can be observed in deciduous and permanent teeth of individuals who experienced significant stress at the initial stages of teeth development. Enamel hypoplasia can aid the reconstruction of intra-vitam stress or malnutrition that disrupted growth in past populations [12,41]. The relatively high expression of enamel hypoplasia in the individuals from Begho, Ntereso, and

Yapei posits significant nutritional/environmental stress. That said, 20th-century groups might have experienced nutritional stress partly from the inadequate supply of nutritional needs during the 1983 famine in Ghana, and its effect might be reflected by the presence of harris lines and hypoplastic lines/pits in their long bones and teeth. The absence of hypoplastic lines or pits on an adult dentition implies only a partial exclusion of developmental stress because the lack of lesions is not the absence of a stress phase. In contrast, its presence in adult dentition may not necessarily mean the prolongation of the stress factor but rather a childhood acquisition at the developmental stages of the permanent crowns in the alveolar.

Some scholars have proposed multi-etiologies to LEH by linking it to various developmental disturbances at the individual or population level. From pre and neonatal to early childhood, especially after weaning – seven years [41,42], stress-related local and systemic events comprising deficient diet, malnutrition, infectious diseases, genetic anomalies, and congenital defects such as amelogenesis imperfecta. Additionally, neonatal disturbances such as neonatal hemolytic anaemia, premature birth, hypocalcaemia (the cause of postnatal hypoplasias), maternal rubella, and diabetes [41,43]. Due to these links, which are equally stressors on growth and development, no one cause can be associated with our remains but the acknowledgment of general stress among the individuals.

Lastly, hypercementosis was recorded in the assemblage. Hypercementosis or cementum hyperplasia is a condition characterized by the thickening (more prominent than average) of the tooth root and canal due to the abnormal deposition of cement [44,45]. It is considered an acute response to infections like caries and periodontal diseases. Irrespective of high caries (30%) and abscesses (9%) in the assemblage, hypercementosis represented only 7% of available teeth (with the exception of unexposed roots of teeth in occlusion). This suggests a multifactorial cause of hypercementosis, for example, an external involvement of the teeth within the different environments, including as a third hand. As gathered from the ethnographic study in the various communities of Sekondi, Begho, Yapei, and Ntereso, (paleo) activities such as fish net mending and basketry sometimes involve the industrial use of teeth (the anterior and $^{first}$ premolar) as a third hand to rip raw materials and supporting elements. This may have posed significant stress on the teeth roots of these past populations. The contemporary inhabitants are at risk of the same or similar threats due to the continuity in practice. Although this pathology can be related to teeth function (masticatory and non-masticatory activities), Thoma and Goldman [46] noted that even non-erupted teeth can develop cementum hyperplasia owing to genetic factors. Other sources of hypercementosis are inflammation, formation of spicules and continuous eruption of the tooth [46], bacterial colonization of teeth [45], and alveolar trauma causing root fracture [47]. In these cases, developmental, nutritional, environmental, and occupational stress can be attributed to hypercementosis. As a consequence, generalized hypercementosis may impact the alveoli and teeth function as well as the effectiveness of tooth root translucency for age estimation in anthropological assessments.

## Conclusion

From a nuanced perspective, the dental pathologies recorded and examined from the study samples are interdependent in occurrence and rate of expression. The expression rate of the dental pathologies significantly reflects compromised oral health and stress among the past Ghanaian ancestors. We establish a high correlation between pathognomic carbohydrate proxies like caries and calculus and the dietary pattern of the past populations, which is not vastly different from that of the contemporary population. For an agrarian country like Ghana, its population is accustomed to cereals and tuber crops, consumed in dried, fresh, and fermented stages and supplemented by animal and fish proteins. Therefore, high caries and calculus expression among these Ghanaian ancestors stem largely from the dietary composition, while offshoots such as abscess, enamel hypoplasia, and cementum hyperplasia may be more related to function and developmental stress on the teeth.

The oral health of the individuals was somewhat compromised by the expression rate of the dental pathologies discussed above. Comparing standards of oral hygiene among past and present populations may be unfair, but what is certain is that care and access to dental services were not the same as today. Concerns may have been the availability of services and public education on the importance of dental care. Additionally, socioeconomic status may have impacted the quality of one's oral health: care, and hygiene. Some factors that aggravate dental pathologies and consequently compromise individual health include diet/nutrition, functional stress on teeth, and poor oral hygiene. Currently, the culture of regular dental care in the rural sectors could be improved by ample education on controls for DPs such as balanced diets, early treatment, and preventive measures. For example, diets can have balanced constituents of carbohydrates and proteins, using teeth as 3rd hands can be minimalized, and proper oral hygiene measures targeted at reducing bacteria activity and infections can be enforced. Like bones (another hard tissue), teeth record most intra-vitam experiences: infections, trauma, dietary, and developmental and functional stress, valuable for context-specific reconstructions of life histories. Thus, the examination of dental pathologies among these ancient Ghanaian individuals offered insights into dietary habits, oral health, and possible indicators of developmental and functional stress.

## Disclosure of interest

The authors declare that they have no competing interest.

## References

[1] World Health Organization. ''World Health Organization, African Region,'' 2021. [Online]. Available: https://www.afro.who.int/health-topics/oral-health [Accessed 3 January 2023].
[2] Hillson S. Diet and dental disease. World Archaeol 1979;11:147–62.
[3] Hillson S. The current state of dental decay. Evol Anthropol 2008;53:111–35.

[4] Charlier P, Huynh-Charlier I, Munoz O, Billard M, Brun L, Lorin de La Grandmaison G. The microscopic (Optical and SEM) examination of dental calculus deposits (DCD). Potential interest in forensic anthropology of a bio-archaeological method. Legal Med 2010;12:163–71.
[5] Hardy K, Buckley S, Collins MJ, Estalrrich A, Brothwell D, Copeland L, et al. Neanderthal Medics? Evidence for food, cooking, and medicinal plants entrapped in dental calculus. Naturwissenschaften 2012;99:617–26.
[6] Radini A, Nikita E, Buckley S, Copeland L, Hardy K. Beyond food: the multiple pathways for inclusion of materials into ancient dental calculus. Am J Phys Anthropol 2017;162:71–83.
[7] Radini A, Nikita E. Beyond dirty teeth: integrating dental calculus studies with osteoarchaeological parameters. Quat Int 2023;653:3–18.
[8] Molnar P. Dental wear and oral pathology: possible evidence and consequences of habitual use of teeth in a Swedish neolithic sample. Am J Phys Anthropo 2008;136:423–31.
[9] Lamptey PSNO. Museums and skeletons: prospects and challenges of cataloguing, storing, and preserving human remains in the museum of archaeology, Ghana. Ethics Med Public Health 2022;21:100753.
[10] Buikstra JA, Ubelaker DH. Standards for data collection from human skeletal remains: proceedings of a seminar at the field museum of natural history. Arkansas: Arkansas Archaeological Survey Research Series; 1994.
[11] Ogden AR. Advances in the paleopathology of teeth and jaws. In: Mays S, Pinhasi R, editors. Advances in human paleopathology. Chichester: John Wiley & Sons; 2008. p. 283–307.
[12] Bereczki Z, Reschler-Nicola M, Marcsik A, Meinzer N, Baten J. Growth disruption in children: linear enamel hypoplasias. In: Steckel RH, Larsen CS, Roberts CA, Baten J, editors. The backbone of Europe: health, diet, work, and violence over two millennia. Cambridge: Cambridge University Press; 2019. p. 175–97.
[13] Touger-Decker R, van Louveren C. Sugars and oral health. ILSI Conference: Sugars and Health, 2002; Washington, DC. Am J Clin Nutr 2003;78:8815–25.
[14] Bonsall LA, Pickard C. Stable isotope and dental pathology evidence for diet in late Roman Winchester, England. J Archaeol Sci: Reports 2015;2:128–40.
[15] Lingström P, van Houte J, Kashket S. Food starches and dental caries. Critical reviews. Oral Biol Med 2000;11:366–80.
[16] Keenleyside A. Dental pathology and diet at Apollonia, a Greek colony on the Black Sea. Int J Osteoarchaeol 2008;18:262–79.
[17] Tomczyk J, Szostek K, Komarnitki I, Mańkowska-Pliszka H, Zalewska M. Dental caries and chemical analyses in reconstruction of diet, health and hygienic behaviour in the middle Euphrates Valley (Syria). Arch Oral Biol 2013;58:740–51.
[18] Hillson S. Dental Pathology. In: Katzenberg MA, Saunders SR, editors. Biological anthropology of the human skeleton. New York: Willey; 2008. p. 301–40.
[19] Dodds ML. The cariogenic property of cereal foods. Nutr J 1960;71:317–21.
[20] Turner CG. Dental anthropological indications of agriculture among the Jomon people of Central Japan. X. Peopling of the Pacific. Am J Phys Anthropo 1979;51:619–35.
[21] Laine M, Tenovuo J, Lehtonen OP, Ojanotko-Harri A, Vilja P, Tuohimaa P. Pregnancy-related changes in human whole saliva. Arch Oral Biol 1988;33:913–7.
[22] Prout RES, Hopps RM. Changes in Hyaluronidases in human saliva during the menstrual cycle. J Periodont Res 1973;8:83–93.
[23] Lukacs JR, Largaespada LL. Explaining sex differences in dental caries prevalence: saliva, hormones, and ''life-history'' etiologies. Am J Human Biol 2006;18:540–55.
[24] Goebelsmann U. Protein and steroid hormones in pregnancy. J Reprod Med 1979;23:166–77.
[25] Cockle SM, Harkeness RA. Changes in salivary peroxidase and polymorphonuclear neutrophil leucocyte enzyme activities during the menstrual cycle. Br J Obstret Gynaecol 1978;85:776–82.
[26] Barron RP, Carmichael RP, Marcon MA, Sàndor CG. Dental erosion in gastroesophageal reflux disease. Can Dental Assoc 2003;69:84–9.
[27] Dawes C. What is the critical pH and why does a tooth dissolve in acid? Can Dental Assoc 2003;69:722–5.
[28] Charlier P, Abadie I, Cavard S, Brun L. Ancient calculus egg. Br Dent J 2013;215:489–90.
[29] Charlier P, Gaultier F, Héry-Arnaud G. Interbreeding between Neanderthals and modern humans: remarks and methodological dangers of a dental calculus microbiome analysis. J Hum Evol 2019;126:124–6.
[30] Blatt SH, Redmond BG, Cassman V, Sciulli PW. Dirty teeth and ancient trade: evidence of cotton fibres in human dental calculus from Late Woodland, Ohio. Int J Osteoarchaeol 2011;21:669–78.
[31] Lieverse AR. Diet and the aetiology of dental calculus. Int J Osteoarchaeol 1999;9:219–32.
[32] Lillie MC, Richards M. Stable isotope analysis and dental evidence of diet at the Mesolithic-Neolithic transition in Ukraine. J Archaeol Sci 2000;27:965–72.
[33] Šlaus M, Bedić Ž, Rajić Šikanjić P, Vodanović M, Domić Kunić A. Dental health at the transition from the Late Antique to the early Medieval period on Croatia's eastern Adriatic coast. Int J Osteoarchaeol 2011;21:5–7.
[34] JSidhu JS, Zafar TA. Bioactive compounds in banana fruits and their health benefits. Food Qual Safety 2018;2:183–8.
[35] Lamptey PSNO, Bourdin V, Charlier P. Dental calculus as a useful tool for public health evidence in past populations: the case of two individuals from Begho-Ghana (10th–19th C. AD). Ethics Med Public Health 2023;27:100875.
[36] Blondiaux J, Charlier P. Palaeocytology in skeletal remains: microscopic examination of putrefaction fluid deposits and dental calculus of skeletal remains from French archaeological sites. Int J Osteoarchaeol 2008;18:1–10.
[37] Dumitrescu AL, Kawamura M. Etiology of periodontal disease: dental plaque and calculus. In: Dumitrescu AL, editor. Etiology and pathogenesis of periodontal disease. Berlin Heidelberg: Springer; 2010. p. 1–38.
[38] Goodman AH, Rose JC. Dental enamel hypoplasias as indicators of nutritional status. Adv Dental Anthropol 1991;5:225–40.
[39] White TD, Black MT, Folkens PA. Human osteology. 3rd ed. Elsevier Academic Press; 2011. p. 689.
[40] Teckel RH, Kjellström A. 3: measuring community health using skeletal remains. In: Steckel RH, Larsen CS, Roberts CA, Baten J, editors. The backbone of Europe: health, diet, work, and violence over two millennia, 80. Cambridge: Cambridge University Press; 2018. p. 52–80.
[41] Goodman AH, Martin D. Reconstructing health profiles from skeletal remains. In: Steckel RH, Rose JC, editors. The backbone of history: health and nutrition in the Western Hemisphere, 2. Cambridge: Cambridge University Press; 2002.
[42] Blakey ML, Leslie TE, Reidy JP. Frequency and chronological distribution of dental enamel hypoplasia in enslaved Americans: a test of the weaning hypothesis. Am J Phys Anthropol 1994;95:371–83.
[43] Pindborg JJ. Aetiology of developmental enamel defects not related to fluorosis. Int Dental J 1982;32:123–34.

[44] Pappen FG, Fagonde CD, Martos J, Silveira LFM. Hypercementosis: a challenge for endodontic therapy. Rev Bras Odontol 2011;8:321—8.
[45] Pinheiro BC, Pinheiro TN, Capelozza ALA, Consolaro A. A scanning electron microscopic study of hypercementosis. J Applied Oral Sci 2008;16:380—4.
[46] Thoma KH, Goldman HM. The pathology of dental cementum. J Am Dental Assoc 1939;26:1943—53.
[47] Coolidge ETD. The reaction of cementum in the presence of injury and infection. J Am Dental Assoc 1922;18: 499—525.