Université de Montréal

Palaeoepidemiological analysis of a historical urban population from Montréal: exploring the interactions between vitamin D deficiency and various palaeopathological skeletal manifestations

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Thesis presented to obtain a Master in Anthropology

November 2020

Université de Montréal

Anthropology: Faculty of Arts and Sciences

This thesis entitled

Palaeoepidemiological analysis of a historical urban population from Montréal: exploring the interactions between vitamin D deficiency and various palaeopathological skeletal manifestations

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Abstract

Vitamin D has many functions in the regulation of various systems affecting the whole body. A deficiency of this hormone thus affects many essential systems (e.g. skeletal, dental, immune) and can leave visible lesions on the skeleton and teeth. During the Industrial Revolution, various environmental factors probably contributed to an increase in vitamin D deficiency. Therefore, most studies on vitamin D deficiency have been conducted on European skeletal samples dated to the 19th century. However, the frequency of this type of metabolic disease has not been explored extensively in Canada for the same period. Furthermore, in palaeopathology, the possible interactions between vitamin D deficiency and other diseases have only been poorly explored. To fill this gap, the present study focuses on an historic Euro-Quebecois population (St. Antoine cemetery, Montréal (1799-1854), exploring the frequency and prevalence of pathological lesions and conditions as well as their possible influence on the age and sex categories. More precisely, are investigated the possible link between the occurrence of vitamin D deficiency cases during infancy and health deterioration later in life, as well as the temporal evolution of the cases within the St. Antoine cemetery in relation to the industrialization process. In total, 52 individuals (8 nonadults, 44 adults) from the St. Antoine urban cemetery were used for the study. The data collected during the research is of an empiric nature and was acquired following three steps: i) the basic skeletal inventory (sex, age-at-death); ii) the macroscopic and radiographic observations of the pathological lesions on the skeletons; iii) and the histological examination of selected dental tissues to assess the presence of interglobular dentin (IGD). The results indicate a high prevalence of IGD during infancy indicative of vitamin D deficiency, i.e. 44% (23/52) to 75% (39/52), excluding and including the degree 1 of IGD, respectively. The majority of these episodes occurred at or from birth to up to three years-old (63%, n=33/52) and 12% of the individuals (n=6/52) presented prenatal IGD episodes, raising questions about maternal deficiencies and cultural influences. The presence of IGD during infancy was not correlated with demographic parameters, nor with health status deterioration later in life. A possible link was recorded between the presence of more severe degrees of IGD and LEH. The number of IGD cases during infancy seems to decrease with time, contrary to what might be expected of an industrializing population. However,

this result must be taken with caution because of the small portion of the sample coming from the more recent parts of the cemetery.

Keywords: 19th century, Montréal, palaeoepidemiology, histology, vitamin D, deficiencies, health, cultural practices, industrialization.

Résumé

La vitamine D occupe de nombreuses fonctions dans la régulation de divers systèmes affectant le corps entier. Une déficience de cette hormone peut donc affecter plusieurs systèmes essentiels (ex. squelettique, dentaire, immunitaire) et peut laisser des lésions repérables sur le squelette et les dents. Au cours de la Révolution industrielle, plusieurs facteurs environnementaux ont probablement contribué à l'augmentation des cas de déficience en vitamine D. Ainsi, la majorité des études portant sur la déficience en vitamine D se sont concentrées sur des populations européennes du XIXe siècle. Au Canada, pour la même époque, la fréquence de ce type de maladie métabolique n'a pas été explorée en profondeur. De plus, les possibles interactions entre déficience en vitamine D et autres maladies ont été peu explorées jusqu'à maintenant. Afin de pallier cette lacune, la présente étude se concentre sur une population euroquébécoise (cimetière St-Antoine, Montréal (1799-1854)), explorant la fréquence et prévalence de diverses lésions et conditions pathologiques, en plus d'évaluer leur influence sur les catégories d'âge et de sexe. Plus précisément, cette recherche investigue le possible lien entre la présence de cas de déficience vitamine D durant l'enfance et la détérioration de l'état de santé plus tard au cours de la vie, en plus de l'évolution temporelle des cas au sein du cimetière St-Antoine en lien avec le phénomène d'industrialisation. Au total, 52 individus (8 non adultes, 44 adultes) issus du cimetière urbain St-Antoine à Montréal ont été étudiés. Les données collectées sont de nature empirique et ont été acquises selon les trois phases suivantes : i) l'inventaire squelettique de base (sexe, âge au décès), ii) les observations macroscopiques et radiographiques des lésions pathologiques pour chaque squelette; iii) et l'analyse histologique de tissus dentaires sélectionnés afin d'explorer la présence ou l'absence de dentine interglobulaire (DIG). Les résultats indiquent une proportion élevée de DIG durant l'enfance indiquant une déficience en vitamine D, c'est-à-dire 44% (23/52) à 75% (39/52), excluant et incluant le degré 1 de DIG, respectivement. La majorité de ces épisodes de déficience se sont produits à ou à partir de la naissance, allant jusqu'à trois ans (63%, n=33/52), et 12% des individus (n=6/52) présentent des épisodes prénataux de DIG, ce qui soulève la question de la déficience maternelle et de l'influence des facteurs culturels. La présence de DIG durant l'enfance n'est corrélée ni aux paramètres démographiques ni à une détérioration de l'état de santé plus tard au cours de la vie de l'individu. Un possible lien a été soulevé entre les degrés plus sévères de DIG et l'hypoplasie dentaire. Le nombre d'épisodes de DIG durant l'enfance semble diminuer

avec le temps, contrairement à ce qui pourrait être attendu d'une population en voie d'industrialisation. Toutefois, ce résultat doit être considéré avec prudence en raison de la faible portion de l'échantillon provenant des aires plus récentes du cimetière.

Mots-clés : XIX^{e} siècle, Montréal, paléoépidémiologie, histologie, vitamine D, carences, santé, pratiques culturelles, industrialisation.

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Acronym and Abbreviation List

IGD: Interglobular dentin

DEJ: Dentino-enamel junction

DOHaD: Developmental Origins of Health and Disease

IVD: Intervertebral disk disease

OA: Osteoarthritis

TB: Tuberculosis

LEH: Linear enamel hypoplasia

CV: Cervical vertebrae

TV: Thoracic vertebrae

LV: Lumbar vertebrae

I_1/I^1: First permanent mandibular/maxillary incisor

I_2/I^2: Second permanent mandibular/maxillary incisor

C_1/C^1: Mandibular/maxillary canine

PM_1/PM^1: First permanent mandibular/maxilary premolar

PM_2/PM^2: Second permanent mandibular/maxillary premolar

M_1/M^1: First permanent mandibular/maxillary molar

M_2/M^2: Second permanent mandibular/maxillary molar

M_3/M^3: Third permanent mandibular/maxillary molar

To the little Rose-Ann who collected fossil rocks on the beach.

Acknowledgments

The help of many people and organizations was crucial to achieve this thesis. This research was funded by the Social Sciences and Humanities Research Council (SSHRC) and the ArchéoScience/ArchéoSociale (As^2) Research Group. I thank them both very much for their financial support. I would also like to thank my supervisor, Isabelle Ribot, for all of her constructive comments and her support throughout this whole process, as well as my co-supervisor, Charlotte Roberts, for her crucial help in the analysis of the pathological lesions and conditions on the skeleton. I also want to thank the examiners of my dissertation for their comments and corrections, Megan Brickley and Michelle Drapeau. I could not have successfully made the observations of IGD on my dental slides without the help and expertise of Megan Brickley and Bonnie Kahlon, to whom I am very grateful. I thank the Laboratoire d'écomorphologie et paléoanthropologie, more specifically the technician, Yi Ji Hyun /Daniel, for his help and patience in teaching me how to use the equipment. I am very grateful to Alexandre Bisson, Jean-Baptiste Lemoine and Diane Martin-Moya for their help in the dental 3D modelling that was part of the research project, as I could not have done it without you all. I also want to thank my stepfather, Daniel Beaudoin, for designing a very clever customized mould to embed my sampled teeth in epothin resin. Finally, I am very grateful to my parents, Lucie Groleau and Normand Bigué, my aunt and godmother, Martine Bigué, and my friends and family who supported me throughout this entire journey.

Introduction

Metabolic disorders such as vitamin D deficiency can be caused by biophysical variables, such as geographical latitude, skin pigmentation and bioavailability of vitamin D in food sources (Brickley, Moffat, and Watamaniuk 2014, 48). Cultural factors that influence individual and populational risk such as infant and child feeding practices and behaviours that affect exposure to sunlight can also cause vitamin D deficiency (Brickley, Moffat, and Watamaniuk 2014, 48). According to clinical studies, vitamin D deficiency during infancy can influence the resistance to infections and the development of some cancers and/or rheumatoid arthritis (Holick 2004; Bikle 2009; Baeke et al. 2010). In fact, studies by Manolagas and his group (1985,1986,1991) provided strong evidence that a large quantity of vitamin D receptors are located on activated lymphocytes that play a crucial role in the regulation of the immune system (Deluca and Cantorna 2001; Yu et al. 1991; Manolagas et al. 1986; Manolagas, Provvedini, and Tsoukas 1985). However, in palaeopathology, the possible interactions of metabolic disorders with other diseases have been poorly explored (Roberts and Brickley 2018). A study conducted by Snoddy and colleagues (2016) on two skeletal collections, one dating to the medieval period (St. Mary Graces, 1350-1540 AD) and the other dating to the postmedieval period (St. Benet Sherehog, 16-17th centuries AD) indicate a strong association (95% accuracy) between vitamin D deficiency and chronic infections, although no significant relation between tuberculosis and vitamin D deficiency was noted (Snoddy, Buckley, and Halcrow 2016). The researchers consider relevant further exploration of this issue in future studies (Snoddy, Buckley, and Halcrow 2016).

Evidence of vitamin D deficiency was found in Late Pleistocene individuals as well as in various ancient Roman populations, indicating that this type of disease is not a recent phenomenon (Mays et al. 2018). However, during the Industrial Revolution, various environmental factors probably contributed to an increase in vitamin D deficiency. Therefore, most studies on vitamin D deficiency have been conducted on European skeletal samples dated to the 19th century (Brickley, Kahlon, and D'Ortenzio 2019; D'Ortenzio, Ribot, et al. 2018; Watts and Valme 2018; Mays et al. 2018; Ives 2018; D'Ortenzio et al. 2016; Mays, Brickley, and Ives 2007; 2006). So far, the frequency of metabolic diseases has not yet been explored extensively in Canada for the same

period, with the exception of a reported cases (Brickley, Kahlon, and D'Ortenzio 2019; D'Ortenzio et al. 2018; 2016; Houle-Wierzbicki 2016; Morland 2009).

Therefore, the present research project aims to fill this gap by studying the health status of a Euroquebecois population transitioning from the preindustrial to the industrial period. Although industrialization started later in Quebec in comparison to other regions (Dickinson 2014), vitamin D deficiency might still have affected people depending on various environmental factors such as their access to resources and their way of life (indoor, outdoor or mixed) (Brickley, Moffat, and Watamaniuk 2014).

Objectives

Thus, the analysis of 52 skeletal and dental remains from an urban population of Montréal (Catholic cemetery of St. Antoine) dated to the 19th century (Ethnoscop 2012) aims to answer the following questions:

i) What is the frequency and prevalence of pathological lesions and conditions among the sampled individuals from 19th century St. Antoine cemetery, Montréal?
ii) How do pathological lesions and conditions affect the various categories (sex, age) of the urban population sample from St. Antoine cemetery?
iii) Is there a possible link between the occurrence and severity of vitamin D deficiency episodes during infancy and the health deterioration occurring later in life?
iv) Is there a temporal evolution within the St. Antoine cemetery of vitamin D deficiency cases during infancy, in relation to the industrialization process?

To explore these four questions, which combine both palaeopathology and palaeoepidemiology, the dissertation is organized in three chapters (Fig.1).

- Chapter 1: Literature review on the palaeopathological and paleoepidemiological approaches, as well as the Developmental Origins of Health and Disease (DOHaD);
- Chapter 2: Literature review on the previous clinical and bioarchaeological studies on vitamin D and vitamin D deficiency;
- Chapter 3: Journal article in preparation, which is composed of:
    1. Materials and methods:

i) The basic skeletal inventory (sex, age-at-death) of the skeletal remains;

ii) The macroscopic and radiographic analysis of the pathological lesions for each skeleton;

iii) The photographic recording of the dental tissues before the destruction of the tissues; and

iv) The histological examination of selected dental tissues to evaluate the presence of metabolic lesions;

2. The results of the study and the discussion;
3. The conclusion of the study.

My work on this journal article consists of the data collection, the analysis of the data and the final writing.

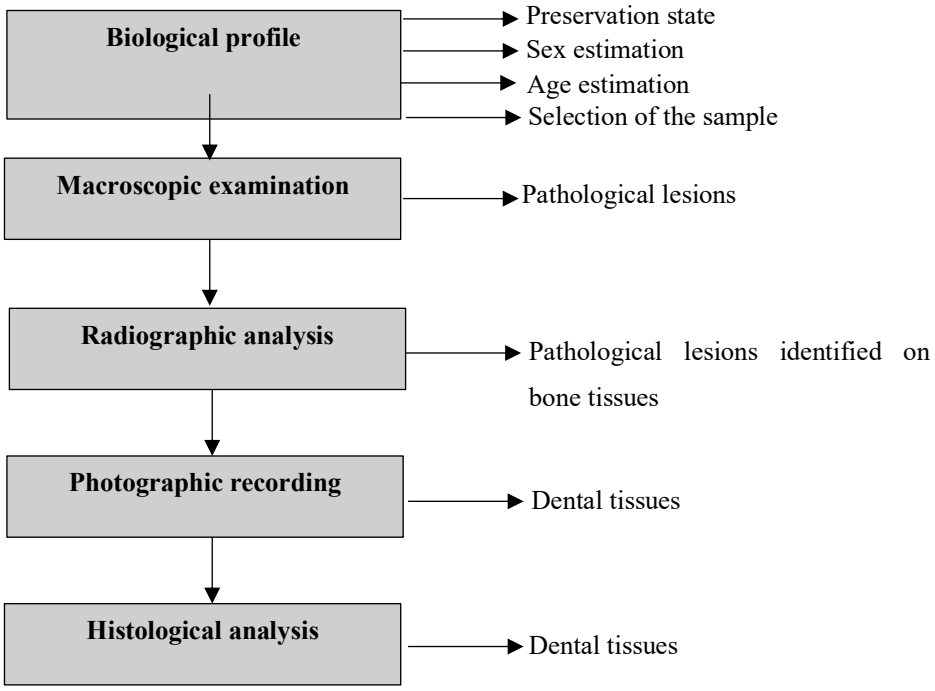

Figure 1. – Main phases of the methodology used in this thesis.

Chapter 1: Theoretical framework-palaeopathological and palaeoepidemiological approach

1.1 Palaeopathological approach

Palaeopathology, simply put, is the field that studies diseases in the past (Grauer 2012, 1). The past might be relatively recent or considerably ancient (Grauer 2012, 1). Indeed, in many states throughout the US, for example, remains are deemed "ancient" if they are over 100 years old and can be thus studied by palaeopathologists (Grauer 2012, 1). Palaeopathology is a multidisciplinary field composed of individuals trained in medicine, dentistry, archaeology or physical anthropology who choose to focus their research on studying the manifestations of diseases on ancient human remains (Grauer 2012, 4). Concerning the scientific methodology, palaeopathology is mainly descriptive but very interdisciplinary and it focuses not only on case studies of single or few individuals but also on population samples to evaluate the general health status within a group (Buikstra and DeWitte 2019, 13).

Differentiating normal variation, taphonomic aspects and pathological lesions is crucial in palaeopathology (Buikstra and DeWitte 2019, 13). Describing lesions and examining the dispersion of the lesions across the skeleton is necessary to achieve a potential diagnosis and evaluate the pathological state of the individuals (Buikstra and DeWitte 2019, 13).

1.2 Palaeoepidemiological approach

Palaeoepidemiology focuses on health and disease patterns at the populational scale according to data mainly derived from skeletal and mummified tissues, applying a more quantitative approach than palaeopathology (Boldsen and Milner 2012, 114; Buikstra and DeWitte 2019, 14). Such studies usually focus on the documentation of changes in disease experience that accompanied major transitions during recent human evolution, such as industrialization in the case of this research (Boldsen and Milner 2012, 114). Palaeoepidemiological research aims to evaluate how common certain diseases were in groups of people that differed according to various variables (e.g. geographical origin, time period, sex, age, socioeconomic position, residential location) (Boldsen and Milner 2012, 114). Ideally, the skeletal collections must be homogenous culturally

and biologically and come from well-documented cemeteries associated with single communities (Boldsen and Milner 2012, 118). The knowledge of the cultural context of a skeletal sample is thus a preliminary step (Boldsen and Milner 2012, 118).

Minimally, palaeoepidemiological studies must take into account the origin and representativeness of skeletal samples, the association between diseases and lesions, the selective effect of mortality in the formation of skeletal assemblages, the causes and consequences of diseases in various cultural and natural environments, and the particular details of local conditions that affect health (Boldsen and Milner 2012, 115). The principal objective is thus to reconstruct the lives of people using data derived from those who died (a mortality sample), which is in part done by analysing the age distribution of people affected and not affected by specific lesions (Boldsen and Milner 2012, 115).

1.2.1 Expected outcome of palaeoepidemiological studies

One of the aims of palaeoepidemiological studies is to assess the impact of diseases or injury on mortality (Milner and Boldsen 2017, 36). Careful considerations of disease processes, heterogeneous frailty, selective mortality and archaeological and cultural contexts have to be undertaken (Milner and Boldsen 2017, 36; Wood et al. 1992).

1.3 Limitations of the approaches and biases

Both palaeopathological and palaeoepidemiological approaches have limitations that can impair the accuracy of any bioarchaeological study. Five of these limitations are presented below.

1.3.1 Selective mortality

The first issue concerns the fact that the skeletons analysed represent people that died for a reason, meaning that the prevalence of lesions of a particular condition in an osteological sample does not directly represent its prevalence in the living population at a given point in time (Wood et al. 1992; Wright and Yoder 2002, 45). It is thus difficult to interpret the absence of lesions: was this person healthy before death or were they weak and died at the first exposure to a pathological agent (Wood et al. 1992; Wright and Yoder 2002, 45)? This makes it difficult to assess the probability that individuals with a pathological condition also present the associated markers, thus often underestimating the frequency of affected people (Milner and Boldsen 2017, 34).

1.3.2 Hidden heterogeneity in risk

Susceptibility to illness varies substantially from one individual to another, a situation called frailty (Wright and Yoder 2002, 45; Wood et al. 1992). The factors contributing to this frailty are generally not identifiable (Wright and Yoder 2002, 45; Wood et al. 1992). During this research, vitamin D deficiency during infancy is a contributing factor that will be assessed, thus reducing the bias. Indeed, according to clinical studies, the occurrence of vitamin D deficiency during infancy can influence the resistance to infections and the development of certain types of cancer and rheumatoid arthritis (Baeke et al. 2010; Bikle 2009; Holick 2004). However, other variables, non-environmental ones, such as genetics, which are not explored here in the present study, might bias the results (Wood et al. 1992, 345).

1.3.3 Representativeness of the sample

A difficult part in examining skeletal remains consists in assessing the representativeness of the sample (Milner and Boldsen 2017, 29). Our sample is constituted of a group of people that have only vaguely known temporal, geographical and social dimensions (Milner and Boldsen 2017, 29). Concerning the representativeness of the skeletal sample from most historic cemeteries in Quebec, it is impossible to know each individual's geographic origin and social status since no gravestones are recovered most of the time (Ethnoscop 2016b; 2016a; 2014; 2012). However, the time frame (i.e. the period of use of a cemetery) is well established according to archaeological data (Ethnoscop 2016b; 2016a; 2014; 2012). Furthermore, people are sampled at the moment of their death and do not reflect the full range of people that would be observed if one could actually analyse a past living population (Milner and Boldsen 2017; Waldron 1994, 12; Wood et al. 1992).

1.3.4 Integrity of the sample

The state of preservation of the bones can alter the identification of pathognomonic lesions associated to a certain condition (Waldron 1994, 13–14). Certain conditions indeed present lesions that appear on specific elements of the skeleton, which might not be visible in fragmented osteological collections (Waldron 1994, 13–14).

1.3.5 Age-at-death estimation

Adequate age-at-death estimation is challenging amongst adults (Roberts 2018, 137). Age estimation after growth has stopped is more difficult because it is based on the senescence process,

which can be influenced by various cultural, occupational and biological factors (Roberts 2018, 131,137-140). Estimation of age-at-death for people older than 50 years is also very difficult and this can result in an underestimation of the age-at-death for some individuals (Roberts 2018, 137).

1.4 Developmental Origins of Health and Disease (DOHaD)

The DOHaD concept, one of the most important theories in biological science, refers to the fact that environmental parameters in early fetal life can impact the expression of genes and might later have effects on health and disease (Suzuki 2018, 266). The DOHaD theory originates from the work of Barker's epidemiological report in 1986 and many following articles by himself and his colleagues, which led to the "Barker hypothesis" (Suzuki 2018, 266; Barker 2007; Osmond et al. 1993; Barker et al. 1993; 1989; Barker and Osmond 1986a; 1986b). This hypothesis suggests that a poor prenatal nutrition can increase the detrimental effects of an opulent diet in adulthood, causing an increased risk of various non-communicable diseases (Suzuki 2018, 266; Barker 2007; Osmond et al. 1993; Barker et al. 1993; 1989; Barker and Osmond 1986a; 1986b). Later, the terminology changed, from the "Barker hypothesis" to the term "DOHaD", because the period of "origin" was extended from the prenatal period to the entire developmental period, up until the maturation from infancy to adolescence (Suzuki 2018, 266; Gillman et al. 2007).

While the causative factors of DOHaD originally started with poor nutritional status during pregnancy, they have now progressed to include various external and internal environmental factors affecting the whole body, such has drugs and chemicals, light and other electromagnetic waves, sounds and oscillations and indigenous microbiota in the gut and elsewhere (Suzuki 2018, 266; Haugen et al. 2015; Rosenfeld 2015). In biological anthropology, the importance of DOHaD has been recognized (e.g. DeWitte and Stojanowski 2015; Gowland 2015). The use of teeth to investigate the subject has emerged in recent years, especially in biomedical research, since teeth can provide permanent biomarkers of metabolic stressors that disrupted mineralization *in utero* or during early infancy (Brickley, Kahlon, and D'Ortenzio 2019, 1–2; Arora et al. 2017; Tvinnereim et al. 2012).

Chapter 2: Previous clinical and bioarchaeological studies on vitamin D and vitamin D deficiency

2.1 Introduction

Vitamin D plays an essential role in the metabolism of the human organism; however, it is affected by a multitude of environmental and cultural factors (Brickley et al. 2017). Its complex aetiology makes it very difficult to pinpoint the causes of its deficiency, even more so in past populations (Brickley, Moffat, and Watamaniuk 2014). Indeed, even in a similar environment, individuals can present different levels of vitamin D depending on a variety of cultural factors, such as clothing, weaning, number of pregnancies, diet and occupation (Brickley, Moffat, and Watamaniuk 2014). In the next chapter, the current clinical studies on vitamin D and vitamin D deficiency will be addressed, followed by an exploration of the different bioarchaeological research on vitamin D deficiency.

2.2 Clinical studies

2.2.1 Metabolism of vitamin D

Vitamin D exists under two forms, i.e. vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol), which are produced differently (Thacher and Clarke 2011, 50). Vitamin D_3 is generated from the reaction of the skin to the UV rays of the sun or is acquired through the diet (ex. fatty fish, egg yolk, liver, mushrooms, supplements) (Thacher and Clarke 2011, 50). Vitamin D_2, found in certain plants and produced commercially through yeast irradiation, is used for fortification and supplementation (Thacher and Clarke 2011, 50). Both vitamin D_2 and D_3 can be used for supplementation (Thacher and Clarke 2011, 51). The metabolism and effects of both forms of vitamin D is explained more extensively in the following sections and it is summarized in Fig. 2.

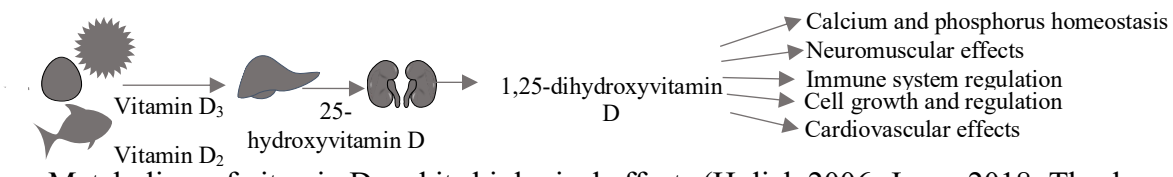

Figure 2. – Metabolism of vitamin D and its biological effects (Holick 2006; Jones 2018; Thacher and Clarke 2011).

The skin of many vertebrate animals naturally produces 7-dehydrocholesterol (provitamin D_3) that resides in epidermal and dermal cells which, during exposure to sunlight, absorb ultraviolet (UVB) radiation (Jones 2018, 3). The absorption of this radiation transforms provitamin D_3 into previtamin D_3 (Jones 2018, 3). By remodelling its double bonds, this element transforms in a more thermodynamically stable structure called vitamin D_3 (Jones 2018, 3).

The vitamin D obtained, through natural light or diet, is, however, an inactive compound which needs to undergo two metabolic processes before becoming an active hormonal compound (Jones 2018; Jones, Strugnell, and DeLuca 1998). The first step, through the liver, produces an intermediary compound 25-hydroxyvitamin D (25(OH)D) (Jones 2018; Blunt, DeLuca, and Schnoes 1968, 3321). The second phase of the activation mainly happens in the kidneys and results in the active form of vitamin D, i.e. 1,25-dihydroxyvitamin D ($1,25(OH)_2$ D) or calcitriol (Jones 2018). When only considering vitamin D_3 absorbed by the skin or acquired through naturally enriched foods, the compounds are referred to as $25(OH)D_3$ and $1,25(OH)_2D_3$ (Jones 2018).

Because few foods contain vitamin D, most people gain their vitamin D intake from sunlight exposure (Webb, Kline, and Holick 1988, 1697). Vitamin D can be stored in fatty tissue during times when sunlight is abundant (e.g. summer), thus providing some stores during times when sunlight exposure is low (e.g. winter) (Webb, Kline, and Holick 1988, 1697). At latitudes where long winters prevail, this storage is insufficient and vitamin D status declines (Webb, Kline, and Holick 1988, 1697).

2.2.2 Effects of vitamin D and vitamin D deficiency on the skeletal and dental systems

The vitamin D thus activated ligates easily to vitamin D receptors (VDR) and generates an increase of calcium and phosphorus absorption through the intestines (Holick 2006, 355). Vitamin D becomes crucial regarding bone formation, resorption and mineralization, as well as maintaining neuromuscular functions (Holick 2006, 355). In case of low vitamin D levels, the small intestine can only absorb 10 to 15% of the dietary calcium, while the intestinal absorption rises up to 30-40% in optimal situation (Holick 2006, 355). Inadequate calcium absorption occurs when vitamin D levels are low, impairing bone health and the majority of metabolic functions and neuromuscular activities (Holick 2006, 355).

Thus, consequences of vitamin D deficiency on skeletal health are multiple. First, an episode of vitamin D deficiency during bone growth and development can cause rickets, i.e. a reduction or absence of endochondral calcification in growth plates, which, under the mechanical pressure of the weight of the individuals, causes bending of the bones (Elder and Bishop 2014, 1665). In adults, remnant bending deformities of nonadult rickets are called residual rickets (Veselka et al. 2018, 69). Vitamin D deficiency in adults can also generate hyperparathyroidism, which can precipitate or increase osteoporosis (Holick 2004, 364). Furthermore, the secondary hyperparathyroidism causes a loss of phosphates in the urine, which results in an imbalance between calcium and phosphate (Holick 2002; 2003; 2004, 364). This inadequate serum composition fails to ensure the mineralization of the osteoid in the bone, resulting in osteomalacia, i.e. nonmineralization of the collagen matrix (Holick 2004, 364). Individuals suffering from osteomalacia thus become at risk of fractures due to a lack of structural support (Holick 2004, 364).

On a microscopical level, vitamin D deficiency can impair dental development and mineralization (D'Ortenzio et al. 2016). During dental growth, the primary dentine, which constitutes the majority of the crown of the tooth, develops before root formation and newly secreted dentine is unmineralized (predentine) (D'Ortenzio, Kahlon, et al. 2018, 102). Both primary dentine and predentine grow in increments during the mineralisation process (D'Ortenzio, Kahlon, et al. 2018, 102; Avery 2002). The mineralization of the dentine requires hydroxyapathite crystals whose spherical regions are named calcospherites, i.e. tiny round spheres containing calcium salts (D'Ortenzio, Kahlon, et al. 2018, 102). A normal mineralization of the dentine results in a homogenous aspect (D'Ortenzio, Kahlon, et al. 2018, 102). However, when an individual has vitamin D deficiency, calcospherites can fail to fuse correctly and can leave badly mineralized patches of dentine, resulting in the presence of IGD or spherical or hemispheric shaped spaces (D'Ortenzio et al. 2016; Dean 2017).

IGD is characterized by a band-like formation that follows the incremental growth lines in the dentine (D'Ortenzio, Kahlon, et al. 2018, 103). If the vitamin D deficiency is long-standing and severe, the IGD can often be found on both sides of the teeth (mesial and distal sides for molars) (D'Ortenzio, Kahlon, et al. 2018, 103). The relative amount of IGD differs in teeth depending on the severity of vitamin D deficiency and the rate of dentinal growth (D'Ortenzio, Kahlon, et al. 2018, 103; Seow, Romaniuk, and Sclavos 1989). The appositional rate is maximal near the growth

initiation centres and decreases progressively towards the apical aspect of the root, which may result in an absence of IGD in the root of the tooth (D'Ortenzio, Kahlon, et al. 2018, 103).

It is important to differentiate IGD from the normal spaces that occur during tooth development, i.e. developmental interglobular dentine (DIGD) (D'Ortenzio, Kahlon, et al. 2018, 105). DIGD appears near the dentinal periphery and consists of poorly mineralized dentine that does not follow the incremental lines, in both the permanent and deciduous dentition of healthy individuals (D'Ortenzio, Kahlon, et al. 2018, 105). DIGD is characterized by a small, sparse area of spaces that can be slightly elongated or wavy (D'Ortenzio, Kahlon, et al. 2018, 105).

Vitamin D deficiency has been recognized as the primary cause of interglobular dentine (IGD), since it ensures the homoeostasis of phosphate and calcium involved in the mineralization of the dentine (D'Ortenzio et al. 2016, 154). Various other vitamin deficiencies (A, C, and E), magnesium deficiency, fluorosis, liver disease and gastrointestinal malabsorption were excluded from the causes of IGD (D'Ortenzio et al. 2016, 154).

2.2.3 Extra-skeletal effects of vitamin D and vitamin D deficiency

Vitamin D is also very involved in extra-skeletal systems. The brain, prostate, breasts and colon, to name a few, as well as immune cells, all possess vitamin D receptors reacting to the presence of $1,25(OH)_2D_3$ (Christakos et al. 2013, 46). According to clinical studies, the occurrence of vitamin D deficiency during infancy can influence the resistance to infections and the development of certain types of cancer and rheumatoid arthritis (Baeke et al. 2010; Bikle 2009; Holick 2004). Indeed, laboratory studies, including animal models, indicate the implication of $1,25(OH)_2D_3$ in numerous systems : inhibition of cancerous progression and certain autoimmune diseases and modulation of innate immunity (Christakos et al. 2013, 46).

First, the vitamin D hormone regulates the expression of certain genes as well as cell differentiation and proliferation (Christakos et al. 2013). According to studies on animals and humans, an increased risk of developing some cancers has been linked to vitamin D insufficiency or deficiency (Snoddy, Buckley, and Halcrow 2016, 187). Research conducted in the United States and Eastern Europe indicate a higher risk of contracting colorectal, breast, prostate, ovaries, bladder, oesophageal, kidney, lung, pancreatic, stomach and corpus uteri cancer in relation with a lesser exposition to UV rays (Garland and Garland 1980; Garland et al. 1990; Gorham, Garland,

and Garland 1990; John et al. 1999; Grant 2002, 1867; Snoddy, Buckley, and Halcrow 2016, 187). These findings have to be taken with precaution since vitamin D deficiency is only one of many risk factors and its specific impact on disease incidence has never been assessed (Peterlik 2012, 784). However, convincing evidence indicate that vitamin D deficiency is a major risk factor for colorectal and breast cancer (Peterlik 2012, 784).

Furthermore, the most common autoimmune diseases, such as type 1 diabetes, rheumatoid arthritis and multiple sclerosis, have all been successfully prevented in models using mice that were susceptible to these diseases if they received $1,25(OH)_2D_3$ early in life (Mathieu et al. 1994; Cantorna, Hayes, and DeLuca 1996; Cantorna, Hayes, and DeLuca 1998; Deluca and Cantorna 2001; Gregori et al. 2002).

It has been revealed that $1,25(OH)_2D_3$ induces macrophages to secrete an antibacterial peptide called cathelicidine involved in the destruction of mycobacteria, including the causal agent of tuberculosis (Lockau and Atkinson 2018; Liu 2006). In fact, many controlled cohort studies of the last decade have found that a low presence of vitamin D is an important independent risk factor for the development of secondary tuberculous infections (Snoddy, Buckley, and Halcrow 2016, 187; Gibney et al. 2008; Nnoaham and Clarke 2008; Ustianowski et al. 2005).

While many clinical studies explore the relation between vitamin D deficiency and various other diseases, all-cause mortality seems to be the most correlated to vitamin D deficiency (Lockau and Atkinson 2018, 5). Indeed, numerous systematic reviews and meta-analyses revealed a decrease of all-cause mortality amongst individuals of cohort and observational studies presenting a higher vitamin D status or amongst individuals of RCT taking supplements (Lockau and Atkinson 2018, 5; Autier 2014; Bolland et al. 2014; Theodoratou et al. 2014; Reid, Bolland, and Grey 2014; Schöttker 2013). Some studies even recorded a mortality reduction of 4 to 8% (Lockau and Atkinson 2018, 5; Autier 2014; Schöttker 2013).

2.2.4 Vitamin D deficiency's biophysical risk factors

The biophysical risk factors include geographical latitude, skin pigmentation, bioavailability of vitamin D in food sources, body fat content, fat malabsorption, gastrointestinal malabsorption and age (Brickley, Moffat, and Watamaniuk 2014, 48; Bikle 2007, V–51; Holick 2006, 356–57).

The latitude and the season can affect the vitamin D metabolism of an individual, since at latitudes above 37°N and below 37°S, sunlight is insufficient to induce cutaneous vitamin D_3 synthesis during winter months (Holick 2006, 356; Holick and Jenkins 2003; Webb, Kline, and Holick 1988; Chen 1999). In northern latitudes, seasons strongly affect circulating levels of $25(OH)D_3$, which decrease during winter and increase during summer (Huotari and Herzig 2008, 172; Rapuri et al. 2002; Maxwell 1994). In Finland (60° N latitude), 328 healthy adults (202 women, 126 men, 31-43 years-old) participated in a study by Lambert-Allardt and colleagues (1998) from February to March. The mean daily intake met the recommendations in the men (5.6 ± 3.2 μg) and in the women (4.7 ± 2.5 μg), but the levels were low (Lamberg-Allardt et al. 2001, 2066). This indicates that even in a normal, healthy, adult population, the vitamin D that has been stored during the summer is not enough to ensure an optimal vitamin D status during winter (Lamberg-Allardt et al. 2001, 2072).

Up to 99.9% of the cutaneous production of vitamin D can be reduced as skin pigmentation increases (Holick 2006, 357; Matsukoa et al. 1995; 1991; Clemens et al. 1982). The studies of Clemens and colleagues (1982), which measured vitamin D itself, indicated that the presence of melanin in high concentrations in skin markedly reduces the amount of vitamin D_3 that is made after one exposure to a relatively small dose of UV rays.

Starting at birth, breastfeeding can put neonates and young children at risk of developing vitamin D deficiency (Holick 2004, 365; Kreiter et al. 2000; Specker 1985). Indeed, breast milk does not contain significant amounts of vitamin D, which puts the infants at risk of developing this kind of metabolic disorder, especially if the mother is already deficient (Holick 2004, 365; Kreiter et al. 2000; Specker 1985). The total vitamin D concentration in human milk is 15 to 50 International Units (IU)/L, which is too low to provide the recommended daily requirement of 400 IU/day (Kreiter et al. 2000, 155; Reeve, Chesney, and DeLuca 1982). Furthermore, the children of women of colour are particularly at risk (Holick 2004, 365; Kreiter et al. 2000; Specker 1985). Dark-skinned mothers require a longer duration of sun exposure than light-skinned mothers to receive sufficient vitamin D concentrations due to the higher concentration of melanin in their skin (Misra et al. 2008, 406; Clemens et al. 1982). As a result, decreased vitamin D levels in the mothers result in a decreased transplacental transfer *in utero*, which reduces the infants' stores of vitamin D at birth (Misra et al. 2008, 408).

It has also been suggested that phytate-derived polyphosphate esters, present in cereal-based products, may be rachitogenic (Robertson et al. 1981, 22; Van Den Berg, Hill, and Stanbury 1972). Lignin, an important component of wheat fibre, combines with bile acids and increases their excretion (Robertson et al. 1981, 22; Eastwood and Hamilton 1968). If vitamin D fuses to the fibre-acid complex, which is chemically likely, it may be transported through the gut (Robertson et al. 1981, 22; Reinhold 1976). A cereal-based diet may thus lead to enough wastage of vitamin D to produce a deficiency (Robertson et al. 1981, 22).

Recent studies indicated that body mass index and body fat content are inversely related to serum 25(OH)D level, which is probably due to the deposition of vitamin D in body fat units (Holick 2006, 357; Snijder et al. 2005; Bell et al. 1985). Individuals with fat malabsorption syndromes, including sprue, cystic fibrosis and Crohn's disease, are at especially high risk of vitamin D deficiency because of the malabsorption of oral vitamin D (Holick 2006, 357; Koutkia et al. 2001; Bell et al. 1985).

There are numerous reasons why gastrointestinal disorders can affect vitamin D absorption. First, vitamin D absorption requires an intact small intestine, pancreas and liver (Bikle 2007, V–51). Partial gastrectomy, chronic pancreatic insufficiency, intrinsic small bowel disease, disorders of the biliary tract and surgical bypass procedures can all cause problems (Bikle 2007, V–51). Second, since vitamin D needs to be metabolized into an active compound through the liver, diseases of the liver can alter the availability of the active metabolites (Bikle 2007, V–51). Third, certain diseases of the liver and small intestine can disrupt the circulation of vitamin D, which is usually conjugated with bile reabsorbed in the small intestine (Bikle 2007, V–51). Fourth, vitamin D has to be transported in the blood through vitamin D-binding proteins which are produced in the liver (Bikle 2007, V–51). Any disorder affecting the liver can decrease the synthesis of these proteins and impair the delivery of the vitamin D metabolites to the target tissues (Bikle 2007, V–51). Finally, the intestine affected by disease, aging or surgical alteration may fail to respond normally to the active vitamin D metabolites concerning calcium and phosphate absorption (Bikle 2007, V–51).

Moreover, aging has been associated with lower 25(OH)D levels regardless of the season (Rucker et al. 2002, 1521). Dietary deficiencies and decreased cutaneous synthesis due to reduced ability of the skin to synthesize vitamin D_3 put the elderly at risk of developing vitamin D

deficiency (Holick 2006, 357). In fact, a 70-year-old produces approximately four times less vitamin D via synthesis compared with a 20-year-old (Holick 2006, 357; Holick, Matsukoa, and Wortsman 1989; MacLaughlin and Holick 1985).

2.2.5 Vitamin D deficiency's biocultural risk factors

The clinical literature is abundant regarding the risk factors associated to vitamin deficiency, both biological and cultural. The Fig. 3 presents an overview of these factors that are presented extensively in the next sections.

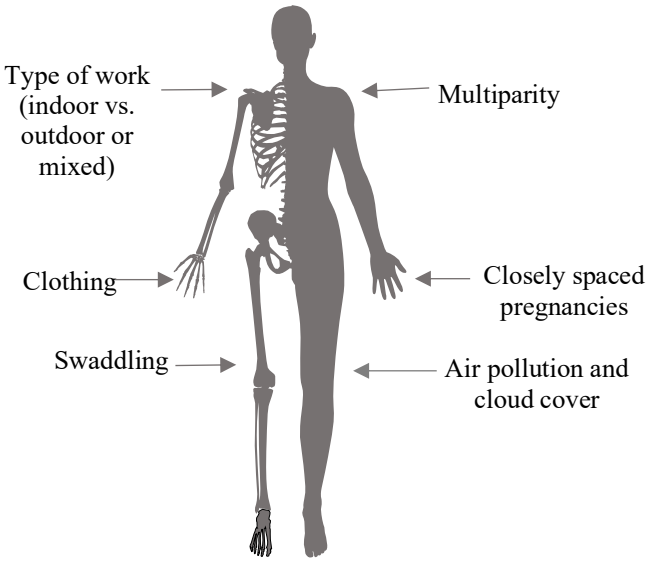

Figure 3. – Biocultural risk factors of vitamin D deficiency. Figure prepared according to the data collected by Brickley, Moffat, and Watamaniuk (2014).

Working young and middle-aged adults who rarely spend any time outdoors or always wear sun protection outdoors are at high risk of vitamin D deficiency (Tangpricha et al. 2002). Tangpricha and colleagues (2002) observed that 32% of healthy adults aged 18 to 29 years-old were vitamin D deficient at the end of the winter in Boston (Tangpricha et al. 2002; Holick 2004, 365–66). The elderly and institutionalized individuals who barely spend any time outside are also at high risk of developing vitamin D deficiency (Pearce and Cheetham 2010, 144).

Clothing also affects the intake of natural UVB light and restricts it, even in regions of the world getting plenty of sun exposure (Holick 2006, 356). The practice of covering the entire body with clothing and of avoiding sunlight adopted by some Saudi Arabian women and children might account for the high prevalence of osteomalacia in the former and of rickets in the latter (Holick

2006, 356). Moreover, a fully clothed infant without a hat requires four times as much sun exposure as an infant in only a diaper to achieve similar 25(OH)D concentrations, which can be problematic if the former is not allowed sufficient time outside due to cultural practices (Specker et al. 1985, 372).

The number of UVB rays that reach the surface of the earth can be impaired by cloud cover and industrial pollution (Misra et al. 2008, 408). Industrial pollution has been associated with a greater prevalence of vitamin D deficiency rickets according to the results of a study of 34 children aged 9-24 months-old living in an area of Delhi, India, renowned for high levels of atmospheric pollution, with a comparable group of children from a less polluted area of the city (Misra et al. 2008, 408; Agarwal 2002).

Swaddling is a cultural practice that might put newborns at risk of developing vitamin D deficiency (Wayse et al. 2004). Indeed, that practice entails that babies are tightly enveloped in layers of cloth from the neck to the feet to minimize body movement (Yurdakok, Yavuz, and Taylor 1990, 873). In India, research conducted by Wayse and colleagues (2004) indicated that unswaddled children had 42,4 nmol/L of serum $25OHD_3$ compared to 26.5 nmol/L for swaddled children when exposed to outdoor sun (Wayse et al. 2004, 556).

Other practices can influence the vitamin D levels in individuals, especially for women. The clinical literature on the possible link between multiple and closely spaced pregnancies is limited (Brickley, Moffat, and Watamaniuk 2014, 55). Sachan and colleagues (2005) indicate that vitamin D deficiency could be due to calcium requirements of the developing fetus but they also remark that factors contributing to vitamin D deficiency are complex (Brickley, Moffat, and Watamaniuk 2014, 55; Sachan et al. 2005). A study of the status of serum (25(OH)D) in a healthy section of the population in Tunisia concluded that multiparity was noted in 68.9% of the subjects with hypovitaminosis D, while in the nonparous group, hypovitaminosis D was only observed in 43.9% of the subjects (Meddeb et al. 2005, 181).

2.3 Bioarchaeological studies

2.3.1 Introduction

Culture and environment-related vitamin D deficiencies have been studied in bioarchaeology among different periods and societies (Mays et al. 2018). However, much of the research has been conducted on European skeletal samples dating to the 19th century A.D., since this period is characterized by the Industrial Revolution where various environmental factors probably contributed to an increase in vitamin D deficiency (Watts and Valme 2018; Mays 2018; Ives 2018; Mays, Brickley, and Ives 2007; 2006). Few bioarchaeological studies have been conducted on North American populations of the same period and mainly consist of vitamin D deficiency-related lesions being recorded in a few individuals (Table 1) (Brickley, Kahlon, and D'Ortenzio 2019; D'Ortenzio et al. 2018; 2016; Houle-Wierzbicki 2016; Morland 2009). Key studies based on 19th-century populations of European descent are presented in Table 1. These sites offer a good comparison to the St. Antoine cemetery, since they are from similar time periods, are composed of populations of European ancestry and are located at northern latitudes. Although the St. Antoine cemetery was situated in an urban environment, it is relevant to consider sites from other environments considering that cultural practices also impact vitamin D deficiency.

Time period	Geographical location	Context	N	Key findings	Source for key findings
Historic, 1771-1860 CE	St. Matthew, Québec city, Quebec, Canada	Urban area	1	Individual 15A-S36 presents 2 episodes of IGD: 1.5-2 and 5.5 years (LM[1])	D'Ortenzio et al. 2016; Brickley, Kahlon, and D'Ortenzio 2019
			1	Individual 15A-S36 presents evidence of residual rickets	Houle-Wierzbicki 2015
			4	Four individuals, two children and two adults, present lesions of rickets and osteomalacia	Morland 2009
Historic, 1748-1878 CE	St. Marie, Beauce, Québec, Canada	Rural area	1	2 episodes: prenatal and ~2.5 years (RM[1])	D'Ortenzio et al. 2016; Brickley, Kahlon, and D'Ortenzio 2019
Historic, 1799-1854 CE	St. Antoine, Montréal, Québec, Canada	Urban area	1	1 episode of IGD: 3 years (LM[1])	D'Ortenzio, Ribot, et al. 2018; Brickley, Kahlon, and D'Ortenzio 2019

Historic, 18th-19th centuries CE	Birmingham, England	Urban area	136 adults, 164 nonadults	5% (7/136) of adults had evidence of osteomalacia, while 12.8% (21/164) of nonadults showed evidence of rickets	Mays, Brickley and Ives 2006; Brickley, Mays and Ives 2007
Historic, 19th century CE	St. John's Church, Redhill, Surrey, UK	Suburban area	79 nonadults	17.7% (14/79) presented rachitic lesions	Watts and Valme 2018
Historic, 19th century CE	Beemster, Netherlands	Rural area	200 adults	14.5% (29/200) of adults had evidence of residual rickets, while 3 others might	Veselka et al. 2018;
			95 nonadults	9.5% (9/95) of nonadults had rickets	Veselka, Hoogland, and Waters-Rist 2015;
			15 adults	14/15 adults presented IGD based on micro-CT and histological data (M^1/C_1)	Veselka et al. 2019
Historic, 17th-19th century CE	Hattem, Netherlands	Urban area	88 adults, 21 nonadults	23.8% (5/21) of nonadults had evidence of rickets, 23.9% (21/88) of adults had evidence of residual rickets	Veselka 2019
			15 adults/nonadults	13/15 adults/nonadults presented IGD based on micro-CT and histological data (M^1/C_1).	Veselka et al. 2019

Table 1. – Vitamin D deficiency-related lesions associated with time period and geographical location. Modified from Brickley et al. (2017).

2.3.2 Lack of natural light exposure

Latitude of living site

The choice of living environment can impact a population's UV rays exposure (Veselka et al. 2019; Veselka, Hoogland, and Waters-Rist 2015; Holick 2003). For example, 29 adults from the rural locality of Beemster, Netherlands (19th century CE), studied by Veselka and colleagues (2018) (N= 200) presented residual rickets (Veselka et al. 2018). Nine nonadults studied by Veselka and colleagues (2015) presented rickets (N=95). 14/15 adults also presented IGD based on micro-CT and histological data of the $M^1/C1$ (Veselka et al. 2019). At the site of Hattem, Netherlands (17th-19th century CE), 5/21 nonadults had evidence of rickets, 21/88 adults showed residual rickets and 13/15 adults and nonadults presented IGD based on micro-CT and histological data of the $M^1/C1$ (Veselka 2019; Veselka et al. 2019). As the sites are located at a latitude of 52°N, the inhabitants had significantly decreased dermal synthesis of vitamin D during winter and early spring, i.e. from November to March, and possibly increased vitamin D deficiency in vulnerable individuals (Veselka et al. 2019; Veselka, Hoogland, and Waters-Rist 2015; Holick 2003). The

rather northern latitudes of the Ste. Marie, St. Antoine and St. Matthew cemeteries in Quebec, Canada (45°N) and of the Birmingham cemetery and the Saint-John's church cemetery (± 50 °N) in England might also have contributed to the vitamin D-related lesions found in some individuals (Watts and Valme 2018; Webb et al. 2018; D'Ortenzio et al. 2016; Gagnon et al. 2010; Brickley, Mays, and Ives 2007; Mays, Brickley, and Ives 2006).

Indoor work and habits

During the Industrial Revolution, in Europe, factory work became increasingly common, which is reflected in the bioarchaeological record (Brickley, Mays, and Ives 2007). The skeletons of 164 nonadults and 143 adults coming from the late 18th and 19th century churchyard of St. Martin's Birmingham, England presented a few cases of vitamin D deficiency (Brickley, Mays, and Ives 2007, 69). None of the adults buried in vault burials (N=59) presented lesions of osteomalacia, while seven of the 84 adults buried in earth-cut graves showed some evidence of this disease (Brickley, Mays, and Ives 2007, 69). The type of burial seemed to suggest that the lesions associated with vitamin D deficiency might be linked to lower status adult individuals working for long periods of time in factories (Brickley, Mays, and Ives 2007, 78).

Suburban areas near industrializing cities were not immune to vitamin D deficiency (Watts and Valme 2018). Indeed, 76 individuals coming from the cemetery of St. John's Church in Redhill, Surrey (19th century) were analysed by Watts and Valme (2018) for vitamin D deficiency lesions. Fourteen nonadults presented rachitic lesions, indicating a prevalence of 17.7% (Watts and Valme 2018, 3). One of the reasons for the high prevalence of rickets in the individuals of the suburban area is the presence at the time of the nearby Reigate Union Workhouse (Watts and Valme 2018, 7). The records from 1881 indicate a list of 193 inmates, including 43 children under 18 years of age (Watts and Valme 2018, 7). Previous bioarchaeological studies into workhouse assemblages have shown that both adults and children presented high rates of pathological lesions, including those associated with metabolic deficiencies (Geber 2016; Geber and Murphy 2012).

In rural areas, vitamin D deficiency was still prevalent among certain groups working indoor (Veselka et al. 2018). The study of Veselka and colleagues (2018) on the rural community of Beemster, Netherlands (19th century) explored the association between vitamin D deficiency and gender-related activities. Out of the 200 adults (18-50+ years) exhumed from the cemetery and

then analysed, 29 presented residual rickets, which was recorded more frequently in the Beemster females than males, suggesting that female nonadults lacked more sunlight exposure compared to male nonadults (Veselka et al. 2018, 73). Up to the 19th century, in most populations of the region and at that time, including Beemster, men worked in the fields, while women mostly conducted work indoor in and around the house, such as spinning, sawing, laundry, churning butter, making cheese and taking care of the children (Veselka et al. 2018, 73). This division of labour started early in life, as young as six years-old, sometimes even younger (Veselka et al. 2018, 73).

Urbanism

According to the study of Mays and colleagues (2018) on human remains pertaining to 18 Roman cemeteries, no association between the settlement type and vitamin D deficiency was found except for one site, Isola Sacra, the necropolis of the port town of Ostia. Both documentary and physical evidence indicate the presence of multi-storey buildings, mainly apartment blocks, in Ostia (Bowman and Wilson 2011; Storey 2003; Packer 1967; Ling 1973). Density of housing at Ostia resulted in a population density possibly even higher than in Rome (Storey 2001; 1997). The lack of sunlight caused by these factors might have influenced the higher prevalence of rickets recorded in individuals buried at Isola Sacra (Mays et al. 2018, 9–10).

As for the period of the Industrial Revolution, the developing urban centres did present a building arrangement improper to sufficient natural light exposure (Brickley, Mays, and Ives 2007, 75). The individuals from St. Martin's churchyard in Birmingham which presented vitamin D deficiency-related lesions might have been affected by living in camped inner city housing with limited and unsanitary outside spaces (Brickley, Mays, and Ives 2007, 75).

Brickley and colleagues (2019) suggest that, as the economic development grew in 18th century Québec city due to shipbuilding and export of agricultural produce, population growth generated cramped housing, which might have limited sunlight exposure (Brickley, Kahlon, and D'Ortenzio 2019, 8; Library and Archives Canada 2016).. The individuals suffering from vitamin D deficiency among the St. Matthew cemetery were thus possibly affected by this predicament (Brickley, Kahlon, and D'Ortenzio 2019, 8; Library and Archives Canada 2016).

Atmospheric pollution

According to Lewis (2002), industrialization is the main factor of health deterioration in children (Lewis 2002, 211). Indeed, in 19th-century London, air pollution created by industrial development limited solar ultraviolet reaching the ground level (Loomis 1970). This might have been a contributing factor to the high rate of rickets in the St. Martin's population (Mays, Brickley, et Ives 2006, 371). The lesions related to vitamin D deficiency in the St. Martin's children indicate that it was a common and frequent condition, i.e. active and healed rickets present in a wide range of ages (six months to four years) and osteomalacia in adults (Watts and Valme 2018, 5; Mays, Brickley, and Ives 2006, 371). On the opposite, the age profile at Redhill is very different, with individuals between six months to 2.5 years of age affected by rickets, and healed cases observed after 2.6 years of age (Watts and Valme 2018, 5).The lack of atmospheric pollution in the Redhill suburban area and the differences in age distribution of active and healed rickets indicate that a completely different set of risk factors are occurring at Redhill (Watts and Valme 2018, 5).

Clothing

Clothing is a cultural factor that highly influences the exposure to sunlight. During the 19th century, the type of clothing worn by Dutch nonadults and adults barely let any skin exposed, which might have increased the risk of developing vitamin D deficiency among the individuals buried in the Beemster cemetery in the Netherlands (Veselka et al. 2018, 72). Clothing also constitutes a contributing factor to the prevalence of vitamin D deficiency in the human remains of the suburban Redhill cemetery (Watts and Valme 2018, 5). Indeed, historical documents indicate that children were clothed from the neck down when playing outside, which only left the head and hands uncovered (Watts and Valme 2018, 5). Lengthy or frequent periods under the sun would have been needed to obtain the required amount of vitamin D, which might have been possible in the summer, but not in winter, Redhill laying at a latitude of 52°N (Watts and Valme 2018, 5).

2.3.3 Lack of or impairment of vitamin D in the diet

The suburban community of Redhill, England (19th century) offers an example of the influence of the diet on the development and healing of vitamin D deficiency-related lesions (Watts and Valme 2018). During the mid to late 19th century, weaning was encouraged once the first teeth erupted at approximately 12-18 months old, introducing a new diet composed of bread, oatmeal and broth (Watts and Valme 2018, 6; Wickes 1953a). The high levels of phytates present in this

type of diet might have impaired calcium and phosphorus absorption, causing rachitic type lesions (Pettifor 2004). However, beyond the age of two, only healed rickets were recorded in the Redhill individuals, which concurs with the integration of a more adult diet composed of accessible vitamin D enriched foods (ex. cod liver oil, eggs, kippers, herring) (Watts and Valme 2018, 5; Clayton and Rowbotham 2008; Moore 1999). Furthermore, cod liver oil was administered in the early 19th century and, at Redhill, it might have been done more specifically to individuals presenting signs of rickets, which might have been more noticeable once the children were walking (Watts and Valme 2018, 7; Fildes 2017; Wickes 1953b).

2.3.3 Perinatal Practices

During the historical period, some of the studied nonadults of the rural community of Beemster (19th century) exhibited short periods of breastfeeding, i.e. a few weeks to a few months, while others indicated no breastfeeding (Veselka et al. 2018, 72; Waters-Rist and Hoogland 2017). The cultural practice of feeding the infants cow milk might have contributed to the development of vitamin D deficiency (Veselka et al. 2018, 72). Indeed, cow's milk differs substantially from human breast milk and predisposes children to many ailments: gastrointestinal bleeding, intestinal blockages, bacterial infections, reduced immune function, gastric symptoms (vomiting, diarrhoea), breathing difficulty and iron deficiency anaemia (Veselka et al. 2018, 72; Thompkinson and Kharb 2007; Butte et al. 2002; Sullivan 1993). When an individual is affected by one or many of these conditions, vitamin D can become less bioavailable (Veselka et al. 2018, 72).

2.3.4 Link between vitamin D deficiency and other diseases

The research by Snoddy and colleagues (2016) is a key study since it is one of the only studies that explored the possible link between vitamin D deficiency cases and other diseases, i.e. chronic infectious diseases. While the goal of the present study is not identical to the research by Snoddy and colleagues (2016), it still provides useful information and background. As Snoddy and colleagues (2016) mention, an important part of the poor vitamin D status spectrum is archaeologically invisible when looking at the lesions on the skeleton (Fig. 4). For example, the adequate vitamin D status can be recovered before skeletal lesions appear and the diagnostic lesions can remodel before the death of the individual (Snoddy, Buckley, and Halcrow 2016, 189).

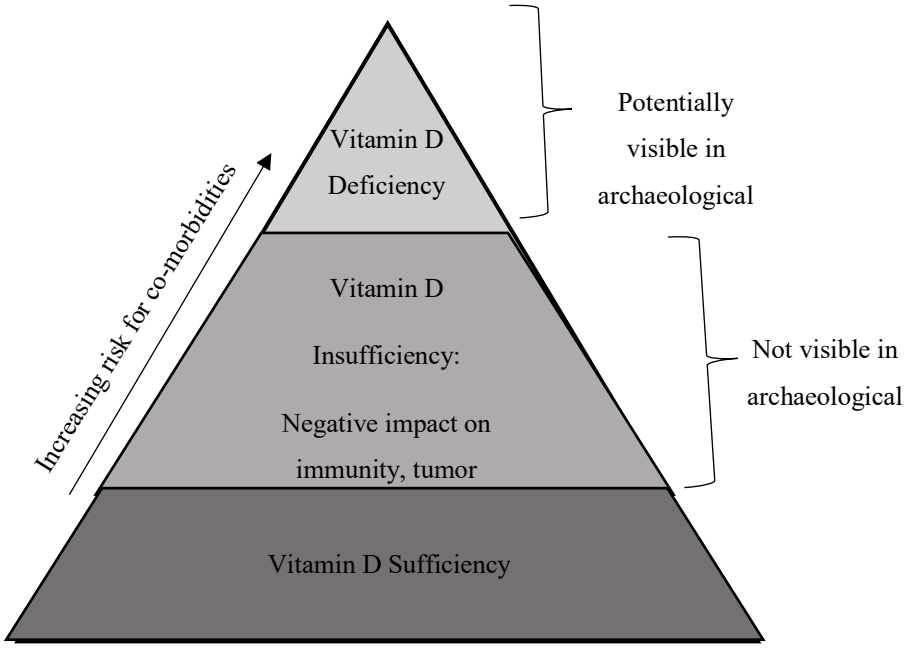

Figure 4. – Scope of vitamin D-related lesions visible in archaeological assemblages (Snoddy et al. 2016).

A key study of Snoddy and colleagues (2016) considered comorbidities from a paleopathological perspective despite the fact that a vast majority of nonskeletal disease processes associated with vitamin D deficiency are archaeologically invisible. Compromised immunity is explored by the authors, meaning that in skeletal assemblages where multiple cases of vitamin D deficiency are present, a similar high prevalence of chronic infectious disease (e.g. treponemal disease and tuberculosis) could also be expected (Snoddy, Buckley, and Halcrow 2016, 190; Pearce and Cheetham 2010; Wayse et al. 2004). Overall mortality also has to be considered according to the researchers, meaning that non-infectious and acute infectious pathologies related to poor vitamin D status should be taken into account as potential contributing factors in populations from regions and eras where people were at high risk of vitamin D deficiency, for example post-colonial North America (Snoddy, Buckley, and Halcrow 2016, 190). The authors refer to "chronic infection" as "any individual exhibiting skeletal lesions attributable to any infectious process (e.g. tuberculosis, nonspecific osteomyelitis, etc.)" (Snoddy, Buckley, and Halcrow 2016, 191). "Nonspecific infection" refers to "individuals exhibiting lesions that are potentially attributable to an infectious process but not to a specific infectious disease" (Snoddy, Buckley, and Halcrow 2016, 191).

The sample used by Snoddy et al (2016) is composed of two cemeteries, St-. Mary Graces (1350-1540 AD; N= 389) and St. Benet Sherehog cemetery (16th-17th centuries AD; N=231)(Snoddy, Buckley, and Halcrow 2016, 19; Miles, White, and Tankard 2008). The results of both analyses are presented in Table 2.

	St. Mary Graces	St. Benet Sherehog
N= 100%	389	231
Vitamin D Deficiency	0.5%	15.0%
Infection (all)	18.0%	54.0%
Tuberculosis	0.8%	2.0%

Table 2. – Prevalence of skeletal evidence of vitamin D deficiency, all chronic infection (including nonspecific infections), and tuberculosis at St. Mary Graces and St. Benet Sherehog (Snoddy, Buckley, and Halcrow 2016).

In both of the populations, these authors found a strong association between the prevalence of vitamin D deficiency and chronic infection at the 95% confidence level (Snoddy, Buckley, and Halcrow 2016, 191). However, a nonsignificant correlation was demonstrated between the prevalence of vitamin D deficiency and tuberculosis alone (Snoddy, Buckley, and Halcrow 2016, 191).

In this research, periosteal new bone formations were considered as lesions of chronic infectious disease, even though trauma, metabolic, neoplastic and autoimmune diseases could also be the cause (Snoddy, Buckley, and Halcrow 2016, 191). However, the authors suggest that future researchers applying this model should only include individuals with lesions that are exclusively related to a systemic infectious process (Snoddy, Buckley, and Halcrow 2016, 191). The lesions exclusively linked to a systemic infectious process refer to a bilateral involvement of multiple skeletal elements and lesion type and patterning which is different from non-infectious pathologies such as scurvy or rickets (Buckley and Tayles 2003; Roberts 2019). Furthermore, although the authors were not able to conclude to an association between the prevalence of vitamin D deficiency and tuberculosis at either site, they still recommend exploring this issue (Snoddy, Buckley, and Halcrow 2016, 192).

In sum, this case concludes to a strong association between the prevalence of vitamin D deficiency and chronic infections, but not tuberculosis alone (Snoddy, Buckley, and Halcrow 2016, 191). It will be interesting to verify whether the same results will be obtained when considering vitamin D deficiency episodes that occurred during infancy. Moreover, as the authors suggested, only individuals with lesions that are exclusively related to systemic infectious process will be integrated to this pathological category (Snoddy, Buckley, and Halcrow 2016, 191).

2.4 Conclusion

To sum up, the current research aims to build on previous clinical and bioarchaeological studies to better understand vitamin D deficiency's manifestation in a North American urban population of the early to mid-19th century. Environmental and demographic parameters studied in various historic populations can serve as comparative references for the sample under study here, i.e. an urban population of Montréal dated to the early to mid-19th century. The issue of the possible association between vitamin D deficiency and other diseases will be explored here using a detailed methodological approach, which is presented in the next section, "Methodology, Results, Discussion and Conclusion-Journal article in preparation".

Chapter 3: Methodology, Results, Discussion and Conclusion-Journal article in preparation

The results of this research are presented as an article (titled *Palaeoepidemiological analysis of a historical urban population from Montréal: exploring interactions between vitamin D deficiency and various palaeopathological skeletal manifestations*) that will be submitted to the *International Journal of Palaeopathology*. My participation resides in the writing of the paper and in the collection as well as the analysis of the data.

Abstract (250 words limit)

Objective: This project aims to explore the possible link between vitamin D deficiency during infancy and mortality, biological sex, industrialization and health deterioration later in life.

Materials: 52 skeletons and M_1 from the St. Antoine cemetery (1799-1854), Montréal, Québec.

Methods: Macroscopic and radiographic observations of pathological lesions and histological examination of interglobular dentine (IGD) in M_1.

Results: Prevalence of IGD of 44% to 75%, excluding and including the degree 1 of IGD, respectively. The episodes occurred mostly at or from birth to up to 3 years-old, six prenatal episodes were recorded. The presence of IGD was not correlated with demographic parameters, nor with health status deterioration later in life, however, a possible link was noted with LEH. IGD cases during infancy seem to decrease with time, although this result is mitigated due to the small portion of the sample coming from recent parts of the cemetery.

Conclusions: Interesting implications for maternal vitamin D deficiency and cultural practices surrounding infants impairing proper vitamin D intake. More severe episodes of IGD and LEH are possibly linked, although the latter is related to various factors.

Significance: This is the first archaeological populational North American study on IGD. It holds implications for epidemiological studies on vitamin D deficiency and the relationship between vitamin D deficiency and health, biological, environmental and cultural factors.

Limitations: The sample is small and first-generation migrants remain unidentified.

Suggestions for Further Research: Use of a bigger sample. Add isotopic and/or aDNA analysis for geographical origins to complement individual health biographies.

Keywords: 19th century, histology, IGD, health, culture, industrialization.

1. Introduction

Metabolic disorders such as vitamin D deficiency can be caused by biophysical variables, such as geographical latitude, skin pigmentation and bioavailability of vitamin D in food sources (Brickley, Moffat, and Watamaniuk 2014, 48). Cultural factors that influence individual and populational risk such as infant and child feeding practices and behaviours that affect exposure to sunlight can also cause vitamin D deficiency (Brickley, Moffat, and Watamaniuk 2014, 48). Vitamin D_3 (cholecalciferol) is generated from the reaction of the skin to the UV rays of the sun or acquired through the diet (e.g. fatty fish, egg yolk, liver, mushrooms) (Thacher and Clarke 2011, 50). The vitamin D obtained, through natural light or diet, is, however, an inactive compound which needs to undergo two metabolic processes before becoming an active hormonal compound (Jones 2018; Jones, Strugnell, and DeLuca 1998). The first step, through the liver, produces an intermediary compound 25-hydroxyvitamin D (25(OH)D) (Jones 2018; Blunt, DeLuca, and Schnoes 1968, 3321). The second phase of the activation mainly happens in the kidneys and results in the active form of vitamin D, i.e. 1,25-dihydroxyvitamin D ($1,25(OH)_2$ D) or calcitriol (Jones 2018). The activated vitamin D ligates easily to vitamin D receptors (VDR) and generates an increase of calcium and phosphorous absorption through the intestines (Holick 2006, 355). Vitamin D becomes crucial regarding bone formation, resorption and mineralization, as well as maintaining neuromuscular functions (Holick 2006, 355). Thus, consequences of vitamin D deficiency on skeletal health are multiple. Skeletal conditions associated with low vitamin D include rickets, hyperparathyroidism, osteoporosis and osteomalacia (Elder and Bishop 2014, 1665; Holick 2004, 364). Vitamin D is also involved in extra-skeletal systems. The brain, prostate, breasts and colon, to name a few, as well as immune cells, all possess vitamin D receptors reacting to the presence of $1,25(OH)_2D3$ (Christakos et al. 2013, 46). According to clinical studies, the occurrence of vitamin D deficiency during infancy can influence the resistance to infections and the development of certain types of cancer and rheumatoid arthritis (Baeke et al. 2010; Bikle 2009; Holick 2004). Indeed, laboratory studies, including animal models, indicate the implication of $1,25(OH)_2D_3$ in numerous systems : inhibition of cancerous progression and certain autoimmune diseases and

modulation of innate immunity (Christakos et al. 2013, 46). Vitamin D's implication in cancer progression is related to the fact that this hormone regulates the expression of cell proliferation and differentiation (Christakos et al. 2013). Convincing evidence indicate that vitamin D deficiency is a major risk factor for colorectal and breast cancer (Peterlik 2012, 784). Studies on mice also indicate that the most common autoimmune diseases (e.g. type 1 diabetes, rheumatoid arthritis, multiple sclerosis) can be prevented when administering $1,25(OH)_2D3$ early in life (Mathieu et al. 1994; Cantorna, Hayes, and DeLuca 1996; Cantorna, Hayes, and DeLuca 1998; Deluca and Cantorna 2001; Gregori et al. 2002). Many cohort and controlled studies also show that low presence of vitamin D is a risk factor for the development of secondary tuberculosis (Ustianowski et al. 2005; Nnoaham and Clarke 2008; Gibney et al. 2008; Snoddy, Buckley, and Halcrow 2016, 2016). While many clinical studies explore the relation between vitamin D deficiency and various other diseases, all-cause mortality seems to be the most correlated to vitamin D deficiency (Lockau and Atkinson 2018, 5). These studies contribute to the DOHaD theory, i.e. the fact that environmental parameters during the developmental period can impact the expression of genes and might later have effects on health and disease, citing vitamin D deficiency as a causative factor for various pathological conditions (Suzuki 2018, 266). In biological anthropology, the importance of DOHaD has been recognized (e.g. DeWitte and Stojanowski 2015; Gowland 2015) and the use of teeth as means to investigate the subject has been emerging in recent years, which is reflected by the use of deciduous teeth in biobanks for biomedical research (e.g. Arora et al. 2017; Tvinnereim et al. 2012) (Brickley, Kahlon, and D'Ortenzio 2019, 2). Indeed, teeth constitute permanent biomarkers of metabolic stressors disrupting mineralization *in utero* or during early infancy (Brickley, Kahlon, and D'Ortenzio 2019, 1).

On a microscopical level, vitamin D deficiency can impair dental development and mineralization (D'Ortenzio et al. 2016). The mineralization defaults present in the dentine called interglobular spaces (IGD) are caused by the inability of the inorganic calcospherites, i.e. tiny round spheres containing calcium salts, to fuse correctly (D'Ortenzio et al. 2016, 153–54). Vitamin D deficiency has been recognized as the primary cause of interglobular dentine (IGD), since it ensures the homoeostasis of phosphate and calcium involved in the mineralization of the dentine (D'Ortenzio et al. 2016, 154). Various other vitamin deficiencies (A, C, and E), magnesium deficiency, fluorosis, liver disease and gastrointestinal malabsorption were excluded from the causes of IGD (D'Ortenzio et al. 2016, 154).

IGD is characterized by a band-like formation that follows the incremental growth lines in the dentine, since both primary dentine and predentine form in increments during the mineralization process (D'Ortenzio, Kahlon, et al. 2018, 102–3; Avery 2002). If the vitamin D deficiency is long-standing and severe, the IGD can often be found on both sides of the teeth (mesial and distal sides for molars) (D'Ortenzio et al. 2018, 103). The relative amount of IGD differs in teeth depending on the severity of vitamin D deficiency and the rate of dentinal growth (D'Ortenzio, Kahlon, et al. 2018, 103; Seow, Romaniuk, and Sclavos 1989). The appositional rate is maximal near the growth initiation centres and decreases progressively towards the apical aspect of the root, which may result in an absence of IGD in the root of the tooth (D'Ortenzio, Kahlon, et al. 2018, 103). IGD differs from the normal spaces that occur during tooth development, i.e. developmental interglobular dentine (DIGD) (D'Ortenzio, Kahlon, et al. 2018, 105). DIGD appears near the dentinal periphery and consists of poorly mineralized dentine that does not follow the incremental lines, in both the permanent and deciduous dentition of healthy individuals (D'Ortenzio, Kahlon, et al. 2018, 105). DIGD is characterized by a small sparse area of spaces that can be slightly elongated or wavy (D'Ortenzio, Kahlon, et al. 2018, 105).

Several biocultural factors can affect the development of vitamin D deficiency in past populations (Thacher and Clarke 2011, 50). Indeed, vitamin D levels depend on natural light exposure and dietary intake, which are highly linked to cultural behaviour (Thacher and Clarke 2011, 50). The main biocultural risk factors (e.g. latitude of living site, indoor work/habits, urbanism/pollution, clothing, perinatal practices, diet) for vitamin D deficiency explored in 19th century European populations are presented in Table 3 (Brickley, Moffat, and Watamaniuk 2014, 48). These cases offer a good comparison to the St. Antoine cemetery, since they are from similar time periods, are composed of populations of European ancestry and are located at northern latitudes. Although the St. Antoine cemetery was situated in an urban environment, it is relevant to consider sites from other environments considering that cultural practices also impact vitamin D deficiency. The findings of these studies indicate that many environmental and cultural parameters can influence the amount of vitamin D deficiency cases, no matter the social class (Brickley et al. 2017). Although the environmental conditions of the Industrial Revolution probably contributed to the most notable increase in vitamin D deficiency cases, this metabolic disorder can be retraced to the Late Pleistocene (Ivanhoe 1982; Hardy 2003; D'Ortenzio, Ribot, et al. 2018; Mays et al. 2018). It is thus not a recent condition, but the majority of bioarchaeological studies on the subject have

been conducted on European skeletal samples dated to the 19th century (Watts and Valme 2018; Mays 2018; Ives 2018; Mays, Brickley, and Ives 2007; 2006).

Key risk factor	Time period, location	Description of main findings	Reference
Latitude of living site	Historic (17-19th CE), Beemster, Netherlands	29/200 adults presented residual rickets at a latitude of 53°N	Veselka et al. 2018.
		9/95 nonadults had evidence of rickets at a latitude of 53°N	Veselka, Hoogland, and Waters-Rist 2015
	Historic (17-19th CE), Beemster and Hattem, Netherlands	4/30 nonadults and adults present IGD episodes consistent with seasonal deficiencies during winter (latitude of 53°N) based on micro-CT and histological data (M^1/C_1)	Veselka et al. 2019
	Historic (18-19th CE), Birmingham, England	5% (7/136) of adults had evidence of osteomalacia, while 12.8% (21/164) nonadults showed evidence of rickets at a latitude of 52°N	Brickley, Mays, and Ives 2007
	Historic (19th CE), Redhill, Surrey	17.7% (14/79) of nonadults presented rachitic lesions at a latitude of 51°N	Watts et Valme 2018
Indoor work and habits	Historic (17-19th CE), Beemster, Netherlands	More residual rickets (N=29/200) recorded in females than males, females working mainly indoors	Veselka et al. 2018.
	Historic (18-19th CE), Birmingham, England	7/84 adults recovered in earth-cut graves present osteomalacia, possibly from working in factories	Brickley, Mays, and Ives 2007
	Historic (19th CE), Redhill, Surrey	14/76 nonadults present rachitic lesions possibly linked to factory work	Watts et Valme 2018
Urbanism	Historic (18-19th CE), Birmingham, England	7/84 adults recovered in earth-cut graves present osteomalacia, possibly in part from the cramped building arrangement	Brickley, Mays, and Ives 2007
Industrialization (atmospheric pollution)	Historic (18-19th CE), Birmingham, England	Lesions indicate that vitamin D deficiency was frequent, i.e. active and healed rickets in nonadults (21/164) and osteomalacia in adults (7/136)	Brickley, Mays, and Ives 2007
Clothing	Historic (17-19th CE), Beemster, Netherlands	Clothing at Beemster barely showed any skin, which might have increased the risk of vitamin D deficiency (29/200 adults had residual rickets, 9/95 nonadults had rickets)	Veselka et al. 2018; Veselka, Hoogland, and Waters-Rist 2015
	Historic (19th CE), Redhill, Surrey	Children's clothing that only let the hands and the head uncovered might have contributed to the vitamin D deficiency cases (14/79).	Watts et Valme 2018
Perinatal practices	Historic (17-19th CE), Beemster, Netherlands	Short periods of breastfeeding followed by cow milk ingestion might have contributed to vitamin D deficiency (29/200 adults had residual rickets)	Veselka et al. 2018.

Diet	Historic (19th CE), Redhill, Surrey	Only healed rickets recorded after the age of two (5/79) might be related to cod liver oil administration	Watts et Valme 2018

Table 3. – Main biocultural risk factors of vitamin D deficiency detected in 19th century European populations.

Except for the recording of vitamin D deficiency-related lesions in a few individuals, the frequency of this type of metabolic disease has not been explored extensively in Canada for the same period (e.g. Brickley, Kahlon, and D'Ortenzio 2019; D'Ortenzio et al. 2018; 2016; Houle-Wierzbicki 2016; Morland 2009). The impact of vitamin D deficiency on past health at a populational level has also been only poorly explored, since much of the research concerned modern societies (Roberts and Brickley 2018).Indeed, according to clinical studies, this deficiency during infancy can influence the resistance to infections and the development of some cancers and/or rheumatoid arthritis (Holick 2004; Bikle 2009; Baeke et al. 2010).

Therefore, the present palaeopathological research project aims to fill this gap by studying the health status of an archaeological population originating from Montréal (Catholic cemetery of St. Antoine) and dated to the end of the pre-industrial period and the very beginning of the industrial period (Ethnoscop 2012). Indeed, the Canadian Industrial Revolution began later in comparison to the European Industrial Revolution and debuted in the 1840s around the Lachine Canal, in Montréal (Dickinson 2014; Brault 1990, 19). At that time, industrial production becomes a significant growth engine for Montréal's economy (Linteau and Robert 1985, 210). In pre-industrial Montréal, however, vitamin D deficiency might still have affected people depending on various environmental factors such as their access to resources and their way of life (indoor, outdoor or mixed) (Brickley, Moffat, and Watamaniuk 2014). In the first half of the 19th century, the population of the city increases due to immigration (Linteau 2007, 65). Indeed, of approximately 9000 residents in 1800, the population increases to 23 000 inhabitants in 1825 and to 58 000 in 1852 (Linteau 2007, 65). English, Scottish and Irish people composed the majority of the immigrants in 1831, a phenomenon that lasted for 35 years (Linteau 2007, 65). From the 1840s and up to 1861, British immigration was gradually replaced by the rural French-Canadian immigration as the main contribution to population growth (Linteau and Robert 1985, 212). During this period, the city is overcrowded and water access and sewage are all inadequate since there is no planning for litter deposition (Bradbury 1982; Brault 1990, 20). During the course of the 19th century,

epidemic diseases spread across the population, including smallpox, typhus, cholera, influenza, yellow fever, plague, leprosy and tuberculosis (Cadotte 1990, 136). Cholera, transmitted by water, milk or other food contaminated by sick people's faeces, hits Montréal in 1832, 1849, 1851, 1852 and 1854, often after the arrivals by boat of infected people (Cadotte 1990, 137). As for the diet, according to a study by Fyson (2008), it is composed of 40-60% of starchy foods among the workers of the Lachine Canal (1822-1823), while the sources on two high-class anglophone families (1816-1824) indicate a diet composed of 29 to 40% of starchy foods. Among starches, bread constitutes 80 to 95% of the caloric intake for Lachine Canal workers (Fyson 2008, 76).

In light of all these historical sources, the present study will address the following research questions:

i) What is the frequency and prevalence of pathological lesions and conditions among the sampled individuals from 19th century St. Antoine cemetery, Montréal?

ii) How do pathological lesions and conditions affect the various categories (sex, age) of the urban population sample from St. Antoine cemetery?

iii) Is there a possible link between the occurrence and severity of vitamin D deficiency episodes during infancy and the health deterioration occurring later in life?

iv) Is there a temporal evolution within the St. Antoine cemetery of vitamin D deficiency cases during infancy, in relation to the industrialization process?

To explore these issues, a detailed archaeological, osteological and palaeopathological profile using macroscopic and radiographic analysis was completed, in parallel to a histological analysis of dental tissues to assess the presence of vitamin D deficiency. The data will be analysed together in order to investigate the possible impact of vitamin D deficiency during infancy, especially of a severe type, on health deterioration and/or early death. Clinical studies tend to indicate a possible link (Holick 2004; Bikle 2009; Baeke et al. 2010). Furthermore, spatial data, i.e. the depth of the burials and the zone of the cemetery where they are situated (Ethnoscop 2012; 2014; 2016b; 2016a; Arkéos 2018), will be added to the palaeopathological data to explore the evolution of the vitamin D deficiency cases during infancy in relation to the industrialization process. The environmental and sanitary deterioration over the course of the 19th century in Montréal could have increased the number of cases of this metabolic disease (Bradbury 1982; Brault 1990, 20).

2. Materials and methods

2.1 Skeletal and dental samples

The selected material for this study consists of 52 individuals from the 19th-century urban cemetery of St. Antoine in Montréal (Québec, Canada). During the period of use of the cemetery, 55 000 people were buried on its grounds, many of them the victims of the cholera epidemics, resulting in the cemetery being called the "cholera cemetery" (Ville de Montréal 2016). For more than 50 years, it constituted the only burying ground of Catholics in Montréal, poor or wealthy, which confers a significant historical importance to this cemetery (Ville de Montréal 2016). In the years following the inauguration, the cemetery expands over the course of a decade (Ethnoscop 2012, 34). The distribution of the graves of the sampled individuals across the St. Antoine cemetery is presented on Fig. 5. According to other archives of the Montréal Fabrique (1868), the cemetery is divided in five areas that were inaugurated at different dates: areas 1 and 2 (1799), area 3 (1800), area 4 (1807), area 5 (1812) and area 6 (1824) (Ethnoscop 2016b, 15). It seems that the opening of a new area did not signify that the older areas ceased to be used (Arkéos 2018, 6). The limits between the zones on Fig. 5 are approximative since they are drawn by the archaeological firm that excavated the site (Ethnoscop 2012) based on the plan of the occupation of the grounds of the St. Antoine cemetery (site BiFj-37) between 1799 and 1824, which does not have a scale. The Annexe D presents a summary of various archaeological data associated with the sampled individuals, i.e. areas of the burials within the cemetery, date of inauguration of the areas, bottom altitude of coffins and artefacts recovered in association with the burials. This spatial data, i.e. the horizontal (area of the burial within the cemetery) and vertical (bottom altitude of coffins) position of the burials in the cemetery, will be used to assess the temporal evolution of vitamin D deficiency cases during infancy in relation to industrialization (Ethnoscop 2012; 2014; 2016b; 2016a; Arkéos 2018). Following the last episode of cholera epidemic, the St. Antoine cemetery is officially closed in 1854 (Ethnoscop 2012, 34). In 2009, 2012, 2014 and 2015, at least 244 burials in total with no gravestones were excavated (Ethnoscop 2012; 2014; 2016b; 2016a; Arkéos 2018). While the exact identity and origin of the recovered individuals are unknown, the Catholic nature of the cemetery (Ville de Montréal 2016) limits the possibilities and tends to indicate mainly French-Canadian ancestry, with also, possibly, Irish ancestry. The St. Antoine cemetery in the Province of Quebec is also located at a latitude of 45°N (Gagnon et al. 2010, 820). This means that virtually no skin

production of vitamin D occurs during winter, which might contribute to seasonal vitamin D deficiency in the sampled individuals from the St. Antoine cemetery (Gagnon et al. 2010, 821).

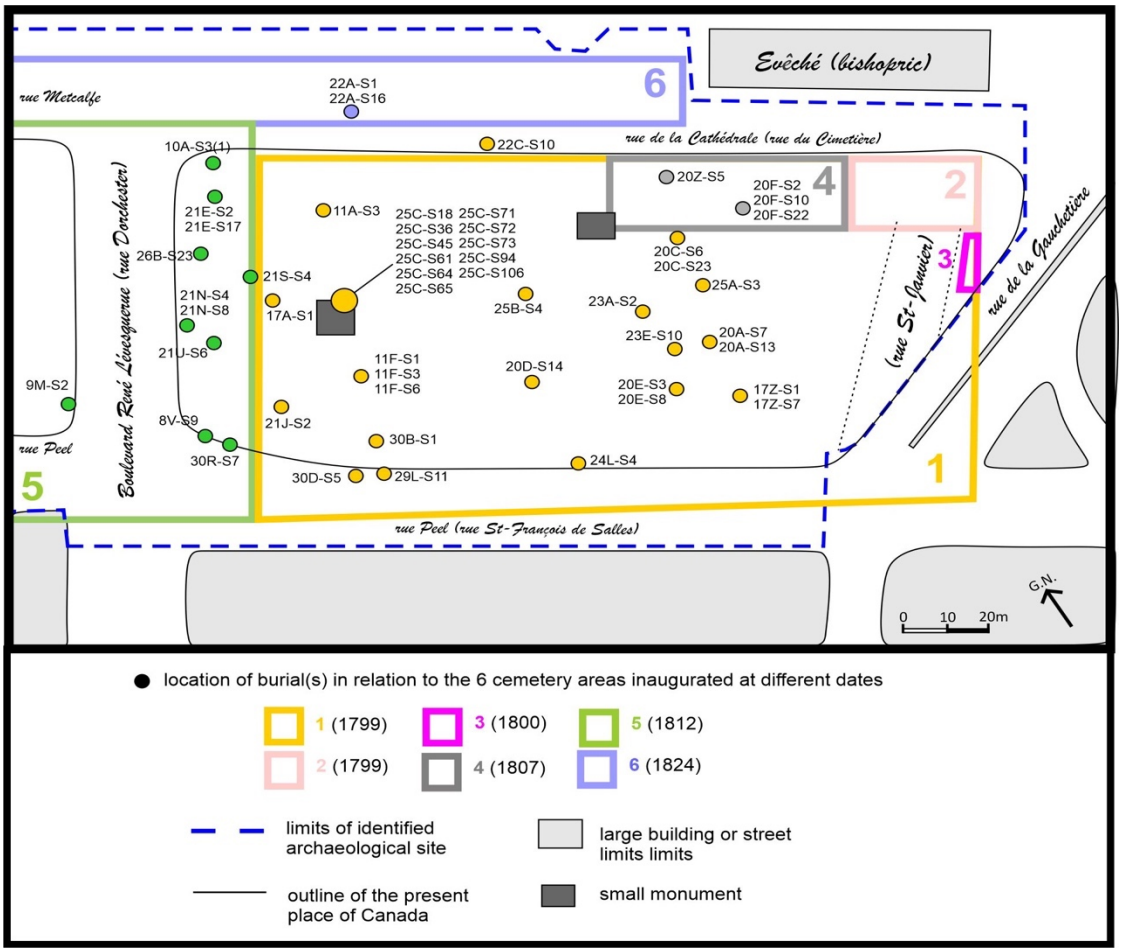

Figure 5. – Location of the burials under study (dots) (N=52) in relation to six areas inaugurated at different dates for the cemetery St. Antoine. The data is superimposed on the land map of Perrault (1867) (modified figure by I. Ribot from Ethnoscop (2016(a): maps 4 & 11), Ethnoscop (2016(b): map 2) and Arkéos (2018: map 5).

A final sample of 52 individuals was selected according to the preservation of at least one of the first permanent mandibular molars because the histological method, developed by D'Ortenzio and colleagues (2016), will be used to assess the presence of vitamin D deficiency-related lesions in the dentine. This type of tooth reflects approximately the first seven years of life and is easier to extract without bone damage than the maxillary molars (AlQahtani, Hector, and Liversidge 2010). Only the left ones were selected for standardization purposes, unless they were not well preserved, in which case the right ones were sampled. We selected individuals for which sex was uncertain to

expand our sample of individuals presenting at least one first permanent mandibular molar (n= 9). Table 4 presents the state of preservation of the sampled individuals. The ACI (Anatomical Conservation Index) of Dutour (1989), presented in Annexe A, was not applied to a few juveniles (n=5) of our sample because the equation is not suitable for juvenile skeletons. Table 4 also presents the composition of the sample according to sex, which was estimated using the methods presented in Annexe B, and according to age-at-death, which was estimated using the methods presented in Annexe C. Individuals were classified into six age categories early childhood (0-6 yrs.), late childhood (6-10 yrs.), adolescence (10-17 yrs.), early adulthood (18-35 yrs.), adulthood (35-51 yrs.) and late adulthood (51 yrs.+). There is a predominance of males in our sample. The age-at-death distribution of the individuals in our sample is mainly composed of young adults. However, the sample is still small and might be biased by the fact that only individuals with first permanent mandibular molars were selected. Thus, older individuals with dental loss and younger individuals with badly preserved skeletal and dental remains might be underrepresented in the sample.

ICA	**Sex**	**Age-at-death**
Bad (n=3, **5%**)	**Female** (n=11, **21%**)	**Early childhood** (n=1, **2%**)
Poor (n=5, **10%**)	**Likely Female** (n=8, **15%**)	**Late childhood** (n=4, **7%**)
Correct (n=13, **25%**)	**Male** (n=11, **21%**)	**Adolescence** (n=3, **6%)**
Good (n=13, **25%**)	**Likely Male** (n=13, **25%**)	**Early adulthood** (n=31, **60%**)
Very good (n=8, **15%**)	**Undetermined** (n=4, **8%**)	**Adulthood** (n=13, **25%)**
Excellent (n=5, **10%**)	**N.A**. (n=5, **10%**)	**Late adulthood** (n=0, **0%**)
N.A. (n=5, **10%)**		

Table 4. – Preservation state, sex and age-at-death composition of the 52 sampled individuals. The Undetermined category refers to the adults whose sex could not be estimated due to preservation issues, while the N.A. (Not Applicable) category concerns the juveniles whose sex or ACI cannot be estimated.

2.2 Data recording

To assess the presence of vitamin D deficiency as well as other pathological lesions and conditions in the sample, macroscopic observations were conducted on the whole skeleton. In this study, pathological lesions are responses in the bones and/or teeth to a disease or a wound (Thomas 1985). By themselves, they can be aetiologically non-specific or pathognomonic, i.e. specifically indicative of a certain pathological condition (Buikstra and DeWitte 2019, 15; Stevenson and

Lindberg 2015). Pathological conditions are defined in this study as abnormal physical disorders that can be estimated by a set of pathological lesions (Buikstra and DeWitte 2019, 15). They can be associated to a disease or be non-specific (Klaus and Lynnerup 2019, 64; Kumar et al. 2014; Kent 2007). The descriptive data of the abnormal bone and dental lesions were recorded in an Excel sheet according to seven criteria recommended by the *Guidance on recording palaeopathology (abnormal variation)* (Roberts 2017) in *Updated Guidelines to the standards for recording human remains* (Mitchell and Brickley 2017) (Table 5).

Recording criteria for palaeopathological lesions
1.Bone/tooth affected (including the side)
2.Part of the bone/tooth and aspect
3.Nature of the lesion (formation or destruction of bone, destruction of the tooth structure or destruction of alveolar bone)
4.Nature of bone formation: « woven » (porous, disorganized and indicating an active pathology at the time of death) or lamellar (soft and organized, indicating a healed lesion and chronic or in the process of healing)
5.Presence or absence of healing signs (ex. Round margins of the lesion)
6.Distribution model of the lesions if more than one bone/tooth is involved
7.Measure and comparison of the abnormality with the normal side

Table 5. – Seven recording criteria for palaeopathological lesions (Roberts 2017).

The visual recording of the pathological lesions allowed to summarize their distribution on the skeletons using a digital skeleton form achieved with the software GIMP (Gutierrez 2017) (Annexe E).

Table 6 presents various pathological lesions and conditions recorded in this study following a differential diagnosis based on the evaluation of the two basic variables previously analysed, i.e. the types of abnormalities and the distribution of the abnormalities within the skeleton (Ortner 2011, 5). This information was afterwards compared with descriptions of known pathological lesions and pathological conditions, to assess which are present (Annexes F-L) (Klaus and Lynnerup 2019, 81). As for the terminology used to indicate the degree of certainty of differential diagnosis in palaeopathology, the *Modified Istanbul Protocol* was chosen for this research (Appleby, Thomas, and Buikstra 2015).

Pathological lesions	References	Pathological conditions	References
Dental calculus	Kinaston et al. 2019	**Dental caries**	Kinaston et al. 2019
Linear enamel hypoplasia (LEH)	El-Najjar, Desanti, and Ozebek 1978; King, Humphrey, and Hillson 2005	**Intervertebral disk disease (IVD)**	Adams and Roughley 2006; Waldron 2019

Cribra orbitalia	Patty Stuart-Macadam 1991; Naveed et al. 2012	**Osteoarthritis (OA)**	Waldron 2019
Porotic hyperostosis	El-Najjar et al. 1976; Mann and Hunt 2013	**Non-specific joint disease**	Waldron 2019
Endocranial lesions	Hershkovitz et al. 2002; Lewis 2004	**Trauma**	Redfern and Roberts 2019
Schmorl's nodes	Faccia and Williams 2008	**Non-specific infection**	Roberts 2019; Davies-Barrett, Antoine, and Roberts 2019
Osteophytes	Alves-Cardoso and Assis 2018	**Tuberculosis (TB)**	Roberts and Buikstra 2019; Davies-Barrett, Antoine, and Roberts 2019; Lewis 2004; Mann and Hunt 2013
		Rickets and Residual Rickets	Brickley, Mays, and Ives 2007; Brickley et al. 2018; Roberts and Buikstra 2019
		Scurvy	Geber and Murphy 2012; Schattmann et al. 2016
		Tumour	Marques 2019
		Syphilis	Roberts and Buikstra 2019
		Sinusitis	Lewis, Roberts, and Manchester 1995; Roberts 2007; 2019
		Osteochondritis dissecans	Anderson 2001; Vikatou, Hoogland, and Waters-Rist 2017

Table 6. – Pathological lesions and conditions recorded in the 52 sampled individuals.

Bones that presented macroscopic lesions that needed to be confirmed for diagnosis were radiographed (for example, bones presenting abnormal curvature, shape, fractures or abnormal growths). The bones were placed at a distance of 70 cm from the X-ray beam and exposed to 8 to 10 pulses depending on the density of the bone and the lesion to observe.

Since it is advisable to take pictures of the tissue in all angles before undertaking a destructive process, photographs of the teeth were taken at the AnthropoLab3D, Université de Montréal (Jean-Baptiste Lemoine, Alexandre Bisson and Diane Martin-Moya), and used to produce a 3D model with the *Metashape* software.

The dental samples were prepared histologically according to the method developed by D'Ortenzio and colleagues (2016) in order to assess the presence or absence of IGD. Prior to

sectioning the teeth using a low-speed saw, the dental tissues were embedded in epothin resin. The dental slides were fixed on a microscope slide using UV activated resin and polished using 400 and 1200 grit paper on an automatic polisher until the sample had a thickness of only 0.3 to 0.4 mm. Microscopic analysis of the dental sample was done using a transmitted light microscope (100X magnification) attached to a digital camera.

Four criteria were recorded per individual: the presence or absence of IGD (including and excluding degree 1), the number of IGD episodes (total and observable), the severity of each IGD episode, the age of onset of each IGD episode and the presence or absence of *in utero* IGD episodes. The data was recorded by Rose-Ann Bigué (RAB), the novice observer, as well as by Megan Brickley (MB) and Bonnie Kahlon (BK), the experienced observers. The observations were compared to obtain a final assessment.

The presence or absence of IGD was noted according to the presence of interglobular spaces following incremental lines and patches of disorganized spaces were considered developmental (DIGD) (Fig. 6) (D'Ortenzio, Kahlon, et al. 2018, 102–3; Avery 2002). The presence or absence of IGD was recorded according to the presence or absence of IGD episodes during infancy, i.e. in the crown portion of the tooth which forms during early childhood (AlQahtani, Hector, and Liversidge 2010). When too much dental wear, diagenesis or post-mortem breakage prevented proper observation of IGD, it was categorized as not observable (N.O.). The number of vitamin D deficiency episodes was recorded according to the number of bands of interglobular spaces following incremental lines present in the dentine (Veselka et al. 2019, 124). Individuals presenting too much dental wear, diagenesis, post-mortem breakage, or incomplete tooth formation, thus impairing proper observation of the total number of IGD episodes that could have occurred, were classified as N.O. The degrees of IGD were scored according to the severity of the phenomenon (Table 7, Fig. 6). Half scores were used (ex. 1.5) when the severity was situated between two categories of degrees.

Grade	Grade 0	Grade 1	Grade 2	Grade 3
Interglobular spaces	Normal: no interglobular spaces	Minimal interglobular spaces (IGD-).	Moderate interglobular spaces (IGD+).	Large interglobular spaces (IGD++)
Description	Dentine is homogeneous; interglobular dentine is absent	Interglobular spaces present but small; spaces are <25% relative to surrounding normal dentine	Interglobular spaces moderately large and more numerous than grade 1; spaces are 25-50% relative to surrounding normal dentine	Interglobular spaces are large and very numerous with a clear scalloped or bubbled appearance; spaces are >75% relative to surrounding normal dentine
Defect in dentine mineralization	Absent	Mild	Moderate	Severe

Table 7. – Scoring system for IGD (interglobular dentine) (D'Ortenzio et al. 2016).

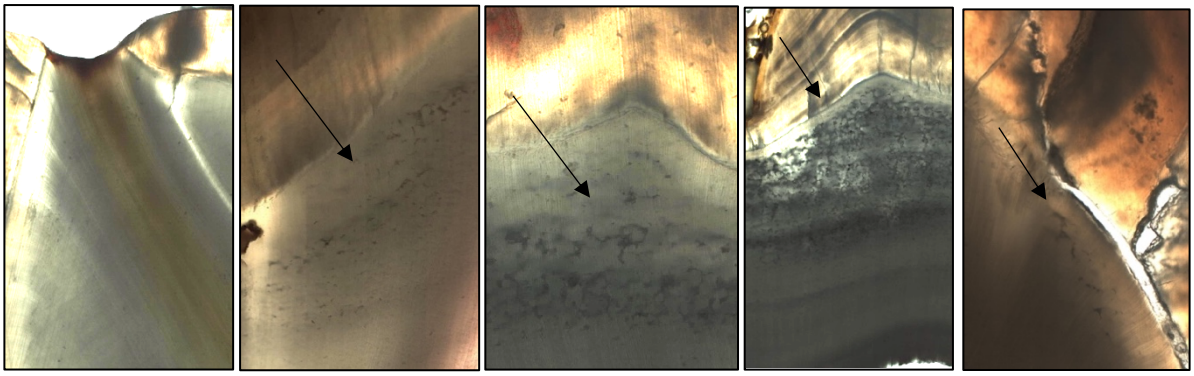

Figure 6. – From left to right: grade 0 of IGD, homogenous dentine (22A-S16), grade 1, IGD <25% (20D-S14), grade 2, IGD 25-50% (17Z-S7), grade 3, >75% (30R-S7), developmental IGD (DIGD) (21U-S6) (D'Ortenzio et al. 2016) (Photos by Rose-Ann Bigué).

As for the age of onset of each IGD episode and the presence or absence of *in utero* IGD episodes, the approximate age of mineralization for the 1rst permanent mandibular molar using the incremental pattern of growth was used, according to the data compilation by Brickley and colleagues 2019 (Fig.7). The various age categories for onset of vitamin D deficiency episodes in the 1rst permanent mandibular molar are presented in Fig.7. *In utero* and at birth episodes of IGD were estimated by identifying the neonatal line in the enamel (Brickley, Kahlon, and D'Ortenzio 2019, 4; Schour 1936). This accentuated microstructure separates the enamel and dentine developed *in utero* from the dental tissue formed after birth (Brickley, Kahlon, and D'Ortenzio 2019, 4). It can be localized in enamel as a dark, sharp band that is larger than the rest of the incremental growth lines in enamel (Brickley, Kahlon, and D'Ortenzio 2019, 4; Eli, Sarnat, and Talmi 1989). In dentine, while not always obse11/06/2021 19:21:00rvable depending on the thickness of the histological slide or the presence of IGD, the neonatal line is characterized by a narrow white line equidistant from the dentino-enamel junction (DEJ) and the neonatal line in

enamel (see Fig.7, dotted blue line) (Brickley, Kahlon, and D'Ortenzio 2019, 4). The final assessment of the ages of onset was decided according to the observations of BK, more experienced in the localization of the neonatal line in the enamel.

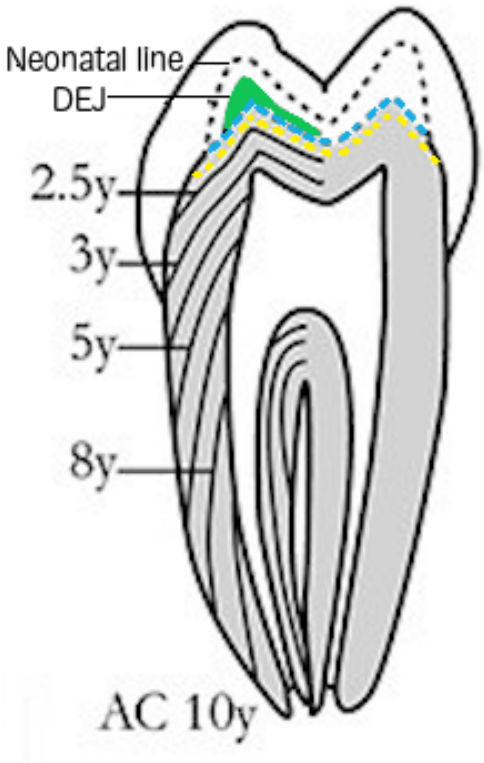

Figure 7. – Ages of mineralization for the first permanent mandibular molar. Enamel = white, dentine= grey, AC= apical closure, black dotted line= neonatal line in the enamel, green zone= *in utero* IGD episode, blue dotted line=IGD episode at birth, yellow dotted line= IGD episode at 1-1.5 years-old. Modified from Brickley, Kahlon, and D'Ortenzio (2019).

2.3 Statistical analysis

Fisher's test was used to explore the links between pathological lesions, conditions and the demographic parameters (age, sex). Because of small sample size, age groups were subdivided in only two groups (i.e. nonadults, adults), as were sex groups (i.e. female/likely female, male/likely male). The age groups for onset of IGD episodes are treated differently (Fig. 7), since these are events that happened during the life of the individuals. As for IGD, the statistical analysis was conducted including and excluding the degree 1, to assess if there might be a difference in the results when exclusively considering the more severe degrees (i.e. more than degree 1). The conventional line is drawn at a p-value below 0.05 for a significant association between

characteristic and outcome (Biau, Jolles, and Porcher 2010, 886). Fisher's test is appropriate in 2X2 tables with small sample sizes and when one of the four cells has fewer than five observations (UCLA Statistical Consulting Group 2020a; 2020b). This test was selected because of the small size of our sample and the inability, in some cases, to conduct tests using several sub-categories of variables with more than five observations.

3. Results

3.1 Frequency and severity of the various pathological lesions and conditions

Annexe M presents all the observations with the individual skeletal forms including various detailed information (i.e. ACI, age-at-death, sex, description of the lesions, differential diagnosis). Annexe N also presents a summary of the differential diagnosis combined with the individuals' code, age-at-death, sex, ACI and histological data. The possible link between various pathological lesions, conditions and demographic parameters (age group, sex) are tested below.

3.1.1 Relationship between pathological lesions and various factors (age and sex groups)

Table 8 shows the frequency of pathological lesions according to sex (the undetermined and N.A. categories were not taken into account for the statistical test). According to the results, no pathological lesion is associated with sex.

	Dental calculus		**LEH**		***Cribra orbitalia***		**Porotic hyperostosis**		**Endocranial lesions**		**Osteophytes**		**Schmorl's nodes**	
	P	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**
Female/likely female	5	14	1	18	1	18	1	18	4	15	3	16	3	16
Male/likely male	14	10	2	22	5	19	1	23	1	23	7	17	9	15
Total	19	24	3	40	6	37	2	41	5	38	10	33	12	31
p-value[a]	0.063		1.000		0.205		1.00		0.153		0.470		0.174	
Odds ratio	0.255		0.611		0.211		1.278		6.133		0.455		0.313	
Significant	No		No		No		No		No		No		No	

Table 8. – Frequency of pathological lesions (P = present, A= absent) according to sex (Fisher's tests) (n=43).

a significant p value below 0.05.

According to the results (Table 9), *cribra orbitalia* is the only pathological lesion to be significantly associated with age (p-value of 0.004, odds ratio value of 13.000). In fact, it is predominant among nonadults' category (5/8).

	Dental calculus		**LEH**		***Cribra orbitalia***		**Porotic hyperostosis**		**Endocranial lesions**		**Osteophytes**		**Schmorl's nodes**	
	P	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**
Nonadults	1	7	1	7	5	3	0	8	1	7	0	8	1	7
Adults	21	23	4	40	5	39	2	42	4	40	10	34	11	33
Total	22	30	5	47	10	42	2	50	5	47	10	42	12	40
p-value[a]	0.118		1.000		0.004		1.000		1.000		0.328		0.663	
Odds ratio	0.156		1.429		13.000		1.048		1.429		1.294		0.429	
Significant	No		No		**Yes**		No		No		No		No	

Table 9. – Frequency of pathological lesions (P= present, A= absent) according to two age groups (Fisher's tests) (N=52).

a Significant p-value below 0.05.

Relationship between pathological skeletal lesions that may occur in vitamin D deficiency cases and demographic parameters (age, sex)

According to Fisher's tests, no link was found between the pathological skeletal lesions that may occur in vitamin D deficiency cases and sex (Table 10).

Pathological lesions that may occur in vitamin D deficiency cases										
	Skull				**Long bones**					
	Cranial vault porosity		**Orbital roof porosity**		**Cranial vault thickening**		**Bending deformity**		**Coxa vara**	
	P	**A**	**P**	**A**	**P**	**A**	**P**	**A**	**P**	**A**
Female/likely female	1	18	1	18	0	19	0	19	0	19
Male/likely male	1	23	5	19	1	23	1	23	1	23
Total	2	41	6	37	1	42	1	42	1	42
p-value[a]	1.000		0.205		1.000		1.000		1.000	
Odds ratio	1.278		0.211		1.043		1.043		1.043	
Significant	No		No		No		No		No	

Table 10. – Frequency of specific and non-specific pathological lesions that may occur in vitamin D deficiency cases according to sex (Fisher's tests) (n=43).

a Significant p-value below 0.05

Except for orbital roof porosity, no link was found between the pathological lesions that may occur in vitamin D deficiency cases and the age groups (Table 11).

Pathological lesions that may occur in vitamin D deficiency cases										
	Skull				Long bones					
	Cranial vault porosity		Orbital roof porosity		Cranial vault thickening		Bending deformity		Coxa vara	
	P	A	P	A	P	A	P	A	P	A
Nonadults	0	8	5	3	0	8	1	7	0	8
Adults	2	42	5	39	1	43	1	43	1	43
Total	2	50			1	51	2	50	1	51
p-value[a]	1.00		0.004		1.000		0.287		1.000	
Odds ratio	1.048		13.000		1.023		6.143		1.023	
Significant	No		**Yes**		No		No		No	

Table 11. – Frequency of specific and non-specific pathological lesions that may occur in vitamin D deficiency cases according to two age groups (Fisher's tests) (N=52).

a Significant p-value below 0.05

3.1.2 Relationship between pathological conditions and age and sex

Table 12 presents the frequency of pathological conditions according to sex along with the Fisher's tests. The results indicate that there is a significant relationship between biological sex and OA, the p-value between the presence of OA and male sex being 0.012 and the odds ratio 1.412.

	Dental caries		IVD		OA		Osteochondritis dissecans		Non-specific joint disease		Trauma		Non-specific infection		Tuberculosis		Sinusitis		Syphilis		Rickets		Residual rickets		Scurvy		Tumour	
	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A
Female/ likely female	4	15	0	19	0	19	1	18	6	13	6	13	5	14	0	19	0	19	0	19	0	19	0	19	1	18	2	17
Male/likely male	10	14	4	20	7	17	0	24	10	14	3	21	4	20	1	23	1	23	1	23	0	24	2	22	1	23	1	23
Total	12	26	4	39	7	36	1	42	16	27	9	34	9	34	1	42	1	42	1	42	0	43	2	41	2	41	3	40
p-value[a]	0.199		0.118		0.012		0.442		0.542		0.153		0.477		1.00		1.000		1.000		-		0.495		1.000		0.575	
Odds ratio	0.373		1.200		1.412		0.947		0.646		3.231		1.786		1.043		1.043		1.043		-		1.091		1.278		2.706	
Significant	No		No		**Yes**		No		No		No		No		No		No		No		-		No		No		No	

Table 12. – Frequency of pathological conditions (P= present, A= absent) according to sex (Fisher's tests) (n=43).

a Significant p-value below 0.05

Table 13 presents the frequency of pathological conditions according to two age groups and the results of Fisher's test. Only the non-specific joint disease category presents a significant association with the adults age group (p-value of 0.042, odds ratio of 1.630).

	Dental caries		IVD		OA		Osteochondritis dissecans		Non-specific joint disease		Trauma		Non-specific infection		Tuberculosis		Sinusitis		Syphilis		Rickets		Residual rickets		Scurvy		Tumour	
	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A	P	A
Nonadults	3	5	0	8	0	8	0	8	0	8	1	7	2	6	0	8	0	8	0	8	1	7	0	8	2	6	0	8
Adults	15	29	5	39	8	36	1	43	17	27	9	35	9	35	1	43	1	43	1	43	0	44	2	42	2	42	3	41
Total	18	34	5	47	8	44	1	51	17	35	10	42	11	41	1	51	1	51	1	51	1	51	2	50	4	48	3	49
p-value[a]	1.00		1.000		0.330		1.000		0.042		1.000		1.000		1.00		1.000		1.000		0.154		1.000		0.107		1.000	
Odds ratio	1.160		1.128		1.222		1.023		1.630		0.556		1.296		1.023		1.023		1.023		0.875		1.048		7.000		1.073	
Significant	No		No		No		No		**Yes**		No		No		No		No		No		No		No		No		No	

Table 13. – Frequency of pathological conditions (P= present, A= absent) according to two age groups (Fisher's tests) (N=52).

a Significant p-value below 0.05

3.2 Microscopic analysis of the teeth: identification of IGD, indicative of vitamin D deficiency

The frequency of IGD, the number of IGD episodes (total and observable), the degree of severity of each IGD episode, the age of onset of each IGD episode and the frequency of *in utero* IGD episodes for each individual are presented in Annexe N, along with other information (individuals' code, ACI, sex, age-at-death, differential diagnosis).

3.2.1 Recording of the data according to various observers

Overall, the three observers were in agreement concerning the presence or absence of IGD. The identification of the observable number of IGD episodes varied between observers, RAB and MB tending to identify multiple episodes, while BK tended to record single, longstanding IGD episodes. The recording of the total number of IGD episodes was mostly impaired by diagenesis and dentine chipping in the roots. The degrees of IGD varied greatly between the observers, although RAB and MG tended to be in agreement. As for the ages of onset and the presence or

absence of *in-uterine* IGD episodes, BK's assessment was selected as the reliable one, since the localization of the neonatal line in the enamel requires more experience.

3.2.2 Frequency and prevalence of IGD

Table 14 presents the final results regarding IGD, i.e. presence or absence of IGD (including and excluding degree 1), number of IGD episodes (total and observable), degree of severity of each IGD episode, age of onset of each IGD episode and presence or absence of *in utero* IGD episodes. Out of the 52 sampled individuals, one (20Z-S5) presents diagenesis on the totality of the dentine surface and was thus excluded in all statistical analysis. Another individual, 25C-S94, presents diagenesis in parts of the crown's dentine and was excluded from the statistical analysis when excluding the degree 1 of IGD. Seventy-five percent of the individuals (39/52) present IGD (degrees 1-3) during infancy but this percentage decreases to 44% (23/52) when excluding the degree 1 of IGD and only considering more severe cases. The total number of IGD episodes could only be observed for 4 individuals since diagenesis and dentine chipping in the roots impaired proper visualization. As for the observable number of IGD episodes, 56% of the individuals (29/52) present only 1 episode of IGD during infancy, 15% (8/52) 2 episodes, 2% (1/52) 4 episodes, 23% (12/52) no episode at all and 4% (2/52) are N.O. Fifty-six percent (29/52) of the individuals present longstanding episodes of IGD, ranging from *in utero* to up to 4 years. Twelve percent (6/52) of the individuals present *in utero* IGD and 63% (33/52) present IGD episodes occurring at or from birth to up to 3 years of age.

Individual	**Histology**						
	IGD (degrees 1 to 3)	IGD (excluding degree 1)	N IGD episodes (total)	N IGD episodes (observable)	Grade	Age of onset	*In utero* episodes
10A-S3 (1)	P	P	N.O.	2	**Episode 1**: grade 1 **Episode 2**: grade 2	**Episode 1:** around birth **Episode 2**: around 1.5 yrs.	No
11A-S3	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: less than 1 yr.	No
11F-S1	P	P	N.O.	2	**Episode 1**: grade 2 **Episode 2**: grade 2	**Episode 1**: *in utero* to around 1 yr. **Episode 2**: around 2 yrs.	Yes
11F-S3	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around birth	No
11F-S6	A	A	N.O.	N.A.	N.A.	N.A.	No
17A-S1	P	P	N.O.	2	**Episode 1**: grade 2 **Episode 2**: grade 1	**Episode 1**: *in utero* to 1yr **Episode 2**: c. 2 yrs.	Yes
17Z-S1	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around 1.5 yrs.	No

17Z-S7	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 2 yrs.	No
20A-S7	P	P	2	2	**Episode 1**: grade 2. **Episode 2**: grade 1	**Episode 1**: near birth **Episode 2**: around 1.5 yrs.	No
20A-S13	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around 1-2.5 yrs.	No
20C-S6	P	P	N.O.	2	**Episode 1**: grade 2. **Episode 2**: grade 1	**Episode 1**: birth **Episode 2**: around 3 yrs.	No
20C-S23	P	A	N.O.	1	**Episode 1**: less than grade 1	**Episode 1**: 3 yrs.	No
20D-S14	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to around 2 yrs.	No
20E-S3	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to about 1yr.	No
20E-S8	A	A	N.O.	N.A.	N.A.	N.A.	No
20F-S2	A	A	N.O.	N.A.	N.A.	N.A.	No
20F-S10	P	P	N.O.	1	**Episode 1**: grade 1-2	**Episode 1**: birth to around 2.5 yrs.	No
20F-S22	A	A	N.O.	N.A.	N.A.	N.A.	No
20Z-S5	N.O.	N.O.	N.O.	N.O.	N.O.	N.O.	N.O
21E-S2	P	A	N.O.	1	**Episode 1**: less than 1-1	**Episode 1**: birth to around 1 yr.	No
21E-S17	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: in utero to about 2 yrs.	Yes
21J-S2	P	A	N.O.	1	**Episode 1**: grade less than 1-1	**Episode 1**: around 1 yr.	No
21N-S4	A	A	N.O.	N.A.	N.A.	N.A.	No
21N-S8	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 1 yr.	No
21S-S4	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: *in utero* to about 1 yr.	Yes
21U-S6	A	A	N.O.	N.A.	N.A.	N.A.	No
22A-S1	A	A	N.O.	N.A.	N.A.	N.A.	No
22A-S16	A	A	N.O.	N.A.	N.A.	N.A.	No
22C-S10	P	P	N.O.	1	**Episode 1**: grade 1-2	**Episode 1**: birth to around 3 yrs.	No
23A-S2	P	P	N.O.	1	**Episode 1**: grade 1-2	**Episode 1**: birth to 1.5 yrs.	No
23E-S10	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to around 1.5 yrs.	No
24L-S4	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: around birth to 1 yr.	No
25A-S3	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: close to birth to less than 1 yr.	No
25B-S4	A	A	N.O.	N.A.	N.A.	N.A.	No
25C-S106	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to around 2 yrs.	No
25C-S18	P	P	4	4	**Episode 1**: grade 1-2. **Episode 2**: grade 1. **Episode 3**: grade 1. **Episode 4**: grade 2	**Episode 1**: birth to c. 1.5 yrs. **Episode 2**: around 5 yrs. **Episode 3**: around 7 yrs. **Episode 4**: around 9 yrs.	No
25C-S36	A	A	N.O.	N.A.	N.A.	N.A.	No
25C-S45	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 2 yrs.	No

25C-S61	A	A	N.O.	N.A.	N.A.	N.A.	No
25C-S64	P	P	1	1	**Episode 1**: grade 2	**Episode 1**: birth to around 1.5 yrs.	No
25C-S65	P	P	N.O.	2	**Episode 1**: grade 1.5 **Episode 2**: grade 1.5	**Episode 1**: birth to around 2 yrs. **Episode 2**: around 4 yrs.	No
25C-S71	P	P	N.O.	2	**Episode 1**: grade 2 **Episode 2**: grade 2	**Episode 1**: birth to 1.5 yrs. **Episode 2**: around 2 yrs.	No
25C-S72	P	P	N.O.	1	**Episode 1**: grade 1.5	**Episode 1**: around birth	No
25C-S73	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 2.5 yrs.	No
25C-S94	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around birth to 1.5 yrs.	No
26B-S23	A	A	N.O.	N.A.	N.A.	N.A.	No
29L-S11	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 1 yr.	No
30B-S1	P	P	N.O.	1	**Episode 1**: grade 1.5	**Episode 1**: *in utero* to 1 yr.	Yes
30D-S5	P	P	1	1	**Episode 1**: grade 2	**Episode 1**: around birth to less than 1 yr.	No
30R-S7	P	P	N.O.	1	**Episode 1**: grade 3	**Episode 1**: *in utero* to around 4 yrs.	Yes
8V-S9	P	A	N.O.	1	**Episode 1**: grade 1.5	**Episode 1**: birth to around 2 yrs.	No
9M-S2	P	A	N.O.	2	**Episode 1**: grade 1 **Episode 2**: grade 1	**Episode 1**: around 2 yrs. **Episode 2**: around 4 yrs.	No

Table 14. – Recording of IGD in the 52 sampled individuals. P= present, A= absent, N.O.= not observable, N.A.= not applicable.

3.2.3 IGD and demographic parameters

The results indicate that there is no correlation between IGD and sex or age groups (Tables 15-16).

	IGD	
	Present	**Absent**
Sex		
Female/likely female	15	4
Male/likely male	21	3
p-value[a]	0.680	
Odds ratio	0.536	
Significant	No	

Table 15. – Frequency of IGD (including degree 1) according to sex and Fisher's test (n=43).

a Significant p-value below 0.05

	IGD	
	Present	**Absent**
Age-at-death		
Nonadults	5	2
Adults	34	10
p-value[a]	0.662	
Odds ratio	0.735	
Significant	No	

Table 16. – Frequency of IGD (including degree 1) according to two age groups and Fisher's test (n=51).

a Significant p value below 0.05

To verify if the degree of severity could impact the possible correlation between IGD and sex, only individuals presenting at least one episode of IGD higher than degree 1 were considered. However, the results in Tables 17 and 18 indicate that there is still no significant association.

	IGD	
	Present	**Absent**
Sex		
Female/likely female	6	13
Male/likely male	14	9
p-value[a]	0.072	
Odds ratio	0.297	
Significant	No	

Table 17. – Frequency of IGD (excluding the degree 1) according to sex and Fisher's test (n=42).

a Significant p-value below 0.05

	IGD	
	Present	**Absent**
Age-at-death		
Nonadults	4	3
Adults	19	24
p-value[a]	0.689	
Odds ratio	1.684	
Significant	No	

Table 18. – Frequency of IGD (excluding the degree 1) according to two age- groups and Fisher's test (n=50).

a Significant p-value below 0.05

The frequency of prenatal IGD according to sex and age-at-death was also assessed, including and excluding the degree 1 of IGD, revealing no statistical link (Tables 19-20).

	Prenatal IGD			
	Present (degrees 1-3)	**Absent (degrees 1-3)**	**Present (excluding degree 1)**	**Absent (excluding degree 1)**
Sex				
Female/likely female	1	18	0	19
Male/likely male	4	20	4	20
p-value[a]	0.363		0.118	
Odds ratio	0.278		1.200	
Significant	No		No	

Table 19. – Frequency of prenatal IGD (including and excluding degree 1) according to sex and Fisher's test (n=43)

a Significant p-value below 0.05

	Prenatal IGD			
	Present (degrees 1-3)	**Absent (degrees 1-3)**	**Present (excluding degree 1)**	**Absent (excluding degree 1)**
Sex				
Nonadults	2	5	2	5
Adults	4	40	3	41
p-value[a]	0.186		0.133	
Odds ratio	4.000		5.467	
Significant	No		No	

Table 20. – Frequency of prenatal IGD (including and excluding degree 1) according to age-at-death and Fisher's test (n=51).

a Significant p-value below 0.05

3.2.4 IGD and pathological lesions or pathological conditions: testing the possible link

Pathological lesions

No significant correlation was observed between IGD and seven pathological lesions (Table 21), even when excluding the degree 1 of IGD. The exceptions are dental calculus and the presence of IGD (including degree 1) with a p-value of 0.048, odds ratio of 5.263 and linear enamel hypoplasia and the presence of IGD (excluding the degree 1) with a p-value of 0.016, odds ratio of 2.50. These results indicate that even though a significant link between the "nonadults" age

group and *cribra orbitalia* was found, the individuals presenting *cribra orbitalia* don't seem to present more severe episodes of IGD. Table 22 presents the results for pathological lesions and the presence or absence of prenatal IGD (including and excluding degree 1), revealing no statistical link.

	Interglobular dentine (IGD)					Interglobular dentine (IGD)				
	Present (degree 1 to 3)	Absent (degree 0)	p-value[a]	Odds ratio	Significant	Present (excluding degree 1)	Absent (degree 0 or 1)	p-value[a]	Odds ratio	Significant
Dental calculus										
Present	20	2	0.048	5.263	**Yes**	11	10	0.567	1.558	No
Absent	19	10				12	17			
LEH										
Present	5	0	0.323	1.353	No	5	0	0.016	2.500	**Yes**
Absent	34	12				18	27			
Cribra orbitalia										
Present	6	3	0.424	0.545	No	4	5	1.000	0.926	No
Absent	33	9				19	22			
Porotic hyperostosis										
Present	2	0	1.000	1.324	No	2	0	0.207	2.286	No
Absent	37	12				21	27			
Endocranial lesions										
Present	5	0	0.323	1.353	No	1	4	0.357	0.261	No
Absent	34	12				22	23			
Osteophytes										
Present	10	0	0.092	1.414	No	7	3	0.155	3.500	No
Absent	29	12				16	24			
Schmorl's nodes										
Present	11	1	0.250	4.321	No	7	5	0.508	1.925	No
Absent	28	11				16	22			

Table 21. – Frequency of IGD (including (n=51) and excluding (n=50) degree 1) according to pathological lesions, and Fisher's tests.

a Significant p-value below 0.05

	Prenatal (IGD)					Prenatal (IGD)				
	Present (degree 1 to 3)	Absent (degree 0)	p-value[a]	Odds ratio	Significant	Present (excluding degree 1)	Absent (degree 0 or 1)	p-value[a]	Odds ratio	Significant
Dental calculus										
Present	3	19	1.000	1.368	No	2	20	1.000	0.867	No
Absent	3	26				3	26			
LEH										
Present	0	5	1.000	1.150	No	0	5	1.000	1.122	No
Absent	6	40				5	41			
Cribra orbitalia										
Present	2	7	0.284	2.714	No	2	7	0.209	3.714	No
Absent	4	38				3	39			

Porotic hyperostosis										
Present	0	2	1.000	1.140	No	0	2	1.000	1.114	No
Absent	6	43				5	44			
Endocranial lesions										
Present	0	5	1.000	1.150	No	0	5	1.000	1.122	No
Absent	6	40				5	41			
Osteophytes										
Present	3	7	0.081	5.429	No	2	8	0.250	3.167	No
Absent	3	38				3	38			
Schmorl's nodes										
Present	1	11	1.000	0.618	No	1	11	1.000	0.795	No
Absent	5	34				4	35			

Table 22. – Frequency prenatal IGD (including and excluding degree 1) according to pathological lesions, and Fisher's tests (n=51).

a Significant p-value below 0.05

Pathological skeletal lesions that may occur in vitamin D deficiency cases

Furthermore, it is tested here whether pathological skeletal lesions that may occur in vitamin D deficiency cases are correlated with the presence of IGD (including and excluding degree 1) and the presence of prenatal IGD (including and excluding degree 1). However, Tables 23 and 24 indicate that there is no significant link between the skeletal and histological observations related to vitamin D deficiency.

		Interglobular dentine (IGD)					Interglobular dentine (IGD)				
Pathological lesions that may occur in vitamin D deficiency cases		Present (degree 1 to 3)	Absent (degree 0)	p-value[a]	Odds ratio	Significant	Present (excluding degree 1)	Absent (degree 0 or 1)	p-value[a]	Odds ratio	Significant
Skull	**Cranial vault porosity**										
	Present	2	0	1.000	1.324	No	2	0	0.207	2.286	No
	Absent	37	12				21	27			
	Orbital roof porosity										
	Present	6	3	4.24	0.545	No	4	5	1.000	0.926	No
	Absent	33	9				19	22			
	Cranial vault thickening										
	Present	1	0	1.000	1.316	No	1	0	0.460	2.227	No
	Absent	38	12				22	27			
Long bones	**Bending deformity**										
	Present	1	1	0.419	0.289	No	1	1	1.000	1.182	No
	Absent	38	11				22	26			
	Coxa vara										
	Present	1	0	1.000	1.316	No	1	0	0.460	2.227	No
	Absent	38	12				22	27			

Table 23. – Frequency of IGD (including (n=51) and excluding (n=50) degree 1) according to pathological lesions that may occur in vitamin D deficiency cases and Fisher's tests.

a Significant p-value below 0.05

Pathological lesions that may occur in vitamin D deficiency cases		Prenatal IGD					Prenatal IGD				
		Present (degree 1 to 3)	Absent (degree 0)	p-value[a]	Odds ratio	Significant	Present (excluding degree 1)	Absent (degree 0 or 1)	p-value[a]	Odds ratio	Significant
Skull	**Cranial vault porosity**										
	Present	0	2	1.140	1.324	No	0	2	1.000	1.114	No
	Absent	6	43				5	44			
	Orbital roof porosity										
	Present	2	7	0.284	2.714	No	2	7	0.209	3.714	No
	Absent	4	38				3	39			
	Cranial vault thickening										
	Present	0	1	1.000	1.136	No	0	1	1.000	1.111	No
	Absent	6	44				5	45			
Long bones	**Bending deformity**										
	Present	1	1	0.224	8.800	No	1	1	0.188	11.250	No
	Absent	5	44				4	45			
	Coxa vara										
	Present	0	1	1.000	1.316	No	0	1	1.000	1.111	No
	Absent	6	44				5	45			

Table 24. – Frequency of prenatal IGD (including and excluding degree 1) according to pathological lesions that may occur in vitamin D deficiency cases and Fisher's tests (n=51).

a Significant p-value below 0.05

Various pathological conditions

Table 25 also shows no significant link between the presence or absence of IGD (including and excluding degree 1) and pathological conditions, except for trauma (p-value 0.030, odds ratio 6.667). Indeed, when excluding the degree 1 of IGD, the results seem to indicate that several individuals presenting trauma suffered from vitamin D deficiency during infancy (n= 8/10). Table 26 presents the results for the presence or absence of prenatal IGD (including and excluding degree 1) according to pathological conditions, revealing no statistical link.

	Interglobular dentine (IGD)					Interglobular dentine (IGD)				
	Present (degree 1 to 3)	Absent (degree 0)	p-value[a]	Odds ratio	Significant	Present (excluding degree 1)	Absent (degree 0 or 1)	p-value[a]	Odds ratio	Significant
Dental caries										
Present	15	3	0.502	1.875	No	8	9	1.000	1.067	No
Absent	24	9				15	18			
Intervertebral disk disease										
Present	4	1	1.000	1.257	No	4	1	0.167	5.474	No
Absent	35	11				19	26			
Osteoarthritis										
Present	6	2	1.000	0.909	No	4	4	1.000	1.211	No
Absent	33	10				19	23			

Non-specific joint disease										
Present	15	2	0.293	3.125	No	10	7	0.239	2.198	No
Absent	24	10				13	20			
Trauma										
Present	10	0	0.092	1.414	No	8	2	0.030	6.667	**Yes**
Absent	29	12				15	25			
Non-specific infection										
Present	8	3	0.706	0.774	No	4	6	0.736	0.737	No
Absent	31	9				19	21			
Tuberculosis										
Present	0	1	0.235	4.545	No	0	1	1.000	1.885	No
Absent	39	11				23	26			
Rickets										
Present	1	0	1.00	1.316	No	1	0	0.460	2.227	No
Absent	38	12				22	27			
Residual rickets										
Present	1	1	0.419	0.289	No	1	1	1.000	1.182	No
Absent	38	11				22	26			
Scurvy										
Present	2	1	0.561	0.595	No	1	2	1.000	0.568	No
Absent	37	11				22	25			
Tumour										
Present	2	1	0.561	0.595	No	2	1	0.588	2.476	No
Absent	37	11				21	26			
Syphilis										
Present	1	0	1.000	1.316	No	0	1	1.000	1.885	No
Absent	38	12				23	26			
Sinusitis										
Present	1	0	1.000	1.316	No	1	0	0.460	2.227	No
Absent	38	12				22	27			
Osteochondritis dissecans										
Present	1	0	1.000	1.316	No	1	0	0.460	2.227	No
Absent	38	12				22	27			

Table 25. – Frequency of pathological conditions according to IGD (including (n=51) and excluding (n=50) degree 1) and Fisher's tests.

a Significant p-value below 0.05

	Prenatal IGD					**Prenatal IGD**				
	Present (degree 1 to 3)	Absent (degree 0)	p-value[a]	Odds ratio	Significant	Present (excluding degree 1)	Absent (degree 0 or 1)	p-value[a]	Odds ratio	Significant
Dental caries										
Present	3	15	0.652	2.000	No	3	15	0.331	3.100	No
Absent	3	30				2	31			
Intervertebral disk disease										
Present	1	4	0.480	2.050	No	1	4	0.416	2.625	No
Absent	5	41				4	42			
Osteoarthritis										
Present	0	8	0.572	1.162	No	0	8	0.580	1.132	No
Absent	6	37				5	38			
Non-specific joint disease										

Present	3	14	0.397	2.214	No	2	15	1.000	1.378	No
Absent	3	31				3	31			
Trauma										
Present	1	9	1.000	0.800	No	1	9	1.000	1.028	No
Absent	5	36				4	37			
Non-specific infection										
Present	0	11	0.319	1.176	No	0	11	0.572	1.143	No
Absent	6	34				5	35			
Tuberculosis										
Present	0	1	1.000	1.136	No	0	1	1.000	1.111	No
Absent	6	44				5	45			
Rickets										
Present	1	0	0.118	10.000	No	1	0	0.098	12.500	No
Absent	5	45				4	46			
Residual rickets										
Present	0	2	1.000	1.140	No	0	3	1.000	1.116	No
Absent	6	43				5	43			
Scurvy										
Present	0	3	1.000	1.143	No	1	2	1.000	0.568	No
Absent	6	42				22	25			
Tumour										
Present	0	3	1.000	1.143	No	0	3	1.000	1.116	No
Absent	6	42				5	43			
Syphilis										
Present	0	1	1.000	1.136	No	0	1	1.000	1.111	No
Absent	6	44				5	45			
Sinusitis										
Present	1	0	0.118	10.000	No	1	0	0.098	12.500	No
Absent	5	45				4	46			
Osteochondritis dissecans										
Present	0	1	1.000	1.136	No	0	1	1.000	1.111	No
Absent	6	44				5	45			

Table 26. – Frequency of pathological conditions according to prenatal IGD (including and excluding degree 1) and Fisher's tests (n=51).

a Significant p-value below 0.05

3.2.5 Temporal evolution of vitamin D deficiency in the St. Antoine cemetery

Figure 8 and figure 9 present the presence of IGD, including and excluding the degree 1, respectively, and absence of IGD recorded in the sampled individuals according to the bottom altitude of coffins (meters, above mean sea level) and the area of burial within the cemetery. The bottom altitude of coffin of one individual is unknown. It is difficult to assess whether there really is an increase in the number of IGD cases during infancy in the zones 1 and 5 because of the very small sample size in the lower altitudes (33-34 m abmsl). Overall, the results tend to indicate a decrease in the number of IGD cases during infancy with time. Figure 10 presents the distribution of IGD cases according to the severity across the excavated areas of the St. Antoine cemetery.

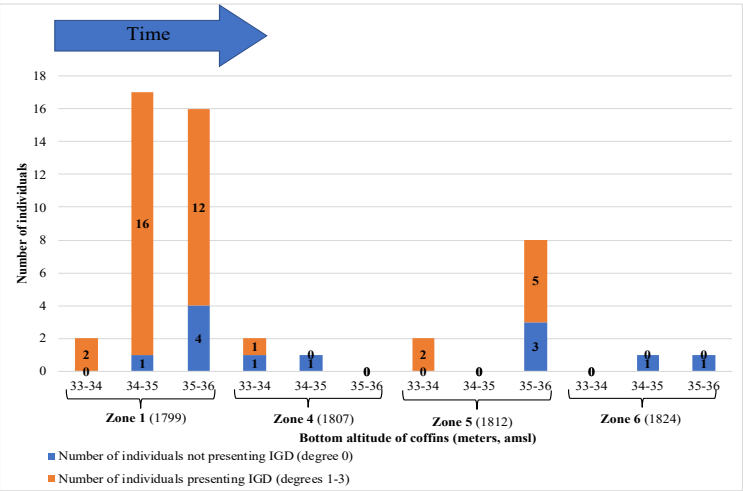

Figure 8. – Frequency of individuals presenting IGD (including degree 1), according to the bottom altitude of coffins (meters, amsl) (n= 50).

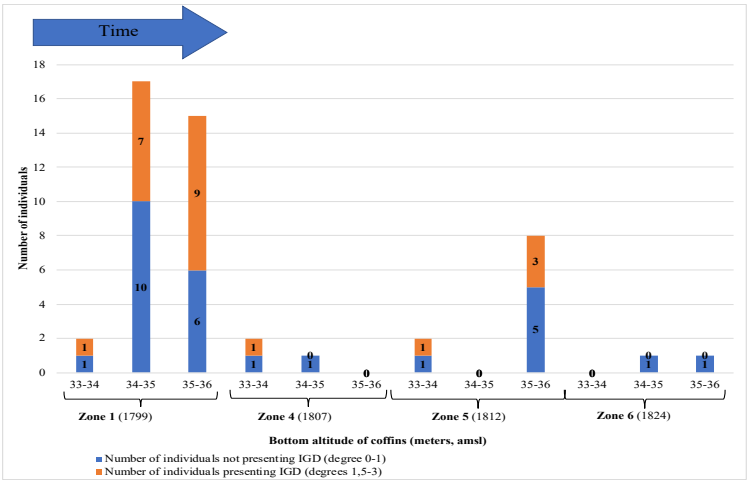

Figure 9. – Frequency of individuals presenting IGD (excluding degree 1), according to the bottom altitude of coffins (meters, amsl) (n= 49).

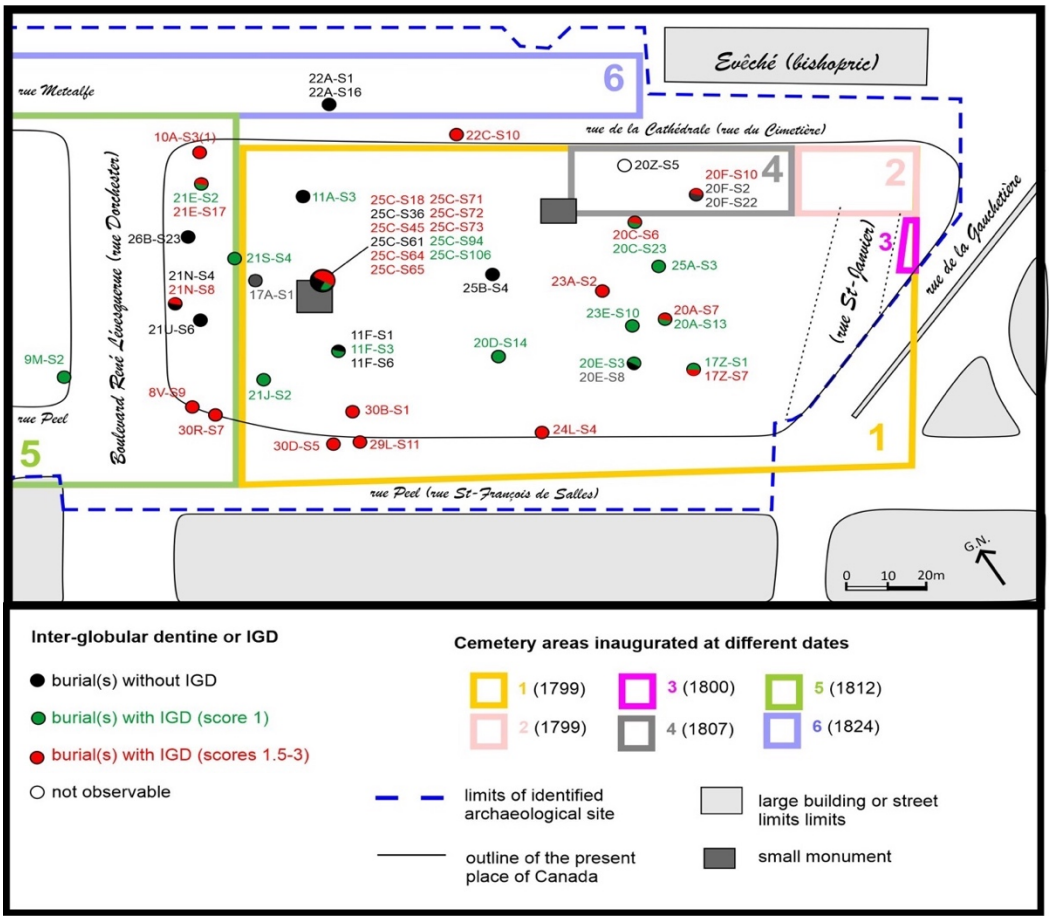

Figure 10. – Location of the burials under study (dots) (N=52) in relation to six areas inaugurated at different dates for the cemetery St. Antoine. Individuals presenting a degree 1 of IGD are green dots, a degree 1.5-3 are red dots and no IGD are black dots. The data is superimposed on the land map of Perrault (1867) (modified figure by I. Ribot from Ethnoscop (2016(a): maps 4 & 11), Ethnoscop (2016(b): map 2) and Arkéos (2018: map 5).

4. Discussion

This palaeoepidemiological and palaeopathological analysis of 52 sampled individuals from the largest 19th-century Catholic cemetery in Montréal provides a preliminary portrait of the health status during the transition to the industrial era (Ville de Montréal 2016). The results of this study are discussed below, first according to the preliminary methodological aspects and then according to the main research objectives, focused on the following questions:

i) What is the frequency and prevalence of pathological lesions and conditions among the sampled individuals from 19th century St. Antoine cemetery, Montréal?

ii) How do pathological lesions and conditions affect the various categories (sex, age) of the urban population sample from St. Antoine cemetery?

iii) Is there a possible link between the occurrence and severity of vitamin D deficiency episodes during infancy and the health deterioration occurring later in life?

iv) Is there a temporal evolution within the St. Antoine cemetery of vitamin D deficiency cases during infancy, in relation to the industrialization process?

4.1 Preliminary methodological aspects

Observers discrepancies

Concerning the discrepancies between the various recordings of degrees of IGD, a possible explanation would be as follows: it is difficult to systematically apply the various degrees of IGD to the observations of the lesions, since it is subjective and based on a visual assessment. As for the differences in the recording of the observable number of IGD episodes, longstanding IGD episodes are sometimes difficult to assess, depending on the thickness of the IGD bands, and require more experience to properly identify them. This explains why BK, the most experienced observer, recorded more longstanding episodes while RAB and MB recorded mostly separate episodes.

4.2 Synthesis of the results in relation to the key questions

Table 27 presents the summary of the results associated with the recording of IGD. These results indicate that the prevalence of vitamin D deficiency cases is underestimated when only observing skeletal lesions. Indeed, when only taking into account rickets and residual rickets, the prevalence in the sample is of 5.8% (3/52). The histological analysis thus reveals a more extensive and comprehensive profile of vitamin D deficiency throughout the lives of the individuals.

Presence of IGD	Maternal deficiency	Number of observable IGD episodes	Age of onset
75% (39/52) including degree 1, **44%** (23/52) excluding degree 1	**12%** (6/52) present *in utero* IGD	**56%** (29/52) = 1 episode **15%** (8/52) = 2 episodes **2%** (1/52) = 4 episodes **23%** (12/52) = 0 episode **4%** (2/52) = N.O. **56%** (29/52) = longstanding episodes (from *in utero* to up to 4 yrs.)	**63%** (33/52) or **85%** (33/39 individuals presenting IGD) = at or from birth

Table 27. – Main results about IGD (N=52). N.O.= not observable.

Table 28 also summarizes the frequencies of pathological lesions and conditions in the 52 studied individuals. Differential diagnosis often remains hypothetical; thus, the frequencies indicate possible pathological lesions and conditions. Dental pathological lesions are most frequent in the sample, as well as non-specific joint diseases and infections among the recorded pathological conditions.

Pathological lesions	n=	Pathological conditions	n=	Pathological conditions	n=
Dental calculus	22	Dental caries	18	Syphilis	1
LEH	5	IVD	5	Rickets	1
Cribra orbitalia	10	OA	8	Residual rickets	2
Porotic hyperostosis	2	Osteochondritis dissecans	1	Scurvy	4
Endocranial lesions	5	Non-specific joint disease	17	Tumour	3
Osteophytes	10	Trauma	10		
Schmorl's nodes	12	Non-specific infection	11		
		Tuberculosis	1		
		Sinusitis	1		

Table 28. – Frequencies of pathological lesions and conditions in the 52 sampled individuals.

4.3 Mortality and sex: possible link to pathological lesions and conditions

First, the statistical analysis indicated that *cribra orbitalia* was predominant in the nonadults, with 5 out of 8 juveniles presenting this type of lesion. This result concurs with other palaeopathological studies indicating that higher frequencies of *cribra orbitalia* were noted in children (Piontek and Kozlowski 2002). According to the results, OA is more likely to be found in male individuals (7/17 for males, 0/19 for females). One of the main precipitants of OA is age, since a positive correlation has been recorded between the prevalence of OA and increasing age (Waldron 2019, 724; Felson et al. 2000). One male individual is 19-20 years-old, three are 20-29 years-old, one is ±30 years-old and two are 30-59 years-old. A few younger male individuals thus present OA. According to clinical studies on modern populations by Felson (2000), before 50 years of age, male individuals are more likely to develop OA in most joints than women. The men presenting OA in our sample are almost all likely to be under 50 years of age, as well as the women not presenting OA (3/19 are 30-59 years-old, 1/19 is more than 40 years-old). This correlates to sex-specific differences of OA recorded by clinical research (Felson et al. 2000). Statistical analysis

revealed that non-specific joint diseases are more likely to be present in adult individuals (17/27 for adults, 0/8 for nonadults), which is congruent with the ageing process of joints (Waldron 2019).

According to the results of the statistical analysis, no correlation can be established between the demographic parameters (sex, age-at-death) and the presence of IGD episodes during infancy or only prenatal IGD episodes (including and excluding the degree 1). These episodes do not seem to affect the population in a selective manner (sex, age). The impact of vitamin D deficiency episodes during infancy on mortality could be explored more efficiently if the sample was bigger and comprised more age categories. It would allow for a better evaluation of the risk factors and survival rates in relation to vitamin D deficiency during infancy.

4.4 *In utero* and at birth vitamin D deficiency episodes: mother-infant link

Several cases of *in utero* IGD episodes were recorded in our sample (n= 6/52), indicating maternal deficiencies transmitted to the offspring. Such episodes were previously recorded in two individuals (15A-S36 and 2E4) from sites dated to a similar time period in Quebec: the urban cemetery of St. Matthew, Quebec City (1771-1860) and the rural one of St. Marie, Beauce (1748-1878) (Brickley, Kahlon, and D'Ortenzio 2019; D'Ortenzio, Ribot, et al. 2018; D'Ortenzio et al. 2016). During winter, pregnant women would be unlikely to receive enough vitamin D through sunlight exposure since Montréal lies at a latitude of 45°N (Gagnon et al. 2010, 820). This fact raises the question of the seasonality of the *in utero* IGD episodes. However, even during summer, when sunlight exposure would be optimal, fully covering clothing could impair proper intake of vitamin D in pregnant women (Brickley, Moffat, and Watamaniuk 2014). Clothing for noble women in the first half of the 19th century included long dresses with long sleeves and varying necklines, sometimes covering the chest and shoulders and sometimes exposing part of the shoulders and the chest (Beaudoin-Ross 1992). A bonnet was also often worn, partially providing shade for the face (Brett 1967). The only available information on common fashion for a women in the 19th century Montréal is that they wore both the blouse and the skirt, in the city as well as in the countryside (Beaudoin-Ross 2013). Furthermore, exposure of pregnant women to natural light might have been reduced by the cramped housing resulting from overcrowded life conditions in the city (Bradbury 1984; Brault 1990, 20). All such factors might have contributed to the *in utero* IGD episodes observed in foetuses.

At birth, infants hold vitamin D reserves transferred *in utero* from the mother to the child through the placenta (Misra et al. 2008, 408). Serum 25(OH)D levels in mothers are the same as the levels found in infants (Misra et al. 2008, 408). Infants born of mothers with healthy vitamin D levels would have reserves above the accepted threshold of deficiency (37.5 nmol/L) (Misra et al. 2008, 408). Thirty-three out of the 52 studied individuals from our sample present at or from birth IGD episodes to up to 3 years of age. This rapid appearance of IGD at birth indicates that these infants had no reserves and were unable to synthesize vitamin D (Misra et al. 2008, 408). The lack of vitamin D synthesis could be linked to the two main sources of vitamin D: sun's UV-rays exposure and diet (Thacher and Clarke 2011, 50). Breast-feeding is unlikely to be the main cause. Maternal milk does not contain enough vitamin D to prevent vitamin D deficiency in breastfed infants without supplementary sunlight exposure, suggesting that both cultural (e.g. clothing, time spent outside *versus* indoor) and seasonal factors might have caused vitamin D deficiency in the individuals presenting IGD at or from birth, regardless of the type of housing (urban *versus* rural) (Brickley, Ives, and Mays 2020, 83; Mays et al. 2018; Ives and Humphrey 2018; Kovacs 2011; Holick 2004, 365; Kreiter et al. 2000; Specker 1985). In the middle of the 19th century, breast-feeding was still widespread in Montréal across several groups, such as the French and Irish Catholics, and most of the mothers were breastfeeding to some extent (Thornton and Olson 1991, 409). It could thus be a common practice between individuals presenting and not presenting vitamin D deficiency at birth, suggesting that other causal factors than breastfeeding were at work for the IGD. For example, several modern cases showing a high prevalence of rickets in breast-fed infants report cultural practices preventing exposure to sunlight, such as swaddling and keeping the infants in a cradle all day (Mays et al. 2018, 8; Shin, Ghotbi, and Claster 2009; Tserendolgor et al. 1998).

4.5 Longstanding and multiple vitamin D deficiency episodes: a link to cultural and environmental factors?

The results indicate that 29 individuals out of 52 individuals our sample suffered continuously from vitamin D deficiency, ranging from the *in utero* period to up to 4 years of age. This tends to indicate that sunlight exposure during infancy was too low, even during summer, to provide sufficient vitamin D synthesis through the skin. It is therefore likely that cultural factors impairing sunlight exposure (e.g. clothing, time spent outside *versus* indoor) contributed to vitamin D deficiency Brickley, Moffat, and Watamaniuk 2014). Other biophysical and cultural factors

could have increased the deficiency, still remaining not severe enough to cause death. For example, contracting diseases through contaminated water and/or milk during the weaning period like the cholera might have caused diarrhoea and/or vomiting (Cadotte 1990, 137). Poor intestinal absorption, along with a tendency to stay inside during illness, could have led to vitamin D deficiency on the long term (Bikle 2007, V–51). Historical records indicate that cholera epidemics stroke Montréal's population on at least five years, although not always consecutively (e.g. 1832, 1849, 1851, 1852, 1854) (Cadotte 1990, 137). The nonadult 30R-S7 is a good example of prolonged vitamin D deficiency. From the womb to approximately 4 years-old, this individual suffered from severe vitamin D deficiency. This individual presents lower limb bones curvature at death (8.5 years); however, biomechanical and shape changes can remain after mineralization returns to normal (Brickley and Mays 2019, 541; Elder and Bishop 2014, 1665). Porotic bone beneath the growth plate is indicative of active vitamin D deficiency, however, the proximal and distal epiphysis of 30R-S7's lower limb bones are missing (Brickley and Mays 2019, 540). It is possible that vitamin D deficiency is one of the consequences, but not the only one, of the poor sanitary conditions that ultimately contributed to the individual's death (Cadotte 1990, 137; Brault 1990, 20; Bradbury 1984).

Nine individuals out of the 52 individuals under study present multiple and separate deficiency episodes (see Table 29):

Individual (code)	Age of onset of IGD episodes
10A-S3(1)	Episode 1: c. birth Episode 2: c.1.5 years
11F-S1	Episode 1: *in utero* to c.1 year Episode 2: c.2 years
17A-S1	Episode 1: *in utero* to c.1 year Episode 2: c.2 years
20A-S7	Episode 1: around birth Episode 2: c.1.5 years
20C-S6	Episode 1: c. birth Episode 2: c.3 years
25C-S18	Episode 1: c. birth to c.1.5 years Episode 2: c.5 years Episode 3: c.7 years Episode 4: c.9 years
25C-S65	Episode 1: c. birth to c.2 years Episode 2: c.4 years
25C-S71	Episode 1: c. birth to c.1.5 years Episode 2: c.2 years
9M-S2	Episode 1: c.2 years Episode 2: c.4 years

Table 29. – Ages of onset of multiple IGD episodes recorded in the 52 studied individuals.

While these deficiency episodes are not always separated by 1-year interval, they might be the result of seasonal variations. This concurs with the fact that there is a significant lack of sunlight exposure during winter in Montréal's region as it is located at a rather high latitude (45 °N) (Gagnon et al. 2010, 820). Focusing on other historic populations from northern Europe (17^{th}-19^{th} century, Netherlands), Veselka et al. (2019) also revealed that vitamin D deficiency occurred in recurrent episodes following seasonal variations. This result was supported by both micro-CT and histological evaluations of IGD on the mandibular molars of 30 individuals of various ages and sex (Veselka et al. 2019). Vitamin D synthesis through sunlight exposure might have been totally absent during the winter months in these historic Dutch communities living at a northern latitude of 53 °N (Veselka et al. 2019, 129). Our results for Montréal, although located at a slightly more southern in latitude, also tend to support the seasonal variations as one of the causal factors of these deficiencies.

4.6 Vitamin D deficiency and health deterioration: a possible link?

LEH and IGD

A possible link between vitamin D deficiency during infancy and LEH was detected. Indeed, the p-value for the link between the presence of LEH and more severe degrees of IGD (excluding degree 1) is 0.016 and the odds ratio 2.500, which is significant. Linear enamel hypoplasia is linked to systemic stress that occurred during the period of tooth crown formation, either pathological or nutritional (Goodman and Rose 1990; Goodman 1996; Duray 1996; King, Humphrey, and Hillson 2005). It is thus logical that individuals presenting vitamin D deficiency during infancy, which is caused by environmental or nutritional stress (Brickley, Moffat, and Watamaniuk 2014, 48), would also present LEH. It is possible that more severe episodes of vitamin D deficiency are necessary to induce LEH. These results concur with research conducted on dogs that concluded to an increase in interglobular spaces and linear enamel hypoplasia linked to vitamin D deficient diets (Mellanby 1928; Mellanby 1934; D'Ortenzio et al. 2016). However, this link is hypothetical and not definitive since our sample is small and further research needs to be undertaken to explore the subject.

Pathological skeletal lesions that may occur in vitamin D deficiency cases and IGD

The lack of correlation between vitamin D deficiency during infancy and skeletal lesions associated with vitamin D deficiency in the sampled individuals seems logical. Indeed, the presence of IGD refers to deficiency episodes that happened during infancy, while the skeletal lesions were developed later at the time of death of the individuals. What the results indicate, at the most, is that the presence of vitamin D deficiency episodes during infancy does not seem to predispose the individuals to develop lesions that may occur in vitamin D deficiency cases later in life.

Other pathological lesions and conditions and IGD

No link was found between other pathological lesions and conditions and the presence of IGD, except for traumas (excluding degree 1 of IGD) and dental calculus (including degree 1 of IGD). However, since no explanation can justify these correlations, they might be random. The lack of correlation between pathological lesions and conditions and IGD is most likely related to the small size of our sample.

4.7 Spatiotemporal evolution of vitamin D within the cemetery of St. Antoine

The distribution of the cases of vitamin D deficiency (including and excluding degree 1 of IGD) according to the bottom altitude of the coffins (meters, above mean sea level) and the different areas of burial within the cemetery reveals that the cases of vitamin D deficiency seem to decrease with time. This might be due to the fact that the majority of the individuals were recovered from the oldest part of the cemetery (zone 1) (Ethnoscop 2012; 2014; 2016b; 2016a; Arkéos 2018). This means that there is an over-representation of individuals buried in the oldest parts of the cemetery and thus of the most ancient burials. This decrease through time might be the result of a bias of the sample. Moreover, during the 1850s and 1860s, several graves were exhumed and transferred to the new cemetery Notre-Dame-des-Neiges, which might account for the higher number of ancient graves left behind discovered by the archaeologists (Ethnoscop 2012, 21). The zones 1 to 5 also seem to have been used simultaneously for some periods after their inauguration, which complicates the spatiotemporal analysis (Arkéos 2018, 6).

5. Conclusion

To conclude, there are several key findings that can be drawn from this research on 52 individuals from the St. Antoine cemetery in Montréal (1799- 1854):

- A high prevalence (75%-44%) of vitamin D cases during infancy according to the histological analysis. The results indicate the presence of prenatal IGD episodes, revealing maternal deficiencies, and a high frequency of at or from birth IGD episodes, indicating possible cultural influences. The presence of longstanding IGD episodes also suggests the influence of cultural factors, while the few multiple episodes of IGD recorded might reflect seasonal deficiencies.
- Histology allows the recording vitamin D deficiency episodes that happened during the early life of the individuals and that are not visible on the skeleton anymore, which might explain the high prevalence in our sample. This suggests that a histological analysis of another urban population dating to the Industrial Revolution (e.g. UK, France) would be relevant (for comparisons with our present study) since it could allow the recording of early-life deficiency episodes and reveal a more accurate representation of the frequency of vitamin D deficiency cases.
- *Cribra orbitalia* was associated with the nonadults age group, while non-specific joint diseases were linked to the adults age group. As for OA, it was correlated to male individuals.
- No correlation was noted between vitamin D deficiency cases during infancy and age-at-death or sex, however, having a bigger sample with a wider age-at-death range could help explore this issue further.
- Non-specific infections and tuberculosis were expected to correlate with the prevalence of vitamin D deficiency episodes during infancy according to the previous research by Snoddy and colleagues (2016) but the results indicated no correlation. In the case of tuberculosis, only one potential case was recorded, thus not allowing to explore the subject.
- LEH correlated statistically with the presence of IGD (excluding degree 1). However, various other parameters than vitamin D deficiency during infancy contribute to the pathogenesis process and this hypothetical result needs to be explored further using a bigger sample.
- The temporal analysis of the frequency of vitamin D deficiency cases during infancy revealed that over the course of the use of the cemetery, this condition was more frequent at the beginning and then tended to decrease. However, the majority (67%)

of the sampled individuals comes from the oldest part of the cemetery and studying more individuals from the most recent parts of the cemetery might help to confirm and possibly understand better the increase or decrease of cases.

To complete this study, an isotopic analysis of ^{15}N would be relevant to pinpoint the weaning age of the sampled individuals and evaluate if the cultural practices surrounding this event might have increased vitamin D deficiency. The research is limited by the fact that the exact geographical origin of the sampled individuals is unknown, since no tombstones were recovered during the excavations, as is the case for most historic cemeteries in Quebec (Ethnoscop 2012; 2014; 2016b; 2016a; Arkéos 2018). In the future, an isotopic analysis of strontium and oxygen would be relevant to assess the geographical origin of the individuals and verify if the results really indicate a portrait of early life in 19th century Montréal or reflect another environment, as some people were first generation migrants. Finally, conducting this type of study investigating the possible links between vitamin D deficiency during infancy and health deterioration later in life using a bigger sample would be relevant.

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ANNEXE A

Estimation of the state of preservation of the adult skeletons

The state of preservation is evaluated according to the Anatomical Conservation Index (ACI) developed by Dutour (1989). This index, calculated for each identified individual, constitutes the ratio between the sum of the preservation scores (ΣC) and the total number of bones in the skeleton (N): **ACI = 100 x ΣC / N**

A score C is given to each type of bone or group of bones according to the following 6 degrees of conservation: **complete= 1, almost complete= 0.95, ¾ present = 0.75; half present = 0.50; ¼ present= 0.25; absent = 0.** The table below presents the different categories of conservation that can be inferred by the calculated ACI.

ACI	Preservation category
1 à 9.9	Bad
10 à 19.9	Poor
20 à 39.9	Correct
40 à 59.9	Good
60 à 79.9	Very good
80 à 100	Excellent

ANNEXE B

Methods used for the sex estimation of adult skeletons

The sex estimation of individuals under the age of 18 years old is almost impossible and not recommended (Katzenberg and Saunders 2000; Scheuer 2000). Indeed, sexual dimorphism, i.e. skeletal differences between males and females, is only significant once puberty starts (Roberts 2018, 127). Sex estimation is generally done using the coxal bone and the skull, which are more accurate (Murail et al. 2005; Bruzek 2002; Byers 2016; Buikstra and Ubelaker 1994). However, many other methods were developed using other bones, for example, the patellas, metacarpals/metatarsals, tarsals and long bones (eg. humerus, radius, ulna) (see table below). This increases the possibilities for sex estimation, especially in the case of fragmentary individuals. However, these methods are limited in their accuracy because of the intra-inter observers' errors, the nature of the reference population used for the method (i.e. time period or origin) and the age-at-death of the individuals (Roberts 2018, 126). Thus, we used here six sex estimates: not applicable due to young age (N.A.), female, likely female, male, likely male and undetermined (Und.). Female and male tendencies are terms used when the results of various methods are mixed but predominantly indicate one sex or the other.

Age category	Type of method	Author-reference population	Description of method-based on…
Adult	Metric	**Murail et al. 2005-**Olivier collection (France 20th century), Spitafields collection (England 18-19th centuries), Tamagnini collection (Portugal 19-20th centuries), Garmus collection (Lithuania 20th century), Dart collection (South Africa 20th century), Hamann-Todd collection and Terry collection (the United States 20th century), forensic collection (Thaïland, 20th century)	Probabilistic sexual diagnosis (PSD) using Coxal bones
	Non-metric	**Bruzek 2002-**France (20th century), Portugal (19-20th century)	Coxal bones
		Byers 2016- Unknown.	Sacrum
		Buikstra and Ubelaker 1994- Unknown	Skull
	Metric	**DiMichele and Spradley 2012-**United States (1900-1985).	Calcaneus

	Case and Ross 2007-Terry collection (United States, 20th century)	Metacarpals, metatarsals and phalanges
	Steele 1976-Terry collection (United States, 20th century)	Calcaneus and talus
	Holman and Bennett 1991-Terry collection (United States, 20th century)	Upper long bones.
	Peckmann et al. 2016- (Spain, 21st century)	Patella

ANNEXE C

Age-at-death estimation

The age-at-death was estimated using various metric and non-metric methods. The table below presents the methods in detail. Concerning the nonadults, estimation of age-at-death is more accurate than for adults (Roberts 2018, 131). The principal methods of age-at-death estimation for the juveniles are based on dental calcification and eruption (AlQahtani, Hector, and Liversidge 2010; Ubelaker 1979), as well as on bone development, although often less precise than tooth development (i.e. long bones length, union of primary ossification centres and fusion of epiphyses) (Schaefer, Black, and Scheuer 2009; Black and Scheuer 1996; Scheuer and MacLaughlin-Black 1994; Molleson and Cox 1993; Plato et al. 1984; Fazekas and Kósa 1978; Maresh 1970). Indeed, teeth are less influenced by "environmental factors" than bones, such as poor diet or disease during growth (Roberts 2018, 134; Liversidge 2015; Cardoso 2007).

As age-at-death estimation for adults is based on the ageing processes of dental and bone tissues, the estimations are thus less accurate, with large age intervals because of various factors, i.e. biological sex, geographic origin, genetic, cultural practices and activities (Roberts 2018, 137–40). The methods of estimating age-at-death for adults are mainly based on the coxal bones (Suchey, Brooks, and Katz 1988; Schmitt 2005), the skull (Meindl and Lovejoy 1985), the teeth (Lovejoy 1985), the vertebrae (Albert and Maples 1995) and the ribs (DiGangi et al. 2009; İşcan, Loth, and Wright 1984). Older individuals are also often underestimated, since ageing processes become even more variable with age (Roberts 2018, 137).

Age category	Type of method	Author-reference population	Description of the method- based on…
Juvenile	Non-metric	**AlQahtani, Hector, and Liversidge 2010-** Spitafields collection (England, 18-19th centuries) and Maurice Stack collection	Dental calcification and eruption
		Ubelaker 1979-multiple sources	Dental eruption through gums.
	Metric	**Black and Scheuer 1996-**Spitafields, St. Bride and St. Barnabas collections (United Kingdom, 18th-19th centuries) and Lisbone collection (20rst century) ; **Maresh 1970-**American, 20th century; **Molleson and Cox 1993-**Spitafields collection (England, 18-19th centuries);	Diaphyseal and total length of diverse bones

		Scheuer and MacLaughlin-Black 1994- Spitafields and St. Barnabas collections (England, 18-19th centuries) **Fazekas and Kósa 1978-**Hungary, middle of the 20th century; **Plato et al. 1984-**Chamorro population	
	Non-metric	**Schaefer, Black, and Scheuer 2009-**multiple sources	Union of primary ossification centres.
		Schaefer, Black, and Scheuer 2009-multiple sources	Fusion of epiphysis
Adult	Non-metric	**Schmitt 2005-**18-20th centuries, Spitafields (England), Simon (Switzerland), Madrid Institute of Forensic Medecine (Spain), Hamann-Todd (United States), Dart (South Africa), Faculty of Medecine of Chang Maï (Thaïland) collections	Morphological changes of auricular surface and of retro-auricular area
		Suchey, Brooks, and Katz 1988- United-States, 20th century	Morphological changes of pubic symphysis.
		DiGangi et al. 2009- Kosovo, 20th century	Morphological changes of first rib
		İşcan, Loth, and Wright 1984- not mentioned	Morphological changes of 3rd, 4th or 5th rib
		Albert and Maples 1995- unknown	Fusion stages of vertebral rings and senescence of vertebral bodies
		Meindl and Lovejoy 1985-Hamann-Todd collection (United States)	Fusion stages of cranial sutures
		Lovejoy 1985-United States	Dental wear

ANNEXE D

Horizontal and vertical information on the localization of the sampled individuals amongst the St. Antoine cemetery (Ethnoscop 2012; 2014; 2016b; 2016a; Arkéos 2018).

Individual	Sex	Age-at-death	Area of the cemetery	Inauguration of the area of the cemetery	Bottom altitude-coffin meters (amsl)	Artefact in association with the burial
8V-S9	FT	16-29 yrs.	5	1812	33,78	Leather
9M-S2	F	18-29 yrs.	5	1812	33,76	-
10A-S3 (1)	Und.	16-22 yrs.	5	1812	35,51	Buttons, Fabric, Plaque
11A-S3	MT	16-20 yrs.	1	1799	35,02	-
11F-S1	M	30 yrs. +	1	1799	35,67	Buttons
11F-S3	FT	16-21 yrs.	1	1799	35,53	Pin
11F-S6	FT	20-49 yrs. (20-29 yrs. according to the vertebras)	1	1799	35,52	Wedding rings
17A-S1	M	15-20 yrs.	1	1799	35,76	-
17Z-S1	M	15-20 yrs.	1	1799	34,37	Buttons Pins Ceramic fragment
17Z-S7	FT	+40 yrs.	1	1799	33,64	-
20A-S7	MT	15-23 yrs.	1	1799	34,51	Button (1) Pin with fabric
20A-S13	F	16-29 yrs.	1	1799	34,48	-
20C-S6	FT	20-29 yrs.	1	1799	34,55	Pin Tombstone recovered during the excavation of 20C (1810 C.E.)
20C-S23	F	20-30 yrs.	1	1799	34,52	Tombstone recovered during the excavation of 20C (1810 C.E.)
20D-S14	FT	17-20 yrs.	1	1799	34,14	Pin Staple
20E-S3	MT	20-29 yrs.	1	1799	34,57	Metal button Cufflinks (4) 4 holes-buttons (6)
20E-S8	Und.	18-24 yrs.	1	1799	34,68	-
20F-S2	N.A.	6.5 yrs.	4	1807	34,16	Buttons (3)
20F-S10	M	±30 yrs.	4	1807	33,93	-
20F-S22	M	19-20 yrs.	4	1807	33,85	Pin Buttons (5) Fabric
20Z-S5	N.A.	18 months ± 6 months	4	1807	34,63	Pin
21E-S2	MT	27-66 yrs. (mid. 45.6 yrs.)	5	1812	35,21	-

21E-S17	M	+40 yrs.	5	1812	35,26	-
21J-S2	F	16-22 yrs.	1	1799	34,80	Medal Pearl Pin
21N-S4	FT	15-25 yrs.	5	1812	35,33	Buttons (3) Pins (2)
21N-S8	FT	16-22 yrs.	5	1812	35,33	-
21S-S4	FT	20-36 yrs.	5	1812	35,76	Medal Pin
21U-S6	N.A.	7-9.5 yrs.	5	1812	35,25	Pin
22A-S1	F	18-29 yrs.	6	1824	35,06	Bottle glass
22A-S16	Und.	+30 yrs.	6	1824	34,48	-
22C-S10	MT	20-29 yrs.	1	1799	35,40	-
23A-S2	MT	30-59 yrs.	1	1799	34,41	Buttons (3)
23E-S10	F	16-29 yrs.	1	1799	34,59	-
24L-S4	M	20-49 yrs. (more 20-29 yrs. According to the vertebras)	1	1799	34,52	Iron forged nail Laminated forged nail Steel screw Bone button Metal medal
25A-S3	MT	16-20 yrs.	1	1799	34,32	-
25B-S4	F	34-45 yrs.	1	1799	35,61	Ceramic Glass Bone button Pipe Pearl Bead
25C-S18	F	30-59 yrs.	1	1799	35,11	-
25C-S36	F	30-59 yrs.	1	1799	35,84	-
25C-S45	MT	18-29 yrs.	1	1799	35,90	-
25C-S61	MT	20-29 yrs.	1	1799	35,65	-
25C-S64	F	15-16 yrs.	1	1799	35,61	Brass pin Laminated iron nail Forged iron nail Steel screw
25C-S65	MT	20-29 yrs.	1	1799	35,12	-
25C-S71	F	30-59 yrs.	1	1799	35,73	Brass pin Laminated iron nail Forged iron nail Steel screw
25C-S72	M	30-50 yrs.	1	1799	-	Metal button Laminated iron nail Forged iron nail
25C-S73	N.A.	9-10 yrs.	1	1799	35,58	Laminated iron nail Forged iron nail Steel screw Metal coffin handle
25C-S94	MT	24-35 yrs.	1	1799	35,34	Laminated iron nail Forged iron nail Mother-of-pearl button
25C-S106	M	30-59 yrs.	1	1799	34,86	Laminated iron nail Forged iron nail Bone button

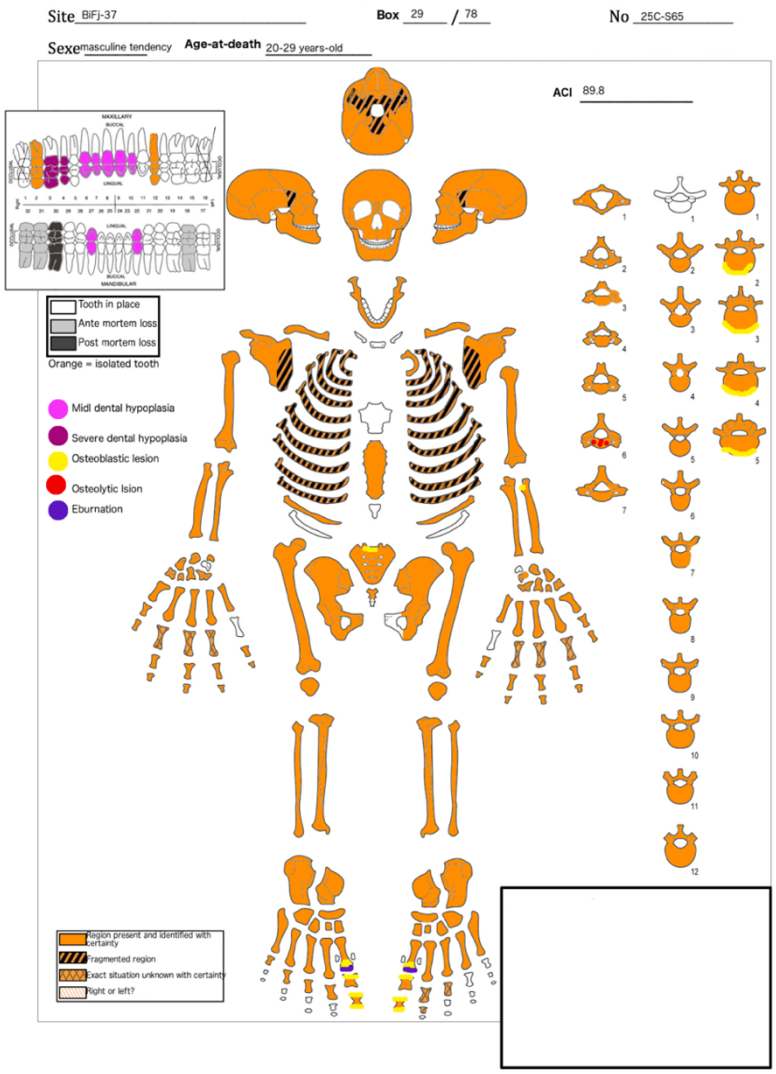
26B-S23	Und.	16-20 yrs.	5	1812	35,46	Button Metal medal Laminated iron nail Forged iron nail
29L-S11	M	+40 yrs.	1	1799	34,19	-
30B-S1	M	18-29 yrs.	1	1799	34,55	Mother-of-pearl buttons (3) Metal plaque
30D-S5	MT	20-39 yrs.	1	1799	34,23	-
30R-S7	N.A.	8.5 yrs.	1	1799	33,82	Pins

F= female, FT= likely female, M= male, MT= likely male, Und. = undetermined, N.A.= not applicable.

ANNEXE E

Example of a digital skeleton form to record various pathological lesions (Modified from Gutierrez 2017)

A colour code is attributed to seven descriptive categories of pathological lesions: **red**= osteolytic lesion, **yellow**= osteoblastic lesion, **light blue**= abnormal size, **deep green**= abnormal shape, **neon green**= fracture, **deep purple**= eburnation, **turquoise**= cavitation. Three more specific pathological lesions were also recorded: **pink**= mild linear enamel hypoplasia (LEH), **deep pink**= severe LEH, **burgundy=** dental calculus. One pathological condition was recorded: **deep blue**= dental caries.



ANNEXE F

Pathological lesions associated with dental caries (Kinaston et al. 2019)

Dental diseases			
Pathology	***Affected body part and aspect***	***Possible lesion(s)***	***Targeted age group***
Dental caries	*Teeth (crown, roots)*	-Necrosis of the dentin and pulp chamber eventually causing a hole or cavity in the enamel.	All age groups

ANNEXE G

Pathological lesions associated with certain bacterial infections (Roberts and Buikstra 2019; Davies-Barrett, Antoine, and Roberts 2019; Mann and Hunt 2013; Lewis 2004)

Bacterial infection			
Pathology	***Affected body part and aspect***	***Possible lesion(s)***	***Targeted age group***
Tuberculosis	*Spine*, mainly the anterior part of the vertebral bodies of the first lumbar vertebra	-Lytic destruction of the vertebral bodies	All ages
		-Cavity formation on the vertebral bodies	All ages
		-Vertebral collapse accompanied by a fracture and a kyphotic deformity of the spine	All ages
	Hips, i.e. the coxal bone and the proximal part of the femur	-Lytic lesions on the femoral head, the femoral neck, the greater trochanter and the acetabulum	Children
		-Destruction of the femoral head and the acetabulum	Children
		-Extension of the lumbosacral focus unilaterally or bilaterally	Young adults
		-Lytic destruction on the pubis	Older children and young adults
	Knee, i.e. the distal part of the femur, the patella and the proximal part of the tibia	-Cortical erosion and destruction of the articular facets	Mainly toddlers, children and adolescents
		-Fibrotic or osseous ankylosis in healed lesions	Mainly toddlers, children and adolescents
	Ankle and tarsals, mainly the tibiotalar articulation and rarely the talocalcanear articulation	-Cavity formation on the talus resulting in destruction	Children
		-Development of tibiocalcanear ankylosis in the case of talus destruction	Children
		-Destruction of the tibial epiphysis and metaphysis	Children
		-Tibiotalar osseous ankylosis	Children
		-Fusion of the talus and calcaneus	Children

	Metacarpals and metatarsals	-*Spina Ventosa,* i.e. the formation of a new bony shell that accounts for the ballooned appearance of the involved bone	Toddler, young children
	Shoulder	-Destruction of the humeral head and glenoid cavity	All ages, more frequent in adults
	Elbow, i.e. the distal part of the humerus, the proximal part of the ulna and the proximal part of the radius.	-Bone destruction -New periarticular bone formations -Fibrous ankylosis in children and osseous ankylosis in adults resulting in healed lesions	Toddler to young adult Toddler to young adult Toddler to young adult
	Wrist and carpals, i.e. the radiocarpal articulations (adults), intercarpal and carpometacarpal (kids). Long bones diaphysis	-Abnormal formation of cavities, usually in the metaphysis -Osseous periosteal formations -Osteoporosis -Osteosclerosis	Children Children Adults Adults
	Ribs	-Lytic lesions, perforation of the cortex -Periostitis on the pleural surface	All ages All ages
	Sternum, mainly the manubrium.	-Lytic lesions that can perforate the cortex	All ages
	Skull	-Round lytic lesions on the cranial vault -Vascular lesions on the endocranial surface -Occasional destruction of the petrous bone and the mastoid process -Facial lesions that can deteriorate in *lupus vulgaris* leading to the destruction of nasal bones	Mainly toddlers and children Mainly toddlers and children Toddlers All ages
Venereal syphilis	*Cranial vault*	-Caries sicca	Adults
	Nasal bones	-Destruction of the nasal bones;	Adults
	Spine (mostly cervical vertebras)	-Osteomyelitis	Adults
	Long bones	-Nongummatous and gummatous osteoperiosis	Adults

ANNEXE H

Pathological lesions associated with certain infectious diseases (Roberts 2019; 2007; Lewis, Roberts, and Manchester 1995)

Infectious diseases			
Pathology	***Affected body part and aspect***	***Possible lesion(s)***	***Targeted age group***
Sinusitis	*Sinuses*	-Bone deposits of trabecular and lamellar bone	All ages
		-Inflammatory pitting	All ages

ANNEXE I

Pathological lesions associated with certain joint diseases (Waldron 2019; Vikatou, Hoogland, and Waters-Rist 2017; Adams and Roughley 2006)

Joint diseases			
Pathology	***Affected body part and aspect***	***Possible lesion(s)***	***Targeted age group***
Osteoarthritis	*Any articulation*	-Eburnation -Sclerosis -Osteophytes -Porosities	Risk increases with age for all lesions
IVD	*Spine* (more common in the cervical vertebras, less in lumbar and rare in the thoracic)	-Pits around the rim of the vertebrae -Marginal osteophytes	Risk increases with age for all lesions
Osteochondritis dissecans	*Any synovial joint*	-Depressed surface resembling a "crater" -Edges smooth and well defined	All ages All ages

ANNEXE J

Pathological lesions associated with certain metabolic diseases (Brickley and Mays 2019; Brickley et al. 2018; Schattmann et al. 2016; Geber and Murphy 2012; Brickley, Mays, and Ives 2007)

Metabolic diseases				
Pathology	***Affected body parts and aspect***	***Possible lesion(s)***	***Active/Healed***	***Targeted age group***
Rickets	*Skull*	-Cranial vault porosity	A	Nonadults for all lesions
		-Orbital roof porosity	A	
		-Cranial vault thickening	H	
	Mandibular	-Deformed mandibular ramus	A/H	
	Ribs	-Rib bending deformity	A/H	
		-Costochondral rib flaring	A1H	
		-Costochondral rib porosity	A/H	
			A	
	Ilium	-Ilium concavity	A/H	
	Long bones	-Bending deformity-upper-limb long bones	A/H	
			A/H	
		-Bending deformity-lower-limb bones	A/H	
			A/H	
		-Long-bone metaphyseal flaring/cupping of ends	A/H	
			A/H	
		-Long-bone general thickening	A/H	
		-Long-bone cortical (especially metaphyseal) porosity	H	
		-Superior flattening femoral metaphysis	A	
			A/H	
		-Coxa vara	A/H	
		-Porosis/roughening on bone underlying growth plates	A/H	
			A/H	
			A/H	
			A	
Residual rickets	*Long bones*	-Bending (upper and lower limb)		Adults for all lesions
	Ribs	-Curvature		
	Scapula	-Curvature		
	Sternum	-Curvature		
	Spine	-Curvature		
	Sacrum	-Curvature		
Osteomalacia	*Skull*	-Extensive porosity and « cardboard-like » texture reported in pathology museum collections. Not observed in archaeological bone to date		Adults for all lesions
		-Invagination of foramen magnum. Not observed in archaeological bone to date		

	Ribs	-Deformation, can be hard to identify in fragmented bone -Pseudofratures, often multiple. Particularly common where corsets were worn, but also noted in noncorset-wearing communities	
	Scapula	-Deformation of blades exaggerated posterior curvature -Pseudofractures noted at the spinous process and lateral border	
	Iliac crest	-Deformation -Pseudofractures.	
	Pubic bone	-Deformation -Pseudofractures.	
	Spine	-Compression fractures of the vertebral body -Pseudofractures noted on transverse processes	
	Long bones	-Deformation, difficult to assess macroscopically -Pseudofractures -Any bone can be affected, but more commonly noted locations include the femoral neck	
	Sternum	-Deformation	
	Sacrum	-Deformation	
Scurvy	*Sphenoid* *Zygomatic bone* *Maxillae* *Mandible* *Orbital walls* *Cranial vault* *Long bones (metaphyses)* *Scapula* *Ilium*	-Porosities and bone formations	All ages for all lesions

ANNEXE K

Injury types related to trauma (Redfern and Roberts 2019)

Trauma		
Body location	***Injury types***	***Excludes***
Head	Superficial injury	Fracture: pathological, with osteoporosis, stress
Neck	Open wound	
Thorax	Fracture: closed	
Abdomen, lower back, lumbar spine, and pelvis	Fracture: open	
Shoulder and upper arm	Malunion of fracture	
Elbow and forearm	Nonunion of fracture	
Wrist and hand	Dislocation, sprain and strain	
Hip and thigh	Injuries to nerves and spinal cords	
Ankle and foot	Injury to blood vessels	
Multiple body regions	Injury to muscle, fascia and tendons	
Unspecified part of the trunk, limb or body region	Crushing injury	
Multiple body regions	Traumatic amputation	

ANNEXE L

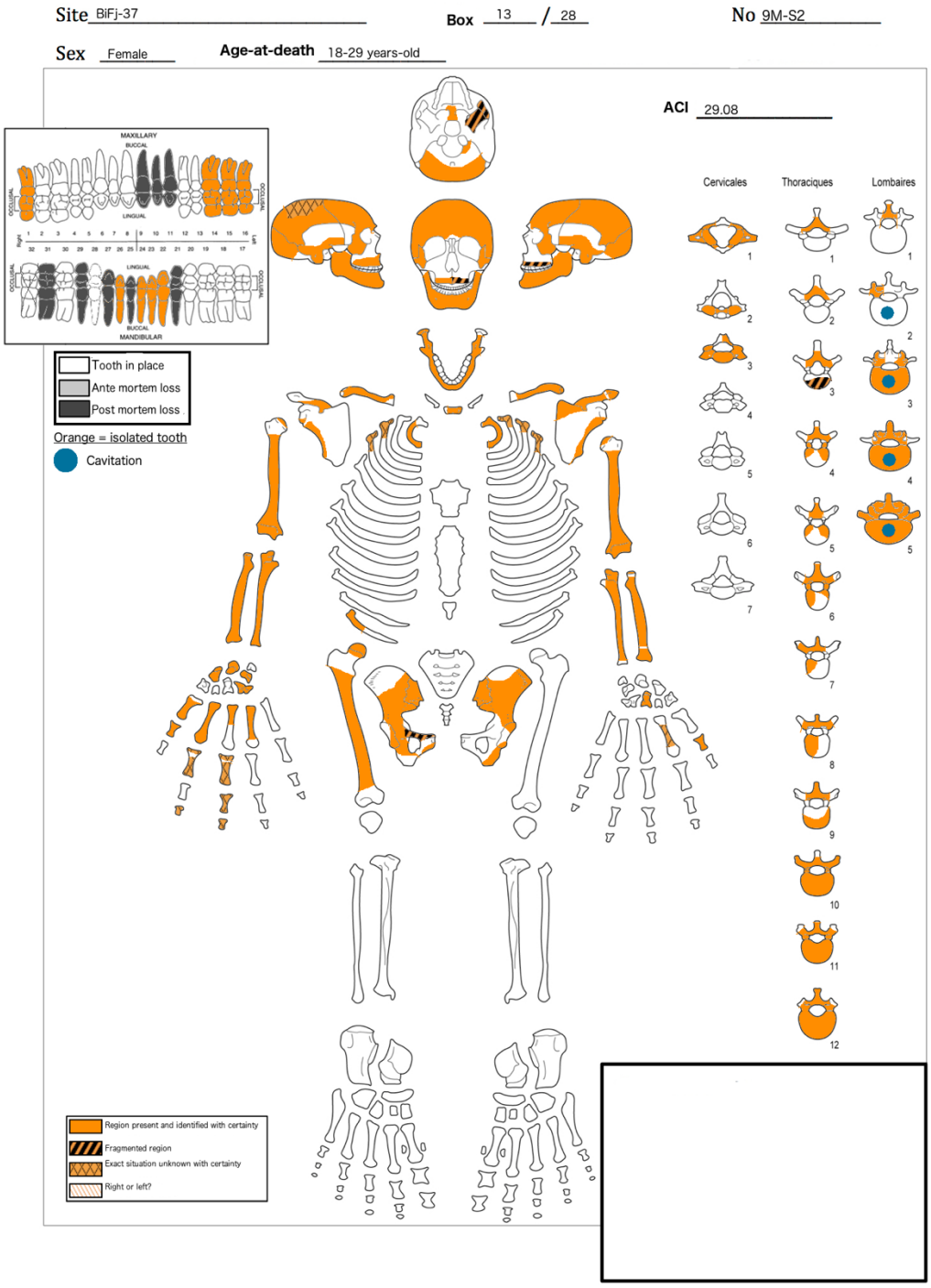
Description of various pathological lesions (Kinaston et al. 2019; Alves-Cardoso and Assis 2018; Mann and Hunt 2013; Naveed et al. 2012; Faccia and Williams 2008; King, Humphrey, and Hillson 2005; Lewis 2004; Hershkovitz et al. 2002; Patty Stuart-Macadam 1991; El-Najjar, Desanti, and Ozebek 1978; El-Najjar et al. 1976)

Pathological lesions

Lesion	*Affected body part and aspect*	*Possible lesion(s)*	*Targeted age group*
Dental calculus	*Teeth (crown)*	-Calcified bacterial plaque	All age groups
Linear enamel hypoplasia (LEH)	*Teeth (enamel)*	-Transverse grooves, lines or pits on the enamel	Juveniles
Cribra orbitalia	*Orbits (roof)*	-Capillary-like impressions on the bone; -Scattered fine foramina -Large and small isolated foramina; -Foramina with trabecular involvement	All age groups for all lesions
Porotic hyperostosis	*Cranial vault*	-Destruction/ thinning of the outer table of the bones; -Widening of the diploe -Hair-on-end striations	All age groups for all lesions
Endocranial lesions	*Cranial vault*	-Porous lesions; -Fiber bone formation -Capillary lesions; -Hair-on-end lesions (frosted appearance)	All age groups for all lesions
Schmorl's nodes	*Spine (vertebral bodies)*	-Small cavitation in the surface	Risk increases with age
Osteophytes	*Several joints*	-Bony outgrowth that develops at the contact point between cartilage and bone	Risk increases with age

ANNEXE M

Recording of pathological lesions in the 52 individuals and differential diagnoses



Individual: 9M-S2

ACI: 35.84

Age-at-death: 18-29 years-old

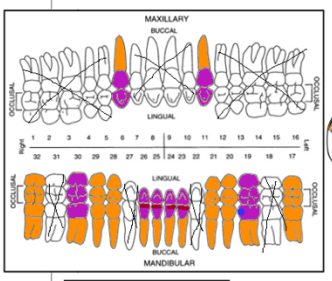
Sex: Female

Description of pathological lesions: Cavitation on two lumbar vertebras (LV) (superior face) (a).

Differential diagnosis: Diagnostic of Schmorl's nodes**, consistent with** degenerative joint disease.

Site BiFj-37 Box 16 / 28 No 10A-S3(1)

Sex Undetermined Age-at-death 16-22 years-old

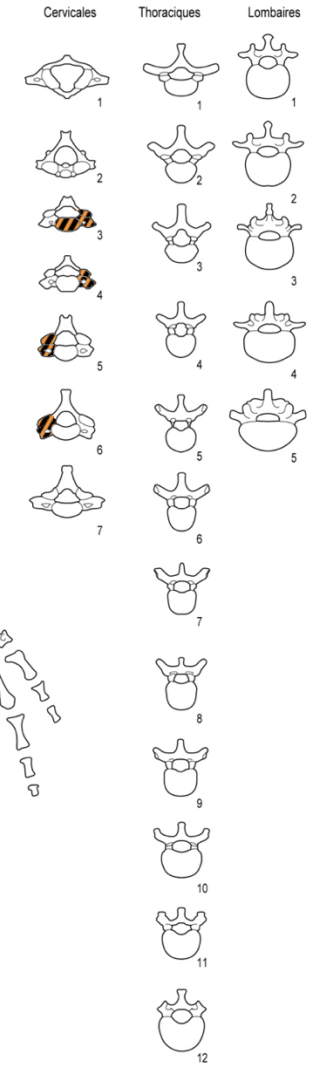


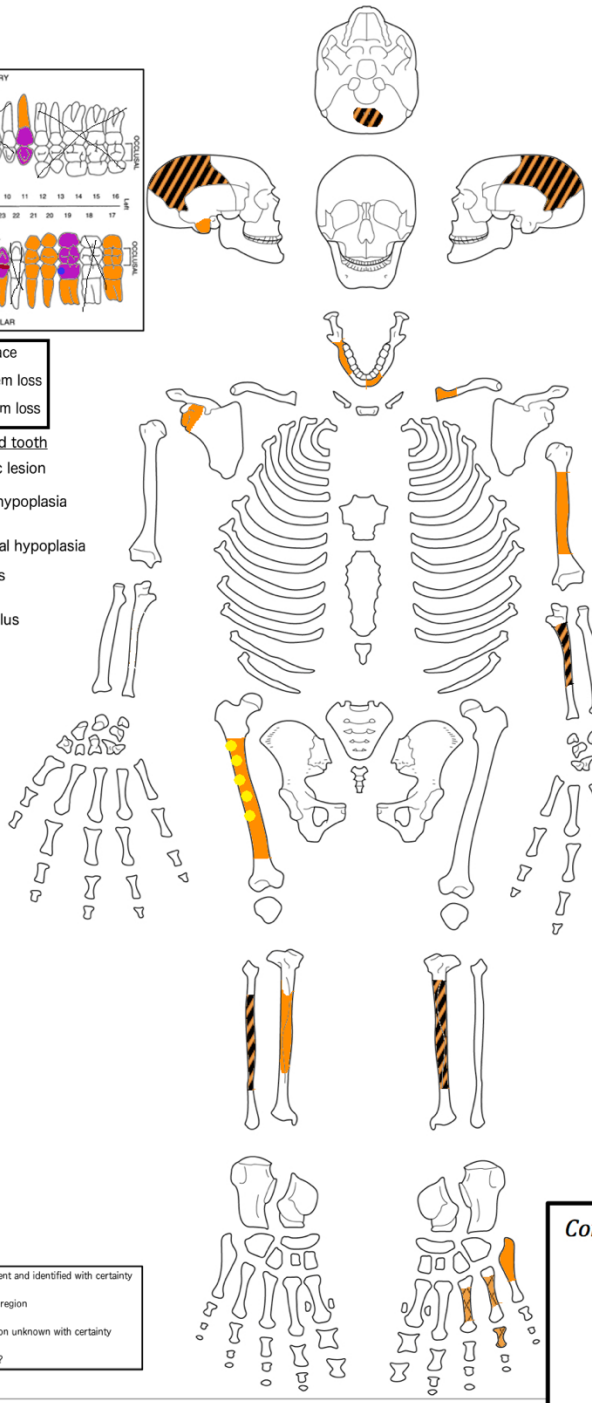
Tooth in place
Ante mortem loss
Post mortem loss

Orange = isolated tooth

Osteoblastic lesion
Mild dental hypoplasia
Severe dental hypoplasia
Dental caries
Dental calculus

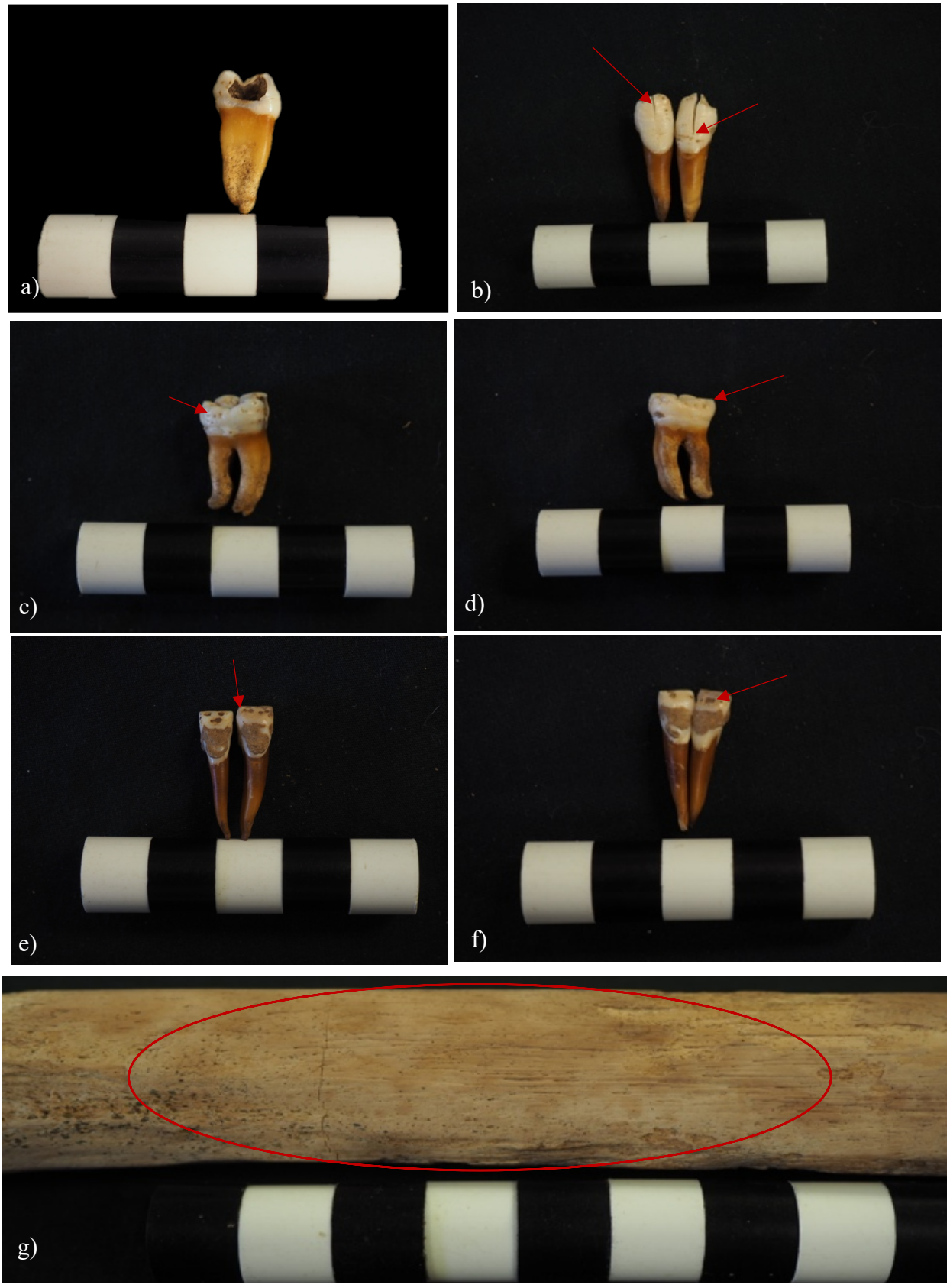
ACI 6.16





Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?

Commentaires :


a)
b)
c)
d)
e)
f)
g)

Individual: 10A-S3(1)

ACI: 6.16

Age-at-death: 16-22 years-old

Sex: Undetermined

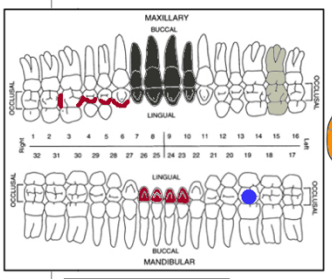
Description of pathological lesions: Cavity on the crown of left the M_1 (mesial view) (a), calcified plaque on the first lower incisors (lingual view/buccal view) (e) (f), pits on upper canines (buccal view) (b), first lower molars (lingual view) (c) (d) and lower incisors (lingual view/buccal view) (e) (f), periosteal lesions on the right femur (lateral view) (g).

Possible aetiologies: Childhood disease-nutritional deficiencies, periapical inflammation or trauma to a deciduous tooth, fever, disease, endocrine dysfunction, infection during odontogenesis.

Differential diagnosis: Diagnostic of LEH, dental caries and dental calculus, **consistent with** trauma.

Site BiFj-37 Box 21 / 28 No 11A-S3

Sex Male tendency Age-at-death 16-20 years-old



Tooth in place
Ante mortem loss
Post mortem loss

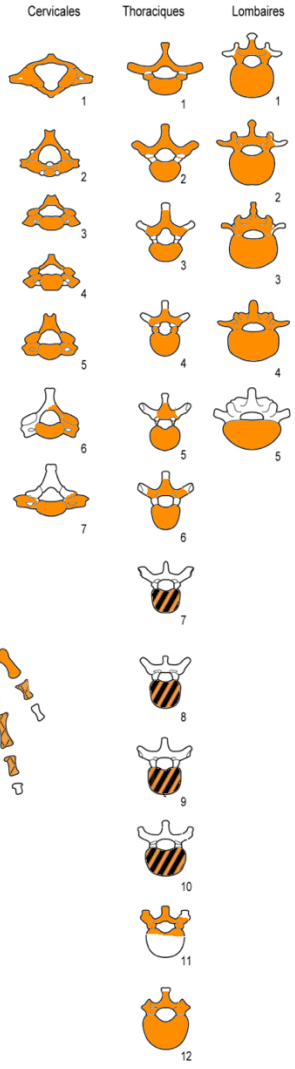
Osteolytic lesion

Dental caries

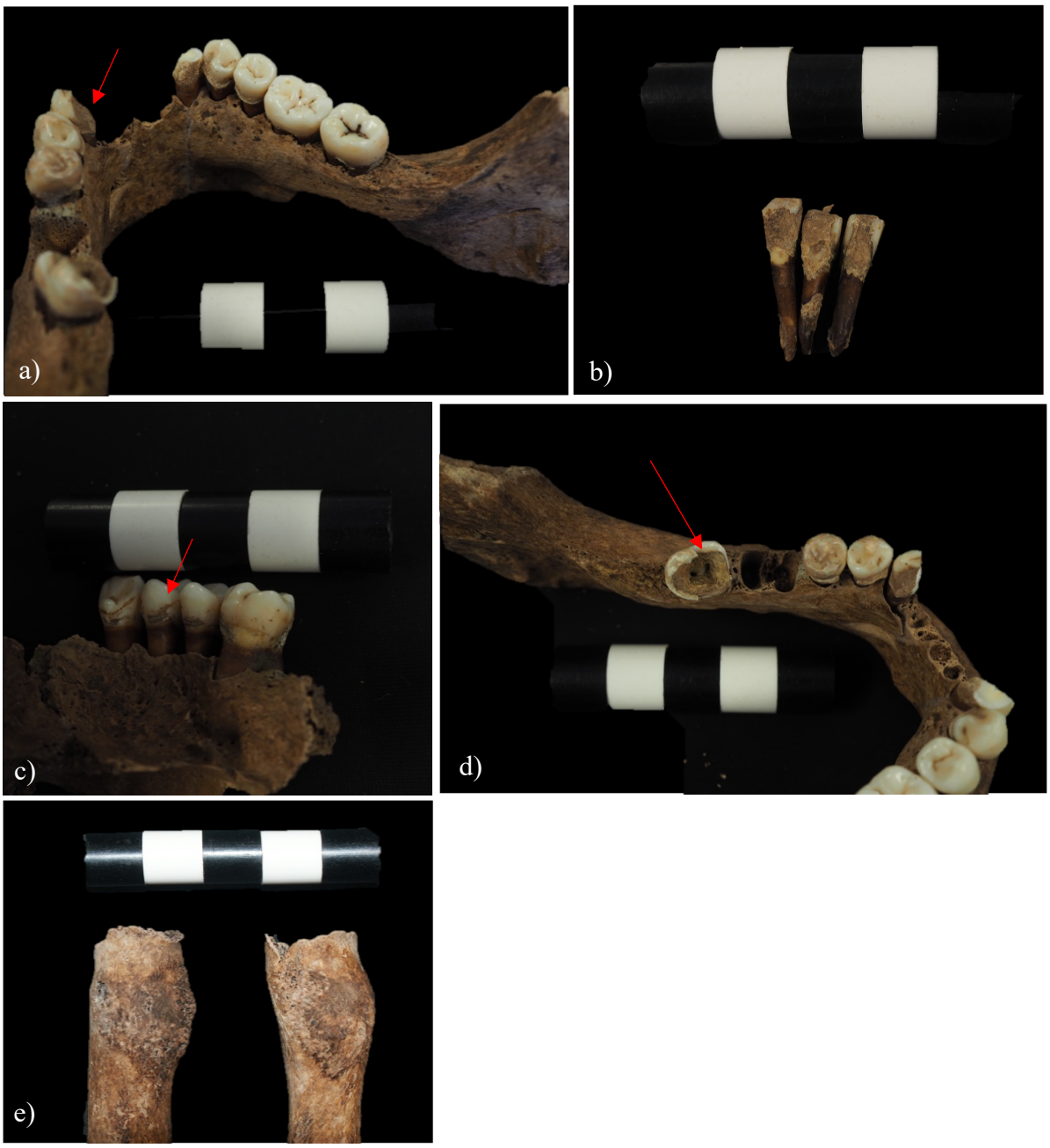
Dental calculus

Inferior M3 not erupted

ACI 47.52



Region present an identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?



Individual: 11A-S3

ACI: 47.52

Age-at-death: 16-20 years-old

Sex: Likely male

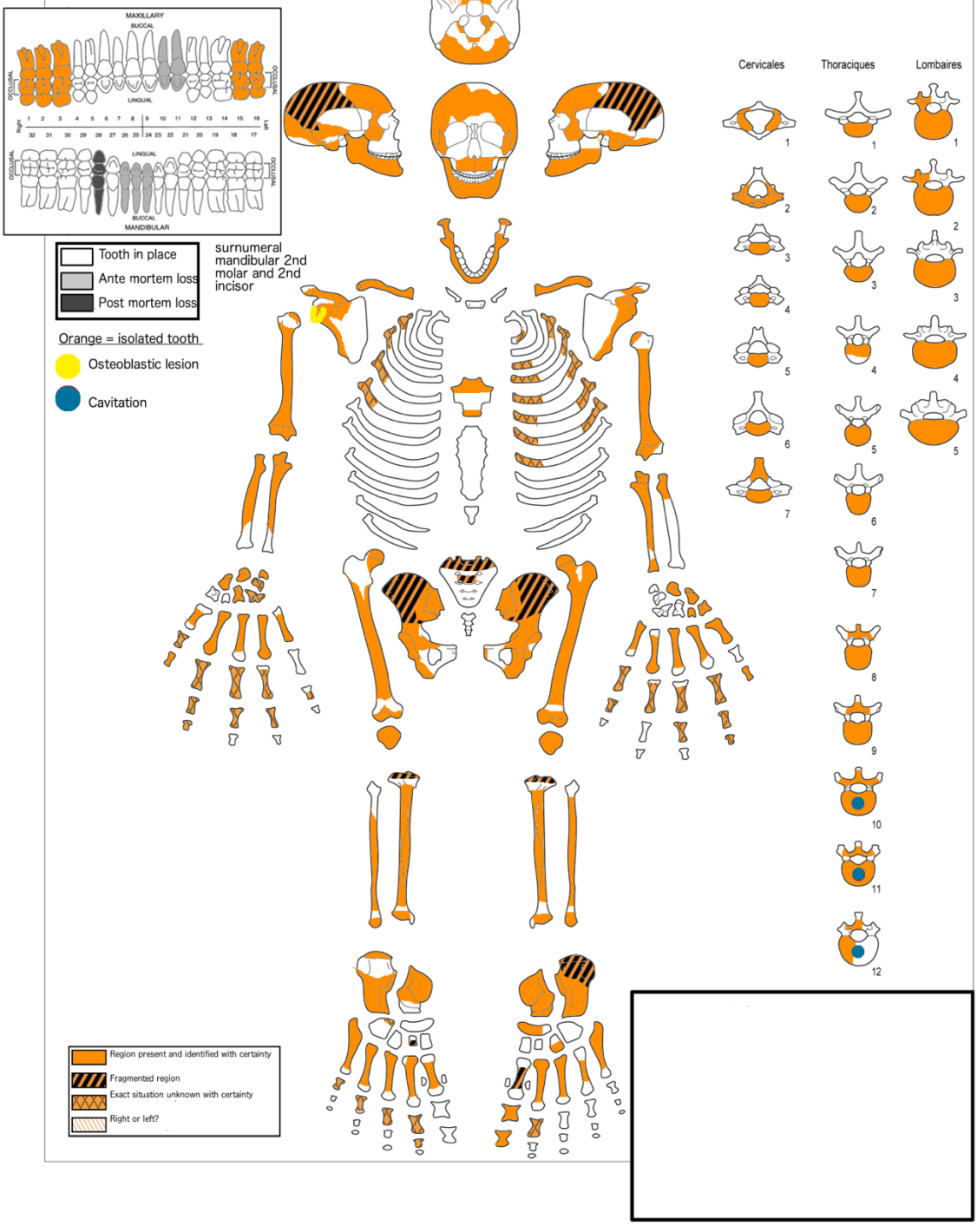
Description of pathological lesions: Calcified plaque on lower (a) (b) and upper teeth (lingual view), cavity on the M_2 (occlusal view) (d), porosities on the radial tuberosities (medial view) (a).

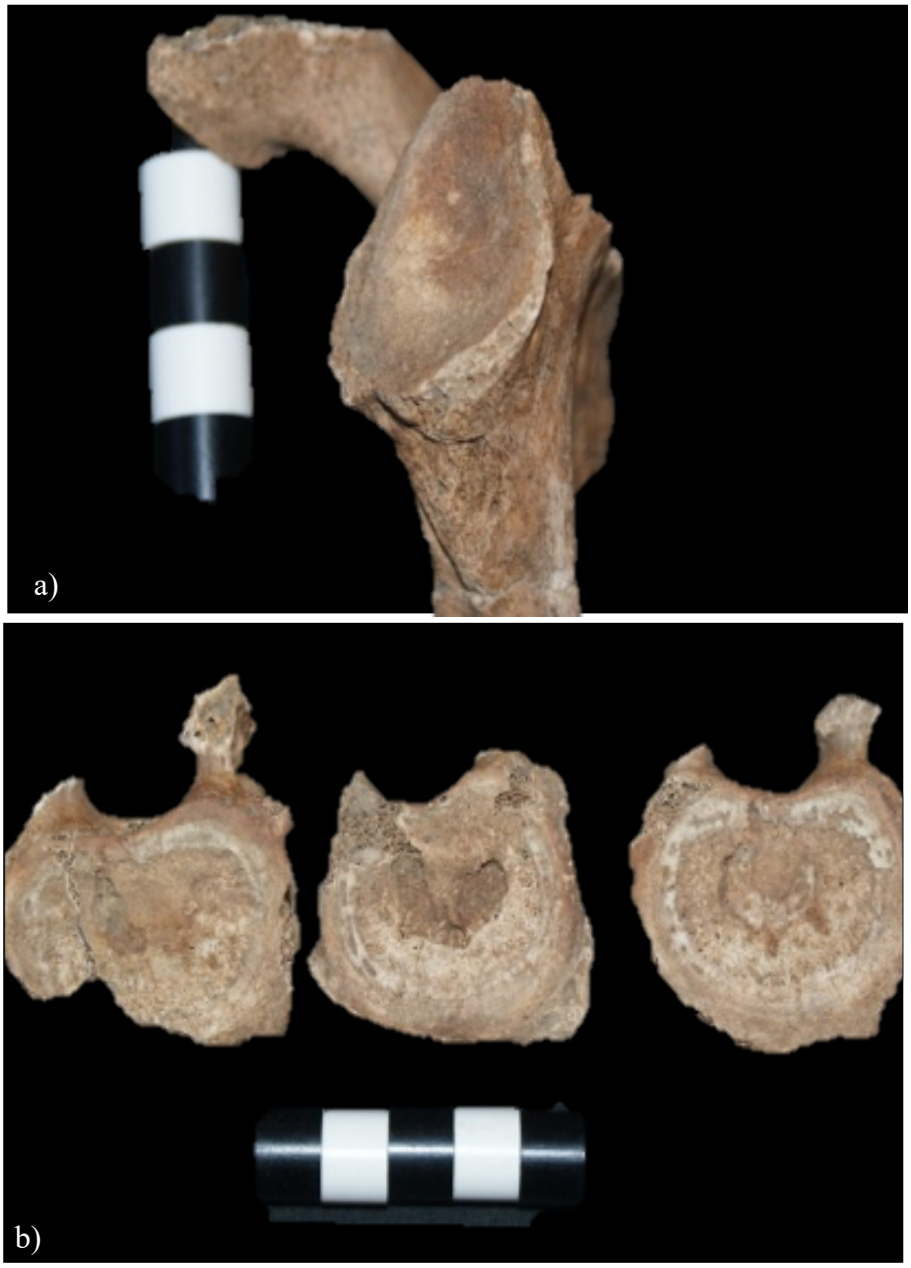
Possible aetiology: Strenuous activity.

Differential diagnosis: Diagnostic of dental calculus, dental caries and inflammation of the biceps muscle insertion.

Site BiFj-37 Box 22 / 28 No 11F-S1

Sex Male Age-at-death 30 years old or more





Individual: 11F-S1

ACI: 57.04

Age-at-death: 30 years-old +

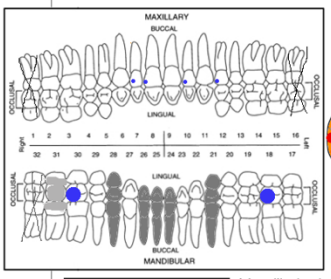
Sex: Male

Description of pathological lesions: Bone formation or “lipping” on the border of the right scapula’s glenoid cavity (medial view) (a), cavitation on thoracic vertebrae (TV) 9,10 and 11 (inferior view) (b).

Differential diagnosis: Diagnostic of osteophytes, Schmorl’s nodes and intervertebral disk disease (IVD).

Site BiFj-37 Box 20 / 28 No 11F-S3

Sex Female tendency **Age-at-death** 16-21 years-old



Tooth in place
Ante mortem loss
Post mortem loss

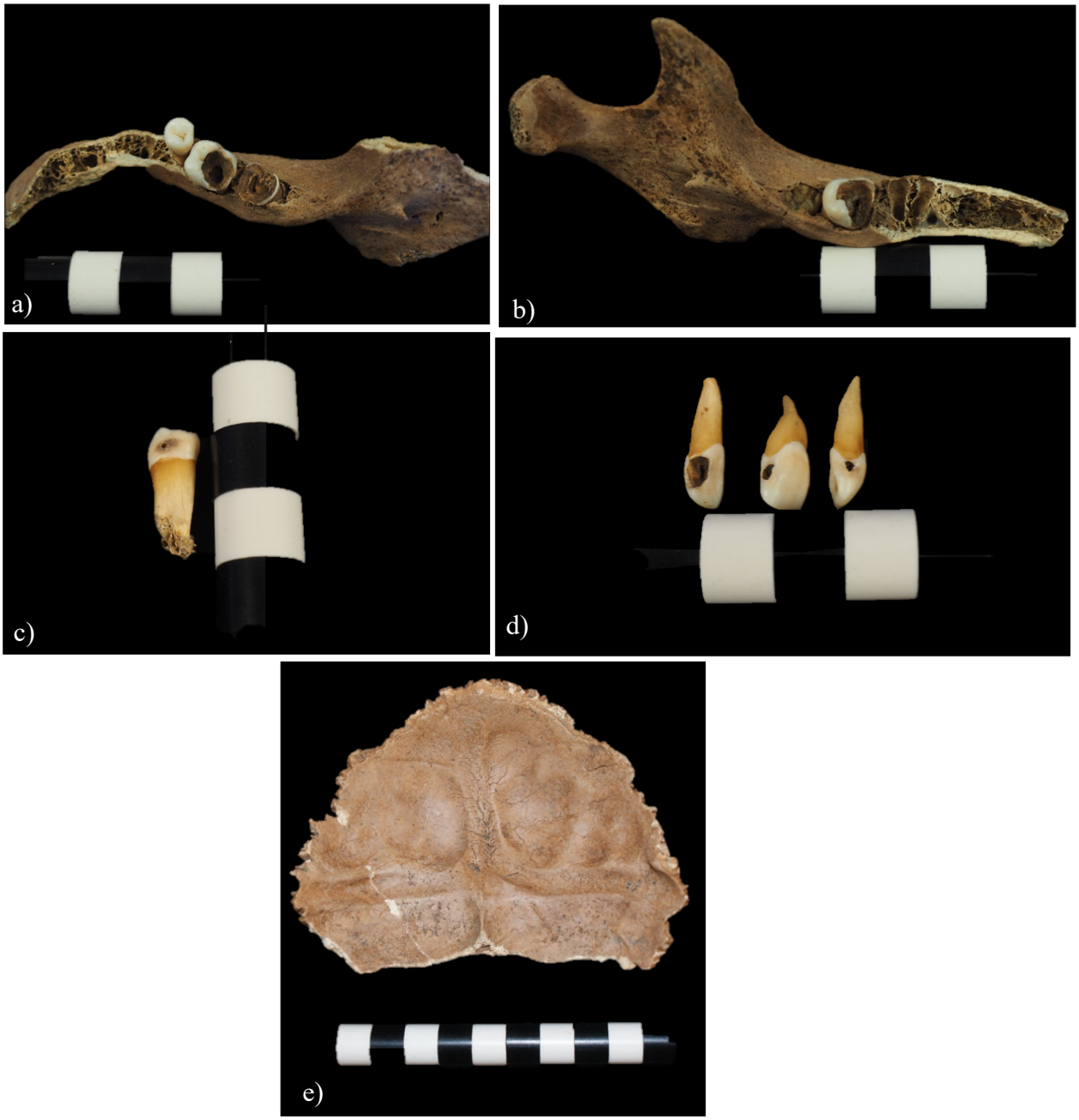
Mandibular left M3 germ not erupted

Osteolytic lesion

Dental caries

ACI 35.68

Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?



Individual: 11F-S3

ACI: 35.68

Age-at-death: 16-21 years-old

Sex: Likely female

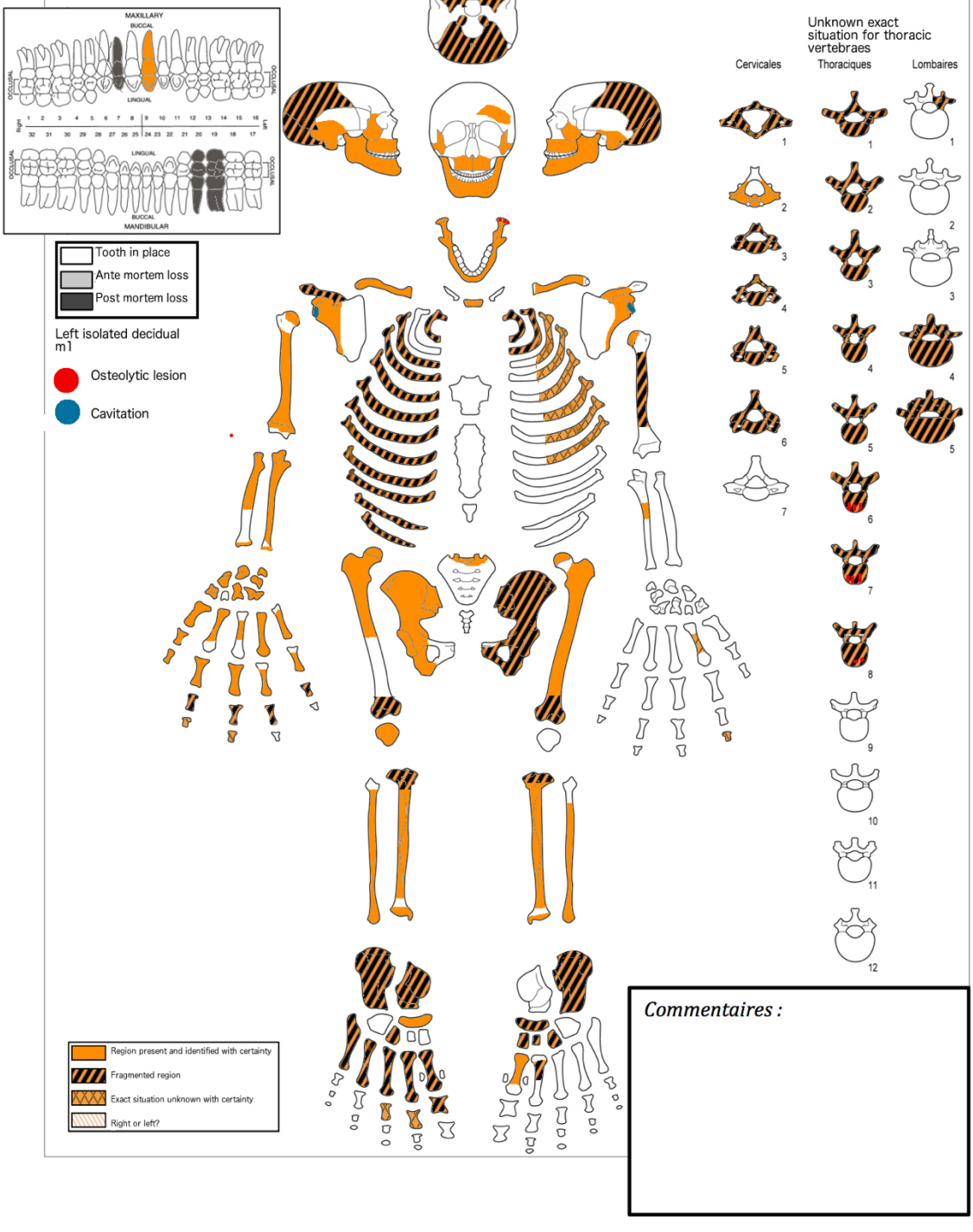
Descrpition of pathological lesions: Cavity on the right M_1 and left M_2 (occlusal view) (a) (b), premolar (c) and right I^1, right and left I^2 (distal/mesial views) (d), capillary-like lesions on the occipital bone (endocranial view) (e).

Possible aetiologies: Trauma, primary and secondary infections of the meninges, tumours, TB, syphilis and vitamin deficiencies of A, C and D.

Differential diagnosis: Diagnostic of dental caries and endocranial lesions.

Site BiFj-37 Box 1 / 16 No 17A-S1

Sex Male Age-at-death 15-20 years-old



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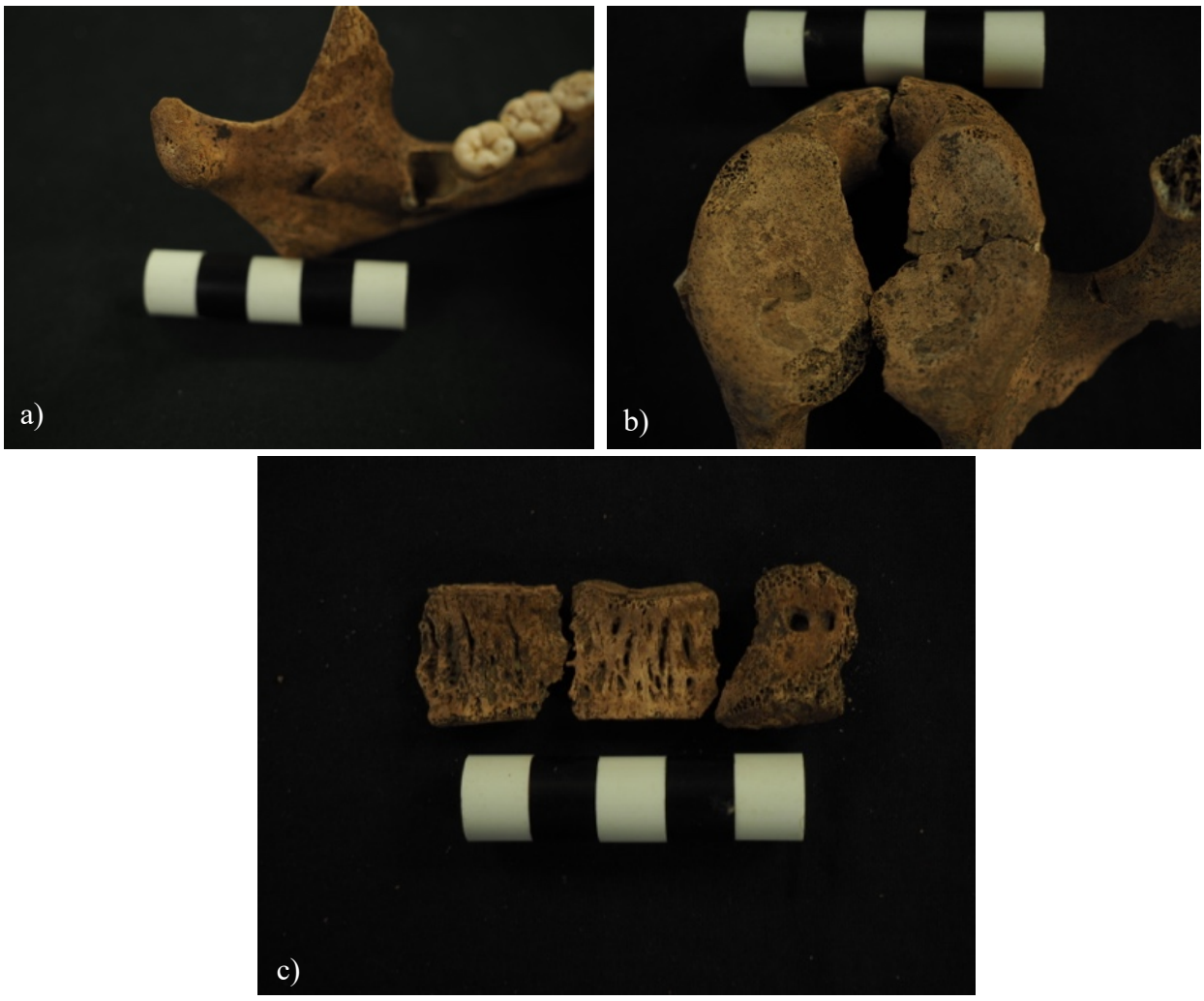


Individual: 17A-S1

ACI: 41.8

Age-at-death: 15-20 years-old

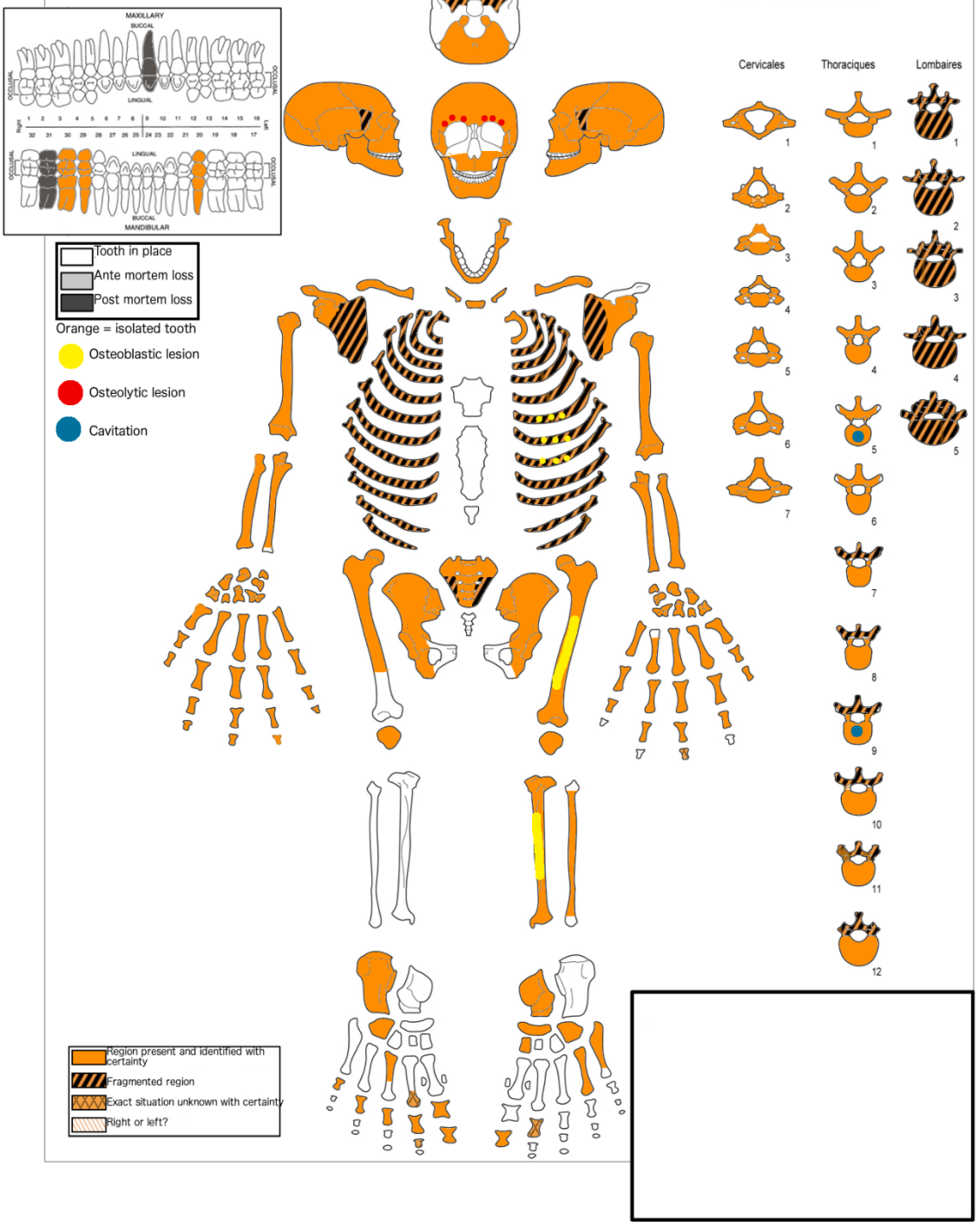
Sex: Male

Description of pathological lesions: Porosities on the left mandibular condyle (a), depressions on the left and right glenoid cavities (b), lytic lesions on three vertebral body fragments which are abnormal compared to the appearance of other vertebral bodies of individuals of the same age (c).

Differential diagnosis: Typical of early degeneration of the left temporomandibular joint.

Site BiFj-37 Box 9 / 18 No 17Z-S1

Sex Male Age-at-death 15-20 years-old




a)
c)
b)
d)
e)
f)

Individual: 17Z-S1

ACI: 70.64

Age-at-death: 15-20 years-old

Sex: Male

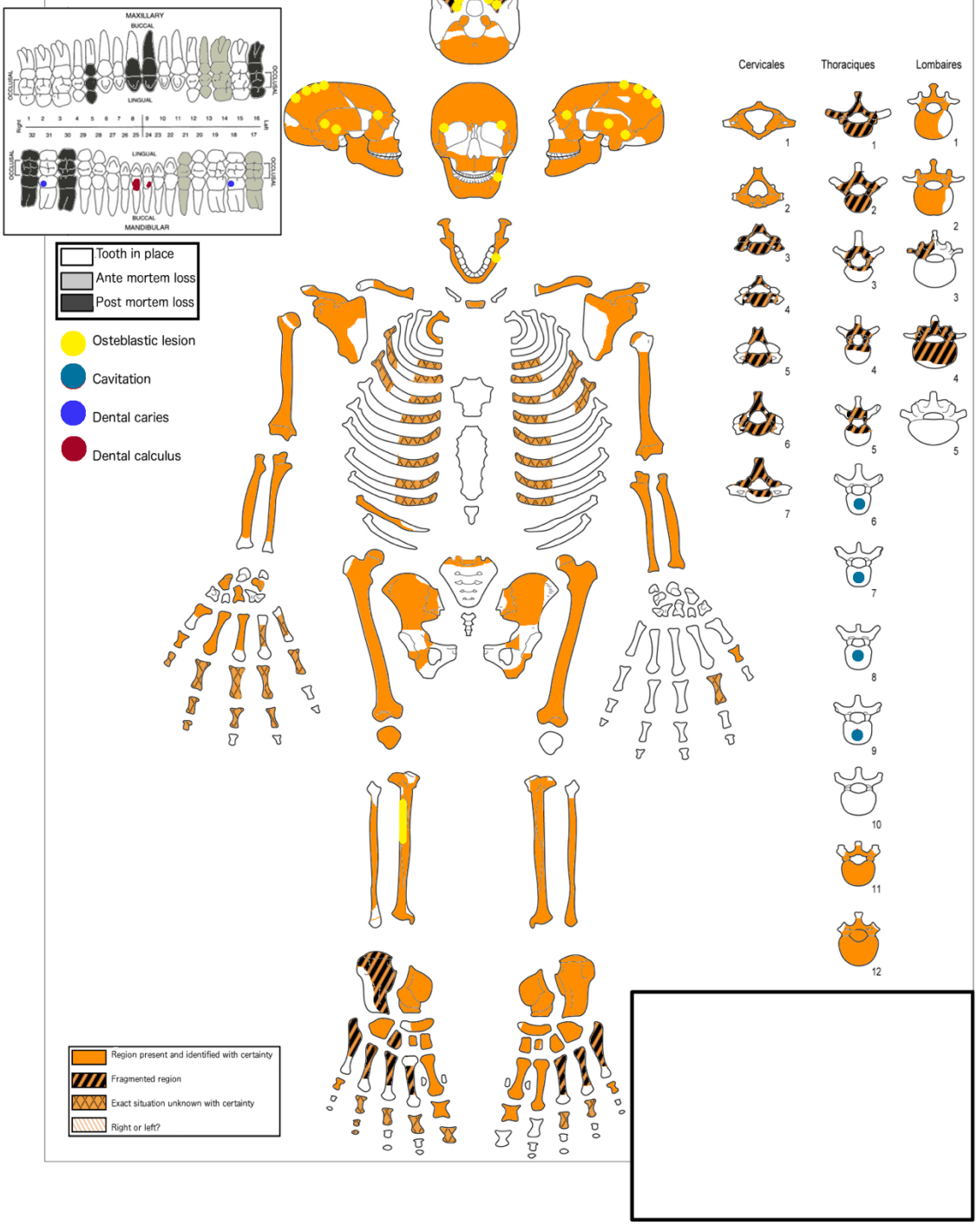
Description of pathological lesions: Orbital roof porosities on the right and left orbits (inferior view) (a), woven bone formations on the visceral side of rib fragments (b), cavitation on TV5 (inferior view) (c) and TV9 (superior view) (d), periosteal lesions on the diaphysis of the left femur (anterior view) (e) and left tibia (anterior view) (f).

Possible aetiologies: Infectious disease, B12 deficiency megaloblastic anaemia, iron-deficiency anaemia;

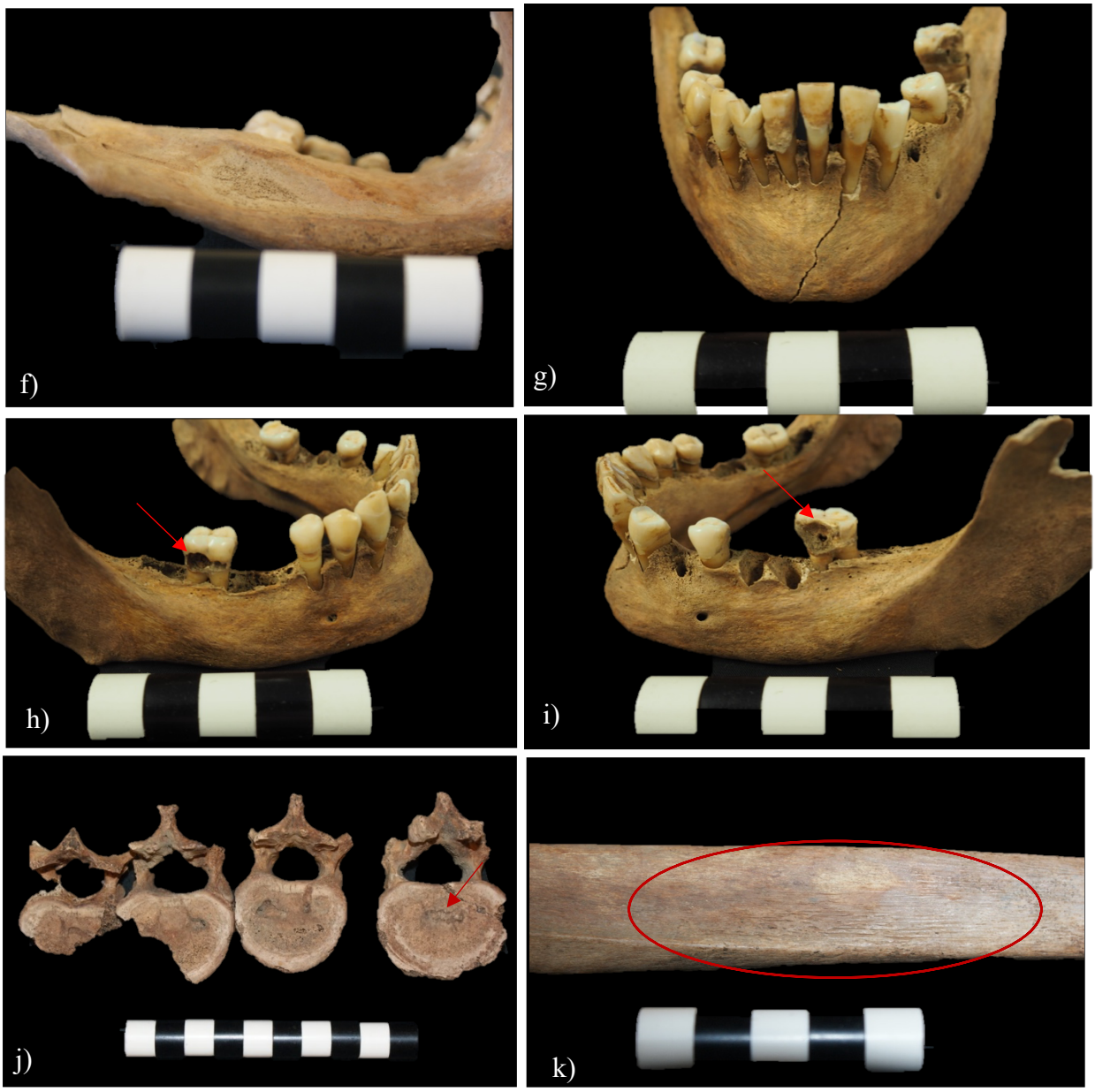
Differential diagnosis: Diagnostic of Schmorl's nodes, non-specific lung infection, *cribra orbitalia*.

Site BiFj-37 Box 10 / 18 No 17Z-S7 (1)

Sex Female tendency **Age-at-death** more than 40 years-old




a)
b)
c)
d))
e)



Individual: 17Z-S7

ACI: 52.34

Age-at-death: more than 40 years-old

Sex: Likely female

Description of pathological lesions: Capillary-like lesions bilaterally in the centre of the cranium (a), new bone formation on the medial face of right and left temporal bones (endocranial view)

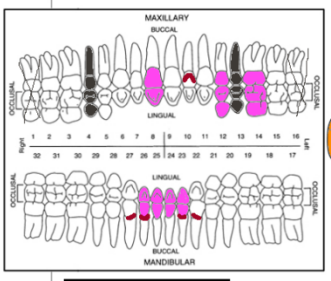
(b), on the right and left sphenoid's greater wings (posterior view) (c), the lateral sides of right and left orbits (inferior view)(d-e) and the left ramus of the mandibula (woven bone) (medial view) (f), calcified plaque on the lower incisors (buccal view) (g), cavity on right and left M2 (buccal view) (h-i), cavitation on four undetermined TV (inferior side) (j) and periosteal lesions on the lateral face of the diaphysis of the right tibia (lateral view) (k).

Possible aetiologies: Trauma, primary and secondary infections of the meninges, tumours, TB, syphilis and vitamin deficiencies of A, C and D.

Differential diagnosis: Diagnostic of endocranial lesions, dental calculus, dental caries, Schmorl's nodes, degenerative joint disease, **typical of** scurvy according to the distribution pattern of periosteal lesions and lytic lesions.

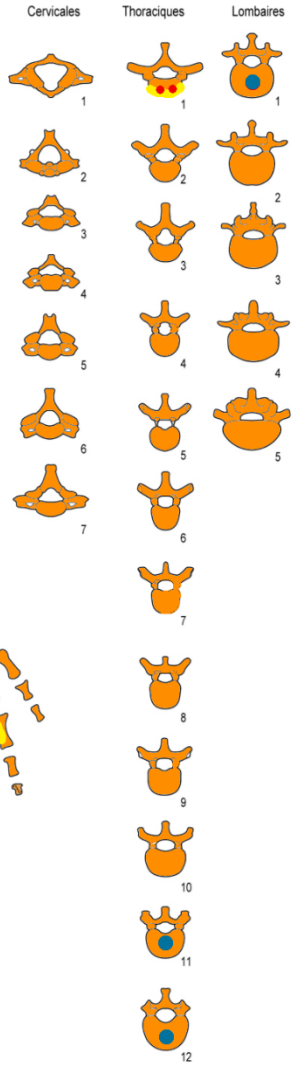
Site BiFj-37 Box 10 / 78 No 20A-S7

Sex Undetermined Age-at-death 20-29 years-old



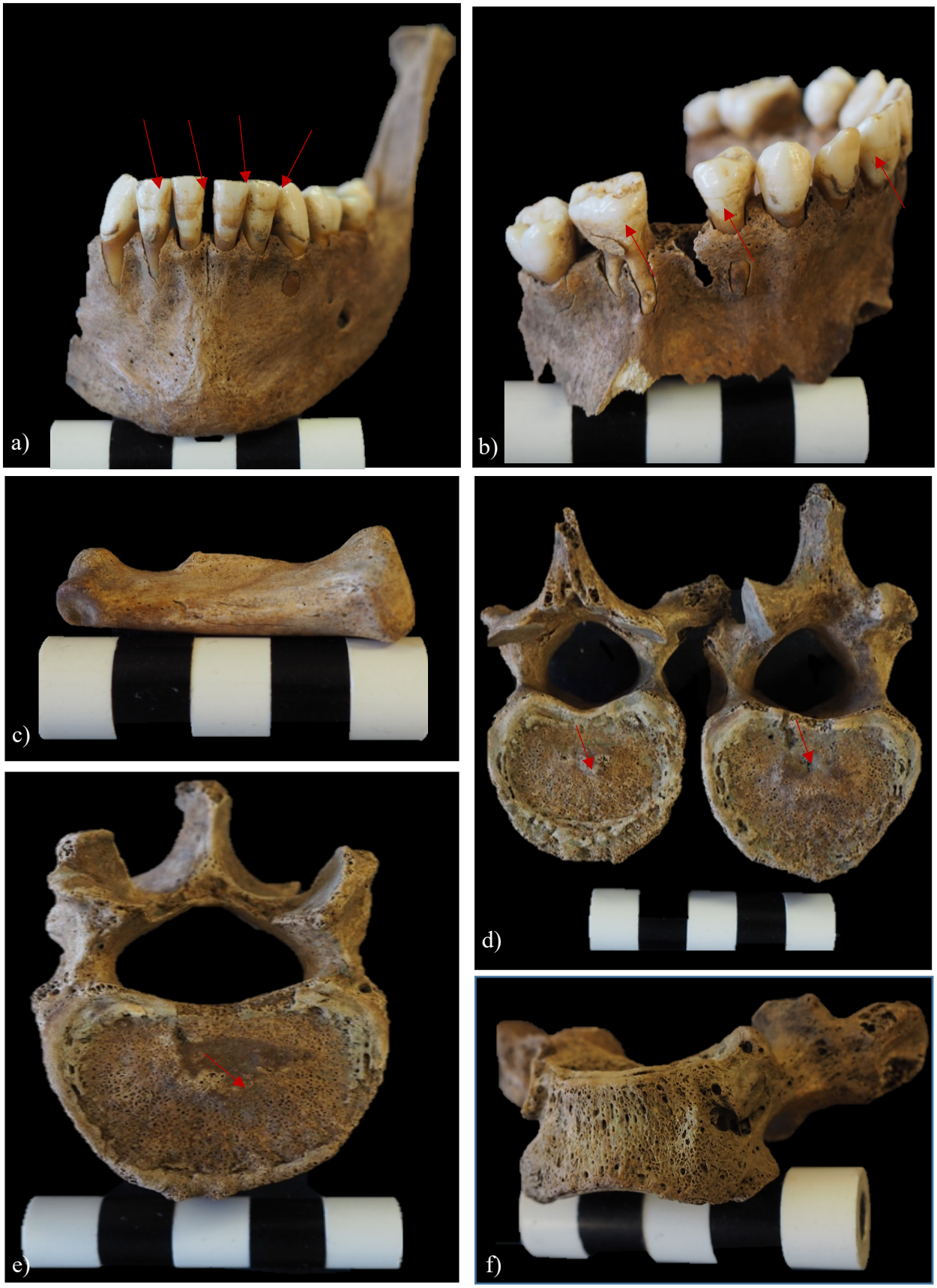
Tooth in place
Ante mortem loss
Post mortem loss
Mild dental hypoplasia
Osteoblastic lesion
Osteolytic lesion
Dental calculus
Cavitation

ACI 80.88



Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?

Commentaires :


a)
b)
c)
d)
e)
f)

Individual: 20A-S7

ACI: 86.92

Age-at-death:15-23 years-old

Sex: Likely male

Description of pathological lesions: Calcified plaque on lower and upper teeth (buccal view) (a-b) lines on the lower incisors (buccal view) (a) and superior left I^1, PM^1 and M^1 (buccal view) (b), healed fracture of a proximal hand phalanx (c), cavitation on TV11-12 (inferior view) (d) and LV1 (superior view) (e), new bone formation on a TV (1rst?) (anterior view) (f).

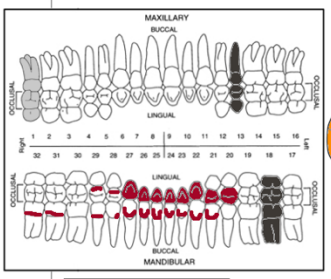
Possible aetiologies: Childhood disease-nutritional deficiencies, periapical inflammation or trauma to a deciduous tooth, fever, disease, endocrine dysfunction, infection during odontogenesis.

Differential diagnosis: Diagnostic of dental calculus, LEH, Schmorl's nodes, degenerative joint disease, trauma.

Site BiFj-37 Box 3 / 52 No 20A-S13

Sex Female Age-at-death 16-29 years-old

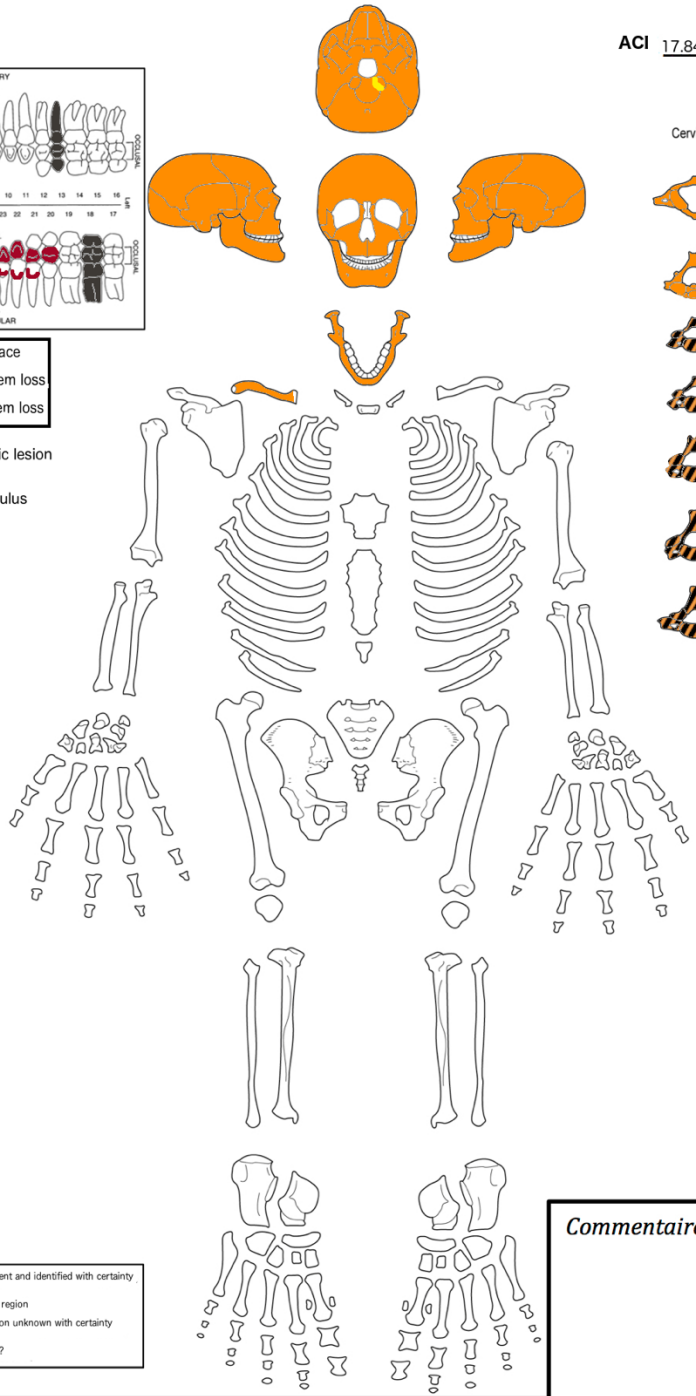
ACI 17.84

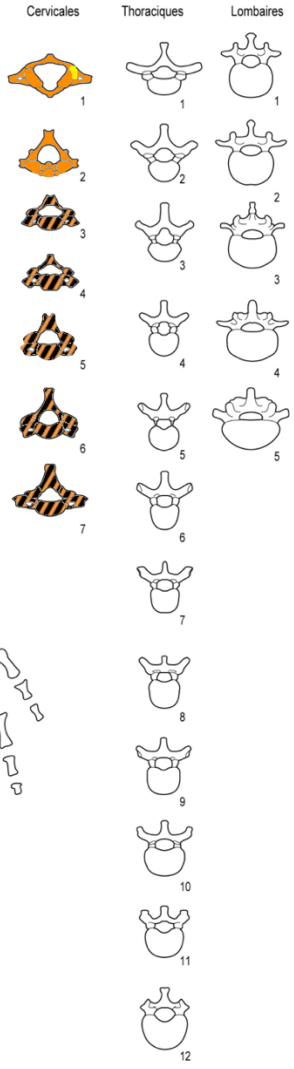


Tooth in place
Ante mortem loss
Post mortem loss

Osteoblastic lesion

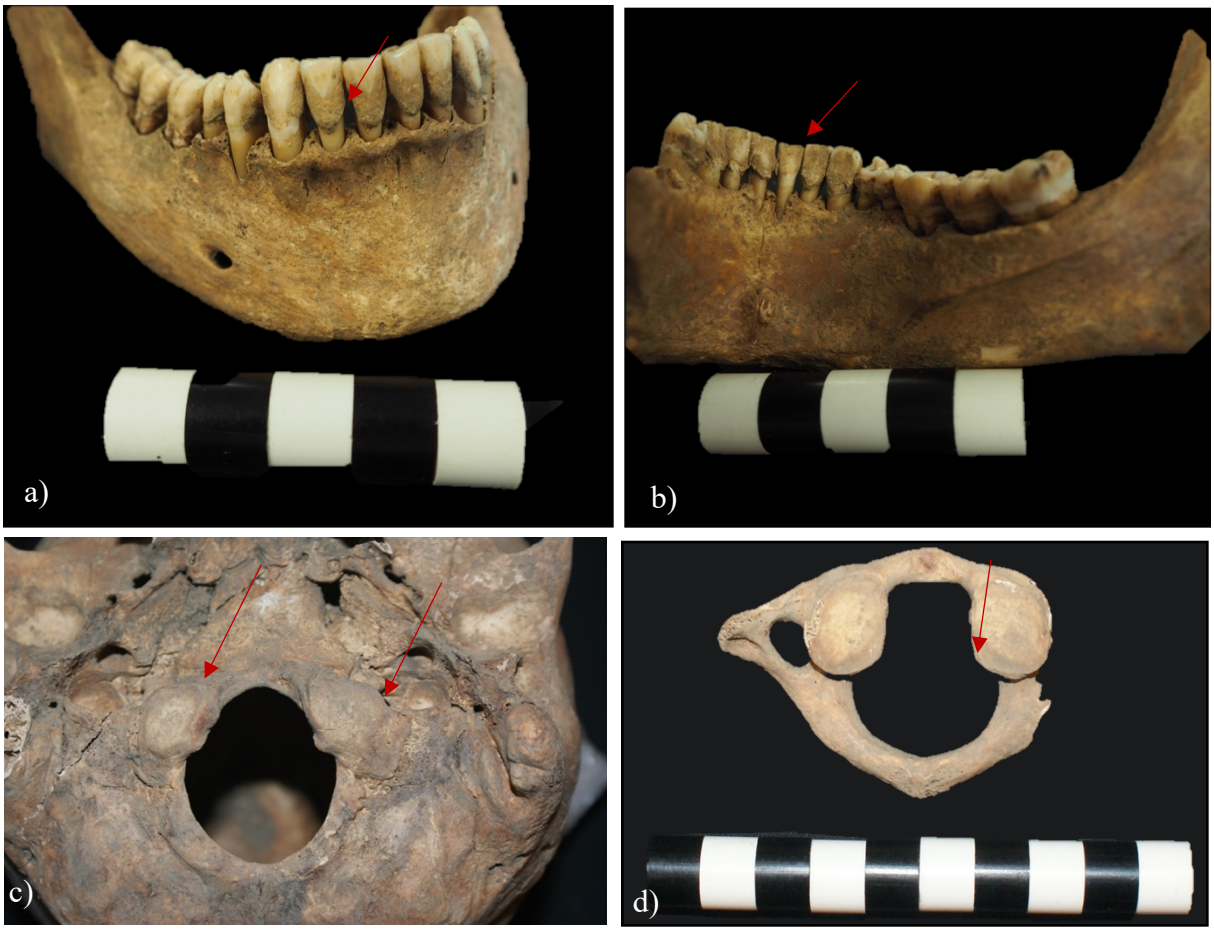
Dental calculus





Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?

Commentaires :



Individual: 20A-S13

ACI: 17.84

Age-at-death: 16-29 years-old

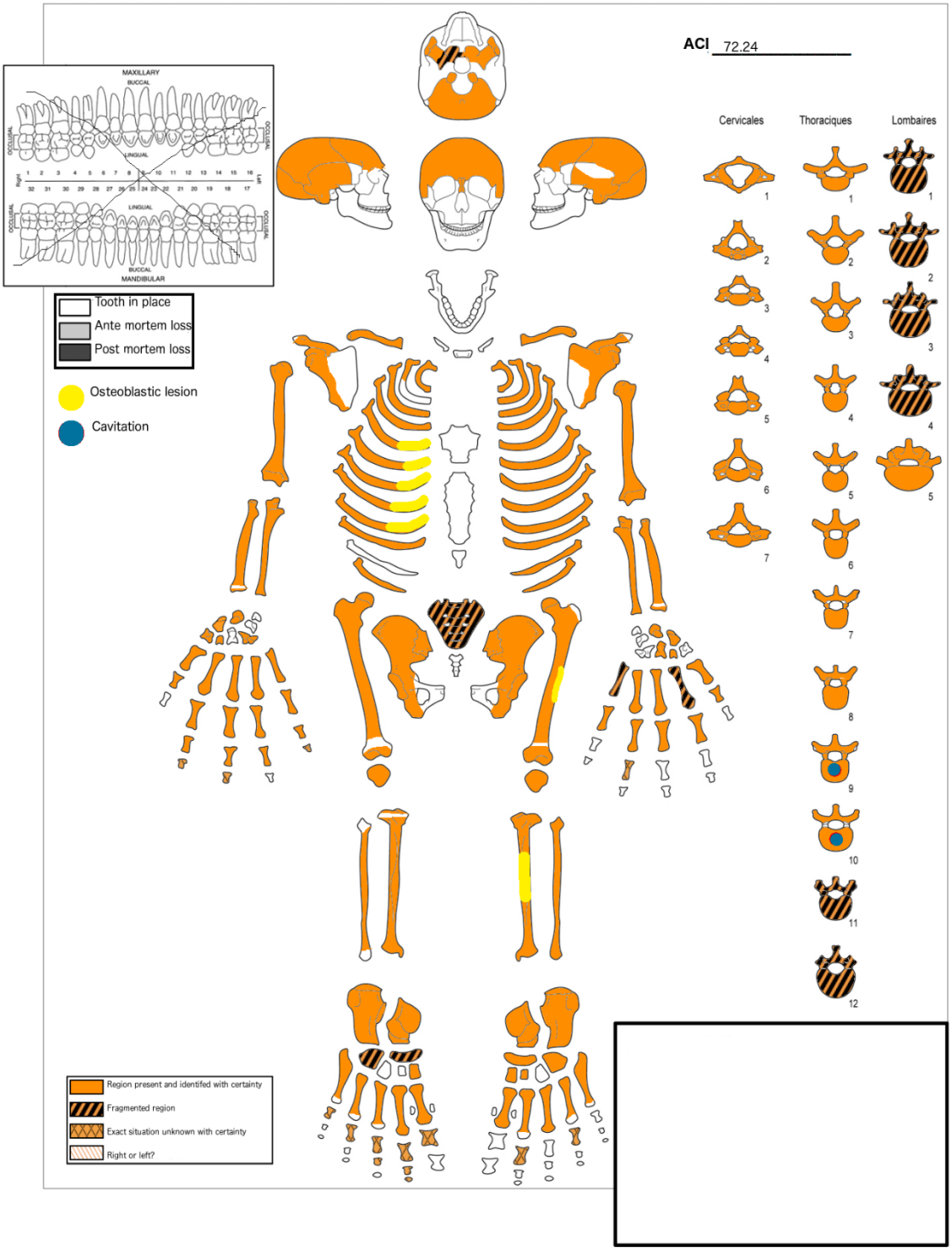
Sex: Female

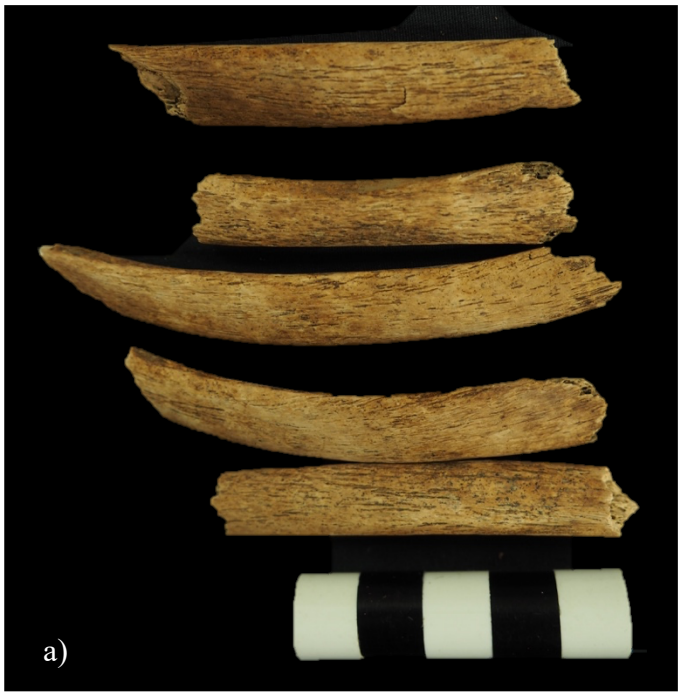
Description of pathological lesions: Calcified plaque on lower teeth (buccal view) (a) lingual view (b), extension of the left occipital condyle and shrinking (inferior view) (c), small bone formation on the left condyle of the atlas (superior view) (d).

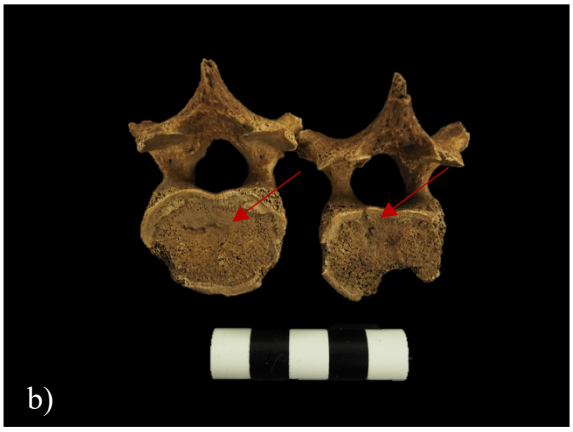
Differential diagnosis: **Diagnostic of** dental calculus, osteophyte, **highly consistent with** degenerative joint disease, unilateral right aplasia and unilateral left hypoplasia.

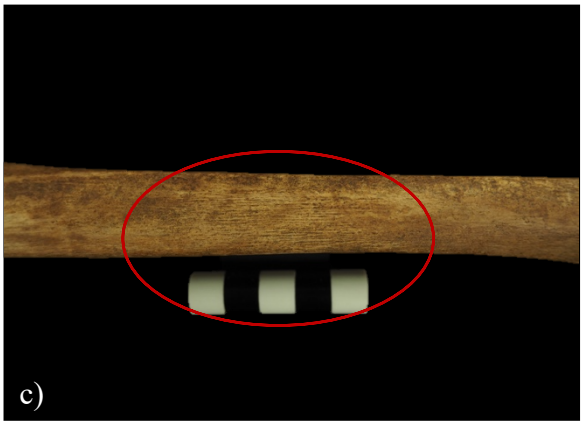
Site BiFj-37 Box 32 / 78 No 20C-S6

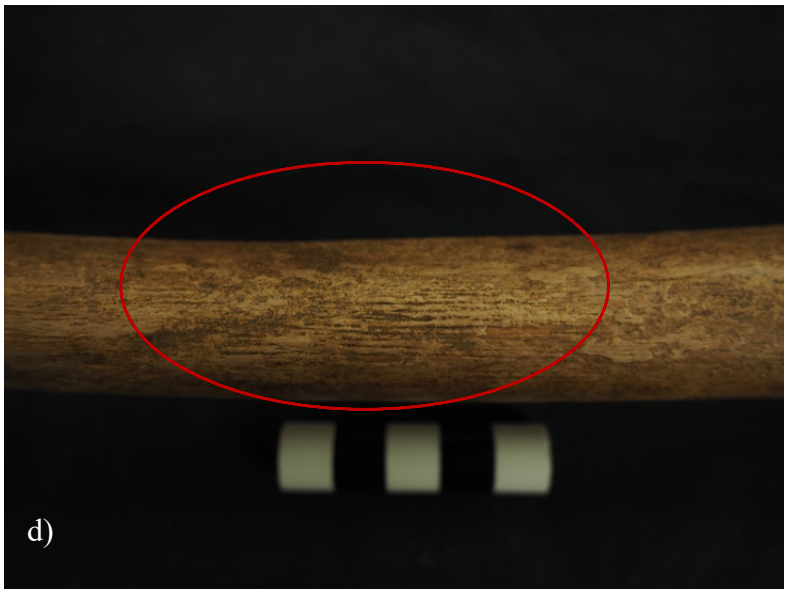
Sex Female tendency Age-at-death 20-29 years-old




a)


b)


c)


d)

Individual: 20C-S6

ACI: 77.24

Age-at-death: 20-29 years-old

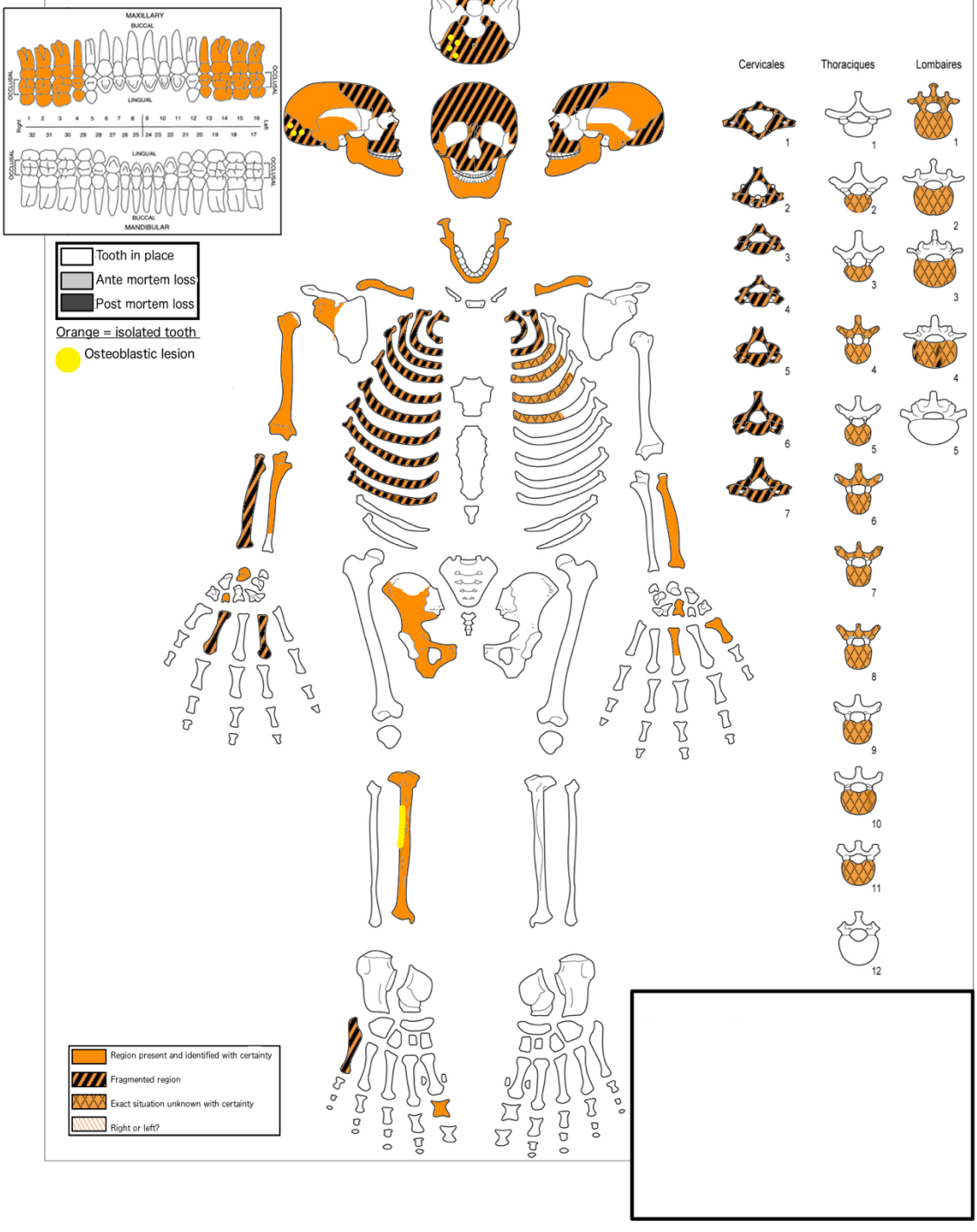
Sex: Likely female

Description of pathological lesions: New bone formation on ribs (lateral view) (a), cavitation on TV9-10 (inferior face) (b), periosteal lesions on the lateral side of left tibia (c) and femur (d) (lateral view).

Differential diagnosis: Diagnostic of Schmorl's nodes, **consistent with** trauma, lung infection, degenerative joint disease.

Site BiFj-37 Box 7 / 52 No 20C-S23

Sex Female Age-at-death 20-30 years-old



Individual: 20C-S23

ACI: 26.28

Age-at-death: 20-30 years-old

Sex: Female

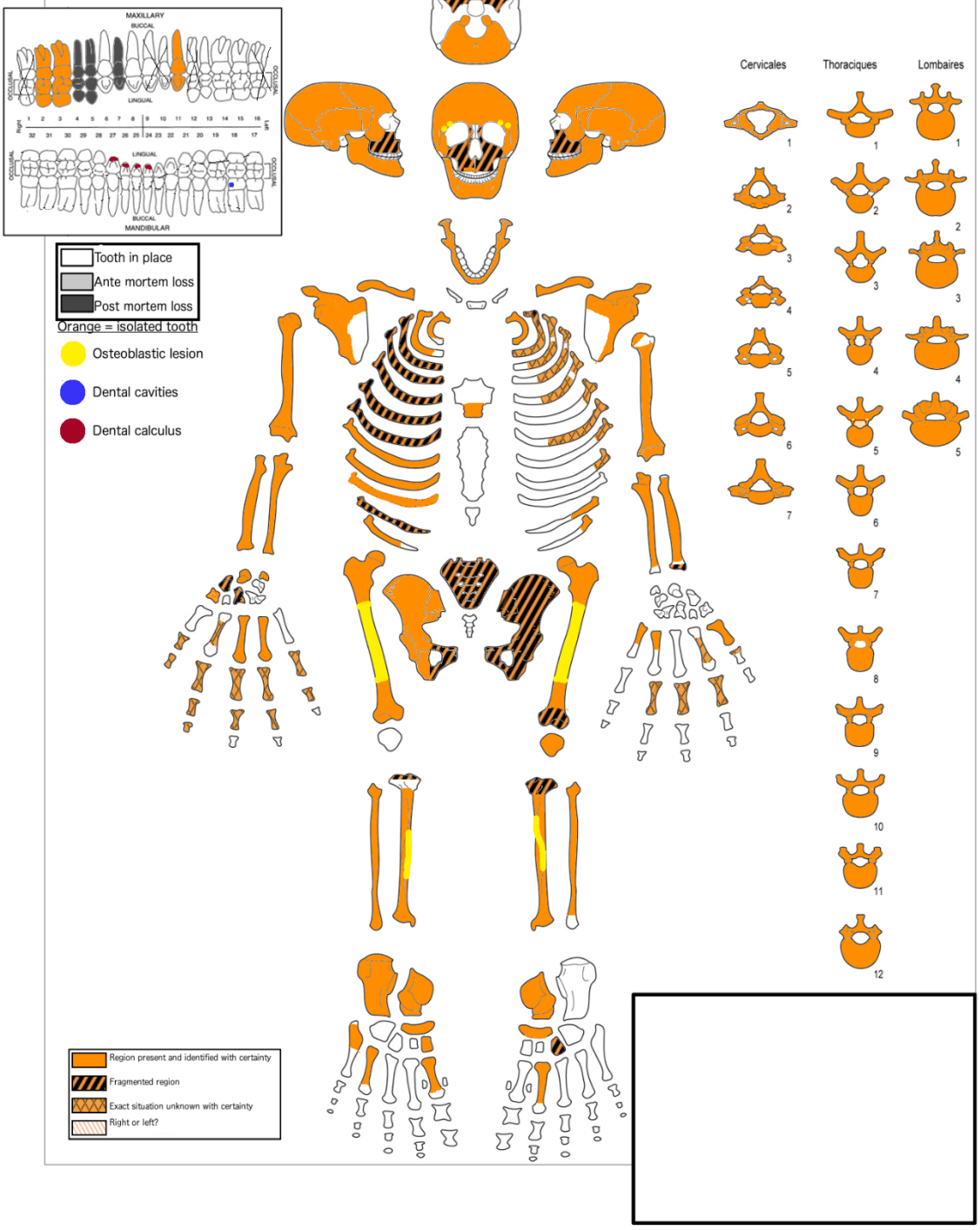
Description of pathological lesions: Endocranial new bone formation (woven) on the occipital bone (a), periosteal lesion on the medial face of the right tibia (medial view) (b).

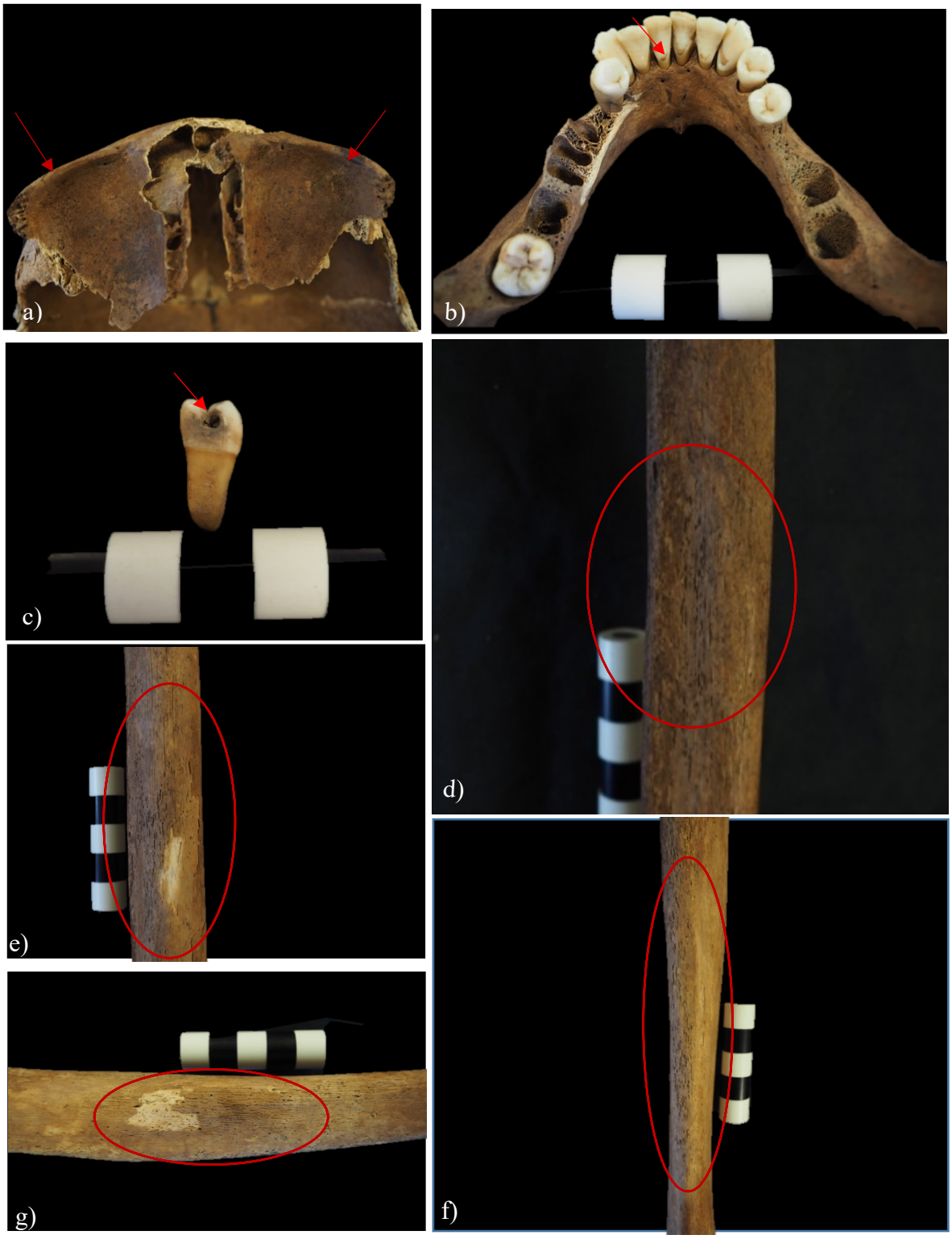
Possible aetiologies: Trauma, primary and secondary infections of the meninges, tumours, TB, syphilis and vitamin deficiencies of A, C and D.

Differential diagnosis: Diagnostic of endocranial lesions.

Site BiFi-37 Box 30 / 78 No 20D-S14

Sex Female tendency **Age-at-death** 17-20 years-old




a)
b)
c)
d)
e)
f)
g)

Individual: 20D-S14

ACI: 57.32

Age-at-death: 17-20 years-old

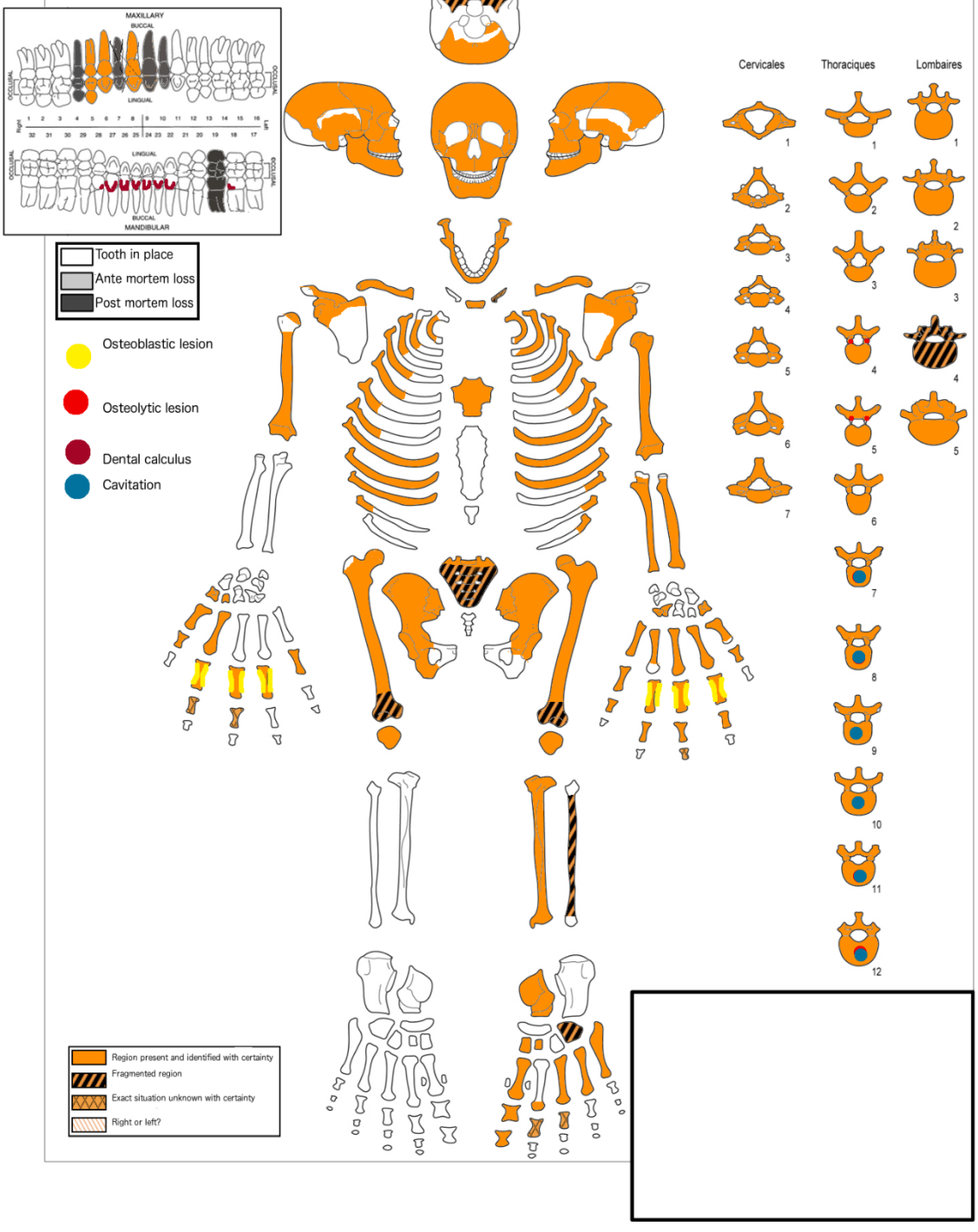
Sex: Likely female

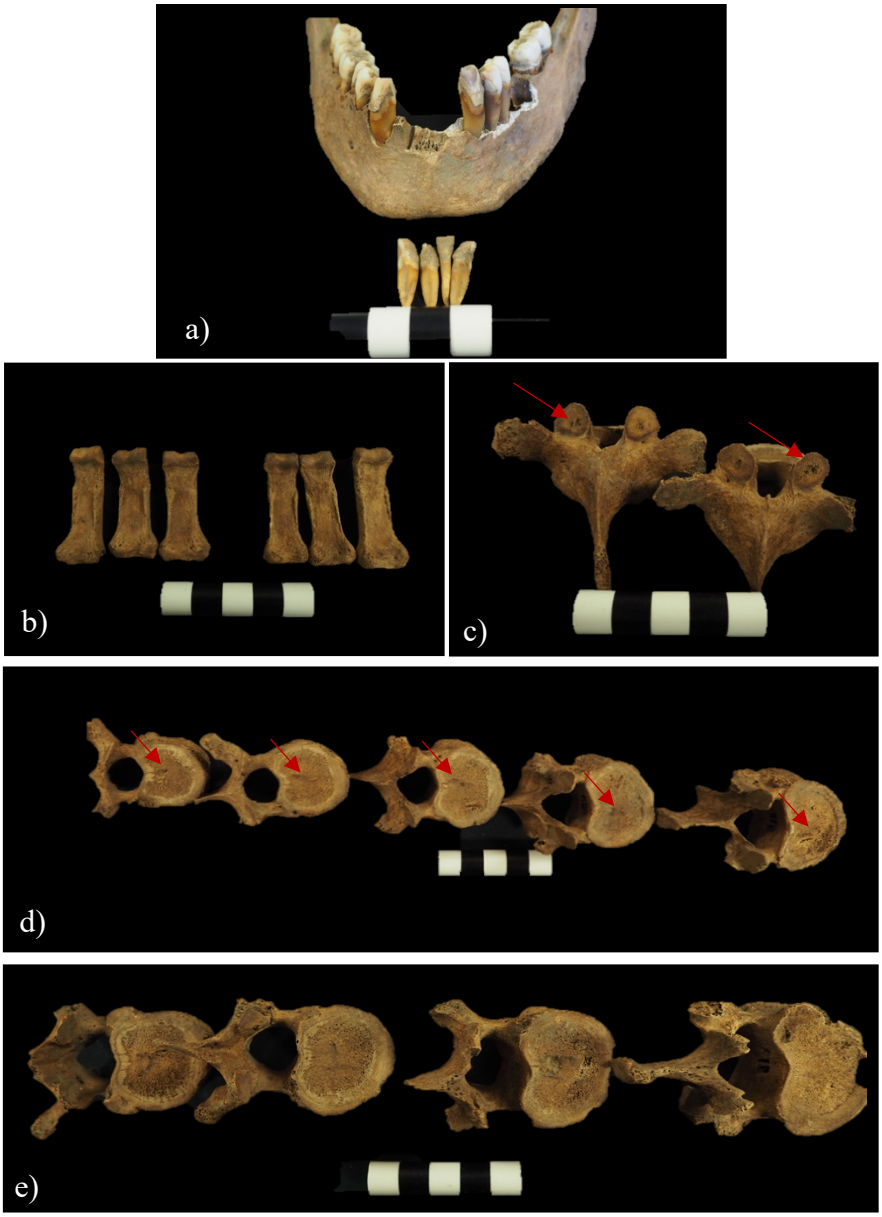
Description of pathological lesions: New bone formation and orbital roof porosities on lateral sides of orbits (inferior view) (a), calcified plaque on lower incisors and canines (b), cavity on left M_2 (mesial view) (c), periosteal lesions on the diaphysis of the right femur (medial view) (d), left femur (medial view) (e), left tibia posterior view) (f) and right tibia (medial view) (g).

Differential diagnosis: Diagnostic of dental calculus, dental caries and *cribra orbitalia*, **typical of** non-specific infection.

Site BiFj-37 Box 12 / 78 No 20E-S3

Sex Male tendency Age-at-death 20-29 years-old





Individual: 20E-S3

ACI: 56.76

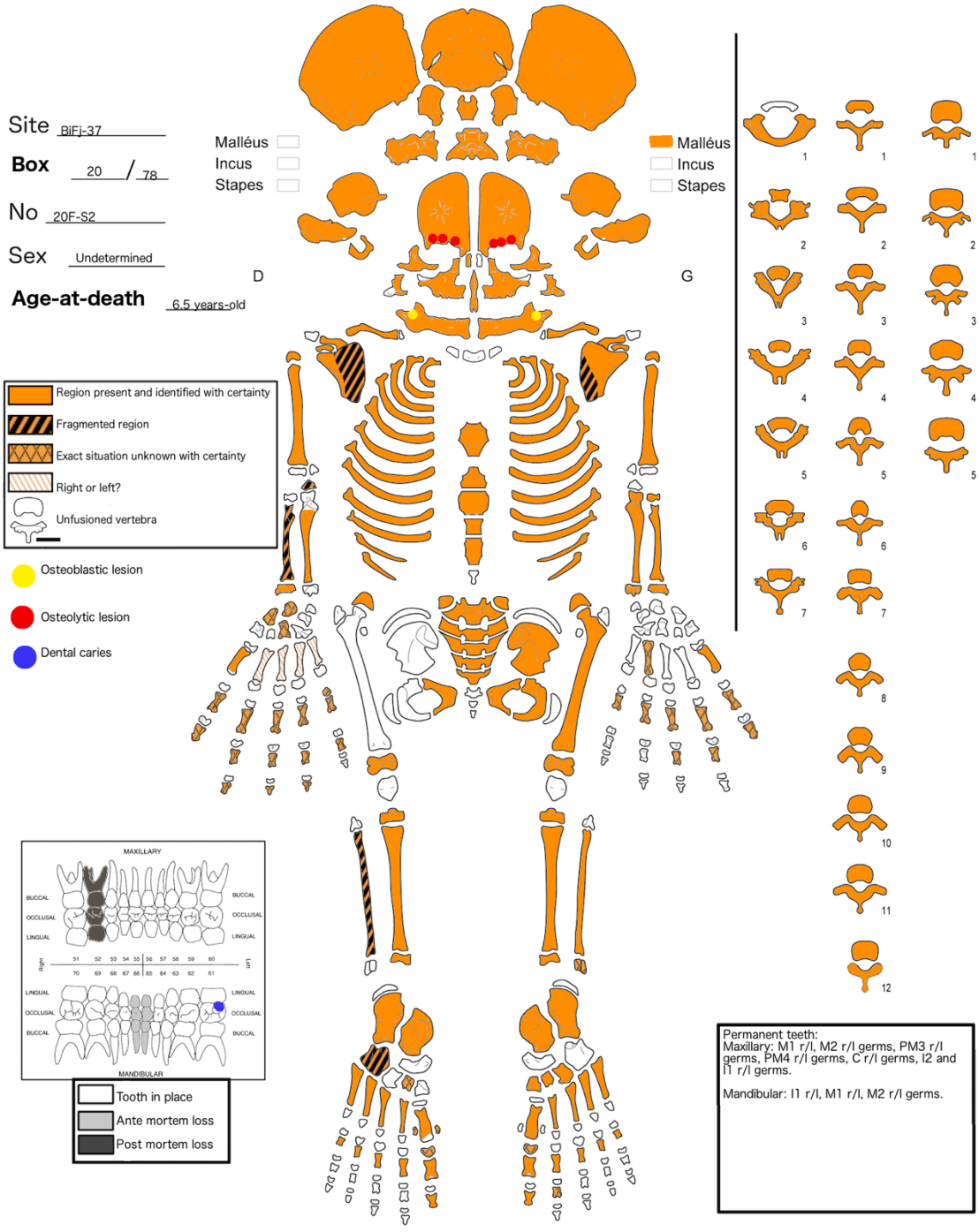
Age-at-death: 20-29 years-old

Sex: Likely Male

Description of pathological lesions: Calcified plaque on lower teeth (buccal view) (a) phalangeal interosseous new bone formations (palmar view) (b), lytic lesions on articular facets of two TV

(posterior view) (c), cavitation on TV 7,8,10,11,12 (superior view) (d) and TV 8,9,10,12 (inferior view) (e).

Differential diagnosis: Diagnostic of dental calculus, Schmorl's nodes, degenerative joint disease, **highly consistent with** osteoarthritis.


Site BiFj-37
Box 20 / 78
No 20F-S2
Sex Undetermined
Age-at-death 6.5 years-old
Malléus
Incus
Stapes
D
G
Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?
Unfusioned vertebra
Osteoblastic lesion
Osteolytic lesion
Dental caries
MAXILLARY
MANDIBULAR
BUCCAL
OCCLUSAL
LINGUAL
Tooth in place
Ante mortem loss
Post mortem loss
Permanent teeth:
Maxillary: M1 r/l, M2 r/l germs, PM3 r/l germs, PM4 r/l germs, C r/l germs, I2 and I1 r/l germs.
Mandibular: I1 r/l, M1 r/l, M2 r/l germs.

Individual: 20F-S2

ACI: N.A.

Age-at-death: 6.5 years-old

Sex: N.A.

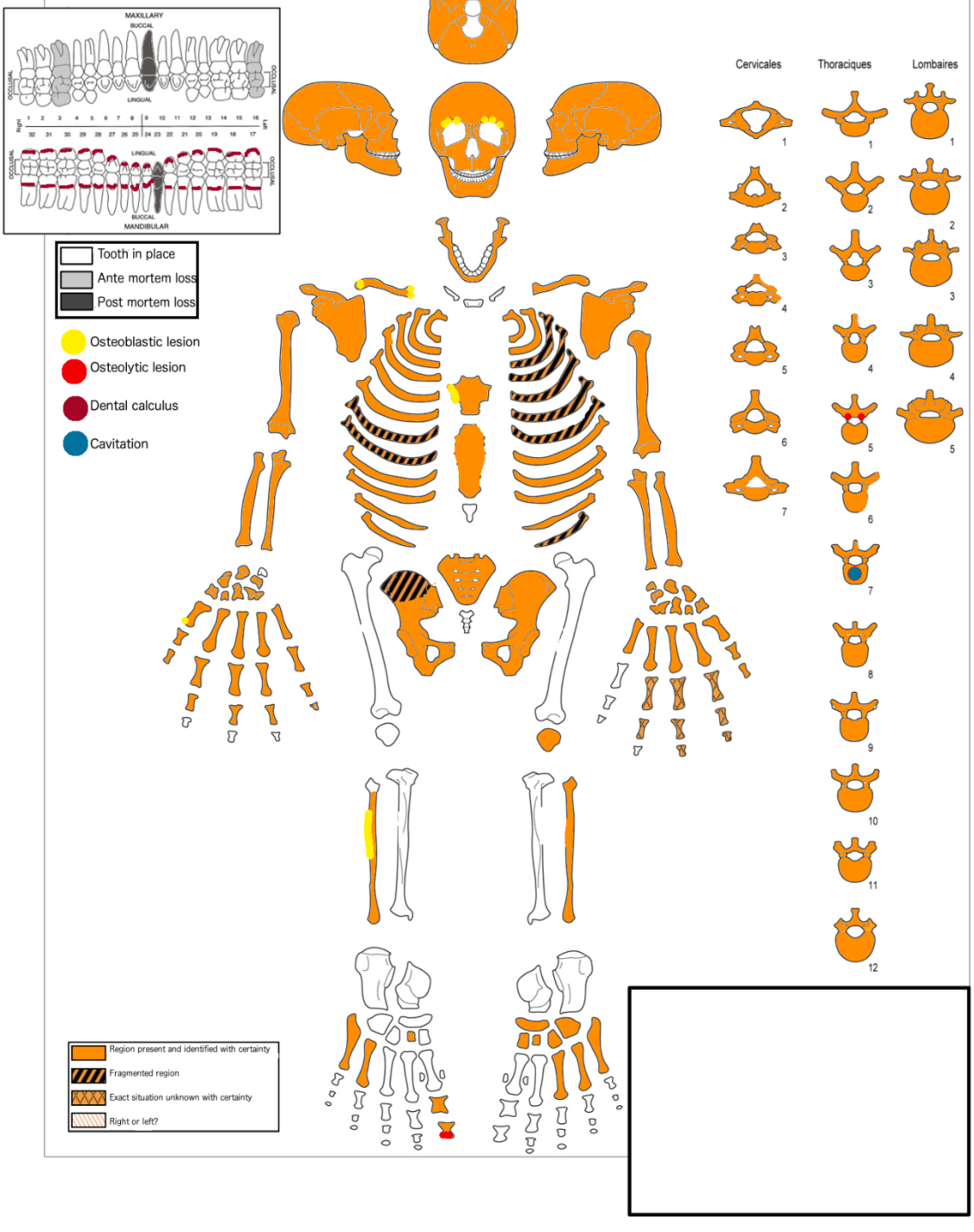
Description of pathological lesions: Orbital roof porosities in both orbits (inferior view) (a), new bone formation on the left (b) and right (c) mandibular ramus (medial view); cavity on the 2nd left decidual molar (occlusal view) (d).

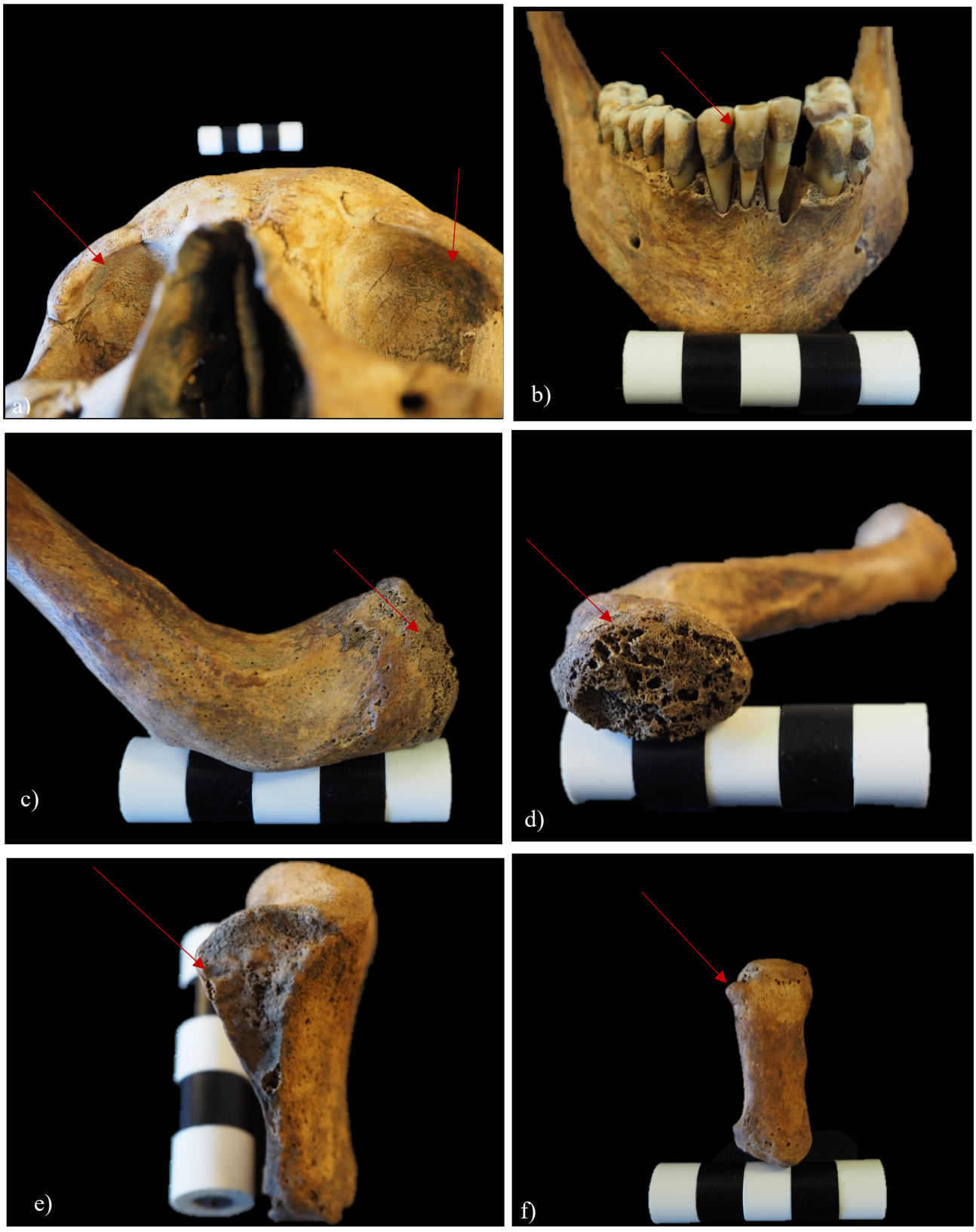
Possible aetiologies: B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

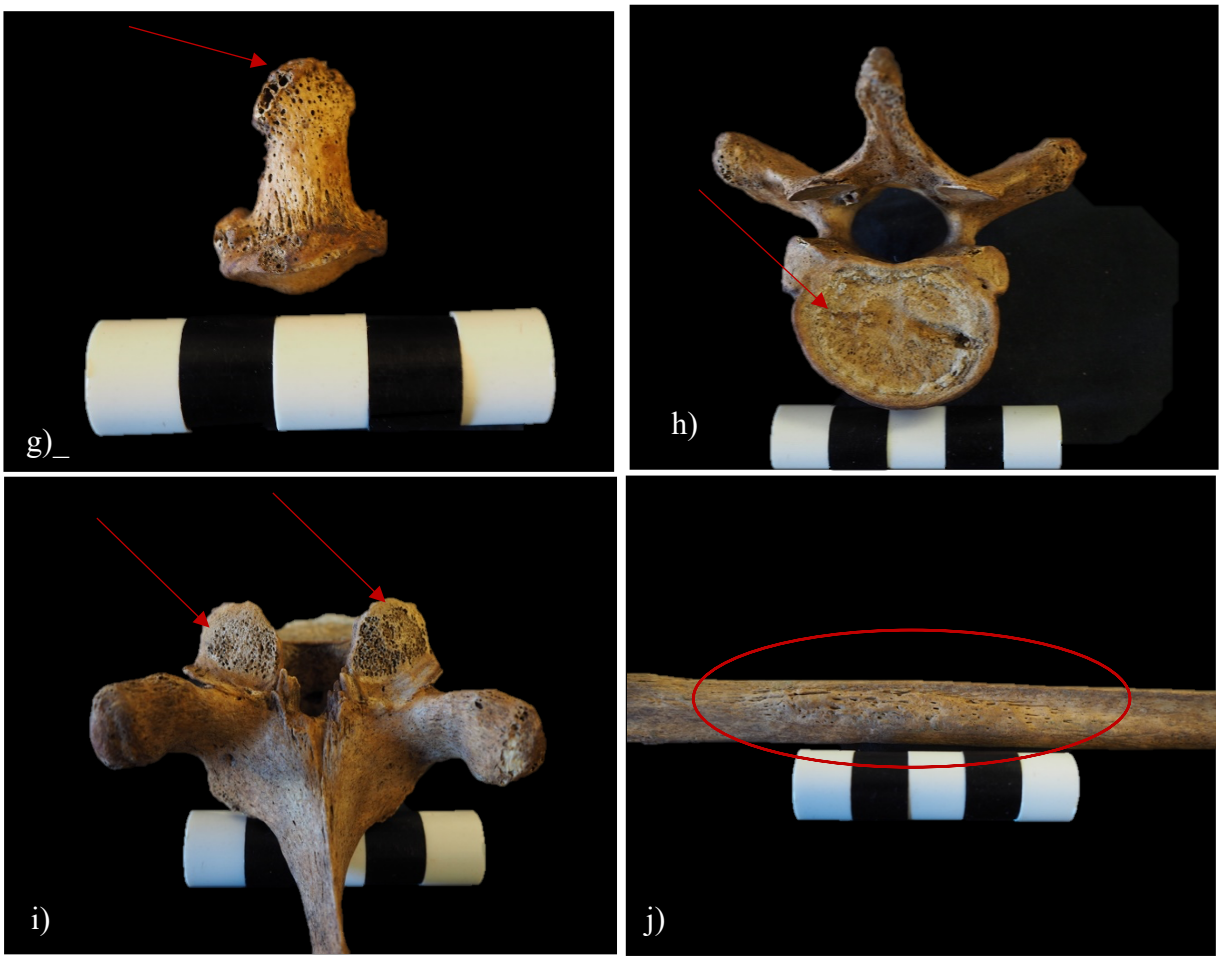
Differential diagnosis: Diagnostic of dental calculus, dental caries and *cribra orbitalia*. **Highly consistent** with scurvy according to the distribution of the lytic and osteoblastic lesions.

Site BiFj-37 Box 30 / 78 No 20F-S10

Sex Male Age-at-death Approximatively 30 years-old




a)
b)
c)
d)
e)
f)



Individual: 20F-S10

ACI: 79.44

Age-at-death: Approximately 30 years-old

Sex: Male

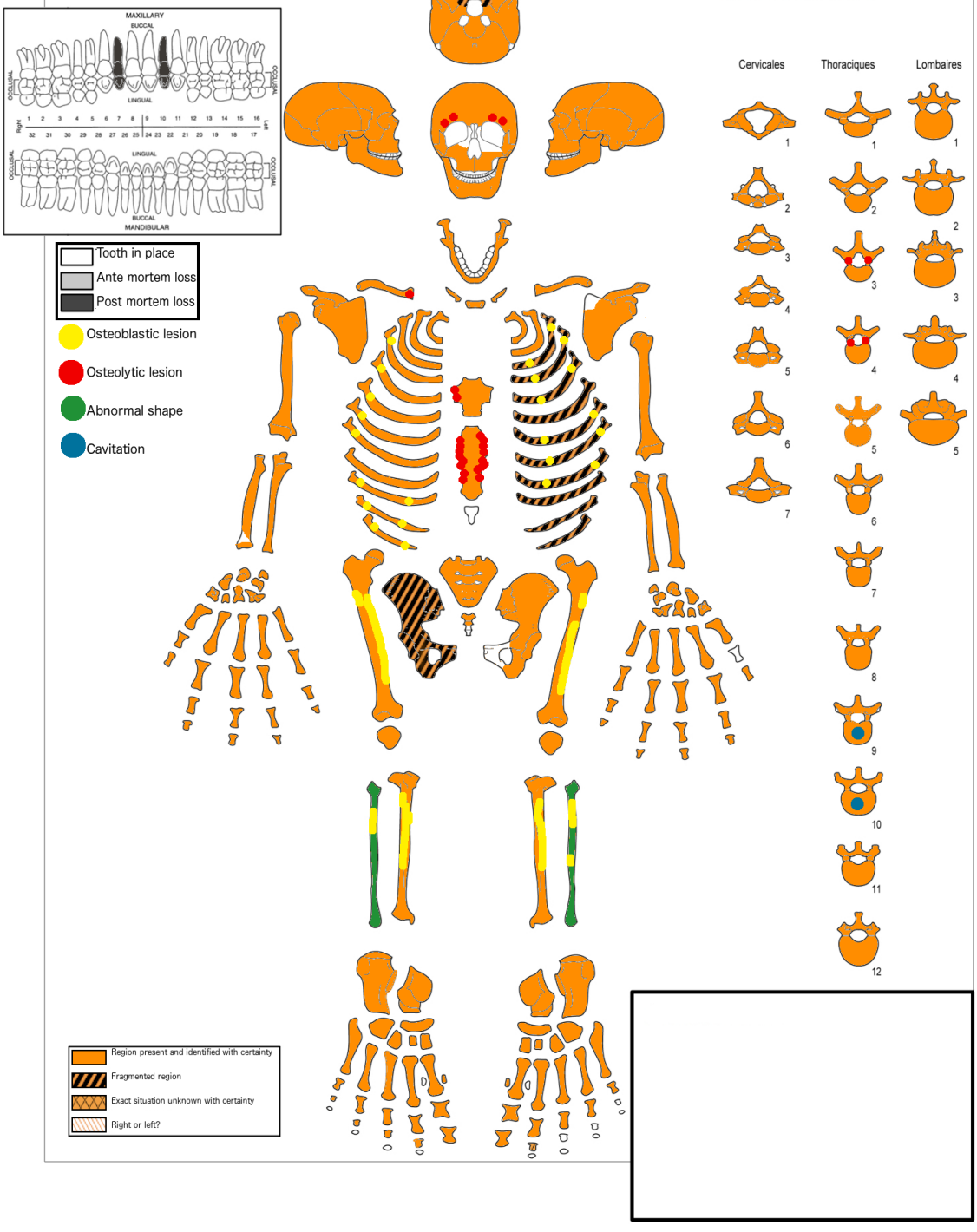
Description of pathological lesions: New bone formation on both orbits (inferior view) (a), calcified plaque on the buccal and lingual sides of lower teeth (buccal view) (b), new bone formation on the lateral end of the right clavicle (superior view) (c), lytic lesions on the sternal end of the right clavicle (medial view) (d), new bone formation on the right clavicular articular facet of the sternum (lateral view) (e), bone nodule on the lateral side of the head of the right MC1 (dorsal view) (f), lytic lesions on the distal part of a first distal foot phalanx (dorsal view) (g), cavitation

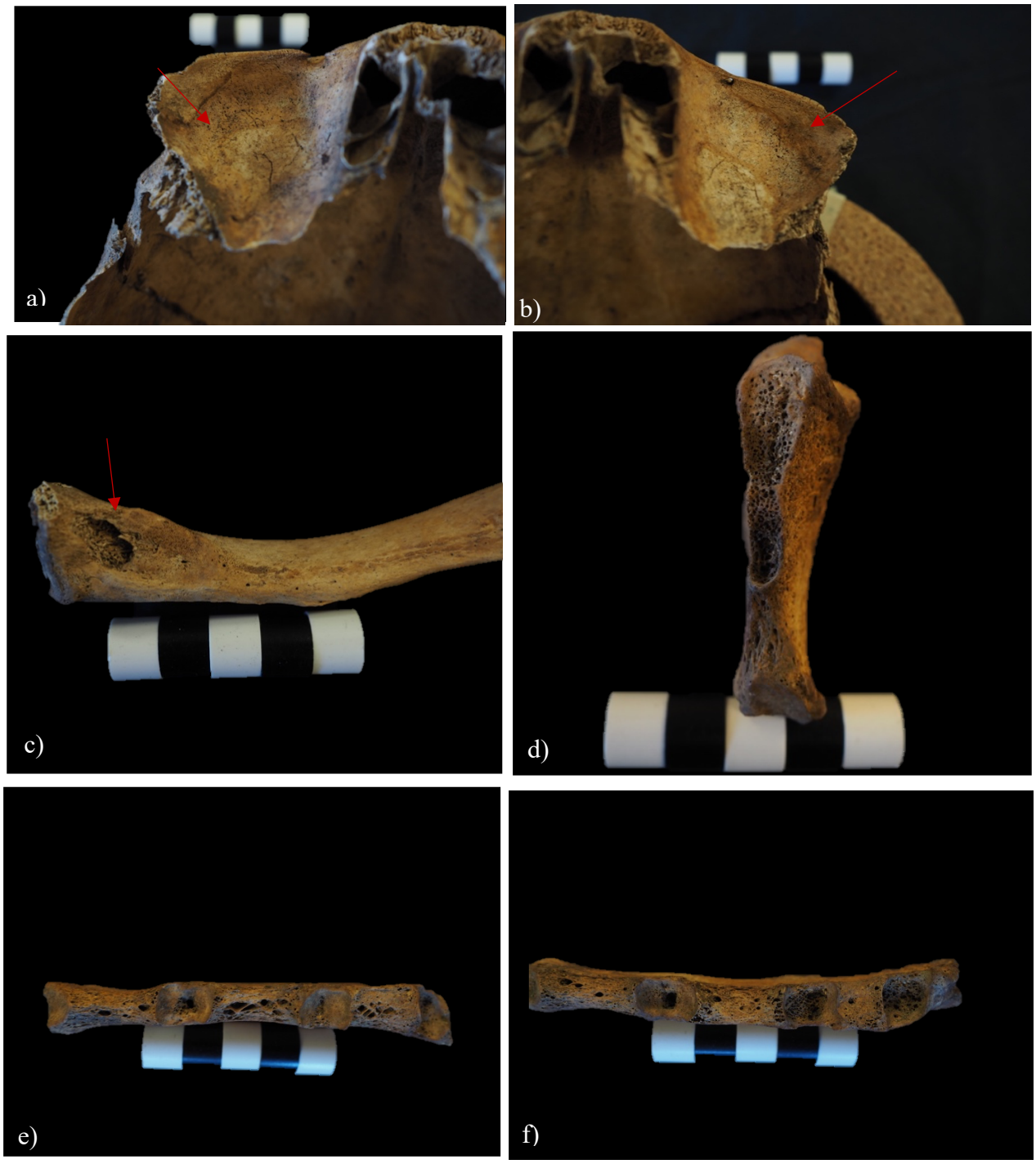
onTV7 (inferior view) (h), lytic lesions on both superior articular facets of TV5 (posterior view) (i) and periosteal lesions on the diaphysis of the right fibula (lateral view) (j).

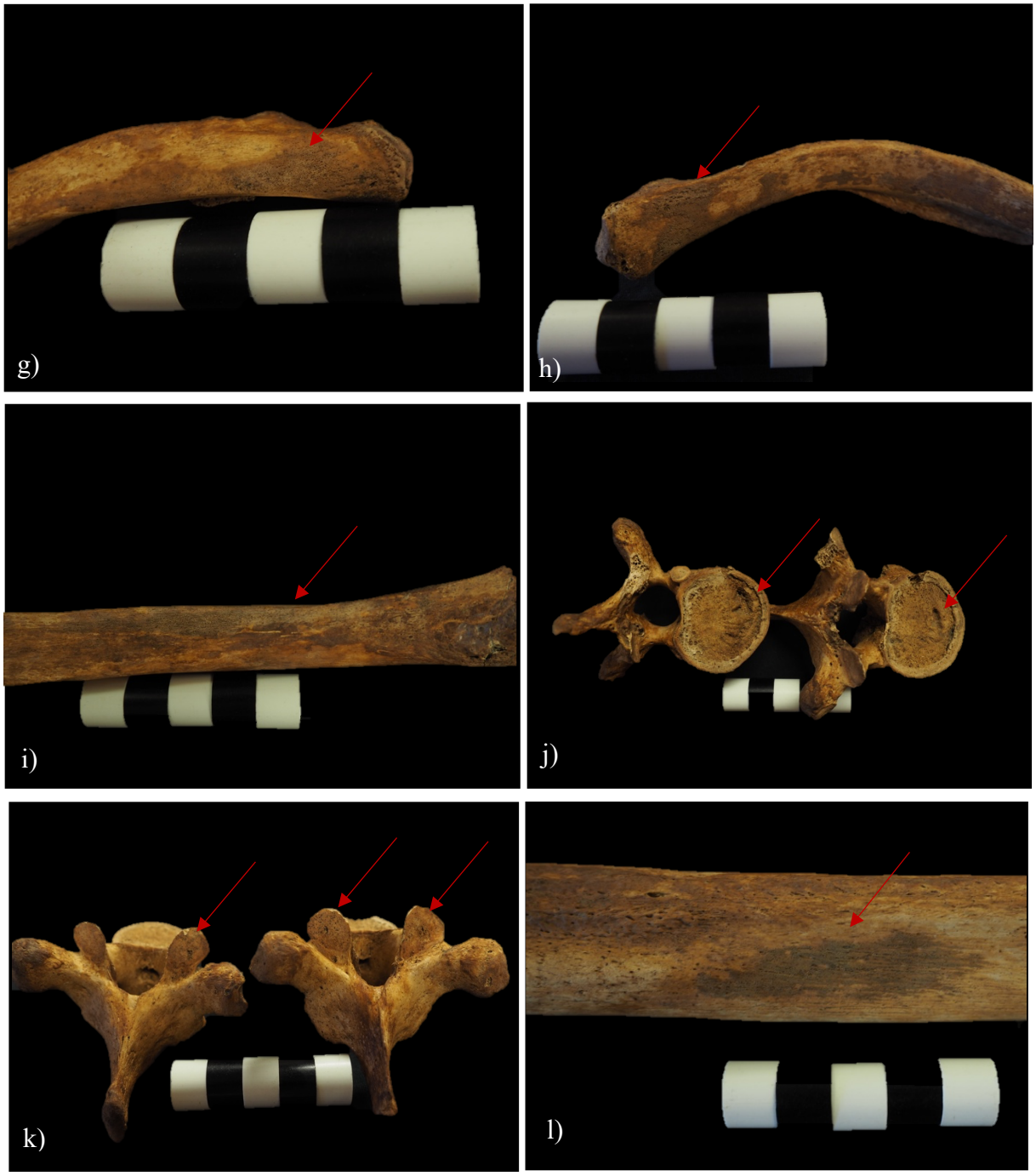
Differential diagnosis: Diagnostic of dental calculus, Schmorl's nodes, degenerative joint disease, osteoarthritis.

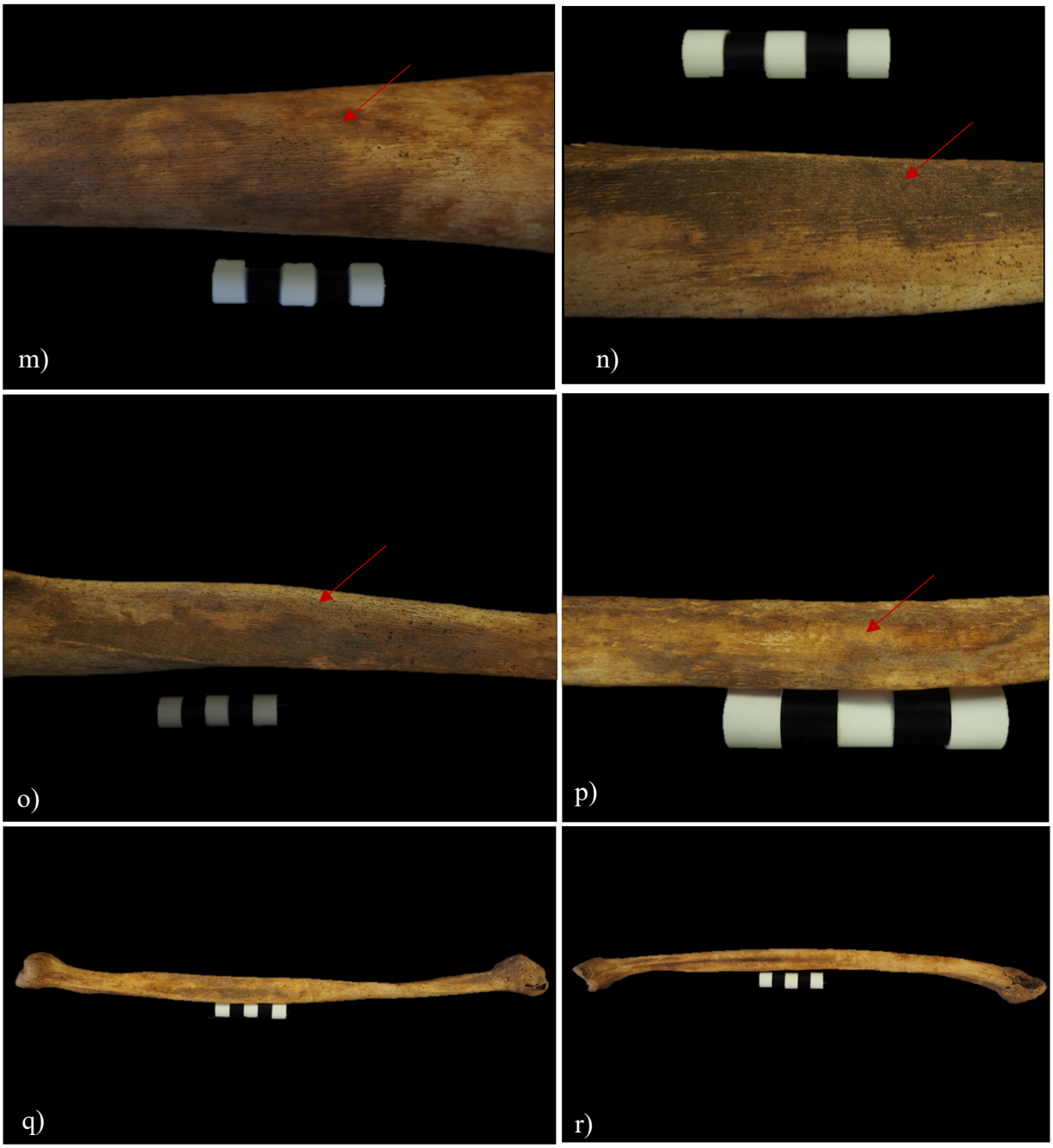
Site BiFj-37 Box 9 / 78 No 20F-S22

Sex Male **Age-at-death** 19-20 years-old




a)
b)
c)
d)
e)
f)


g)
h)
i)
j)
k)
l)


m)
n)
o)
p)
q)
r)

Individual: 20F-S22

ACI: 95.53

Age-at-death: 19-20 years-old

Sex: Male

Description of pathological lesions: Orbital roof porosities on both orbits (inferior view) (a) (b), lytic lesion on the inferior side of the right clavicle's metaphysis (cyst) (c), lytic lesions on the right clavicular articulation of the sternum (lateral view) (d), lytic lesions on the right and left costal articular surfaces of the sternum (lateral view) (e) (f), woven bone on the visceral face of the head of the 4^{rth} right rib (g), woven bone on the visceral face of the 7th left rib (h) (i), cavitation on the bodies of TV9 (inferior view) and TV10 (superior view) (j), lytic lesions on the superior articular facets of TV3 and 4 (posterior view) ((k), periosteal lesions on the left and right femur (anterior view) (l) (m), on the right and left tibia (lateral view) (n) (o), on the left fibula (medial view) (p) and on the right fibula (lateral view) (q), bending of the right (lateral view) (q) and left fibulas' diaphysis (lateral view) (r), slight bending of right and left tibias, the left tibia slightly shorter than the right (370 mm (left), 376 mm (right)) (s).

Possible aetiologies: B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

Differential diagnosis: Diagnostic of Schmorl's nodes, osteoarthritis, degenerative joint disease, *cribra orbitalia*, **typical of** residual rickets, empyema, actinomycosis, or pulmonary TB with haematogenous spread of infection, a combination of respiratory disease and other osteolytic disease.

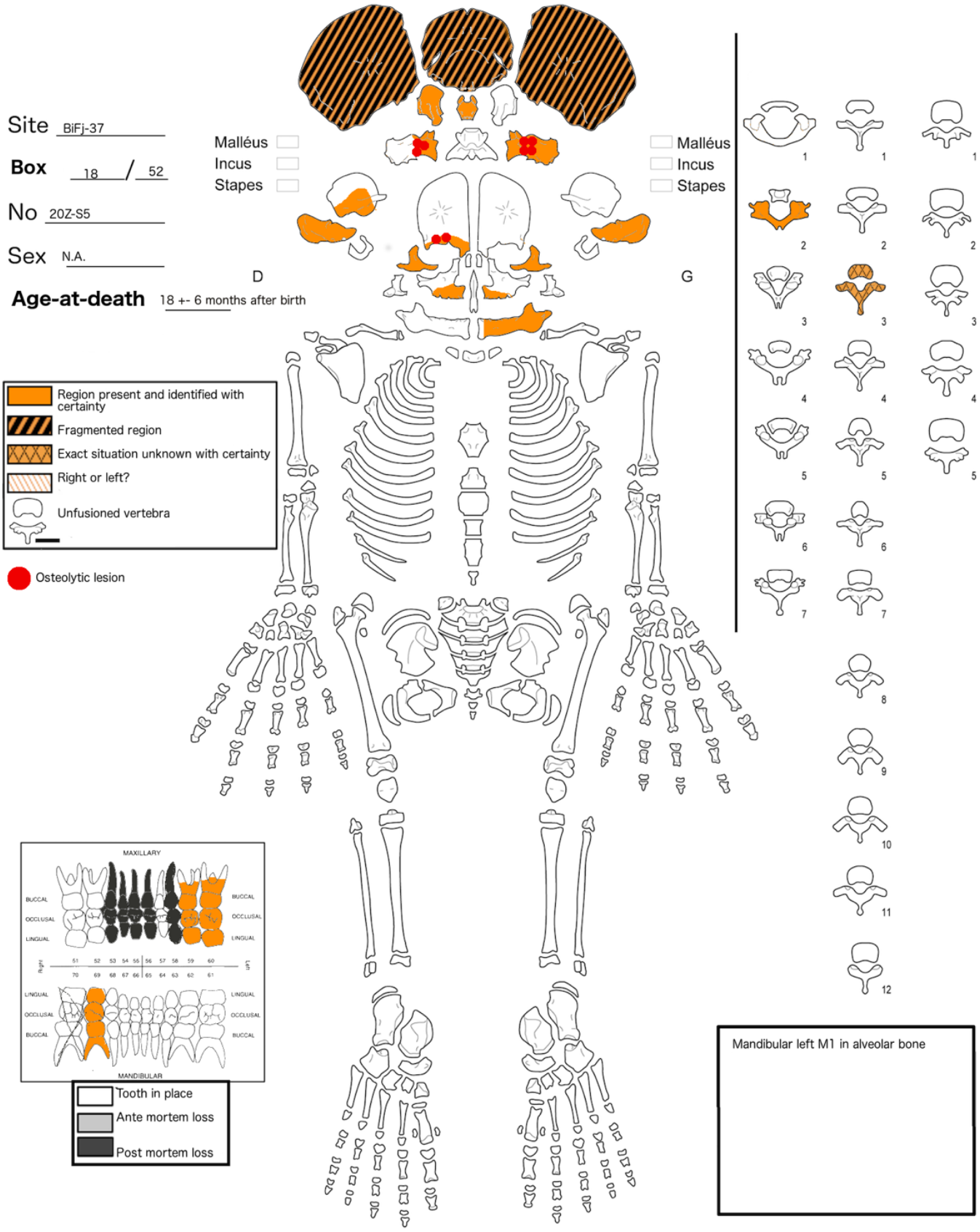
Site BiFj-37

Box 18 / 52

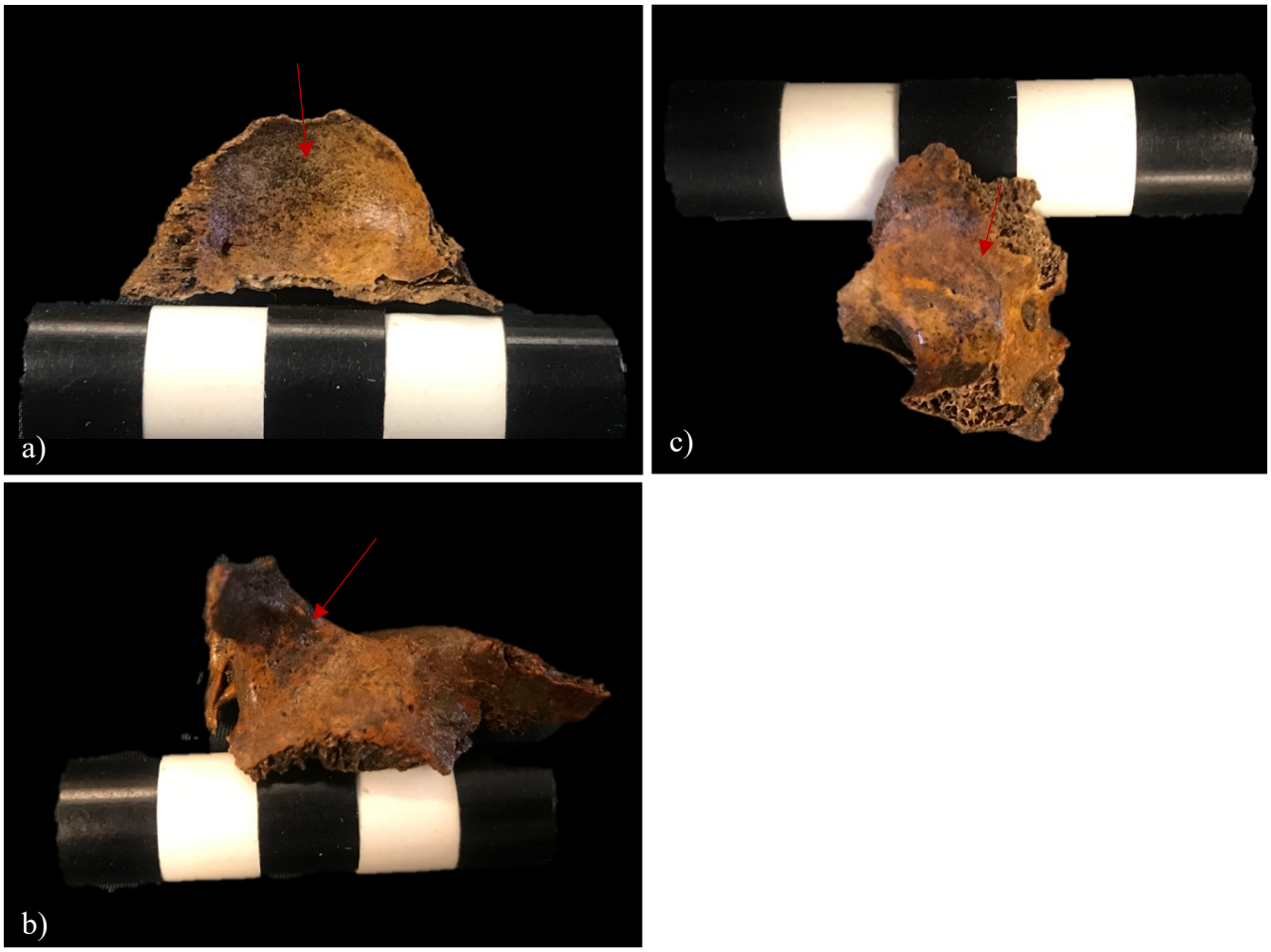
No 20Z-S5

Sex N.A.

Age-at-death 18 +- 6 months after birth



Mandibular left M1 in alveolar bone



Individual: 20Z-S5

ACI: N.A

Age-at-death: 18 months-old +- 6 months

Sex: N.A.

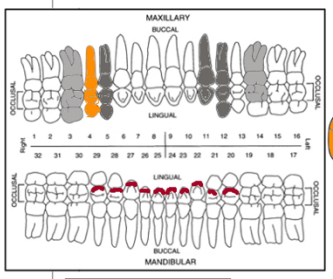
Description of pathological lesions: Orbital roof porosities on the right orbit (inferior view) (a), porosities on the greater wing of the left and right sphenoid bones (inferior view) (b) (c).

Possible aetiologies: Infectious disease, B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

Differential diagnosis: Diagnostic of *cribra orbitalia*, **highly consistent with** scurvy according to the distribution pattern of the lytic lesions.

Site BiFj-37 Boite 33 / 78 No 21E-S17

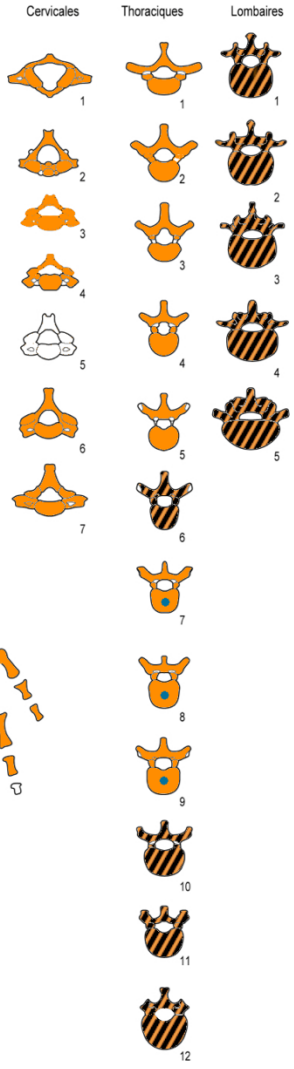
Sexe Male Âge-au-décès More than 40 years Stature Undetermined Isotopes : dent ☐ os ☐

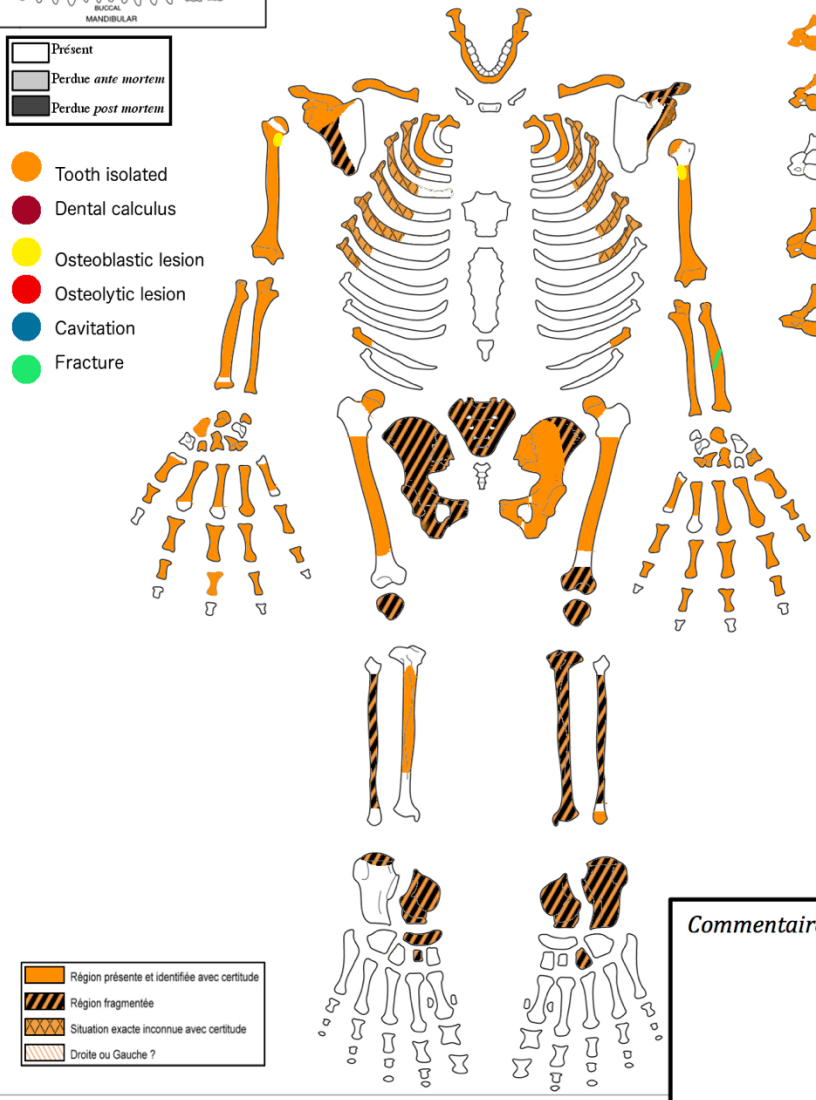


Présent
Perdue *ante mortem*
Perdue *post mortem*

Tooth isolated
Dental calculus
Osteoblastic lesion
Osteolytic lesion
Cavitation
Fracture

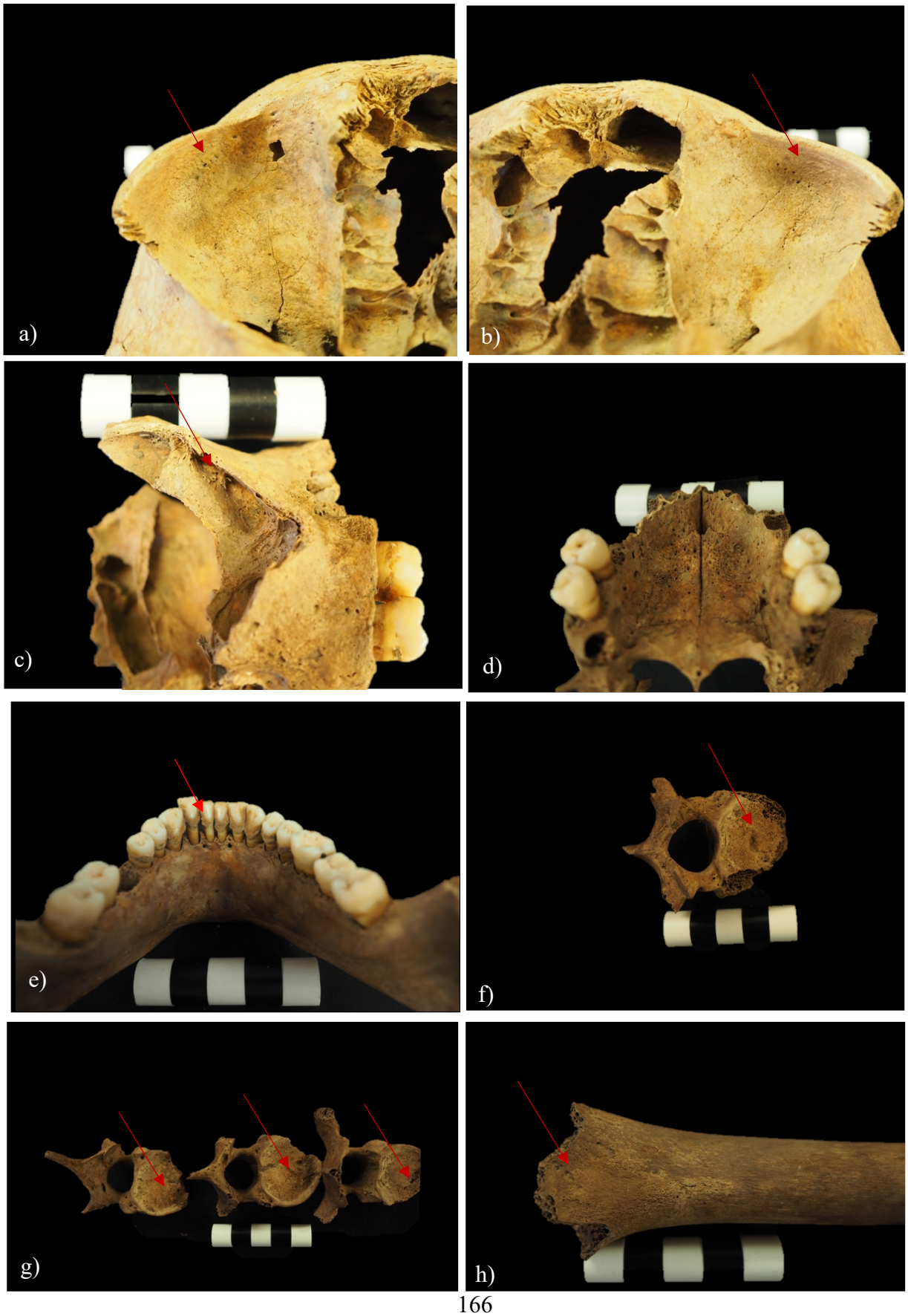
ICA 58.52

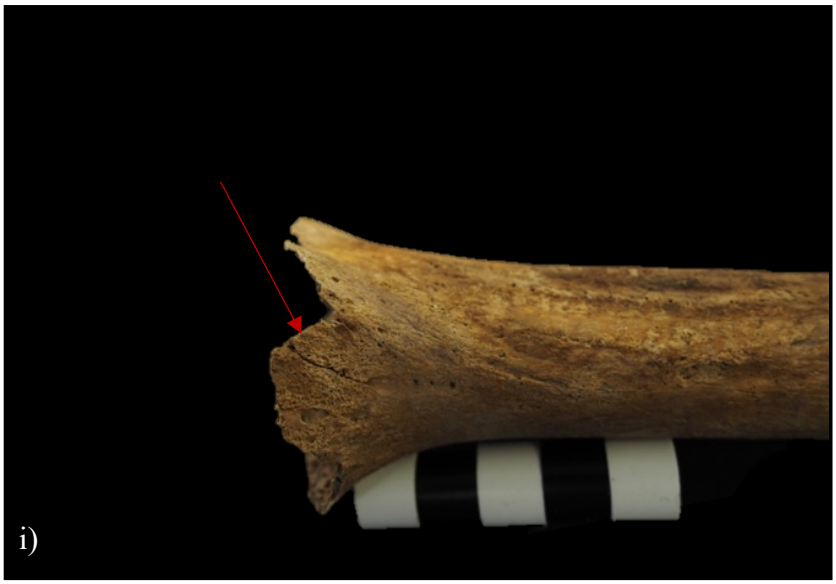




Région présente et identifiée avec certitude
Région fragmentée
Situation exacte inconnue avec certitude
Droite ou Gauche ?

Commentaires :


a)
b)
c)
d)
e)
f)
g)
h)



Individual: 21E-S17

Age-at-death: More than 30 years-old

Sex: Male

ACI: 58.72

Description of pathological lesions: Orbital roof porosities on left and right orbits (inferior view) (a) (b), bone formations in the right sinus (superior view) (c), abnormal porosities on the maxillary bone (inferior view) (d), calcified plaque on the lower front teeth (lingual view) (e), cavitation on TV8 (superior view) (f) and on TV7, 8 and 9 (inferior view) (g), new bone formation on the proximal metaphysis of the right (posterior view) (h) and left humerus (medial view) (i), fracture on the mid-diaphysis the left radius (lateral view) (j).

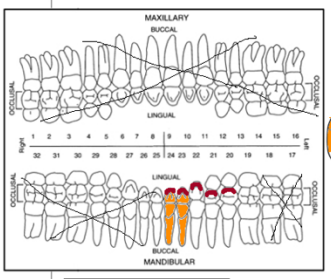
Possible aetiologies: Infectious disease, B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

Differential diagnosis: Diagnostic of dental calculus, Schmorl's nodes, maxillary sinusitis, trauma, degenerative joint disease*, cribra orbitalia*.

Site BiFj-37 Box 38 / 78 No 21S-S4

Sex Female tendency **Age-at-death** 20-36 years-old

ACI 65.56

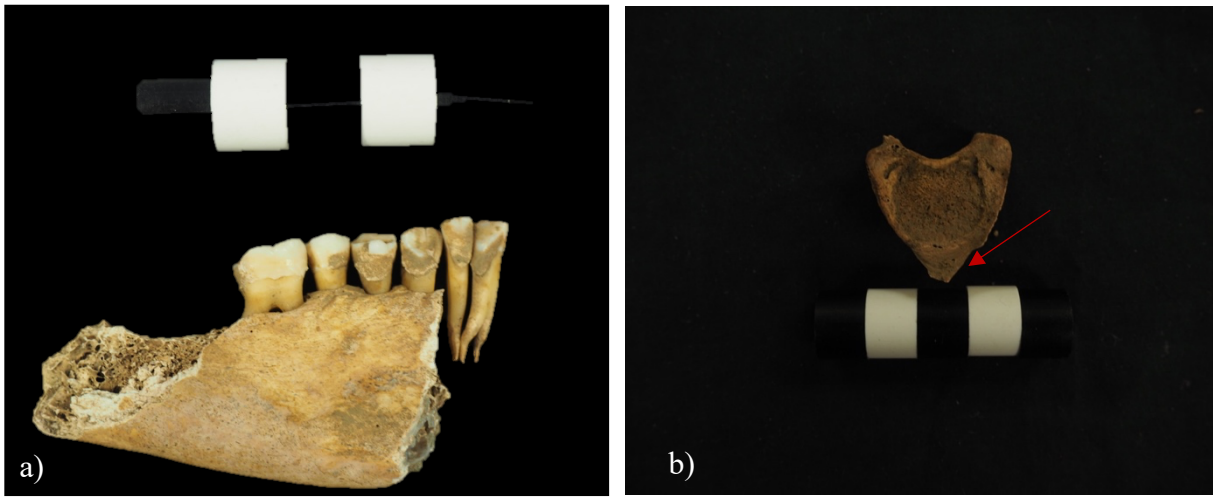


Tooth in place
Ante mortem loss
Post mortem loss

Osteoblastic lesion

Dental calculus

Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?



Individual: 21S-S4

ACI: 65.56

Age-at-death: 20-36 years-old

Sex: Likely female

Description of pathological lesions: calcified plaque on left lower teeth (buccal view) (a), bone formation on the anterior part of an undetermined TV (superior view) (b).

Differential diagnosis: Diagnostic of dental calculus**,** osteophyte, degenerative joint disease.

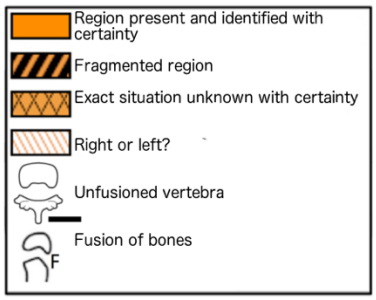
Site BiFj-37

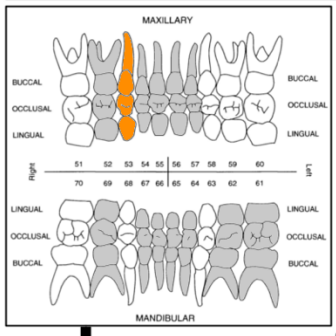
Box 41 / 78

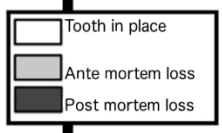
No 21U-S6

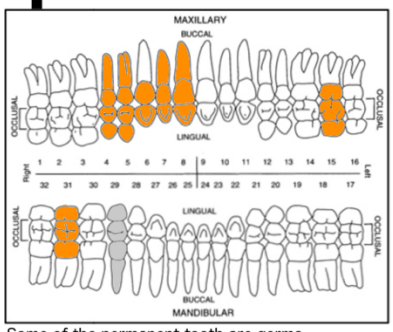
Sexe N.A.

Age-at-death 7-9.5 years-old



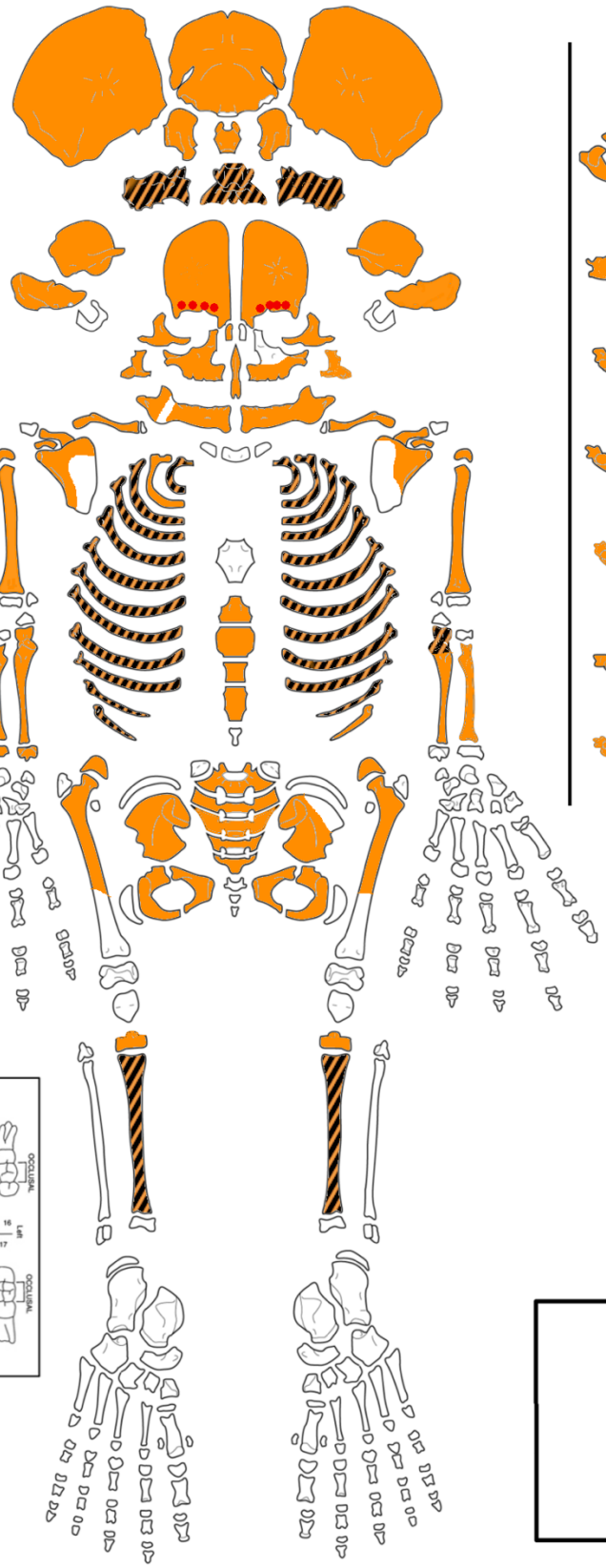


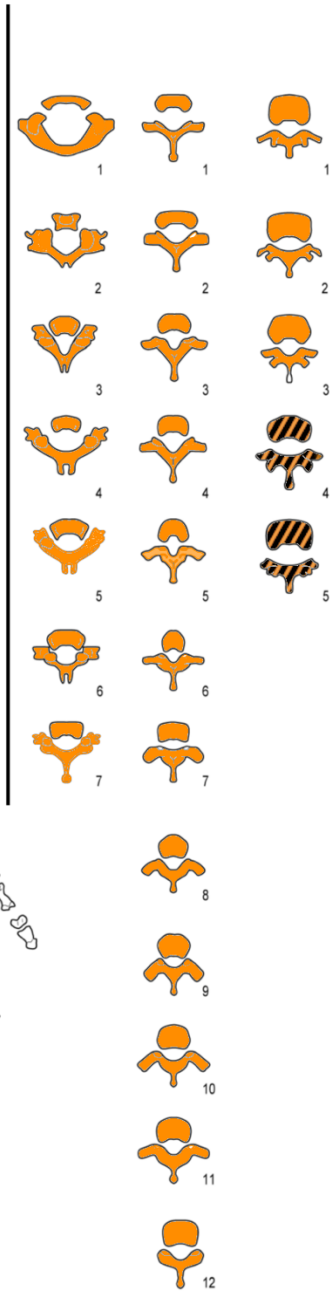


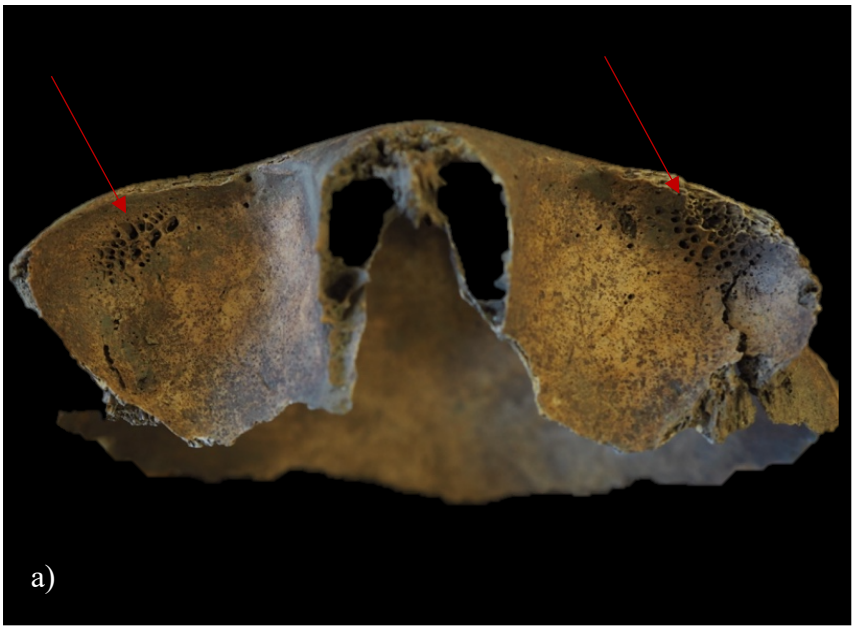


Some of the permanent tooth are germs
Orange = isolated tooth

Osteolytic lesion







Individual: 21U-S6

ACI: N.A.

Age-at-death: 7-9.5 years-old

Sex: N.A.

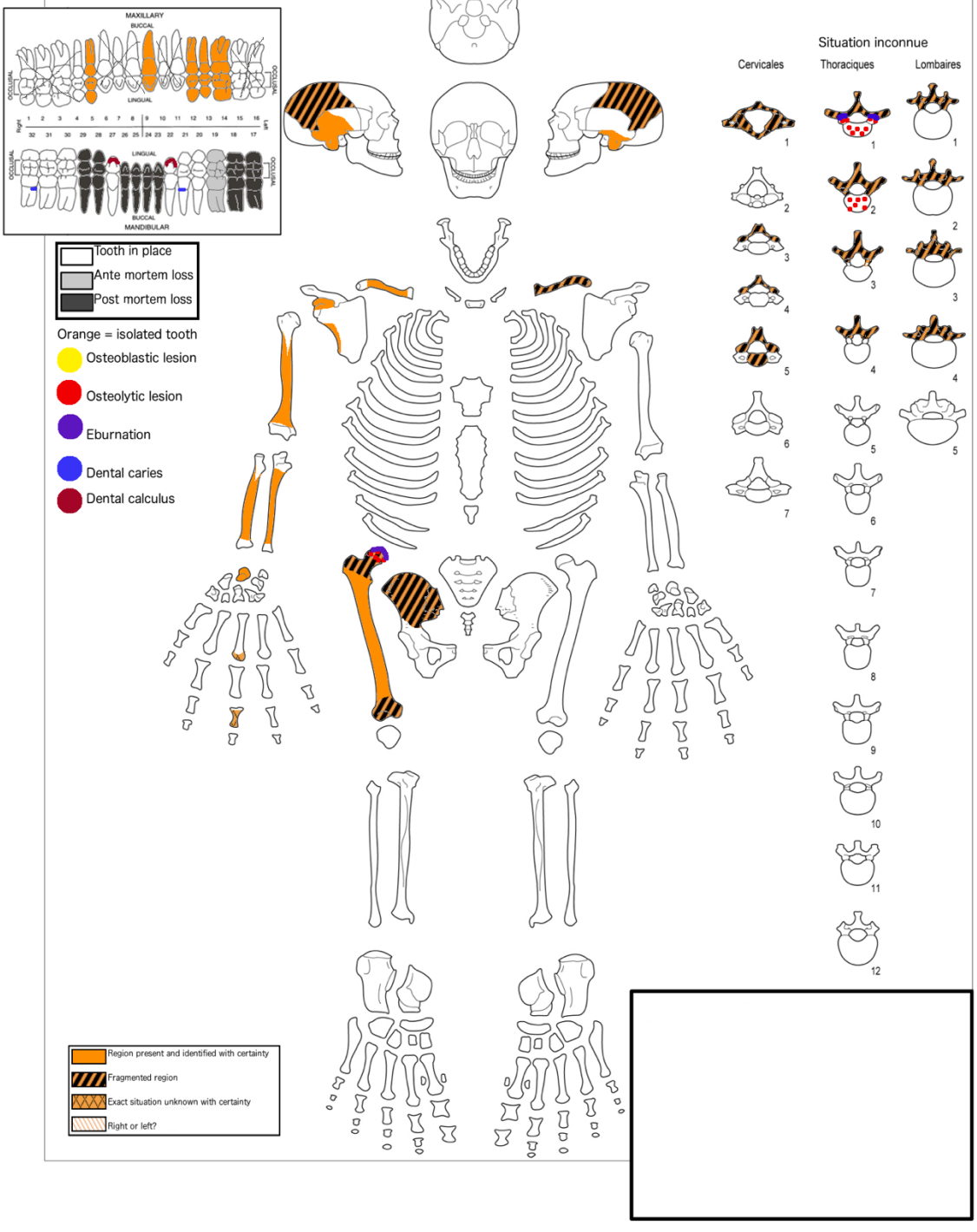
Description of pathological lesions: orbital roof porosities on right and left orbits (inferior view) (a).

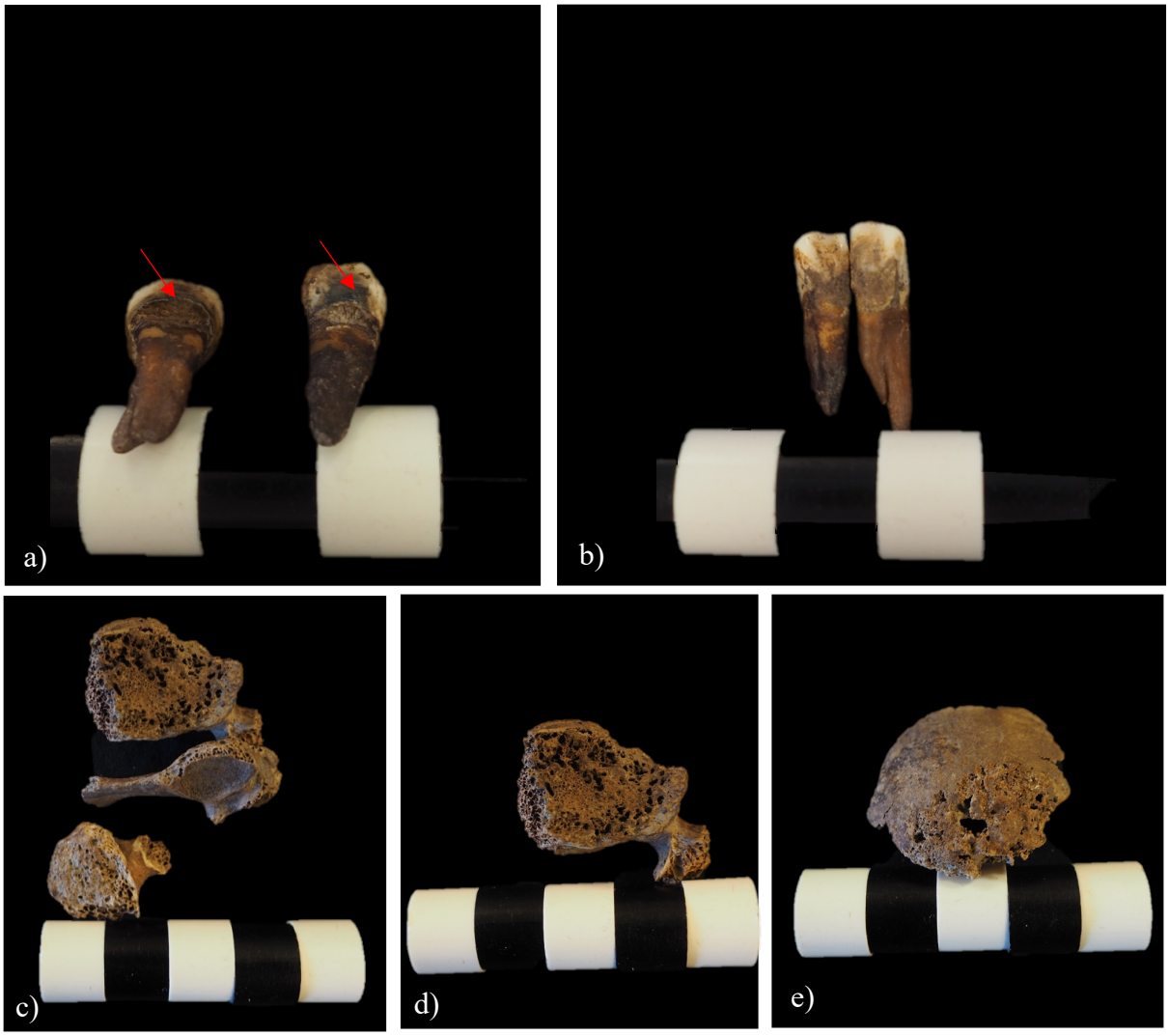
Possible aetiologies: B12 deficiency megaloblastic anaemia, iron-deficiency anaemia, infectious disease.

Differential diagnosis: Diagnostic of *cribra orbitalia*.

Site BiFj-37 Box 59 / 78 No 22A-S16

Sex Undetermined. Age-at-death More than 30 years-old





Individual: 22A-S16

ACI: 13.52

Age-at-death: More than 30 years-old

Sex: Undetermined.

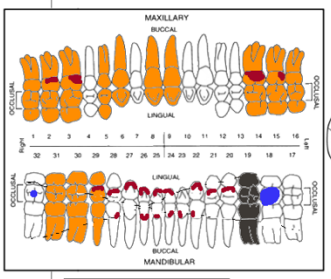
Description of pathological lesions: Cavity on the right M_3 and left PM_1 (mesial view) (a), calcified plaque on right and left mandibular canines (lingual view) (b), lytic lesions on the body of two undetermined cervical vertebrae CV (superior view) (c) (d), lytic lesions and eburnation on the superior articular facet of undetermined CVs (postero-superior view) (c), lytic lesions and eburnation on the right femoral head (superior view) (e).

Differential diagnosis: Diagnostic of dental calculus, dental caries, intervertebral disk disease and osteoarthritis.

Site BiFj-37 Box 16 / 78 No 22C-S10

Sex Male tendency **Age-at-death** 20 -29 years-old

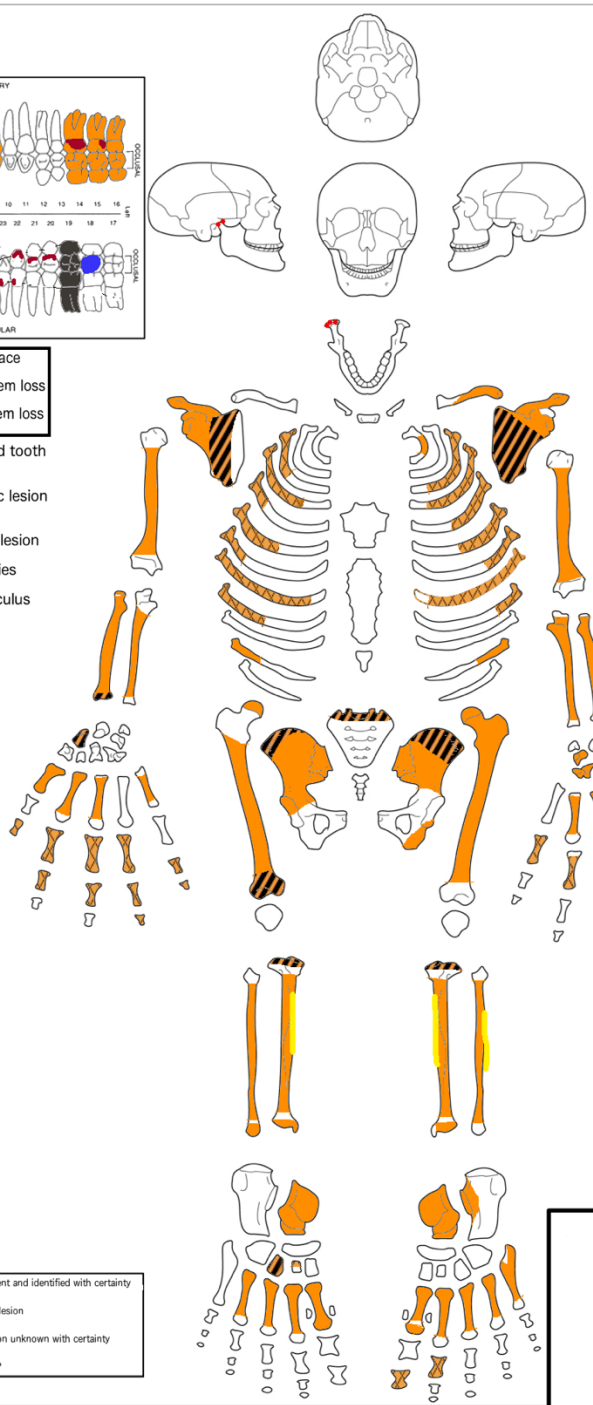
ACI 36.24

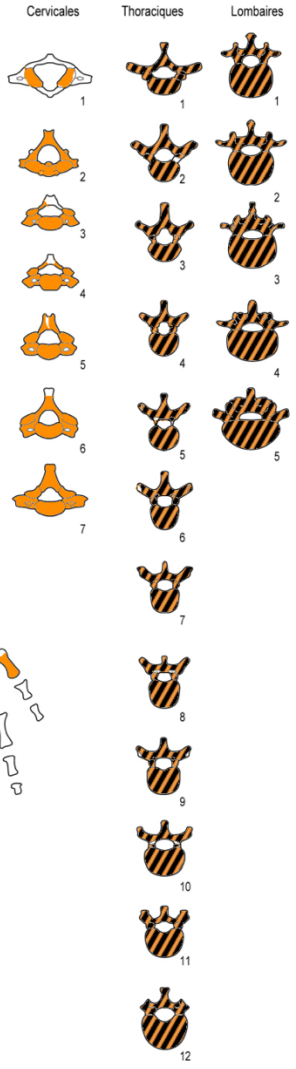


Tooth in place
Ante mortem loss
Post mortem loss

Orange = isolated tooth

Osteoblatic lesion
Osteolytic lesion
Dental caries
Dental calculus





Region present and identified with certainty
Fragmented lesion
Exact situation unknown with certainty
Right or left?


a)
b)
c)

Individual: 22C-S10

ACI: 47

Age-at-death: 20-29 years-old

Sex: Likely male

Description of pathological lesions: Calcified plaque on upper and lower teeth (buccal view) (a-b) and on the lingual view of the lower teeth (c), degeneration of the right temporomandibular joint

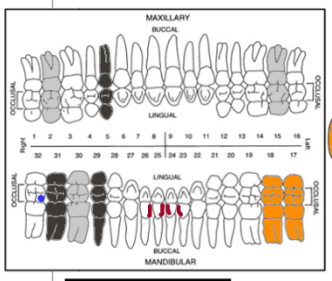
(postero-superior view) (d), new bone formation on the diaphysis of right and left tibias (medial view) (e-f), new bone formation on the diaphysis of the left fibula (lateral view) (g).

Differential diagnosis: Diagnostic of dental calculus, **typical of** non-specific infection, degenerative joint disease.

Site BiFj-37 Box 42 /78 No 23A-S2

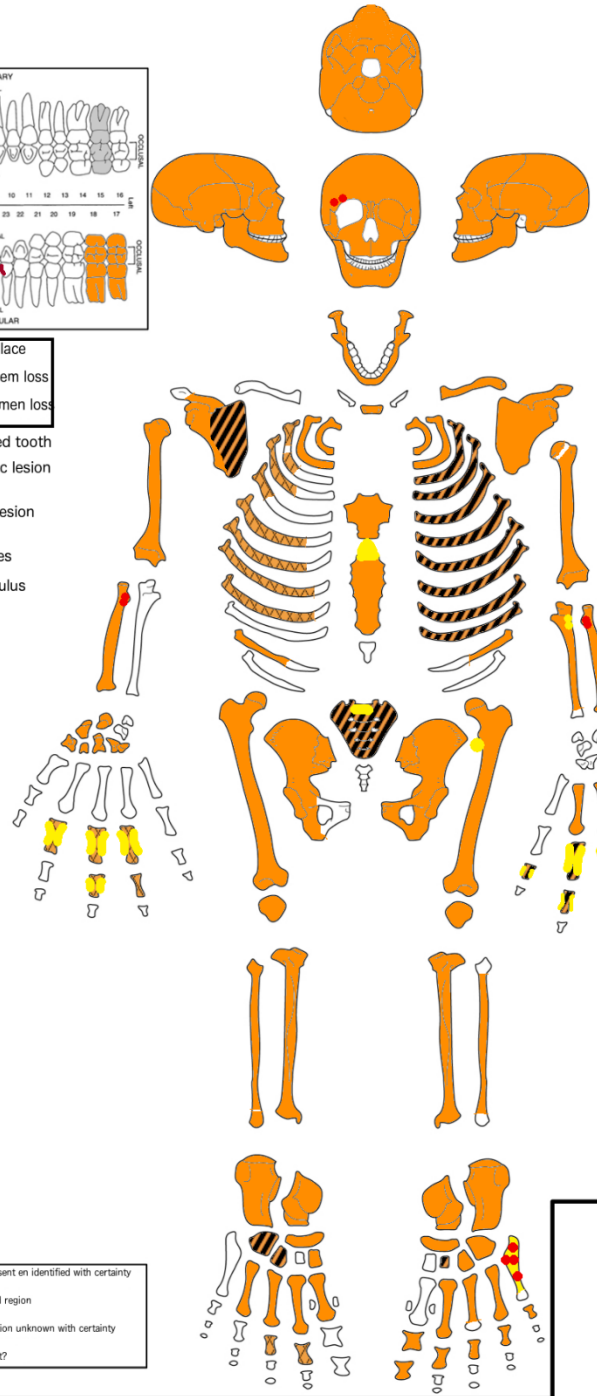
Sex Male tendency Age-at-death 30-59 years-old

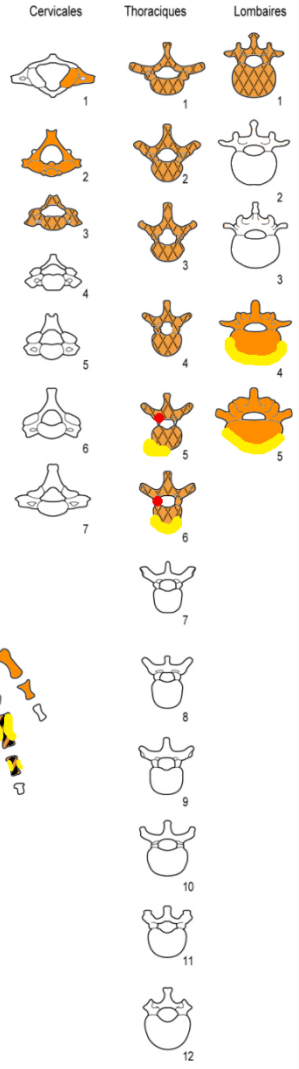
ACI 73.12



Tooth in place
Ante mortem loss
Post mortmen loss

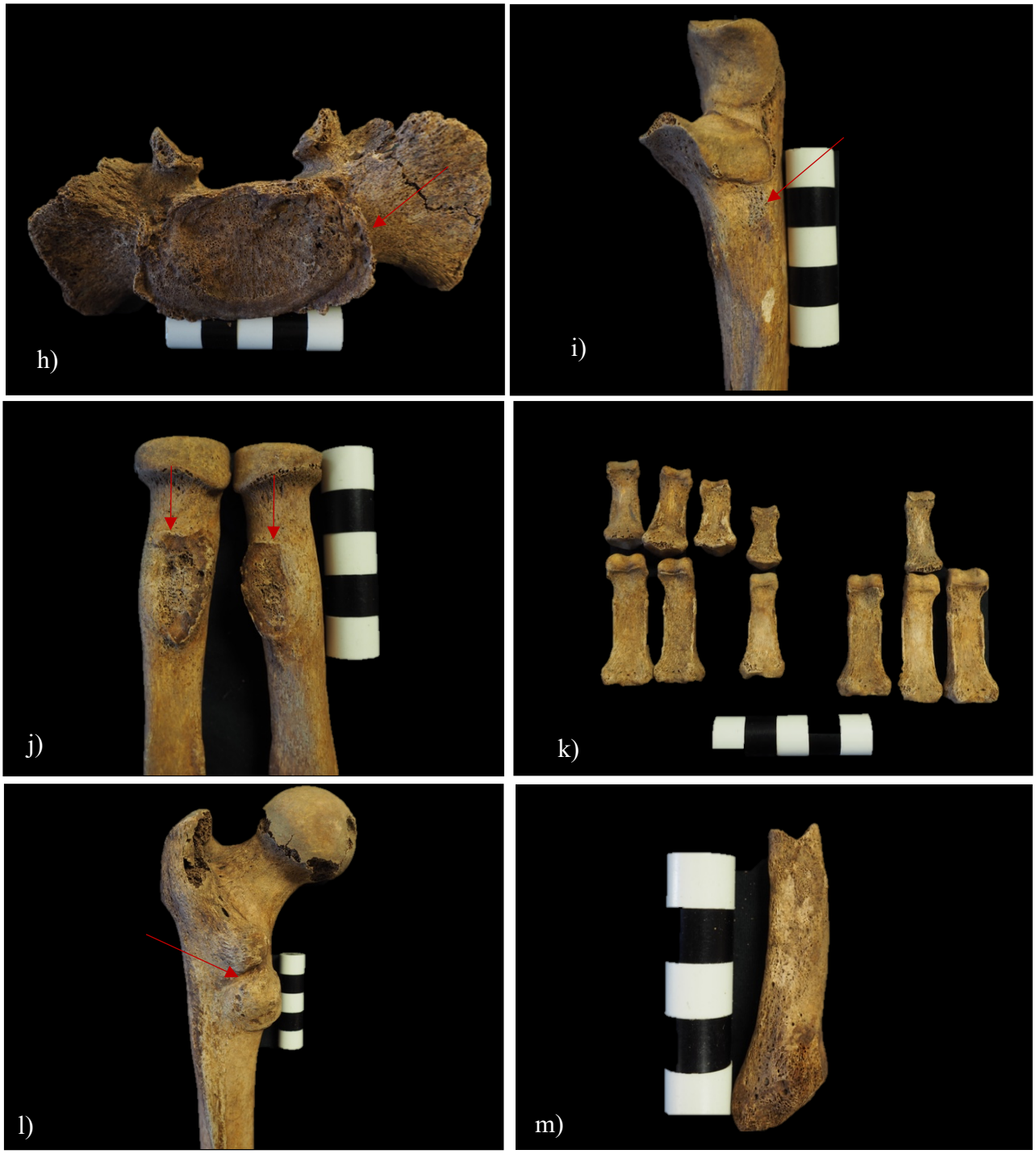
Orange = isolated tooth
Osteoblastic lesion
Osteolytic lesion
Dental caries
Dental calculus





Region present en identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?


a)
b)
c)
d)
e)
f)
g)



Individual: 23A-S2

ACI: 73.12

Age-at-death: 30-59 years-old

Sex: Likely male

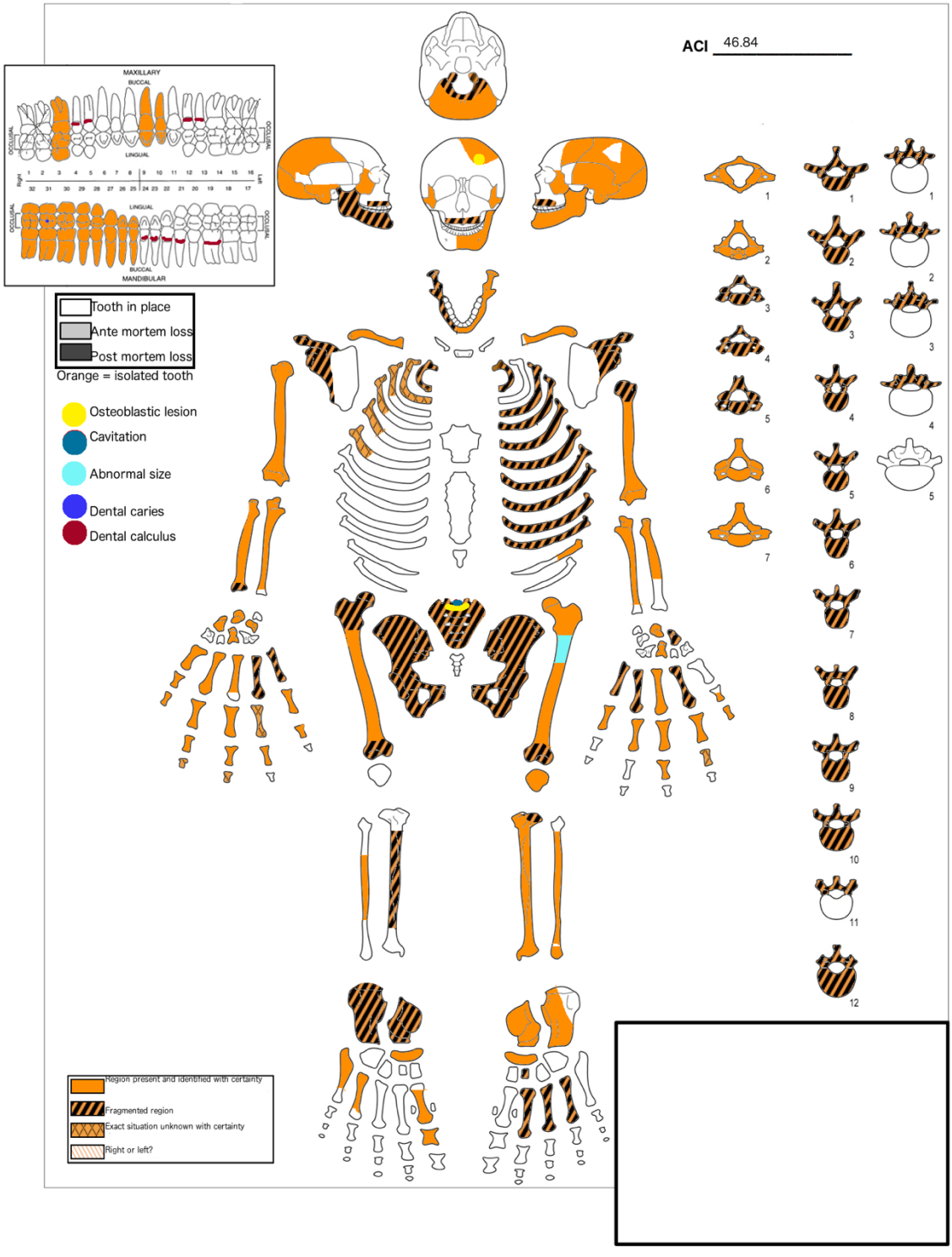
Description of pathological lesions: Calcified plaque on lower incisors (mesial view) (a), cavity on the right M_3 (mesial view) (b), orbital roof porosities on the right orbit (inferior view) (c), compression fracture of an undetermined TV and new bone formation on the inferior border of the body of the TV above (lateral view) (d), lytic lesions on the right inferior articular facet of an undetermined TV (inferior view) (e),), lytic lesions on the right superior articular facet of an undetermined TV (posterior view) (f), bone formations or "lipping" on the superior border of the body of LV4 and on the inferior border of the body of LV5 (anterior view) (g), bone formations or "lipping" on the superior border of the body of S1 (superior view) (h), woven bone near the radial articular facet of the left ulna (antero-lateral view) (i), lytic lesions on the radial tuberosities (medial view) (j), interosseous bone formations of proximal and intermediate hand phalanx (palmar view) (k), round bone formation under the lesser trochanter of the left femur (posterior view) (l), new bone formation on the left MT5 (dorsal view) (m).

Possible aetiologies: Strenuous activity, infectious disease, B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

Differential diagnosis: Diagnostic of dental calculus, dental caries, osteophytes, inflammation of biceps muscle insertion, osteoarthritis, degenerative joint disease, *cribra orbitalia*, **typical of** bone tumour.

Site BiFj-37 Box 10 / 29 No 24L-S4

Sex Male Age-at-death 20-49 years-old



Individual: 24L-S4

ACI: 46.84

Age-at-death: 20-49 years-old (maybe more 20-29 years-old according to the vertebras)

Sex: Male

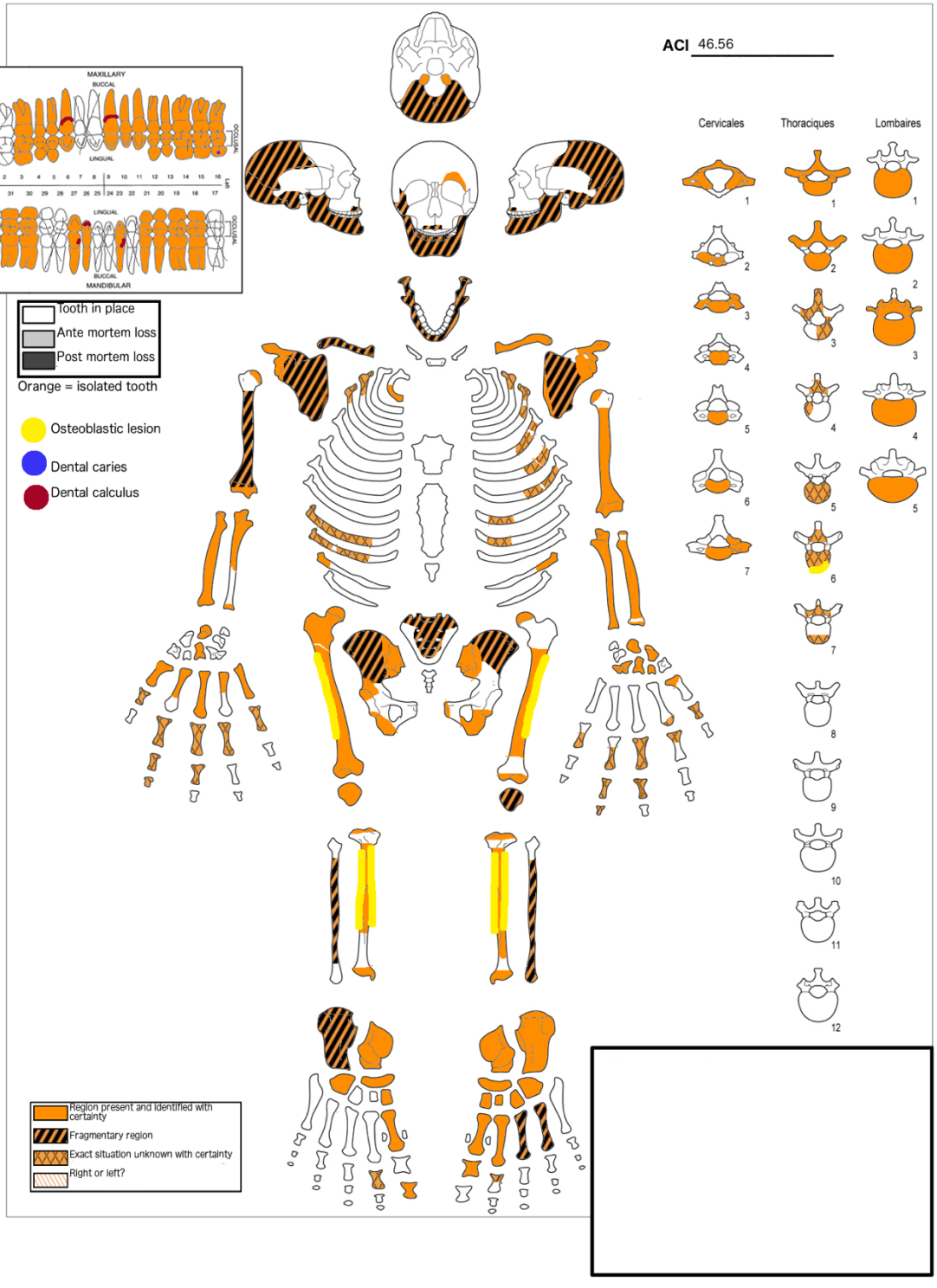
Description of pathological lesions: Calcified plaque on lower and upper teeth (buccal view) (a-b), cavity on the right M_2 (occlusal view) (c), bone nodule on the surface of the frontal bone

(anterior view) (d), cavitation on the superior surface of S1 and bone formations or “lipping” on the border of the body (superior view) (e), *coxa vara* of the left femur (anterior view) (f).

Differential diagnosis: Diagnostic of dental calculus, dental caries, Schmorl’s nodes, osteophytes, intervertebral disk disease, **typical of** osteoma, residual rickets.

Site BiFj-37 Box 23-24 / 78 No 25A-S3

Sex Male tendency Age-at-death 18-34 years-old



Individual: 25A-S3

ACI: 46.56

Age-at-death:16-20 years-old

Sex: Likely male

Description of pathological lesions: Periosteal lesions on the diaphysis of right and left femurs (lateral view) (a) and of right and left tibias (lateral view) (b); bone formations or "lipping" on the

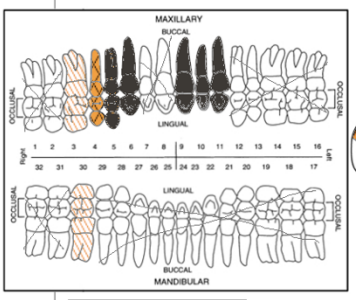
inferior border of the body of an undetermined TV (lateral view) (c), calcified plaque on upper and lower front teeth (buccal, lingual, mesial view) (d), cavity on the left M^3 (lingual side) (e).

Differential diagnosis: Diagnostic of dental calculus, dental caries, osteophyte, **typical of** non-specific infection.

Site BiFj-37 Box 73 / 78 No 25B-S4

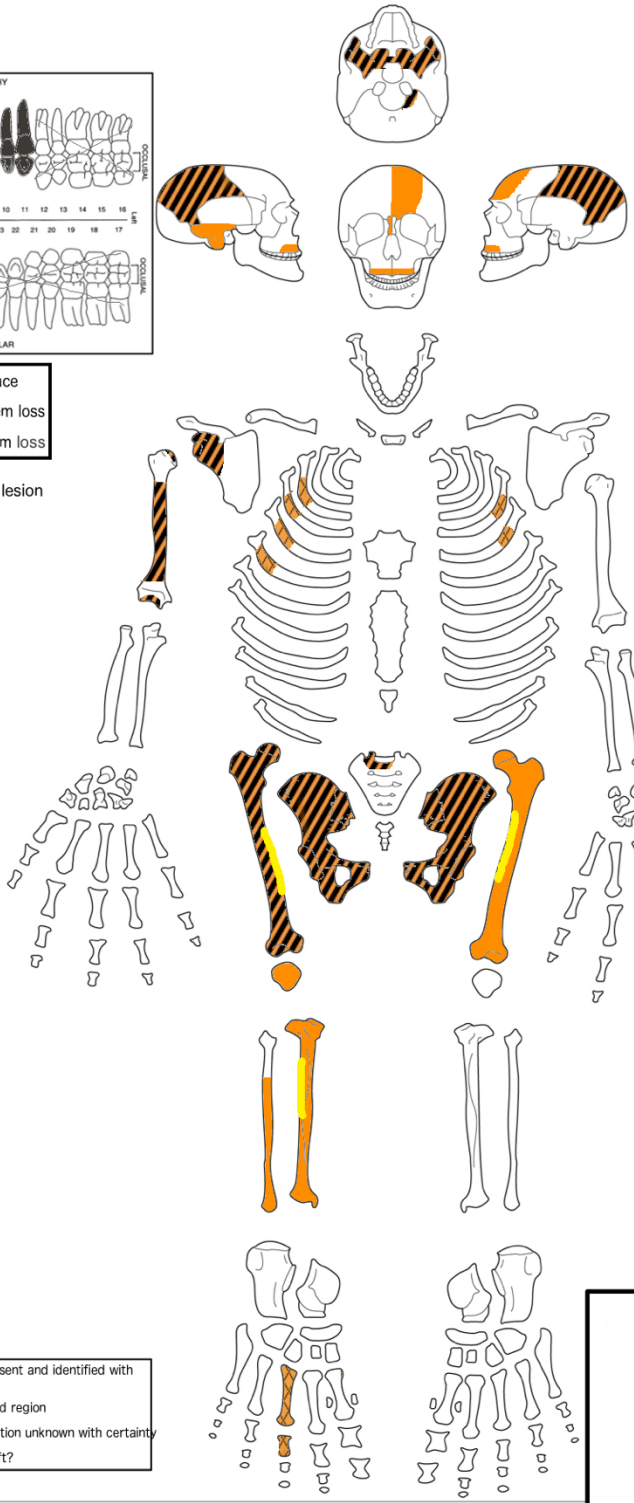
Sex Female **Age-at-death** 35-45 years-old

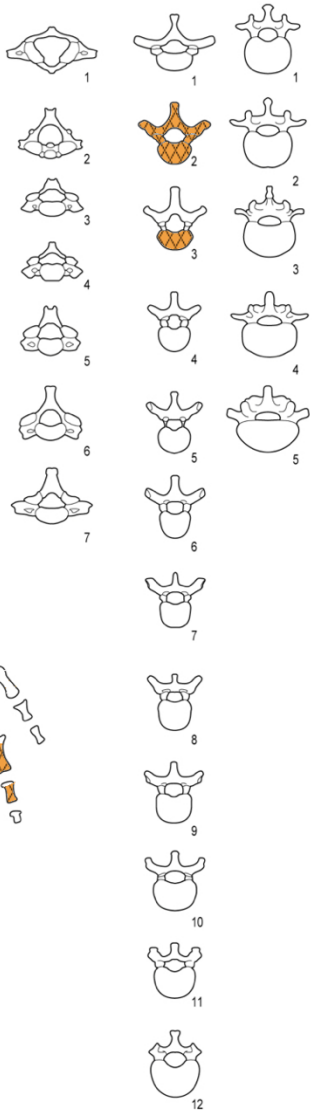
ACI 13.08



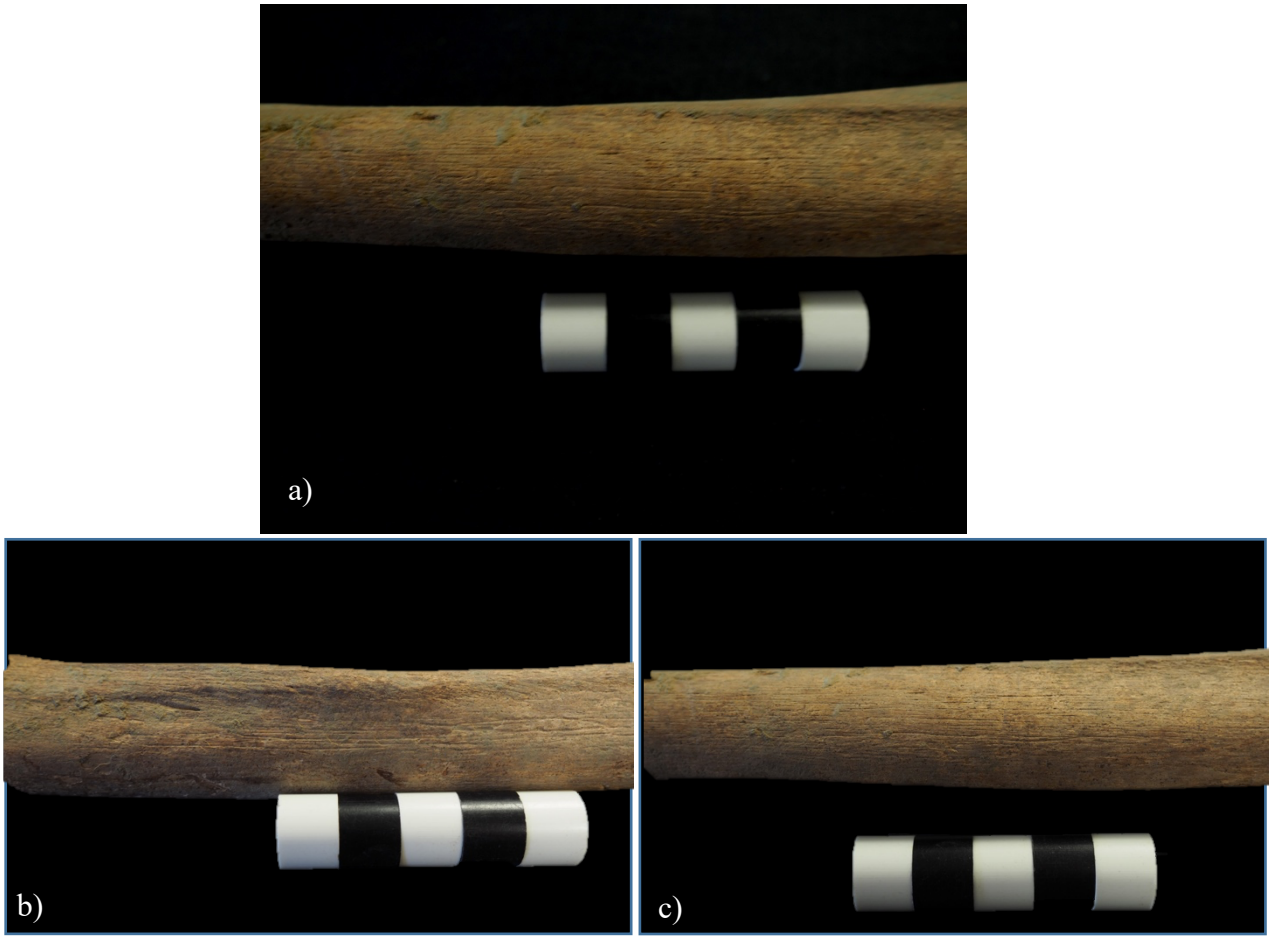
Tooth in place
Ante mortem loss
Post mortem loss

Osteoblastic lesion





Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?



Individual: 25B-S4

ACI: 13.08

Age-at-death: 35-45 years-old

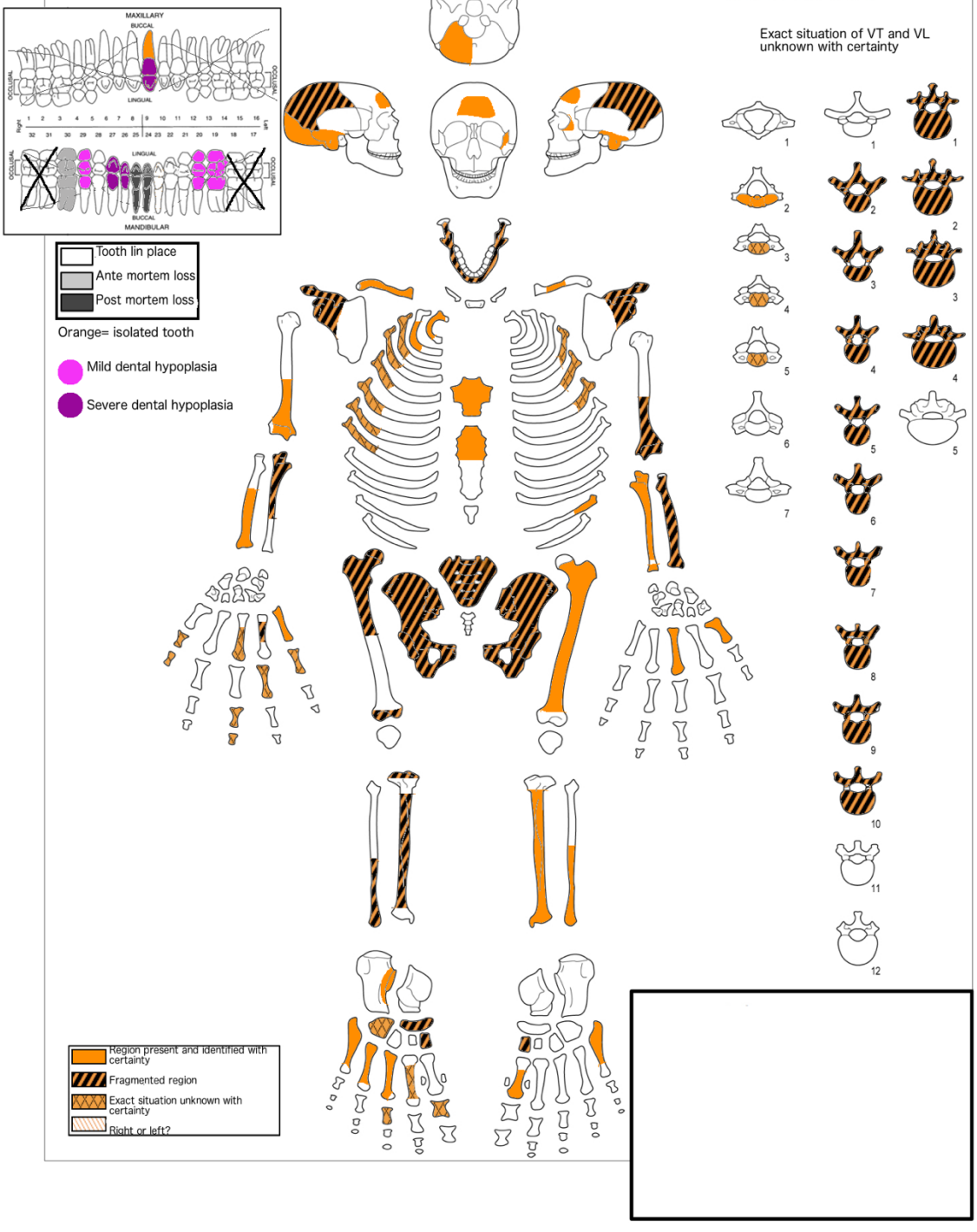
Sex: Female

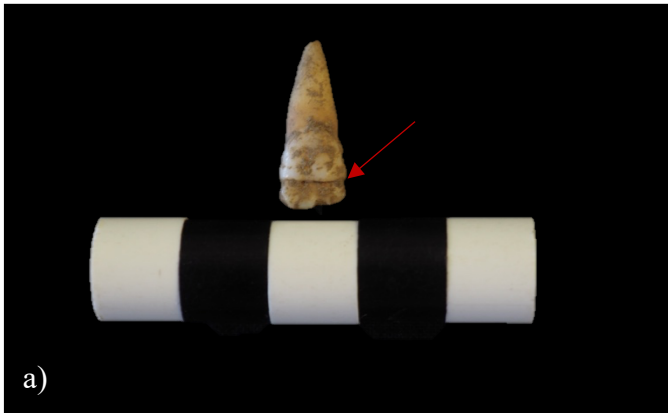
Description of pathological lesions: New bone formation on the medial face of right (a) and left (b) femurs (medial view) and right tibia (lateral view) (c).

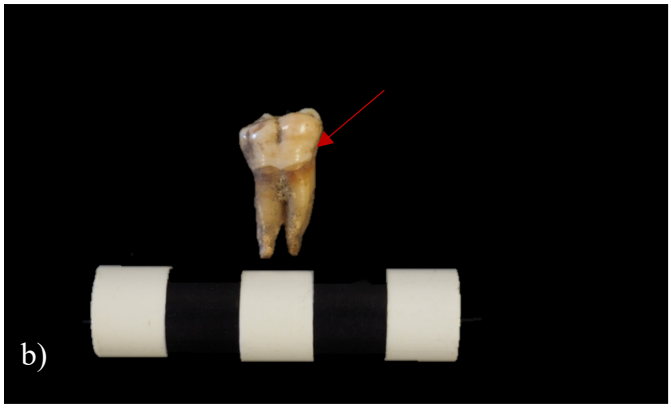
Differential diagnosis: Typical of non-specific infection.

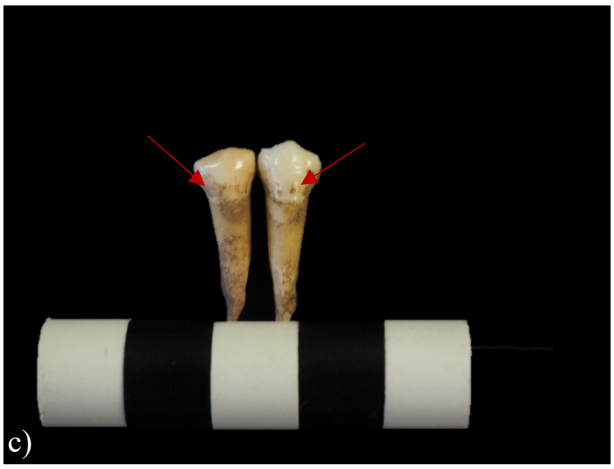
Site BiFj-37 Box 71 / 78 No 25C-S18

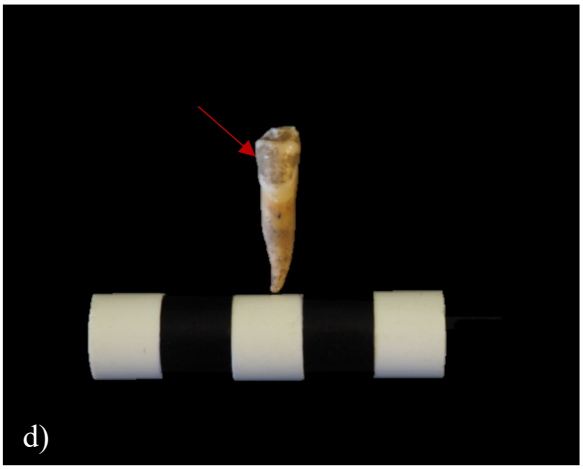
Sex Female Age-at-death 30-59 years-old

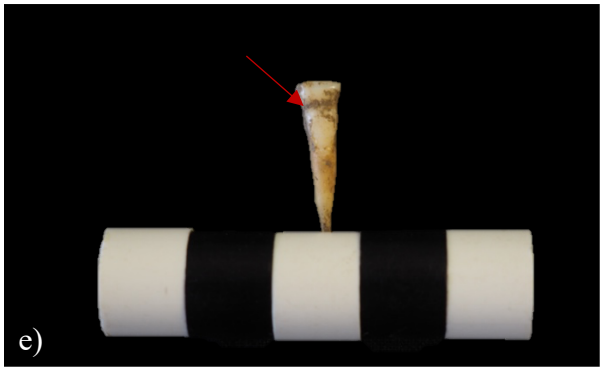



a)


b)


c)


d)


e)

Individual: 25C-S18;

ACI: 28.72;

Age-at-death: 30-59 years-old;

Sex: Female

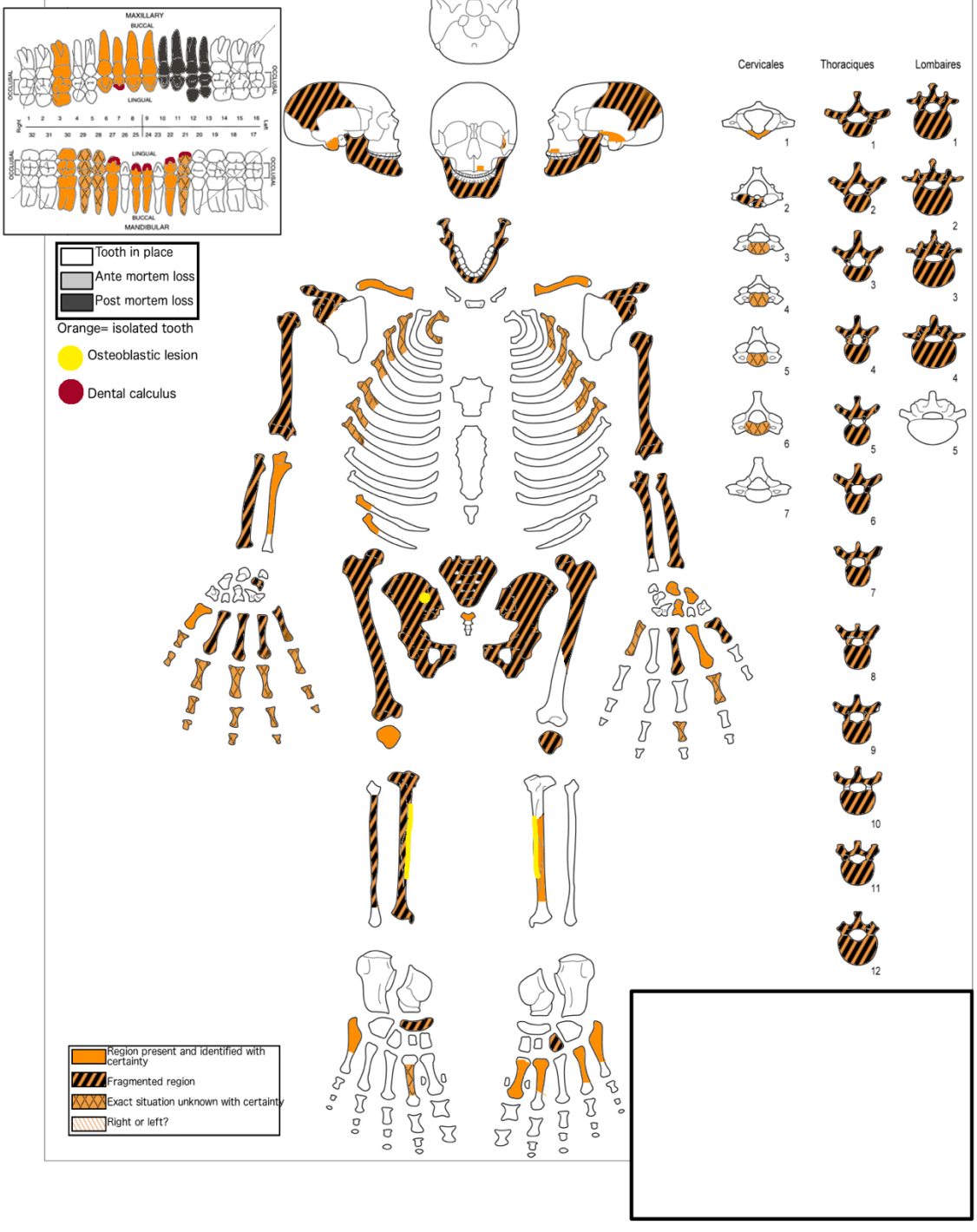
Description of pathological lesions: Lines on left I^1 (severe) (buccal view) (a), left M_1 (mild) (lingual view) (b), left and right PM_2 (buccal view) (c) (mild), right C_1 (severe) (buccal view) (d) and right I_1 (severe) (e) (buccal view).

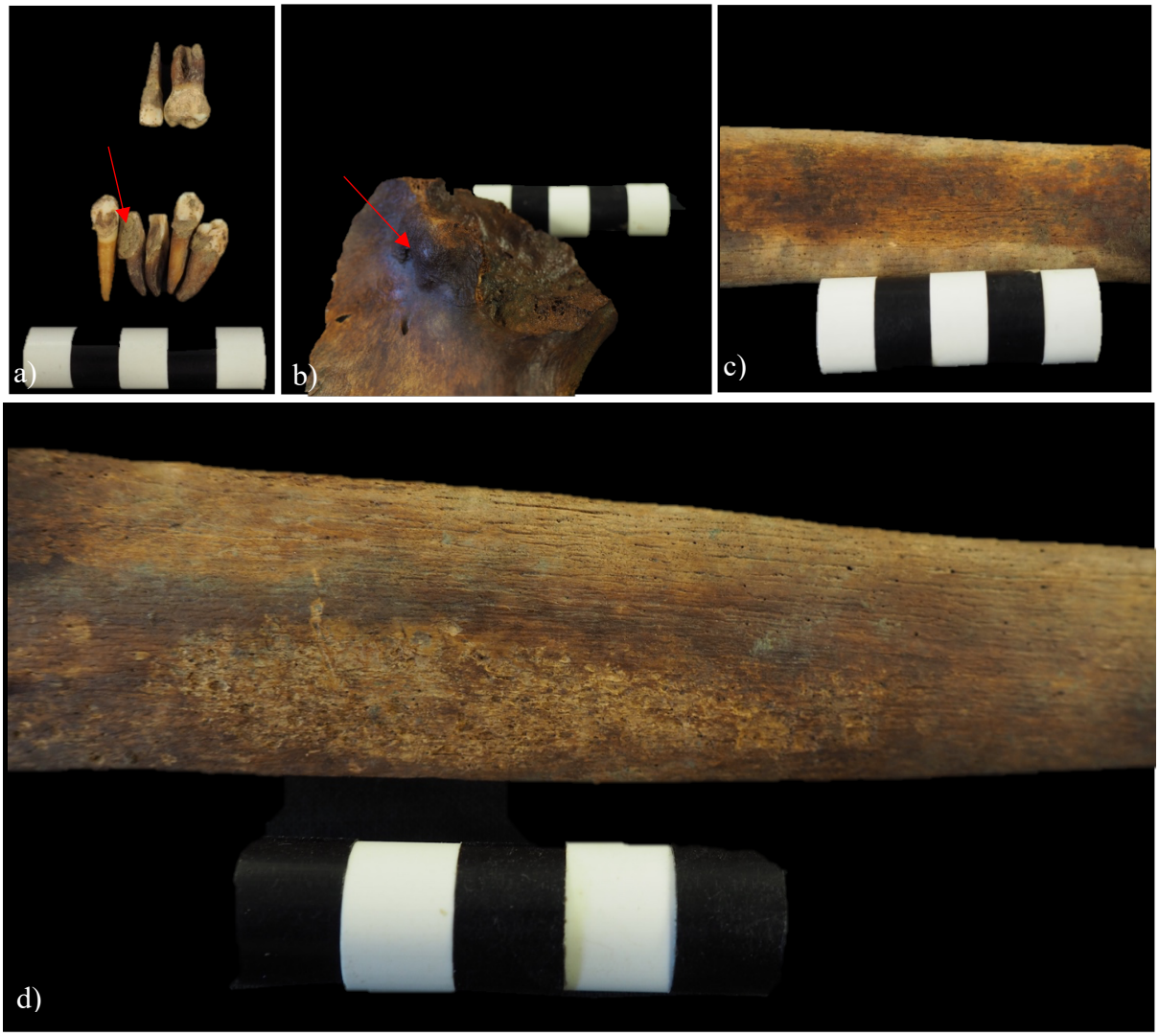
Possible aetiologies: Childhood disease-nutritional deficiencies, periapical inflammation or trauma to a deciduous tooth, fever, disease, endocrine dysfunction, infection during odontogenesis.

Differential diagnosis: Diagnostic of LEH.

Site BiFj-37 Box 75 / 78 No 25C-S36

Sex Female Age-at-death 30-59 years-old





Individual: 25C-S36;

ACI: 33.08;

Age-at-death: 30-59 years-old;

Sex: Female

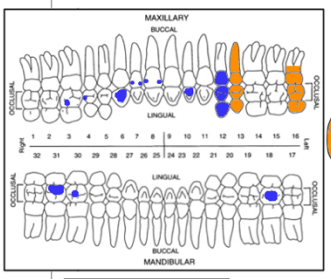
Description of pathological lesions: Calcified plaque on upper and lower teeth (lingual view) (a), bone growth on the anterior edge of the right auricular surface (medial view) (b), periosteal lesions on the diaphysis of the left (c) and right (d) tibias (medial view).

Differential diagnosis: Diagnostic of dental calculus, **typical of** bone tumour, non-specific infection.

Site BiFj-37 Box 13 / 29 No 25C-S64

Sex Female Age-at-death 15-16 years-old

ACI 48.84



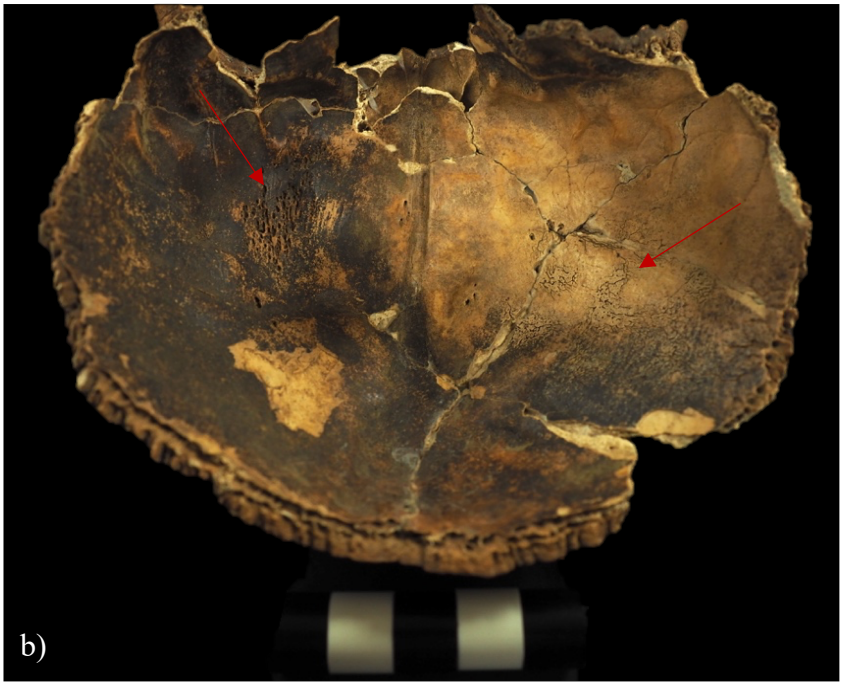
Tooth in place
Ante mortem loss
Post mortem loss

Orange = isolated tooth

Osteoblastic lesion
Osteolytic lesion
Dental caries

Und. ribs

Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?



Individual: 25C-S64

ACI: 48.84

Age-at-death: 15-16 years-old

Sex: Female

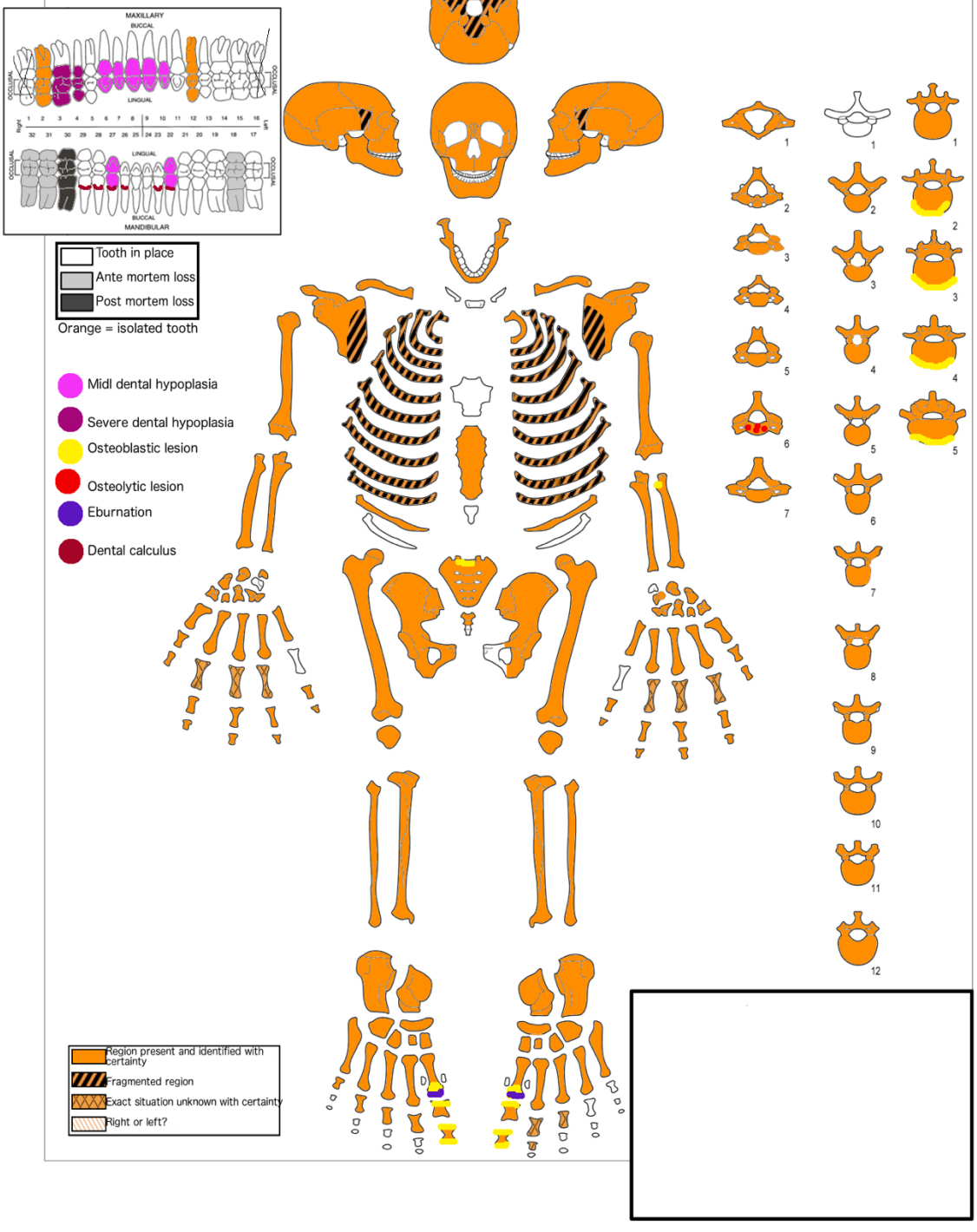
Description of pathological lesions: Cavity on right I^1, C, M^1, right and left I^2, PM^1 and right M_1, right and left M_2 (occlusal view) (a), fibrous bone formations and porous lesions on the frontal bone (endocranial view) (b).

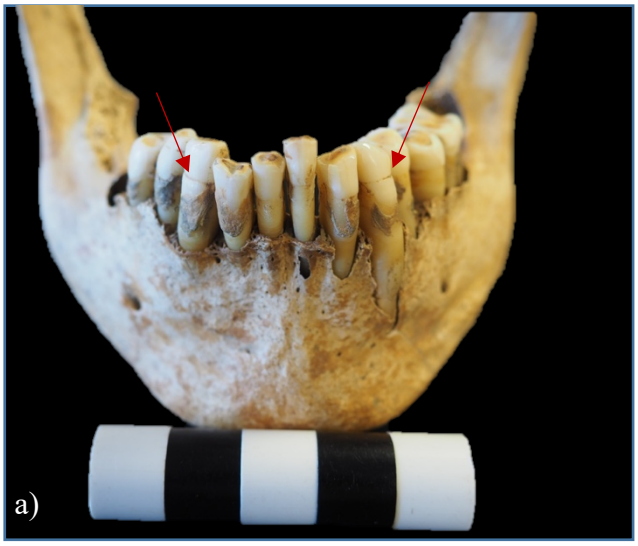
Possible aetiologies: Trauma, primary and secondary infections of the meninges, tumours, TB, syphilis and vitamin deficiencies of A, C and D.

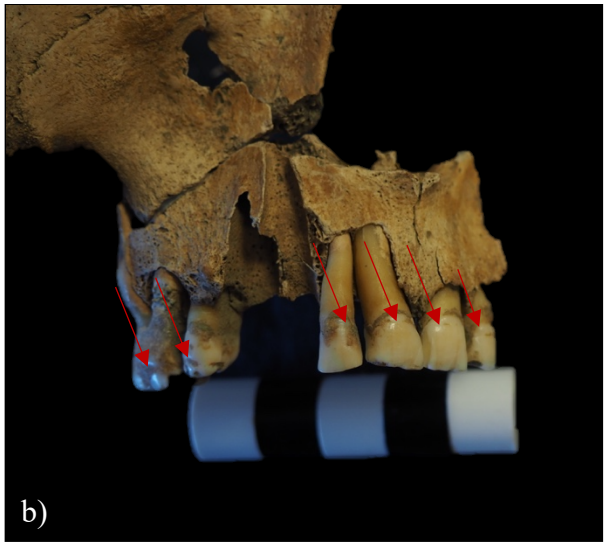
Differential diagnosis: Diagnostic of dental cavities, endocranial lesions.

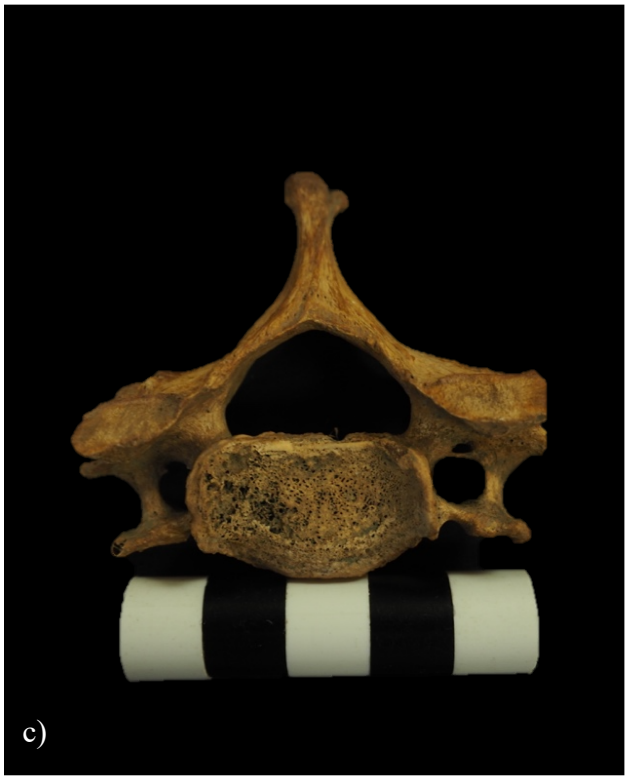
Site BiFj-37 Box 29 / 78 No 25C-S65

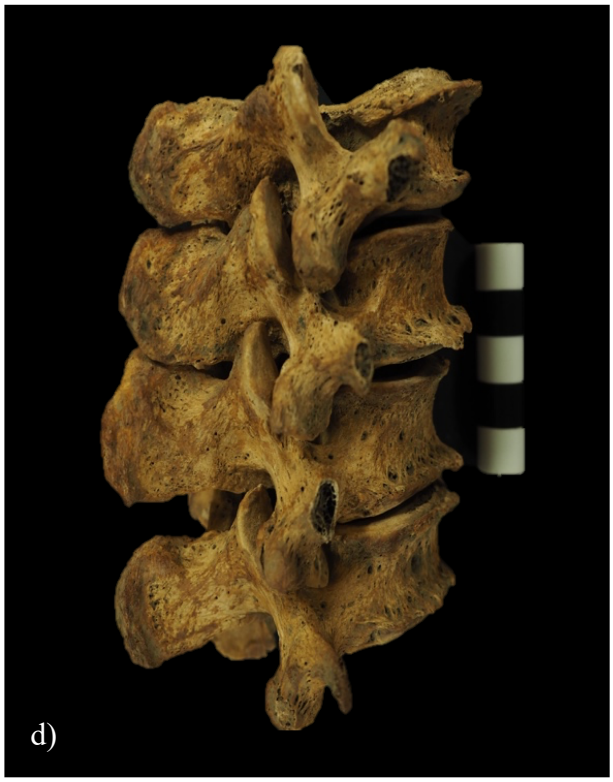
Sexe Male tendency Age-at-death 20-29 years-old




a)


b)

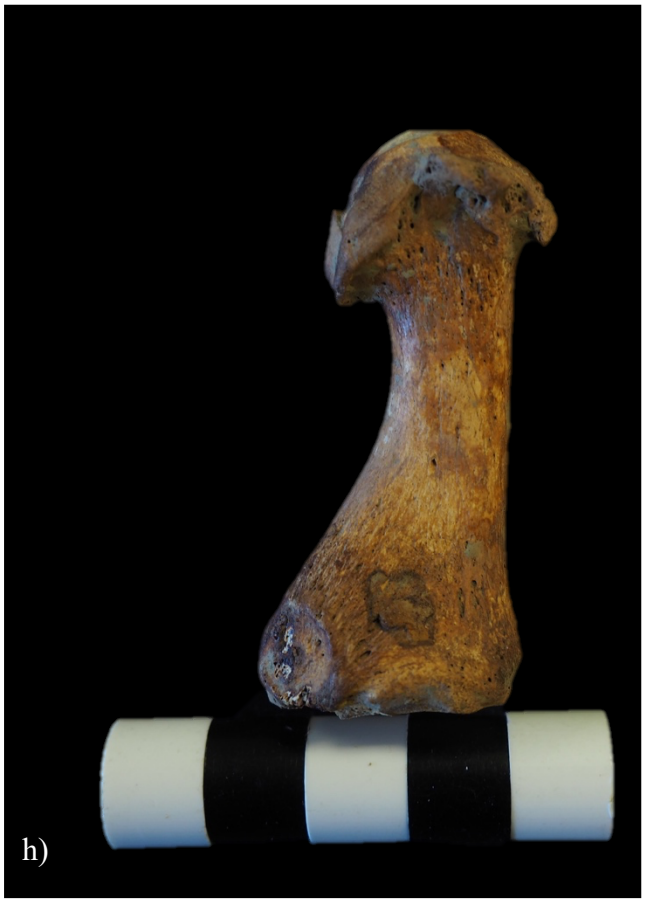

c)

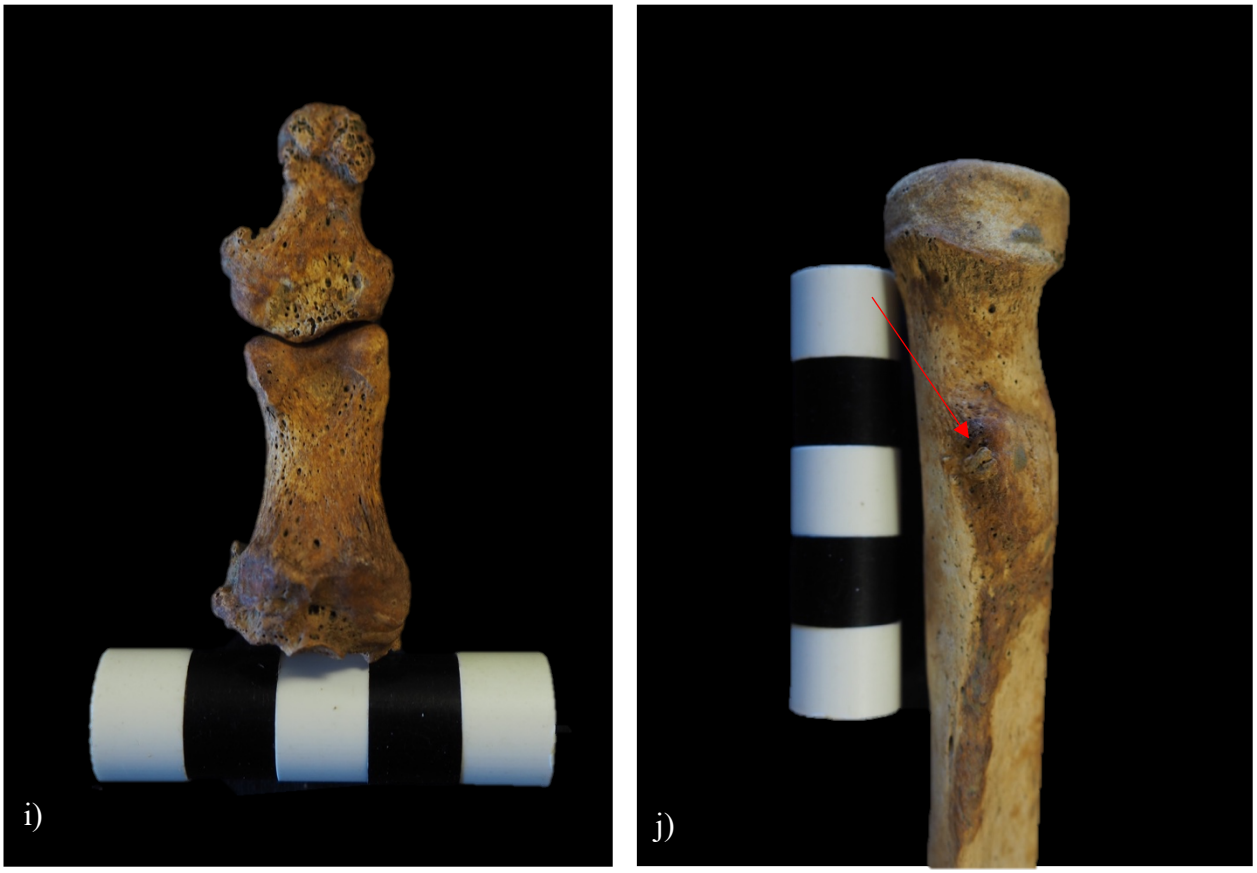

d)


e)


f)


g)


h)



Individual: 25C-S65

ACI: 89.8

Age-at-death:20-29 years-old

Sex: Likely male

Description of pathological lesions: Lines on right and left lower canines (buccal view) (a), on the right C^1, right and left I^1 and I^2 and pits on right PM^2 and M^1 (buccal view) (b), calcified plaque on right/left I2 and C1, right PM_1 and PM_2 (a), lytic lesions on the body of CV6 (superior view) (c), bone formations or "lipping" on the bodies of LV2 to LV5 (lateral view) (d), bone formations or "lipping" on the border of the body of S1 (anterior view) (e), new bone formations and eburnation on the distal epiphysis of the right and left MT1 (lateral view) (f) (h), new bone formation on the proximal epiphysis of the right and left proximal first foot phalanx and new bone

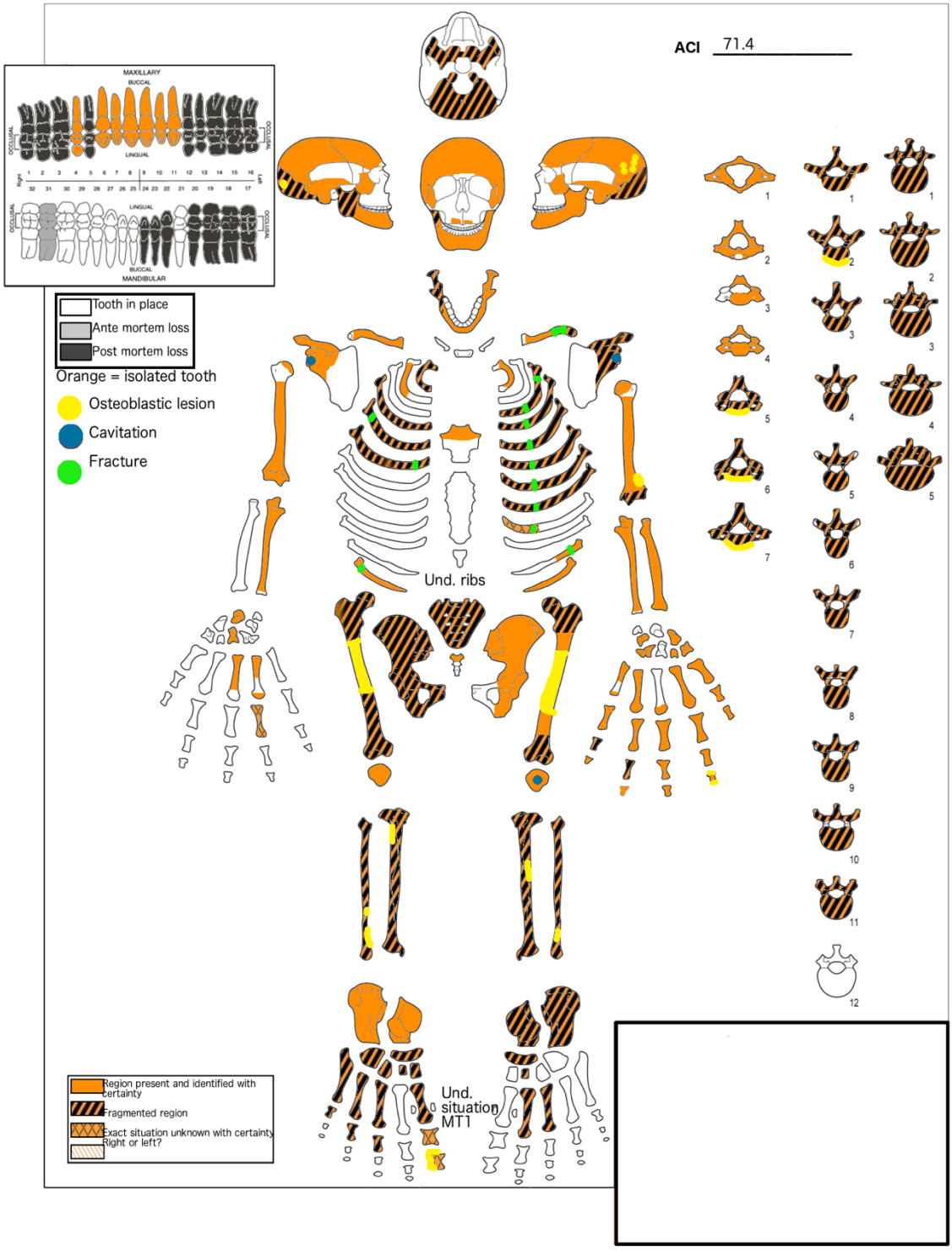
formation on the proximal and distal epiphysis of the right and left distal foot phalanx (g) (i) (plantar view), bone nodule on the radial tuberosity of the left radius (medial view) (j).

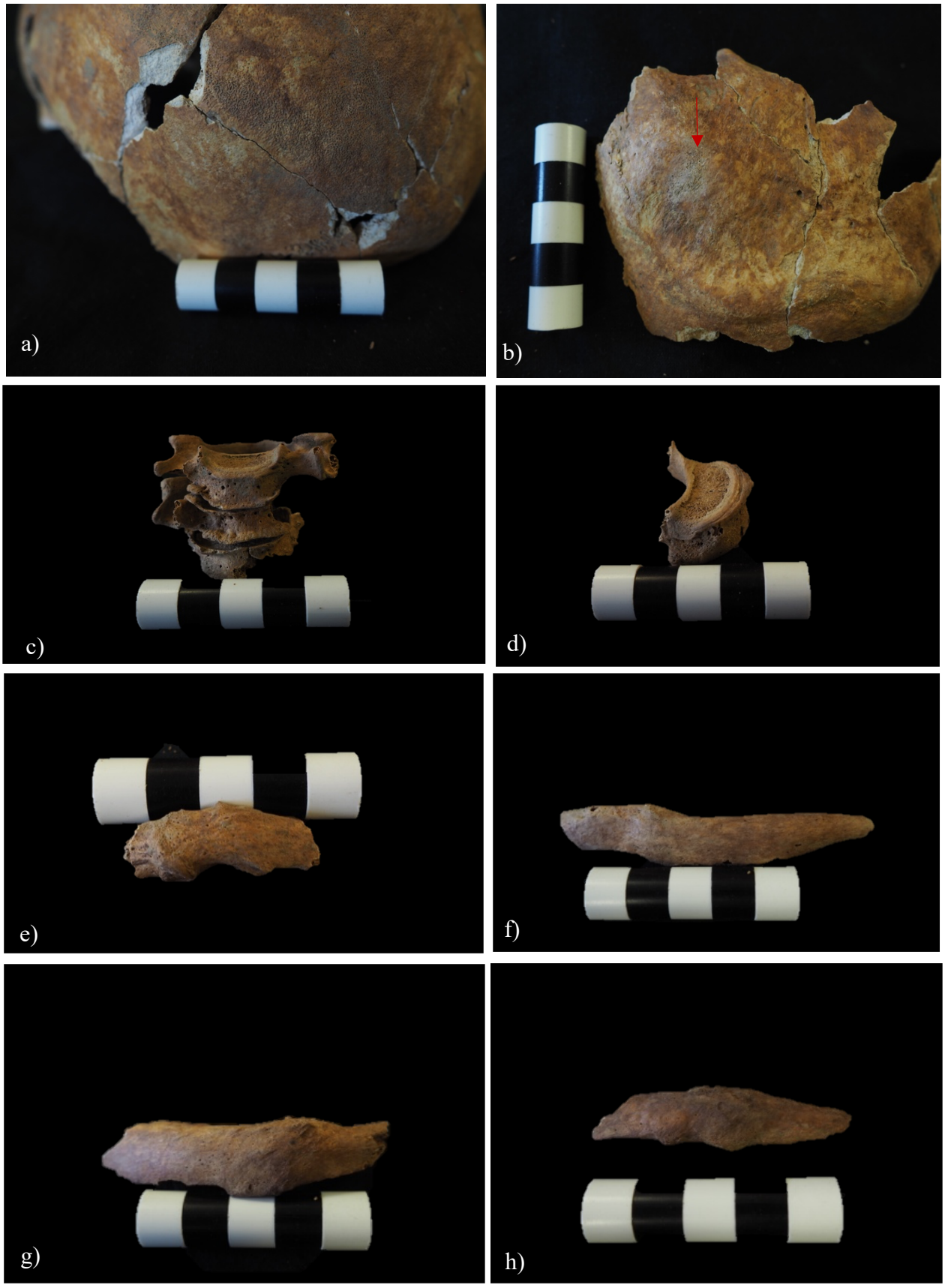
Possible aetiologies: Childhood disease-nutritional deficiencies, periapical inflammation or trauma to a deciduous tooth, fever, disease, endocrine dysfunction, infection during odontogenesis.

Differential diagnosis: Diagnostic of dental calculus, LEH, osteophytes, degenerative joint disease, intervertebral disk disease and osteoarthritis.

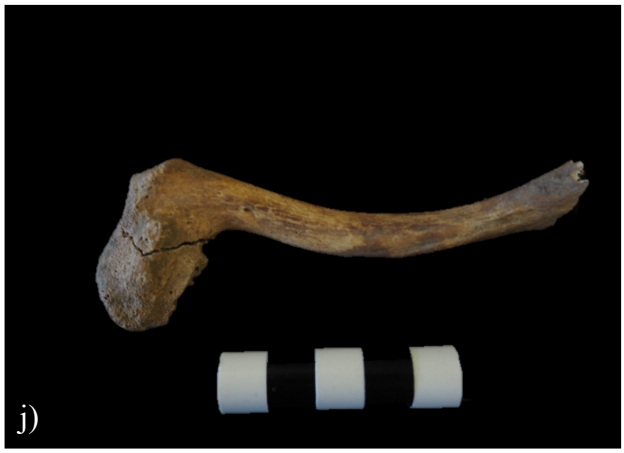
Site BiFj-37 Box 17 / 29 No 25C-S71

Sex Female Age-at-death 30-59 years-old

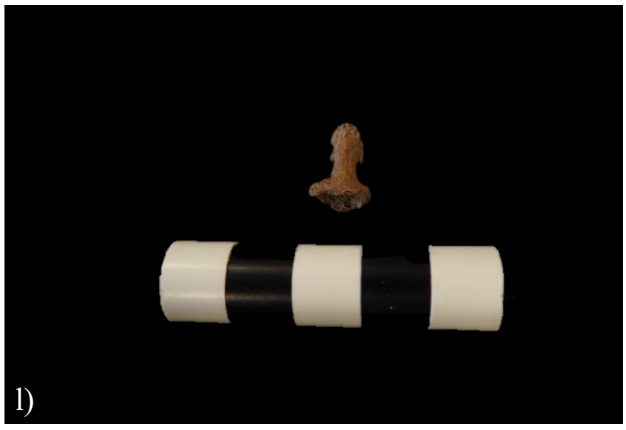



a)
b)
c)
d)
e)
f)
g)
h)


i)


j)


k)


l)


m)


n)

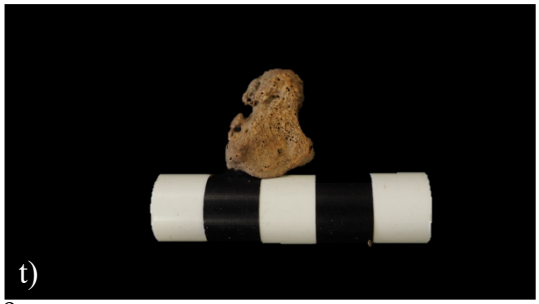

o)


p)


q)


r)


s)


t)

Individual: 25C-S71

ACI: 71.4

Age-at-death: 30-59 years-old

Sex: Female

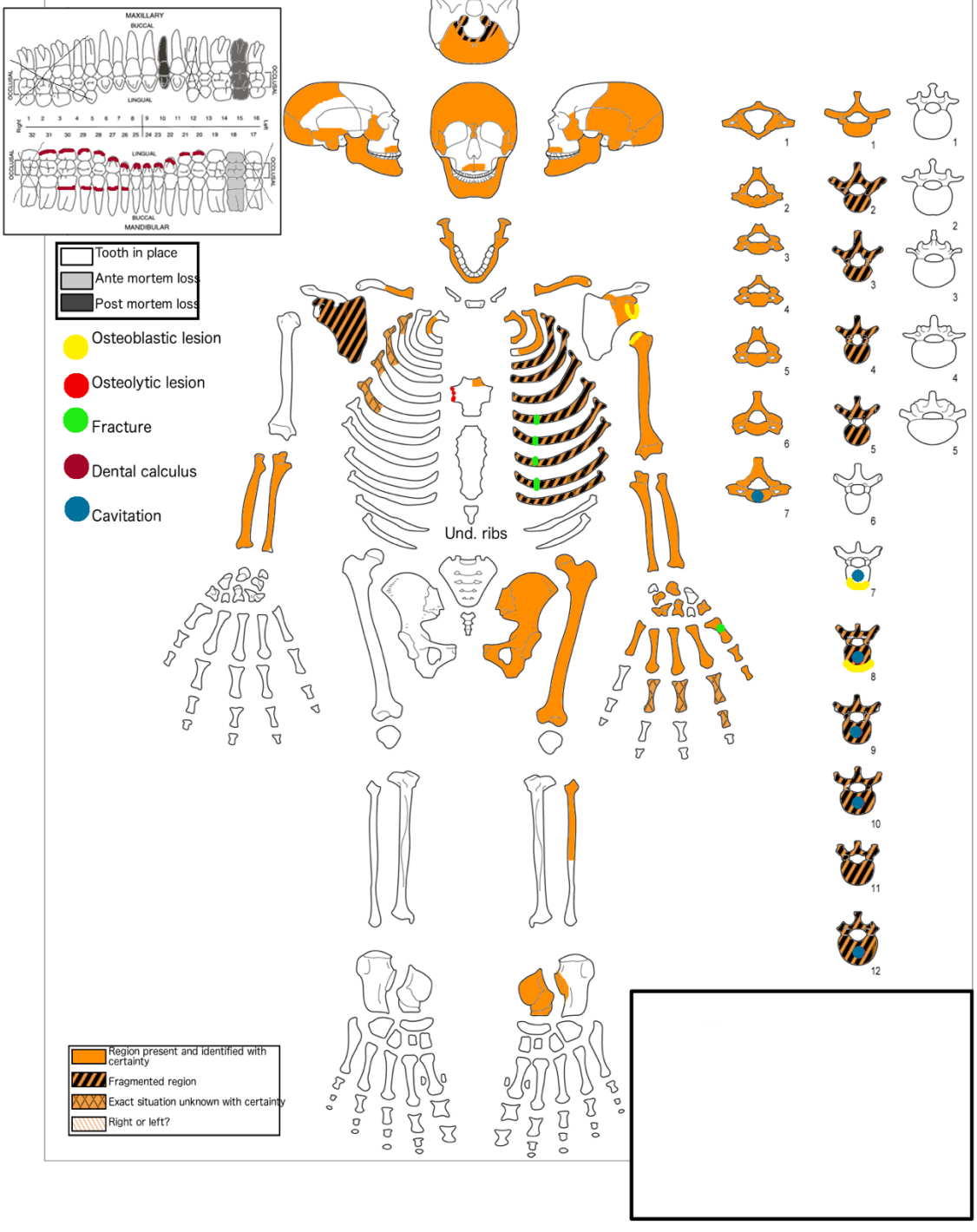
Description of pathological lesions: Porosities on the cranial vault (posterior view) (a), woven bone on the ectocranial surface of the occipital bone (posterior view) (b), bone formations or "lipping" s on the bodies of CV5-7 (anterior view) (c), bone formation or "lipping" on the body of an undetermined TV (lateral view) (d), fractures on 3 right rib fragments (lateral view) (example of one (e)), fractures on 12 rib fragments (lateral view) (example of three (f-h)), degeneration of the right and left glenoid cavities (medial view) (i), possible fracture of the left clavicula (superior view) (j), bone nodule on the lateral side of the distal epiphysis of the left humerus (anterior view) (k), new bone formation on the proximal and distal epiphysis of an undetermined distal hand phalanx (dorsal view) (l), degeneration of the posterior surface of the left patella (posterior view) (m), periosteal lesions on the diaphysis of the right (n) and left (o) femurs (anterior view), on the diaphysis of the right (p) and left (q) tibias, on the diaphysis of the right fibula (medial view) (r), on the distal metaphysis of the left fibula (anterior/lateral view) (s), new bone formations on the proximal and distal epiphysis of the right first distal foot phalanx (plantar view) (t).

Differential diagnosis: Diagnostic of osteophytes, trauma, degenerative joint disease, porotic hyperostosis (confirmed by radiographs), **typical of** osteochondritis dissecans, non-specific infection, bone tumour.

Site BiFj-37 Box 4 / 29 No 25C-S72

Sex Male Age-at-death 30-50 years

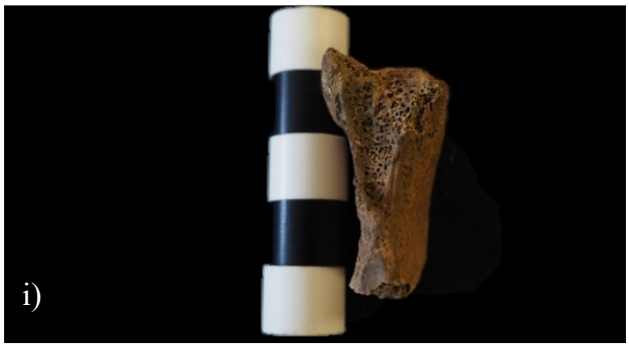
ACI 47.92



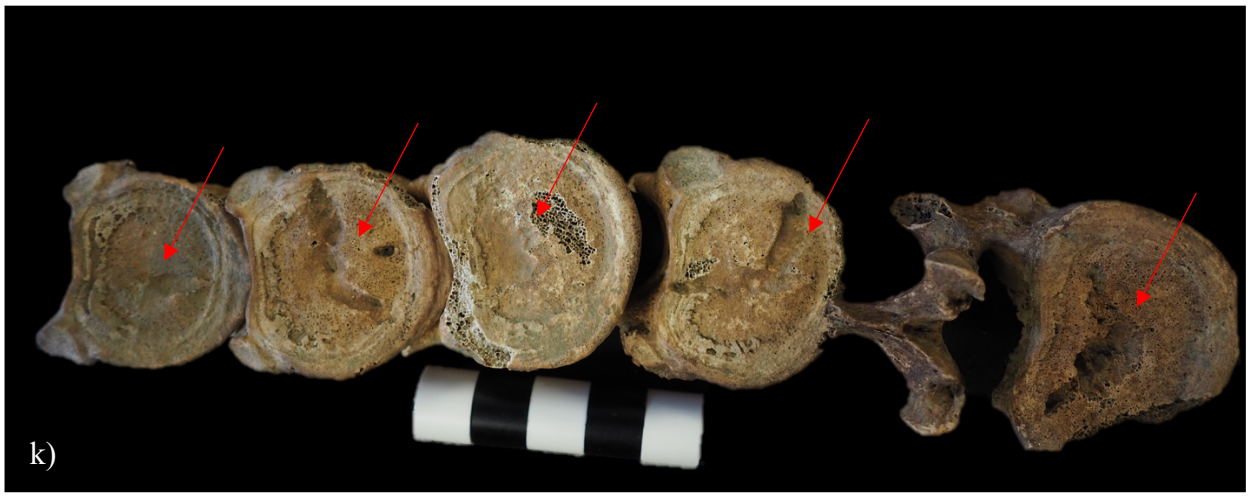

a)
b)
c)
d)
e)
f)


g)


h)


i)


j)


k)

Individual: 25C-S72

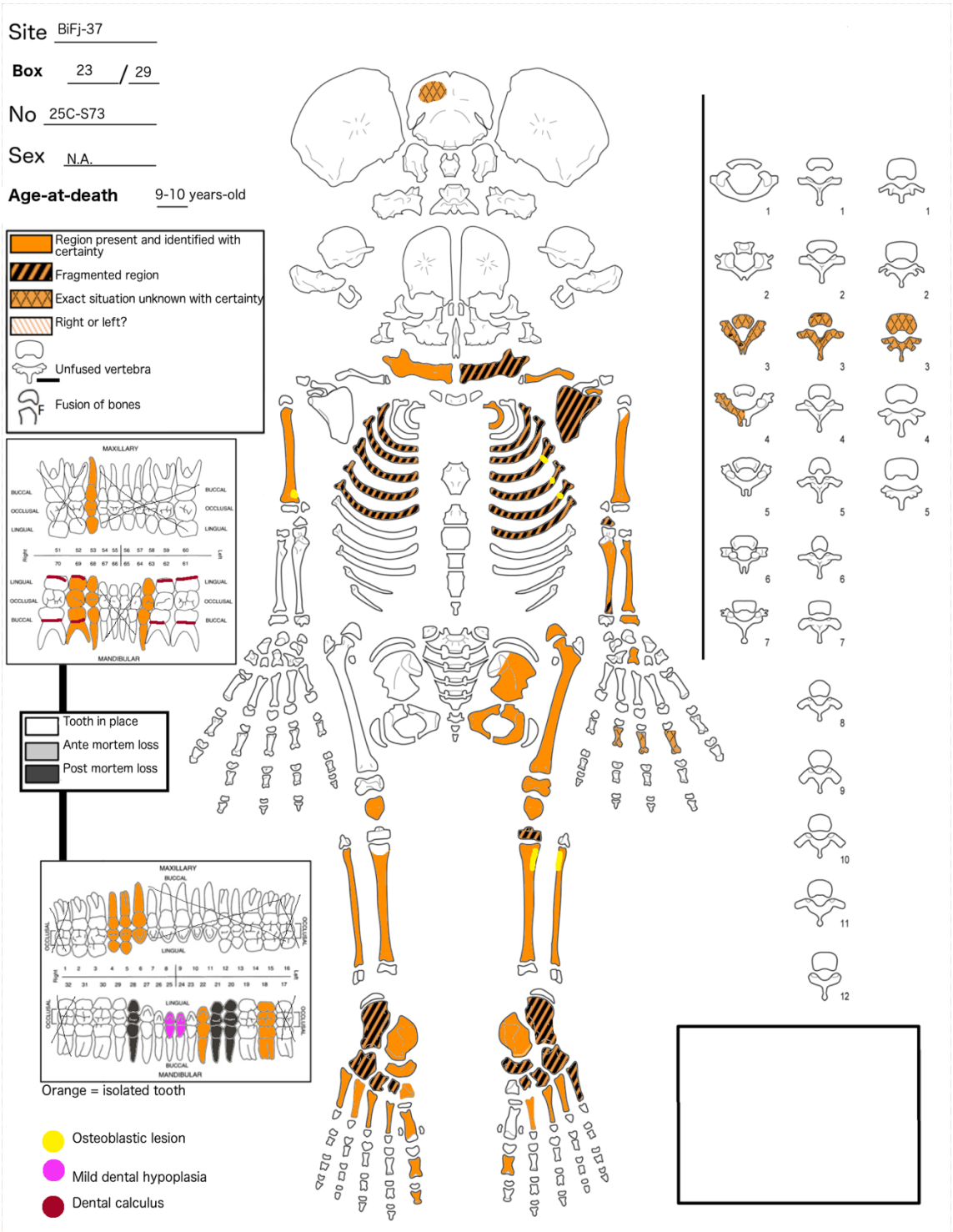
ACI: 47.92

Age-at-death: 30-50 years-old

Sex: Male

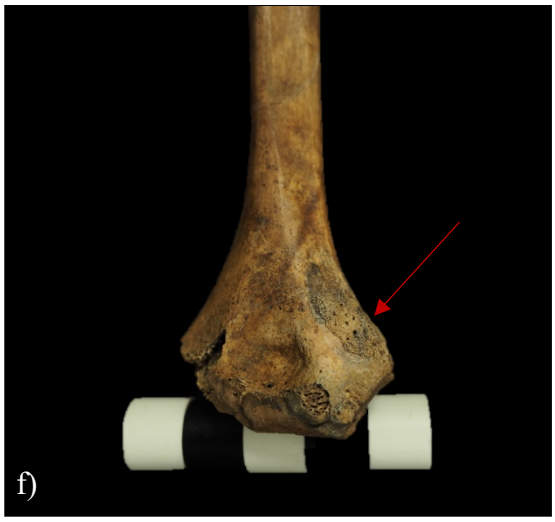
Description of pathological lesions: Calcified plaque on lower teeth (buccal view) (a) (lingual view) (b), osteoblastic and lytic lesions all around the edge of the left glenoid cavity (medial view) (c) (anterior view) (d), new bone formations and eburnation on the left humeral head (medial view) (e), shrinking of the left glenoid cavity and matching new bone formations on the left glenoid cavity and the left humeral head (anterior view) (f), healed fracture of left MC1 (medial view) (g), fractures on 4 rib fragments (lateral view) (h),lytic lesions on the right lateral side of the manubrium (lateral view) (i), cavitation on undetermined CV (superior view) (h), cavitation on TV7-8-9-10-12 (superior view) (j); bone formations or "lipping" on the inferior and superior edges of TV7 and on the inferior edge of the TV8 (superior view) (k);

Differential diagnosis: Diagnostic of dental calculus, Schmorl's nodes, osteophytes, trauma, degenerative joint disease.


Site BiFj-37
Box 23 / 29
No 25C-S73
Sex N.A.
Age-at-death 9-10 years-old
Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?
Unfused vertebra
Fusion of bones
MAXILLARY
MANDIBULAR
Tooth in place
Ante mortem loss
Post mortem loss
Orange = isolated tooth
Osteoblastic lesion
Mild dental hypoplasia
Dental calculus


a)
b)
c)
d)


e)


f)


g)


h)

Individual: 25C-S73

ACI: N.A.

Age-at-death: 9-10 years-old

Sex: N.A.

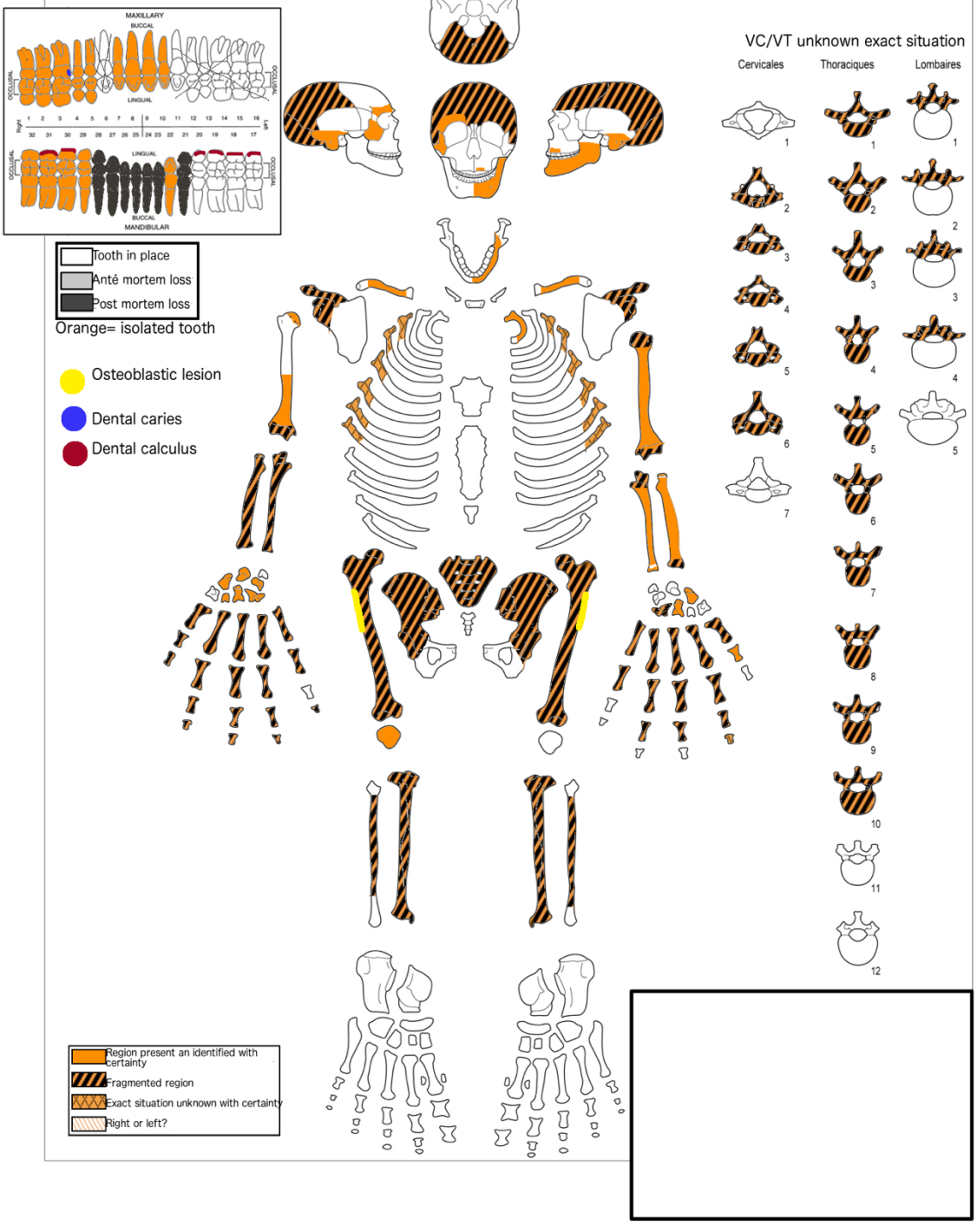
Description of pathological lesions: Lines on the right and left I1 (buccal view) (a), calcified plaque on lower decidual m_1 and m_2 (lingual view) (b), new bone formation on the visceral surface of 3 left rib fragments (visceral view) (c-e), woven bone on the medial side of the right humeral distal metaphysis (anterior view) (f), periosteal lesions on the lateral side of the left tibia's diaphysis (lateral view) (g) and on the left fibula's metaphysis (medial view) (h).

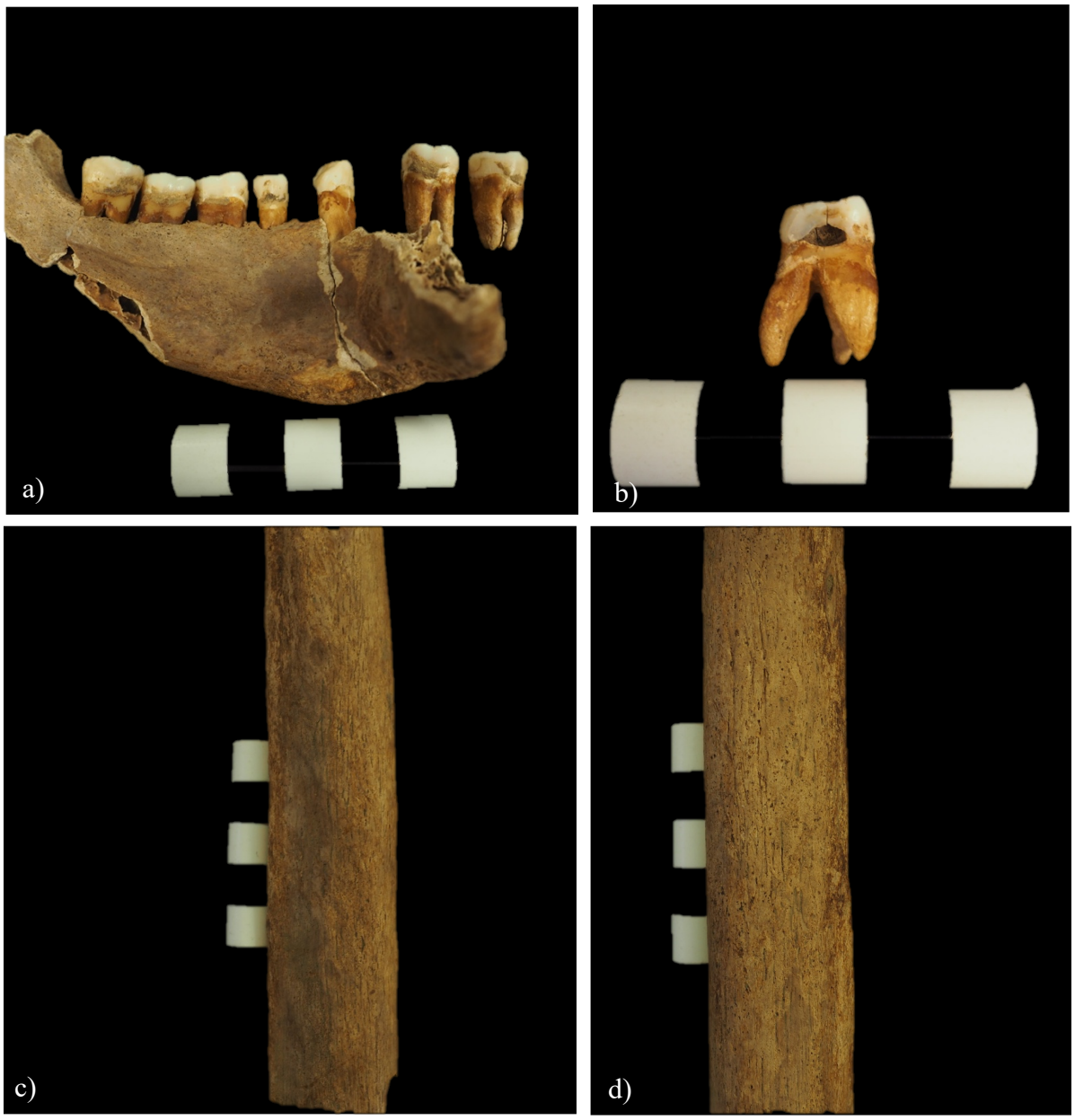
Possible aetiologies: Childhood disease-nutritional deficiencies, periapical inflammation or trauma to a deciduous tooth, fever, disease, endocrine dysfunction, infection during odontogenesis.

Differential diagnosis: Diagnostic of dental calculus, LEH, **highly consistent with** lung infection.

Site BiFj-37 Box 26 / 29 No 25C-S94

Sex Male tendency Age-at-death 24-35 years old





Individual: 25C-S94;

ACI:35.28;

Age-at-death: 24-35 years-old;

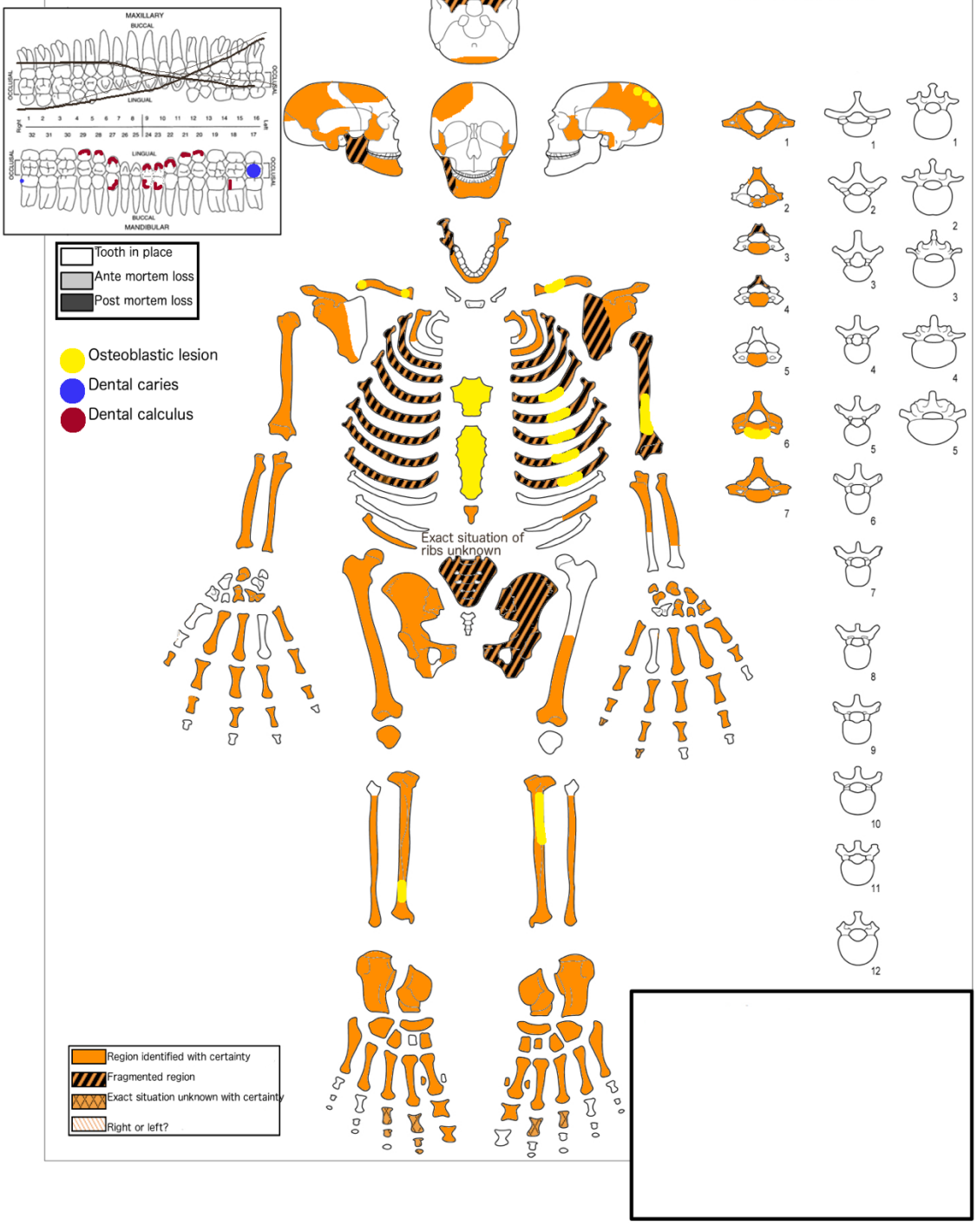
Sex: Likely male

Description of pathological lesions: Calcified plaque on lower premolars and molars (lingual view) (a), cavity on the right M^1 (mesial view) (b), periosteal lesions on the lateral side of the diaphysis of the right (c) and left femurs (d).

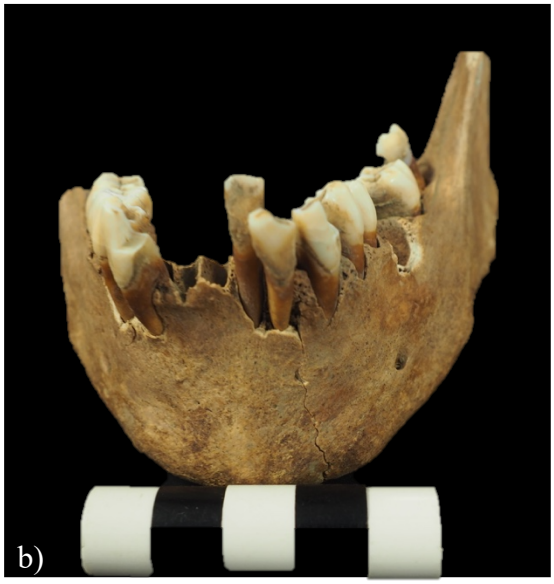
Differential diagnosis: Diagnostic of dental calculus and dental caries**, typical of** non-specific infection.

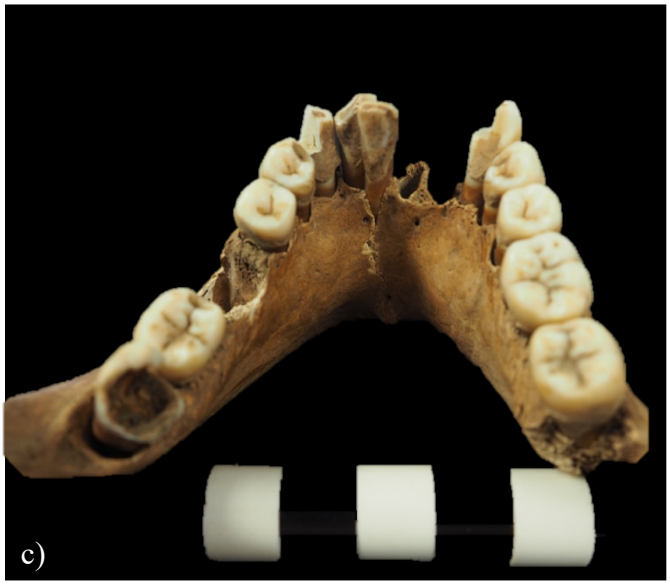
Site BiFj-37 Box 3/12 / 29 No 25C-S106

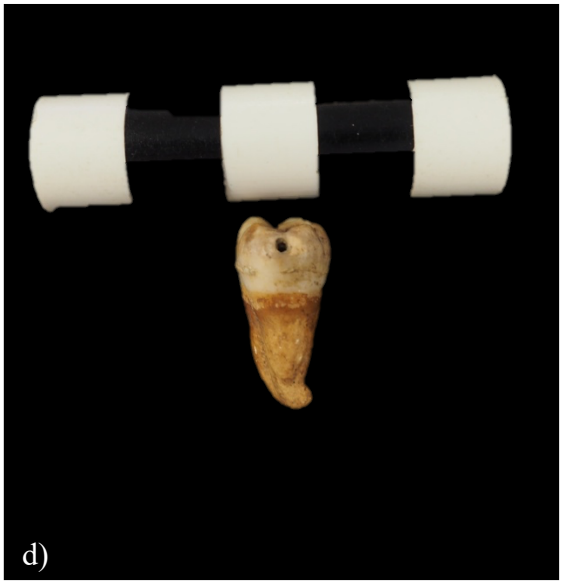
Sex Male Age-at-death 30-59 years-old




a)


b)


c)


d)


e)


f)
g)
h)
i)
j)
k)

Individual: 25C-S106

ACI: 68.24

Age-at-death: 30-59 years-old

Sex: Masculine

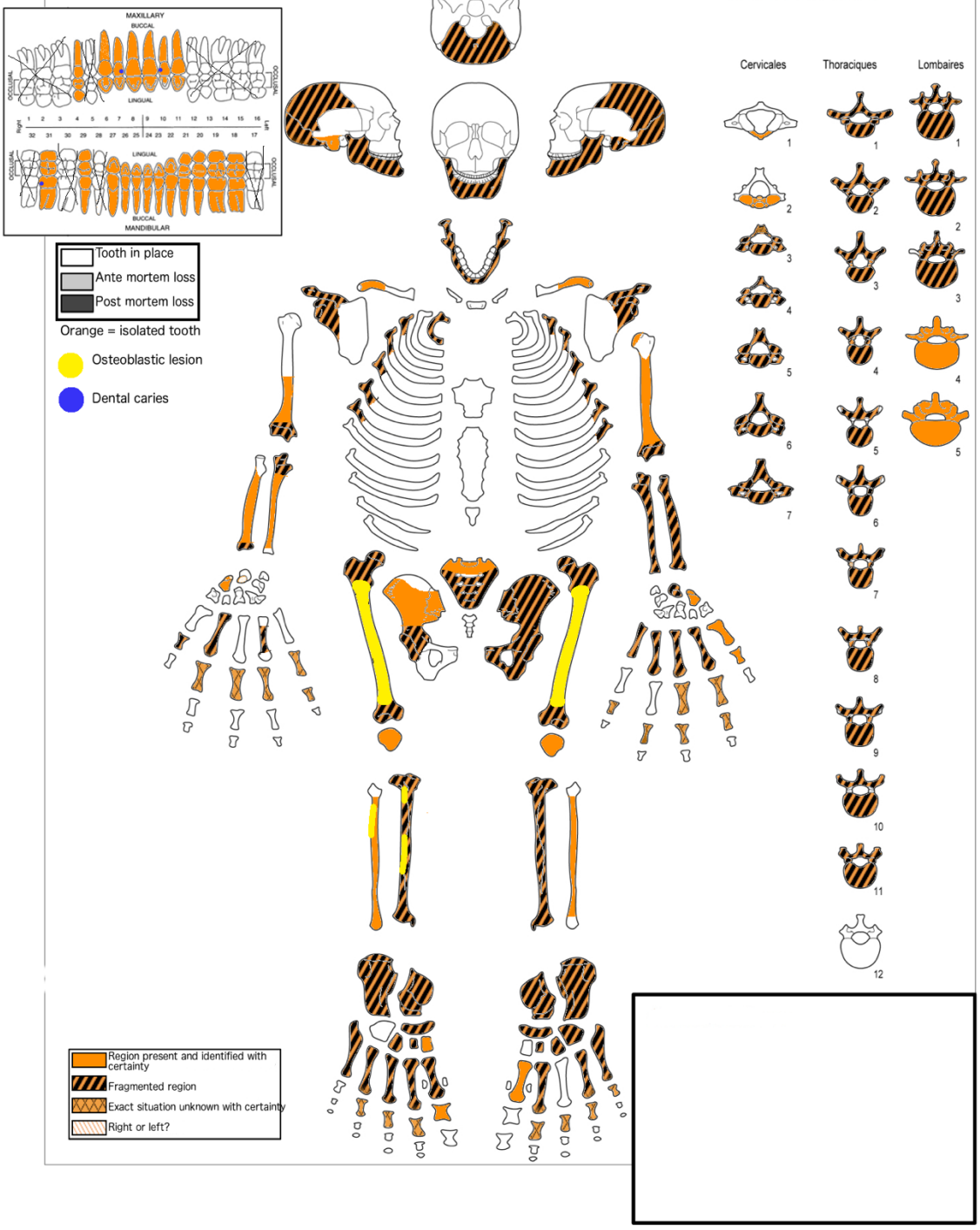
Description of pathological lesions: Hair-on-end lesions on the left parietal bone (endocranial view) (a), calcified plaque on lower front teeth (buccal view) (b) lingual view (c), cavity on left M_3 (occlusal view) (c) and right M3 (distal view) (d), new bone formation on the external surface of 5 left rib fragments (lateral view) (example of one (e)), new bone formation on the sternum (anterior view) (f), new bone formation on the anterior surface of the body of CV6 (anterior view) (g), woven bone on the superior side of the lateral epiphysis of the right clavicle and on the inferior side of the medial epiphysis of the right clavicle (superior view) (h), new bone formation on the anterior side of the diaphysis of the left clavicle (postero-superior view) (i), new bone formation on the diaphysis of the left humerus (posterior view) (j), new bone formation on the metaphysis of the right tibia (posterior view) (k), periosteal lesions on the diaphysis of the left tibia (lateral view) (l).

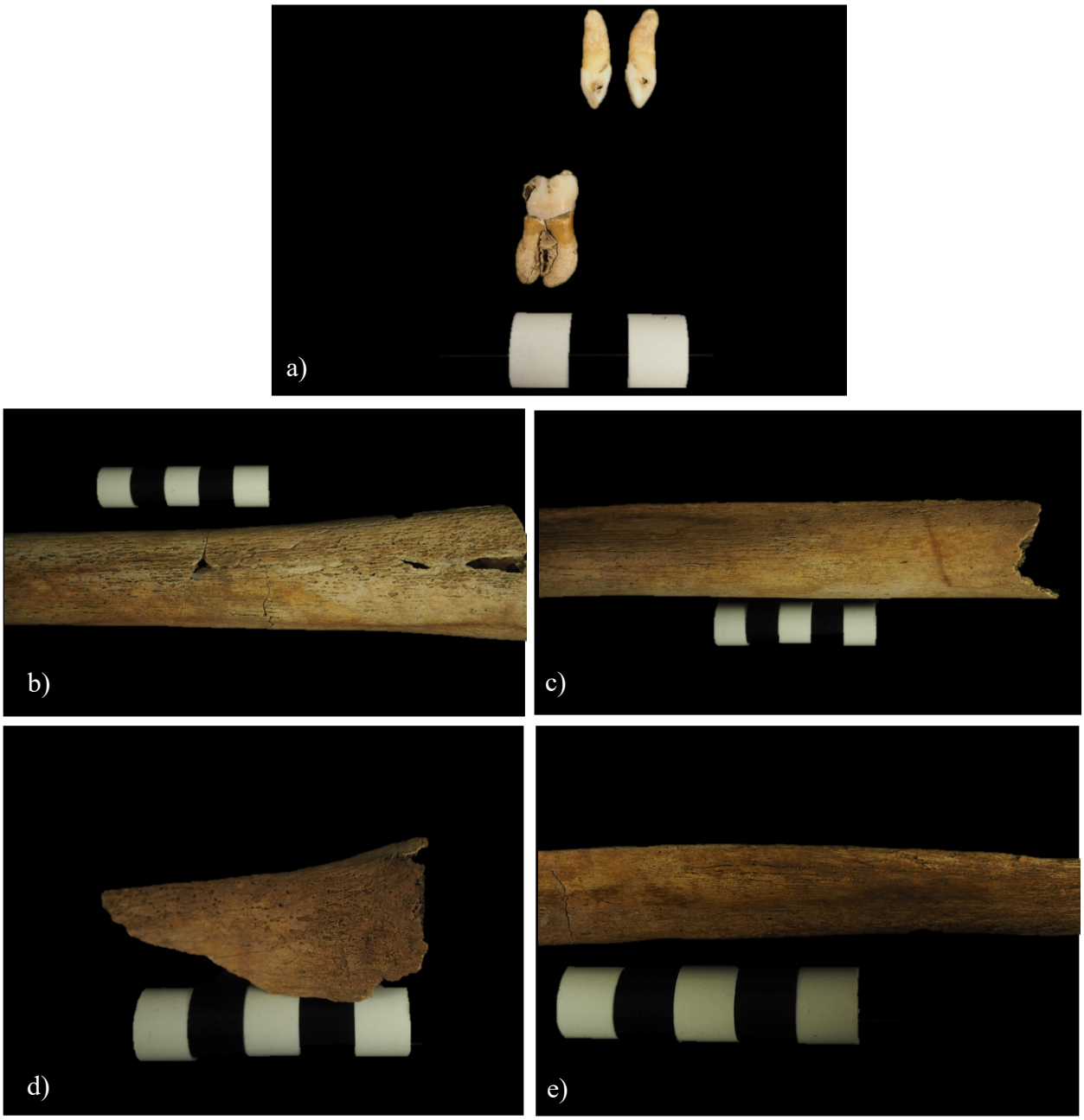
Possible aetiologies: Trauma, primary and secondary infections of the meninges, tumours, TB, syphilis and vitamin deficiencies of A, C and D.

Differential diagnosis: Diagnostic of dental calculus, dental caries, endocranial lesions, **highly consistent** with hypertrophic (pulmonary) osteoarthropathy, **typical of** treponemal disease: venereal syphilis.

Site BiFj-37 Box 25 / 29 No 26B-S23

Sex Undetermined Age-at-death 16-20 years-old





Individual: 26B-S23

ACI: 34.48

Age-at-death: 16-20 years-old

Sex: Undetermined

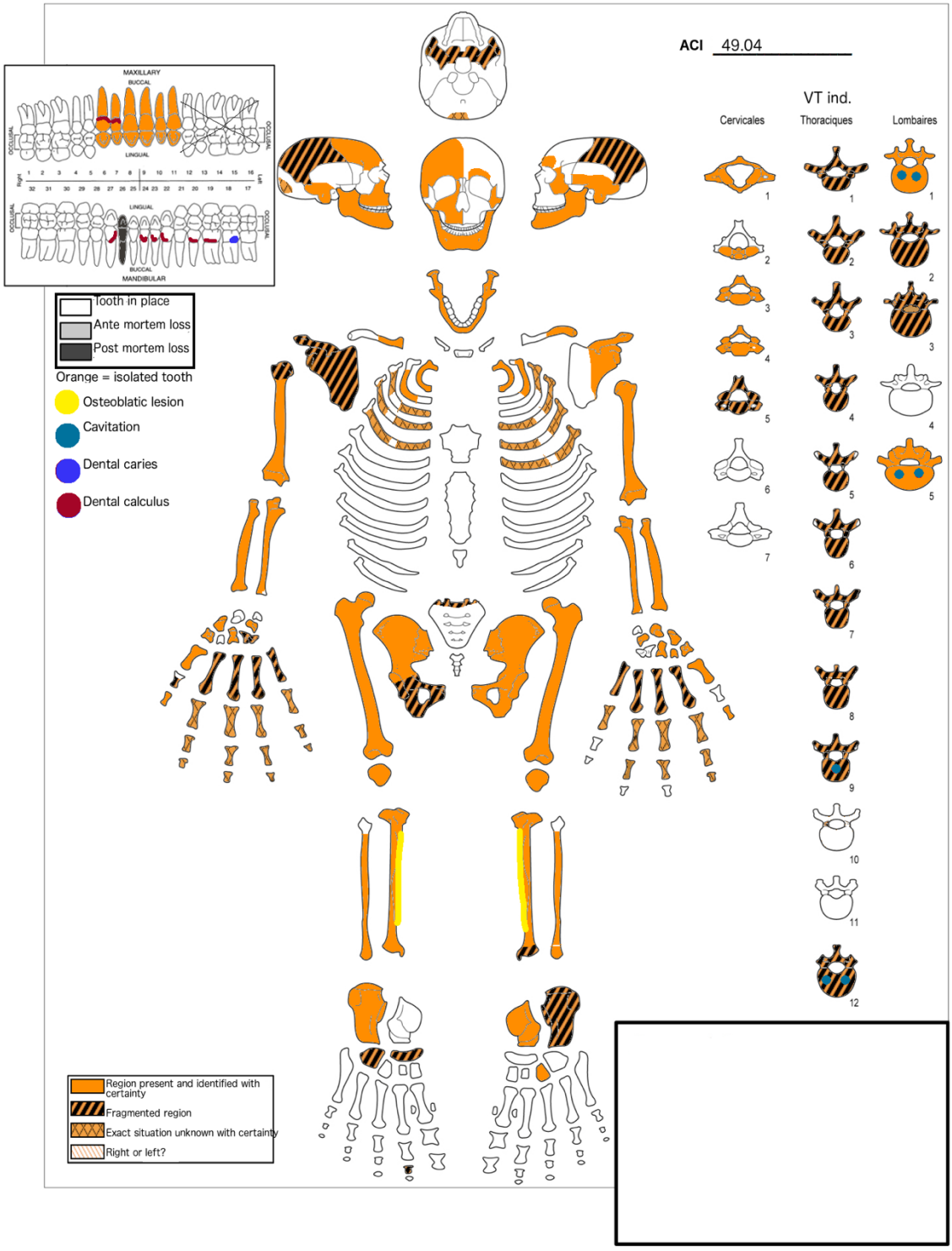
Description of pathological lesions: Cavity on right and left I^2 (mesial view) and on right M_2 (distal view) (a), periosteal lesions on the diaphysis of the right femur (anterior view) (b), periosteal

lesions on the diaphysis of the left femur (lateral view) (c), woven bone on the right tibia (d), periosteal lesions on the diaphysis of the right fibula (lateral/anterior views) (e).

Differential diagnosis: Diagnostic of dental caries, **typical of** non-specific infection.

Site BiFj-37 Box 4 / 13 No 29L-S11

Sex Male **Age-at-death** More than 40 years-old




a)
b)
c)
d)
e)
f)
g)

Individual: 29L-S11

ACI: 49.04

Age-at-death: More than 40 years-old

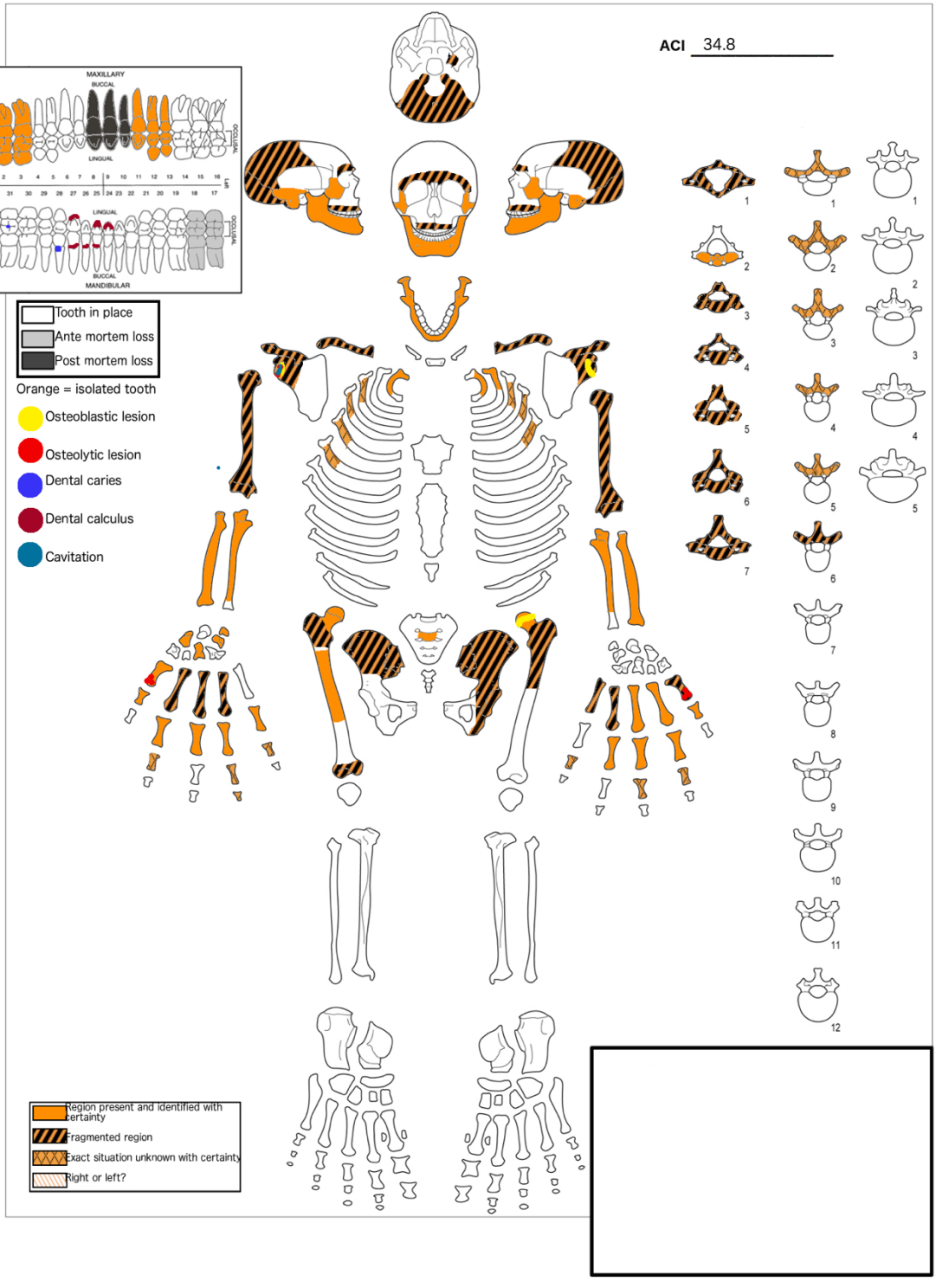
Sex: Male

Description of pathological lesions: Calcified plaque on lower and upper teeth (buccal view) (a-b), cavity on the left M_2 (buccal view) (c), cavitation on the body of an und. TV (superior side) (d), cavitation on the body of TV12, LV1 and LV5 (inferior side) (e), new bone formation on the diaphysis of right (f) and left tibias (g) (medial view).

Differential diagnosis: Diagnostic of dental calculus, dental caries, Schmorl's nodes, degenerative joint disease, **typical of** non-specific infection.

Site BiFj-37 Box 5 / 13 No 30B-S1

Sex Male Age-at-death 18-29 years-old




a)
b)
c)
d)
e)
f)

Individual:30B-S1

ACI: 34.8

Age-at-death: 18-29 years-old

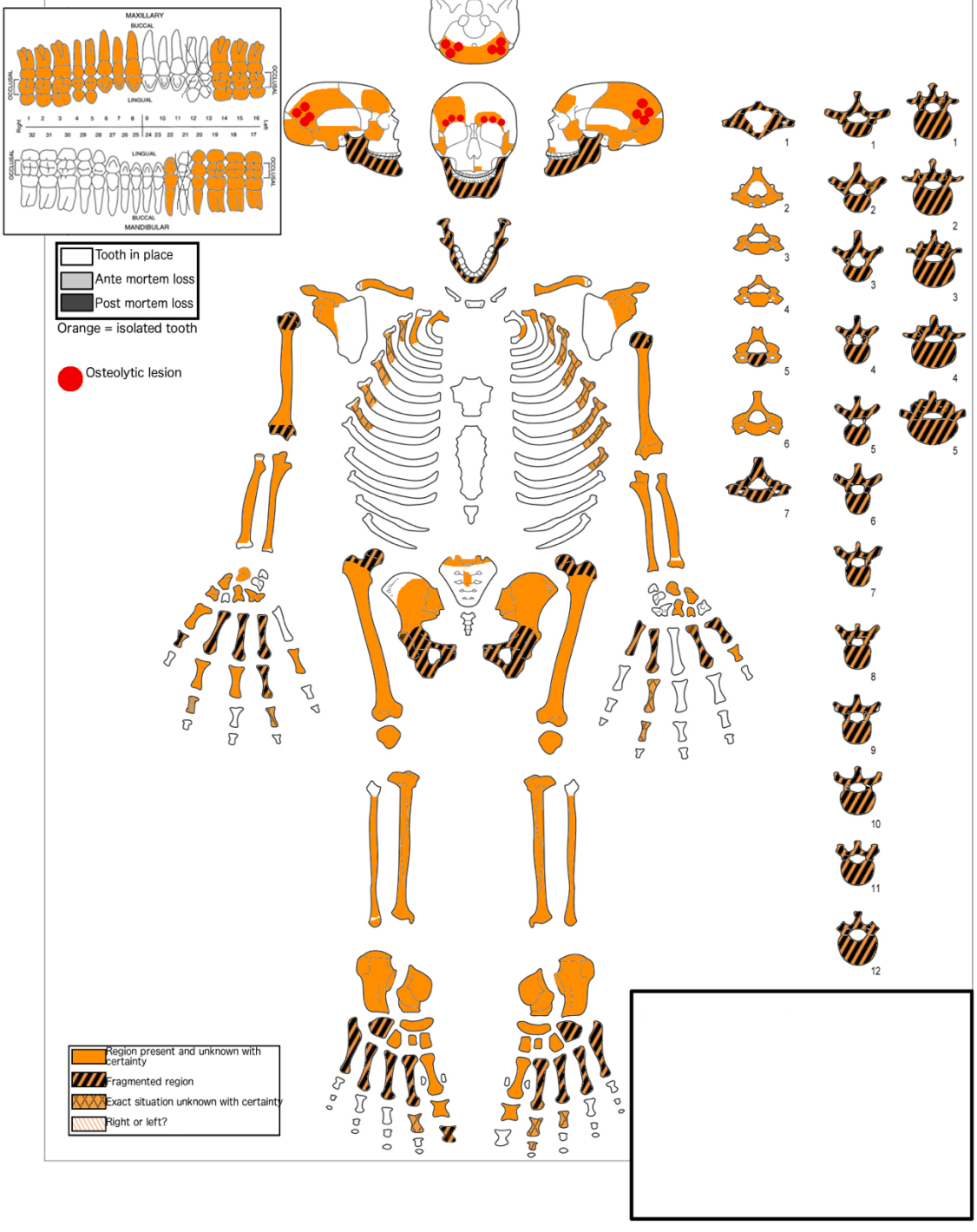
Sex: Male

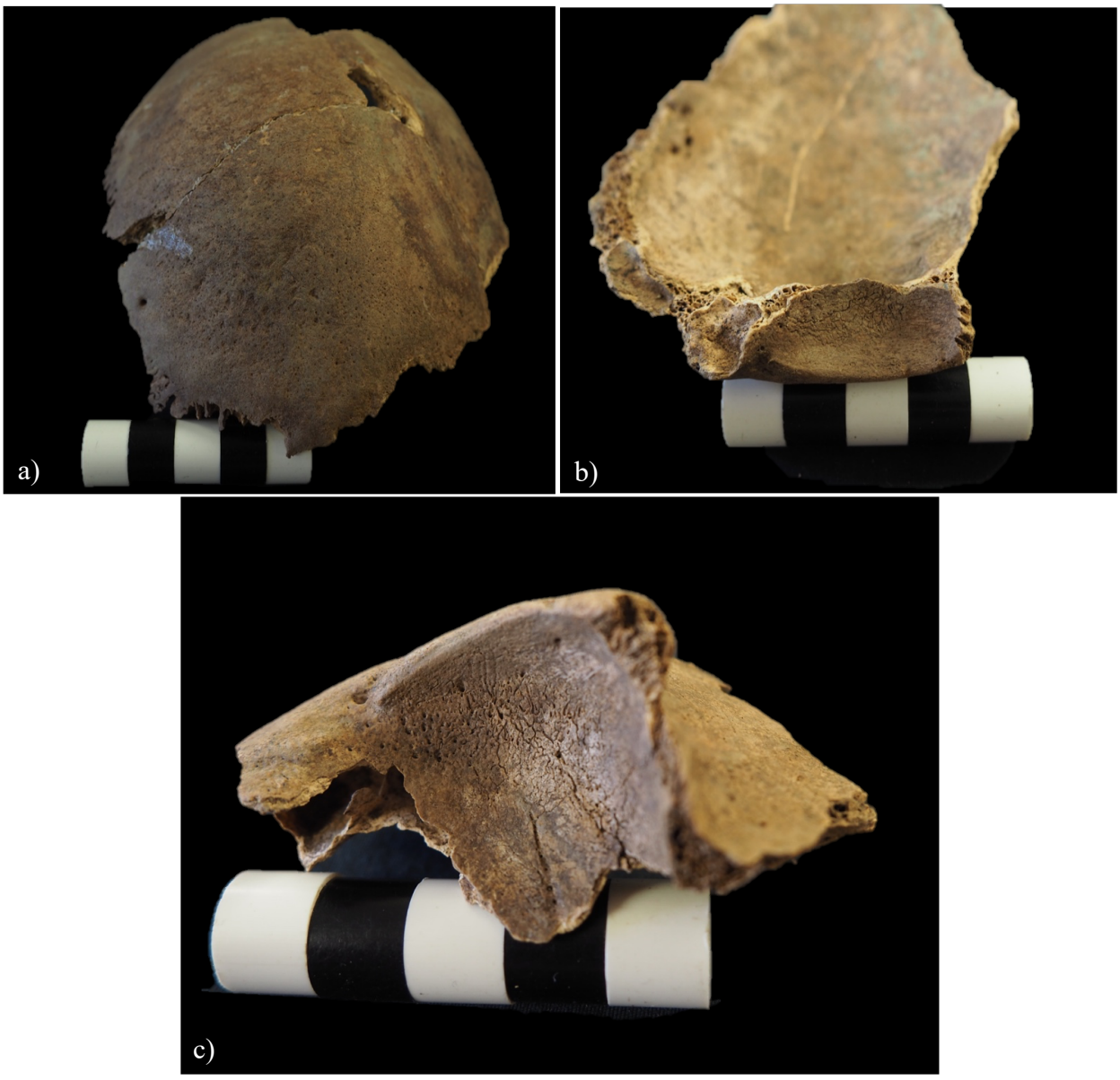
Description of pathological lesions: Cavity on the right PM_1 (buccal view) and calcified plaque on lower teeth (buccal view) (a) (lingual view) (b), cavitation and new bone formation on the edge of the right glenoid cavity (medial view) (c), new bone formation on the edge of the left glenoid cavity (medial view) (d), lytic lesions on the distal epiphysis of right and left MC1 (palmar view) (e) and bone nodules on the lateral side of right and left MC1 (e), new bone formation around the edge of the left femoral head (superior view) (f).

Differential diagnosis: Diagnostic of dental caries, dental calculus, osteophytes, degenerative joint disease.

Site BiFj-37 Box 7 / 13 No 30D-S5

Sex Male tendency Age-at-death 20-49 years-old





Individual: 30D-S5

ACI: 59.8

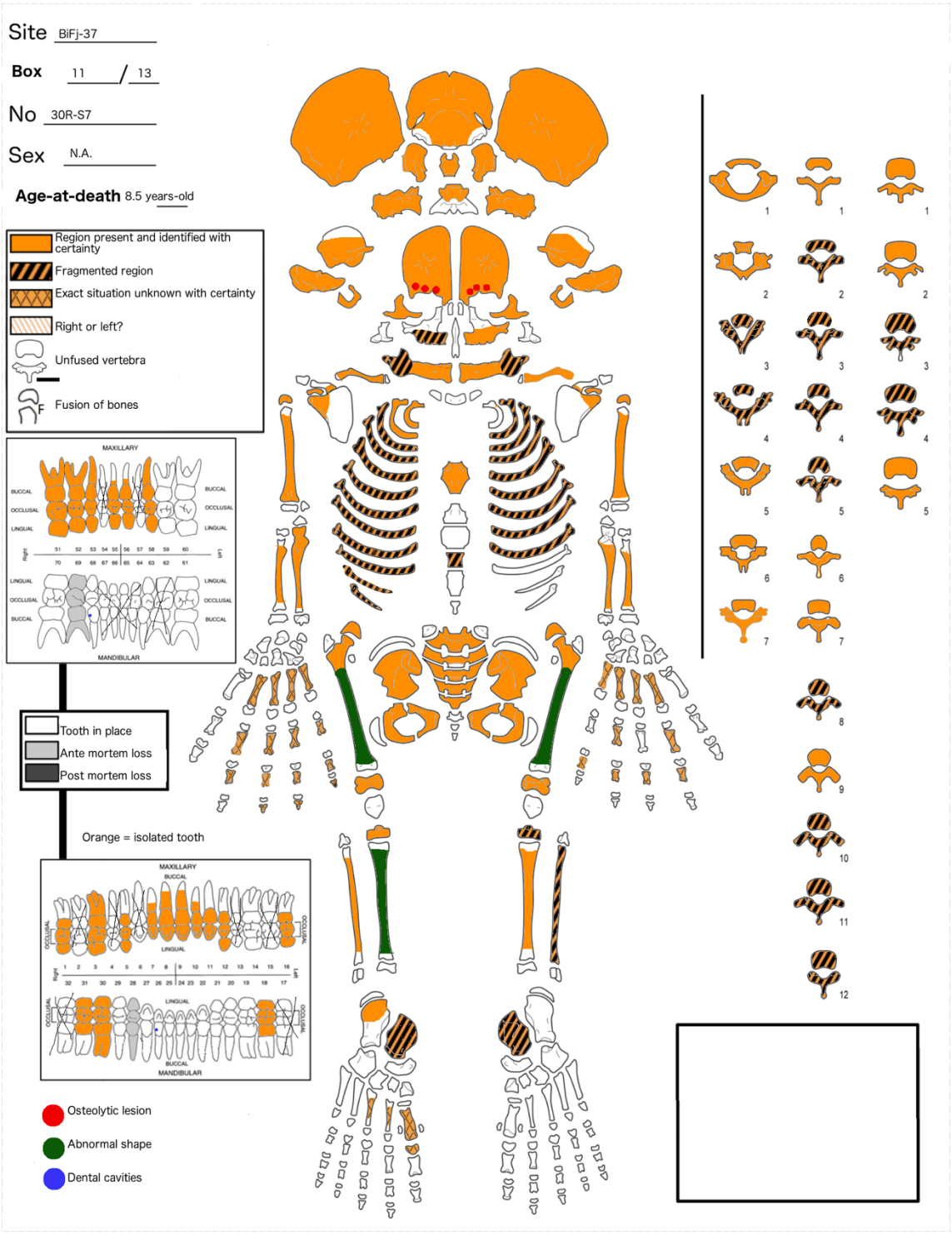
Age-at-death: 20-39 years-old

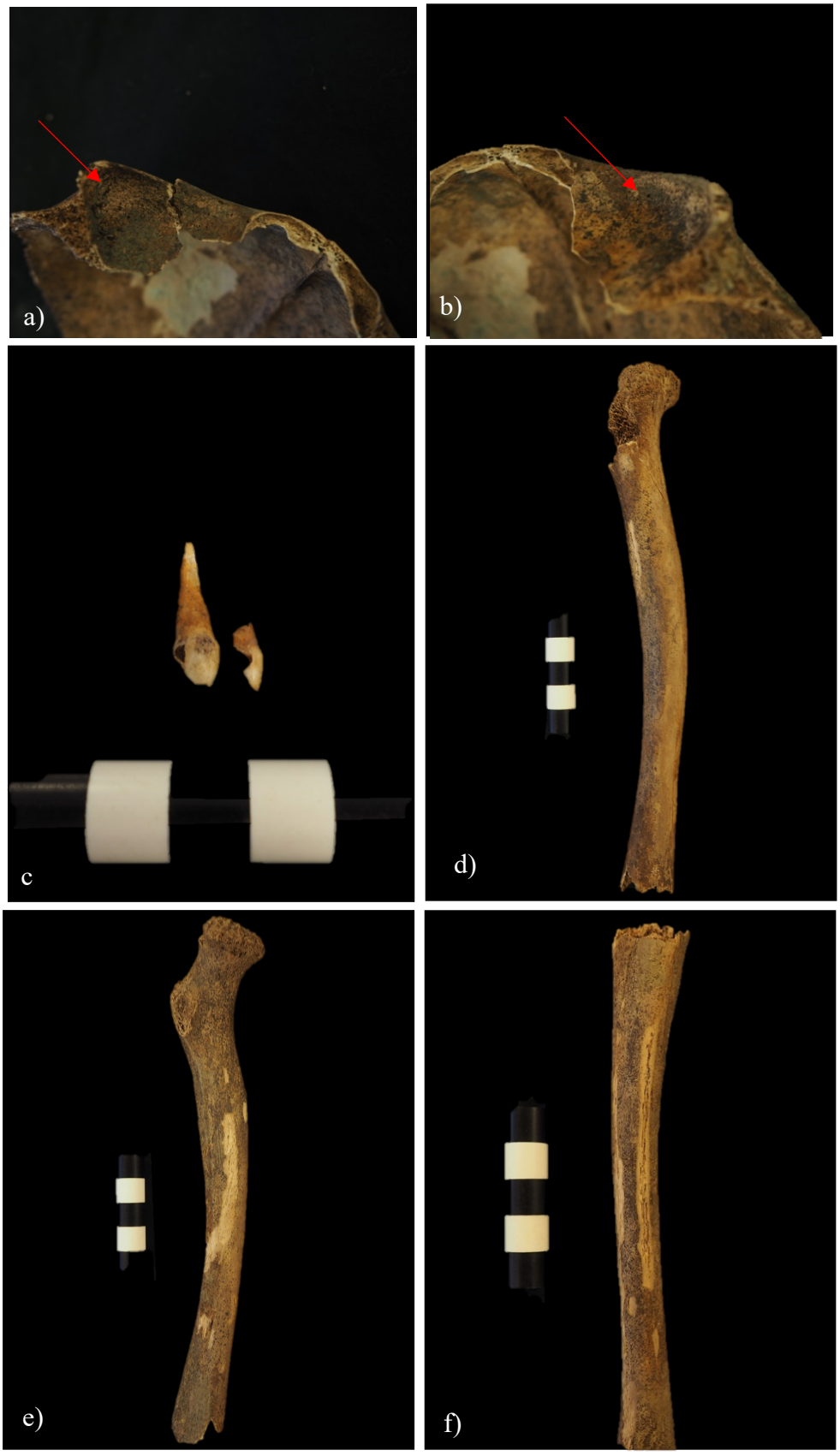
Sex: Likely male

Description of pathological lesions: Porosities on the cranial vault (posterior view) (a), orbital roof porosities on the right (b) and left (c) orbits (inferior view).

Possible aetiologies: Infectious disease, scurvy, B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

Differential diagnosis: Diagnostic of *cribra orbitalia*, porotic hyperostosis (confirmed by radiographs, vault thickening)**.**


Site BiFj-37
Box 11 / 13
No 30R-S7
Sex N.A.
Age-at-death 8.5 years-old
Region present and identified with certainty
Fragmented region
Exact situation unknown with certainty
Right or left?
Unfused vertebra
Fusion of bones
MAXILLARY
MANDIBULAR
Tooth in place
Ante mortem loss
Post mortem loss
Orange = isolated tooth
Osteolytic lesion
Abnormal shape
Dental cavities


a)
b)
c
d)
e)
f)

Individual: 30R-S7;

ACI: N.A.;

Age-at-death: 8.5 years-old;

Sex: N.A.;

Description of pathological lesions: Orbital roof porosities on the right (a) and left (b) orbits (inferior view), cavity on the right decidual lower canine and I_2 (distal view) (c), antero-posterior bending of the right (lateral view) (d) and left (medial view) (e) femurs, lateral bending of the right tibia (anterior view) (f), lateral bending of right and left fibulas (g).

Possible aetiologies: Infectious disease, B12 deficiency megaloblastic anaemia, iron-deficiency anaemia.

Differential diagnosis: Diagnostic of dental caries, *cribra orbitalia*, **typical of** rickets.

ANNEXE N

Summarized data on the palaeopathological analysis (macroscopic and histological) for each individual

Individual (code)	ACI	Sex	Age-at-death	Differential diagnosis	Presence /absence of IGD (including degree 1)	Presence /absence of IGD (excluding degree 1)	N IGD episodes (total)	N IGD episodes (observable)	Severity of each IGD episode	Age of onset of each IGD episode	*In utero* IGD
10A-S3 (1)	6.16	Und.	16-22 yrs.	**Diagnostic of** LEH, dental caries and dental calculus, **consistent with** trauma	P	P	N.O.	2	**Episode 1**: grade 1 **Episode 2**: grade 2	**Episode 1:** around birth **Episode 2**: around 1.5 yrs.	No
11A-S3	47.52	MT	16-20 yrs.	**Diagnostic of** dental calculus, dental caries and inflammation of biceps muscle insertion	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: less than 1 yr.	No
11F-S1	57.04	M	30 yrs. +	**Diagnostic of** osteophytes, Schmorl's nodes and intervertebral disk disease (IVD)	P	P	N.O.	2	**Episode 1**: grade 2 **Episode 2**: grade 2	**Episode 1**: *in utero* to around 1 yr. **Episode 2**: around 2 yrs.	Yes
11F-S3	35.68	FT	16-21 yrs.	**Diagnostic of** dental caries and endocranial lesions	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around birth	No
11F-S6	37.84	FT	20-49 yrs. (20-29 yrs. according to the vertebras)	N.O.	A	A	N.O.	N.A.	N.A.	N.A.	No
17A-S1	41.8	M	15-20 yrs.	**Typical of** early degeneration of the left temporomandibular joint	P	P	N.O.	2	**Episode 1**: grade 2 **Episode 2**: grade 1	**Episode 1**: *in utero* to 1yr **Episode 2:** c. 2 yrs.	Yes
17Z-S1	70.00	M	15-20 yrs.	**Diagnostic of** Schmorl's nodes, non-specific lung infection, *cribra orbitalia*	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around 1.5 yrs.	No

17Z-S7	52.34	FT	+40 yrs.	**Diagnostic of** endocranial lesions, dental calculus, dental caries, Schmorl's nodes, degenerative joint disease, **typical of** scurvy according to the distribution pattern of periosteal lesions and lytic lesions	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 2 yrs.	No
20A-S7	86.92	MT	15-23 yrs.	**Diagnostic of** dental calculus, LEH, Schmorl's nodes, degenerative joint disease	P	P	2	2	**Episode 1**: grade 2. **Episode 2**: grade 1	**Episode 1**: near birth **Episode 2**: around 1.5 yrs.	No
20A-S13	17.84	F	16-29 yrs.	**Diagnostic of** dental calculus, osteophyte, **highly consistent with** degenerative joint disease, unilateral right aplasia and unilateral left hypoplasia	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around 1-2.5 yrs.	No
20C-S6	77.24	FT	20-29 yrs.	**Diagnostic of** Schmorl's nodes, **consistent with** trauma, lung infection, degenerative joint disease	P	P	N.O.	2	**Episode 1**: grade 2. **Episode 2**: grade 1	**Episode 1**: birth **Episode 2**: around 3 yrs.	No
20C-S23	25.88	F	20-30 yrs.	**Diagnostic of** endocranial lesions	P	A	N.O.	1	**Episode 1**: less than grade 1	**Episode 1**: 3 yrs.	No
20D-S14	57.32	FT	17-20 yrs.	**Diagnostic of** dental calculus, dental caries and *cribra orbitalia.*, **typical of** non-specific infection	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to around 2 yrs.	No
20E-S3	56.76	MT	20-29 yrs.	**Diagnostic of** dental calculus, Schmorl's nodes, degenerative joint disease, **highly consistent with** osteoarthritis	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to about 1yr.	No
20E-S8	5.6	Und.	18-24 yrs.	N.O.	A	A	N.O.	N.A.	N.A.	N.A.	No
20F-S2	N.A.	N.A.	6.5 yrs.	**Diagnostic of** dental calculus, dental caries and *cribra orbitalia*. **highly consistent with** scurvy according to the distribution of the lytic and osteoblastic lesions	A	A	N.O.	N.A.	N.A.	N.A.	No
20F-S10	79.44	M	±30 yrs.	**Diagnostic of** dental calculus, Schmorl's nodes, degenerative joint disease, osteoarthritis	P	P	N.O.	1	**Episode 1**: grade 1-2	**Episode 1**: birth to around 2.5 yrs.	No
20F-S22	95.528	M	19-20 yrs.	**Diagnostic of** Schmorl's nodes, osteoarthritis, degenerative joint disease, *cribra orbitalia*, **typical of** residual rickets, empyema, actinomycosis, or pulmonary TB with haematogenous spread of infection, a combination of	A	A	N.O.	N.A.	N.A.	N.A.	No

				respiratory disease and other osteolytic disease							
20Z-S5	N.A.	N.A.	18 months ± 6 months	**Diagnostic of** *cribra orbitalia*, **highly consistent with** scurvy according to the distribution pattern of the lytic lesions	N.O.	N.O.	N.O.	N.O.	N.O.	N.O.	N.O.
21E-S2	33.68	MT	27-66 yrs. (mid. 45.6 yrs.)	N.O.	P	A	N.O.	1	**Episode 1**: less than 1-1	**Episode 1**: birth to around 1 yr.	No
21E-S17	58.72	M	+40 yrs.	**Diagnostic of** dental calculus, Schmorl's nodes, maxillary sinusitis, trauma, degenerative joint disease, *cribra orbitalia*	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: in utero to about 2 yrs.	Yes
21J-S2	9.92	F	16-22 yrs.	N.O.	P	A	N.O.	1	**Episode 1**: grade less than 1-1	**Episode 1**: around 1 yr.	No
21N-S4	27.04	FT	16-25 yrs.	N.O.	A	A	N.O.	N.A.	N.A.	N.A.	No
21N-S8	23.88	FT	17-22 yrs.	N.O.	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 1 yr.	No
21S-S4	65.56	FT	20-36 yrs.	**Diagnostic of** dental calculus, osteophyte, degenerative joint disease	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: *in utero* to about 1 yr.	Yes
21U-S6	N.A.	N.A.	7-9.5 yrs.	**Diagnostic of** *cribra orbitalia*	A	A	N.O.	N.A.	N.A.	N.A.	No
22A-S1	13.48	F	18-29 yrs.		A	A	N.O.	N.A.	N.A.	N.A.	No
22A-S16	13.52	Und.	+30 yrs.	**Diagnostic of** dental calculus, dental caries, intervertebral disk disease and osteoarthritis	A	A	N.O.	N.A.	N.A.	N.A.	No
22C-S10	47.00	MT	20-29 yrs.	**Diagnostic of** dental calculus, **typical of** non-specific infection, degenerative joint disease.	P	P	N.O.	1	**Episode 1**: grade 1-2	**Episode 1**: birth to around 3 yrs.	No
23A-S2	73.12	MT	30-59 yrs.	**Diagnostic of** dental calculus, dental caries, osteophytes, inflammation of biceps muscle insertion, osteoarthritis, degenerative joint disease, *cribra orbitalia*, typical of bone tumour	P	P	N.O.	1	**Episode 1**: grade 1-2	**Episode 1**: birth to 1.5 yrs.	No
23E-S10	61.2	F	16-29 yrs.	N.O.	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to around 1.5 yrs.	No
24L-S4	46.84	M	20-49 yrs. (more 20-29 yrs. According to the vertebras)	**Diagnostic of** dental calculus, dental caries, Schmorl's nodes, osteophytes, intervertebral disk disease, **typical of** osteoma, residual rickets	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: around birth to 1 yr.	No

25A-S3	46.56	MT	16-20 yrs.	**Diagnostic of** dental calculus, dental caries, osteophyte, **typical of** non-specific infection	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: close to birth to less than 1 yr.	No
25B-S4	13.08	F	34-45 yrs.	**Typical of** non-specific infection.	A	A	N.O.	N.A.	N.A.	N.A.	No
25C-S106	68.24	M	30-59 yrs.	**Diagnostic of** dental calculus, dental caries, endocranial lesions, **highly consistent with** hypertrophic (pulmonary) osteoarthropathy, **typical of** treponemal disease: venereal syphilis	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: birth to around 2 yrs.	No
25C-S18	28.72	F	30-59 yrs.	**Diagnostic of** LEH	P	P	4	4	**Episode 1**: grade 1-2. **Episode 2**: grade 1. **Episode 3**: grade 1. **Episode 4**: grade 2	**Episode 1**: birth to c. 1.5 yrs. **Episode 2**: around 5 yrs. **Episode 3**: around 7 yrs. **Episode 4**: around 9 yrs.	No
25C-S36	33.08	F	30-59 yrs.	**Diagnostic of** dental calculus, **typical of bone** tumour, non-specific infection	A	A	N.O.	N.A.	N.A.	N.A.	No
25C-S45	19.44	MT	18-29 yrs.	N.O.	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 2 yrs.	No
25C-S61	88	MT	20-29 yrs.	N.O.	A	A	N.O.	N.A.	N.A.	N.A.	No
25C-S64	48.84	F	15-16 yrs.	**Diagnostic of** dental cavities, endocranial lesions	P	P	1	1	**Episode 1**: grade 2	**Episode 1**: birth to around 1.5 yrs.	No
25C-S65	89.8	MT	20-29 yrs.	**Diagnostic of** dental calculus, LEH, osteophytes, degenerative joint disease, intervertebral disk disease and osteoarthritis	P	P	N.O.	2	**Episode 1**: grade 1.5 **Episode 2**: grade 1.5	**Episode 1**: birth to around 2 yrs. **Episode 2**: around 4 yrs.	No
25C-S71	71.4	F	30-59 yrs.	**Diagnostic of** osteophytes, trauma, degenerative joint disease, porotic hyperostosis (confirmed by radiographs), **typical of** osteochondritis dissecans, non-specific infection, tumour	P	P	N.O.	2	**Episode 1**: grade 2 **Episode 2**: grade 2	**Episode 1**: birth to 1.5 yrs. **Episode 2**: around 2 yrs.	No

25C-S72	47.92	M	30-50 yrs.	**Diagnostic of** dental calculus, Schmorl's nodes, osteophytes, trauma, degenerative joint disease	P	P	N.O.	1	**Episode 1**: grade 1.5	**Episode 1**: around birth	No
25C-S73	N.A.	N.A.	9-10 yrs.	**Diagnostic of** dental calculus, LEH, **highly consistent with** lung infection	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 2.5 yrs.	No
25C-S94	35.28	MT	24-35 yrs.	**Diagnostic of** dental calculus and dental caries, **typical of** non-specific infection	P	A	N.O.	1	**Episode 1**: grade 1	**Episode 1**: around birth to 1.5 yrs.	No
26B-S23	34.48	Und.	16-20 yrs.	**Diagnostic of** dental caries, **typical of** non-specific infection	A	A	N.O.	N.A.	N.A.	N.A.	No
29L-S11	49.04	M	+40 yrs.	**Diagnostic of** dental calculus, dental caries, Schmorl's nodes, degenerative joint disease, **typical of** non-specific infection	P	P	N.O.	1	**Episode 1**: grade 2	**Episode 1**: birth to around 1 yr.	No
30B-S1	34.8	M	18-29 yrs.	**Diagnostic of** dental caries, dental calculus, osteophytes, degenerative joint disease	P	P	N.O.	1	**Episode 1**: grade 1.5	**Episode 1**: *in utero* to 1 yr.	Yes
30D-S5	59.8	MT	20-39 yrs.	**Diagnostic of** *cribra orbitalia*, porotic hyperostosis (confirmed by radiographs, vault thickening)	P	P	1	1	**Episode 1**: grade 2	**Episode 1**: around birth to less than 1 yr.	No
30R-S7	N.A.	N.A.	8.5 yrs.	**Diagnostic of** dental caries, *cribra orbitalia,* **typical of** rickets	P	P	N.O.	1	**Episode 1**: grade 3	**Episode 1**: *in utero* to around 4 yrs.	Yes
8V-S9	30.16	FT	16-29 yrs.	N.O.	P	A	N.O.	1	**Episode 1**: grade 1.5	**Episode 1**: birth to around 2 yrs.	No
9M-S2	35.84	F	18-29 yrs.	**Diagnostic of** Schmorl's nodes, **consistent with** degenerative joint disease	P	A	N.O.	2	**Episode 1**: grade 1 **Episode 2**: grade 1	**Episode 1**: around 2 yrs. **Episode 2**: around 4 yrs.	No

P= present, A= absent, N.O. = not observable, N.A.= not applicable, F= female, FT= likely female, M= male, MT= likely male, Und. =undetermined.