Macroscopic dental enamel hypoplasia in deciduous teeth: health conditions and socio-economic status in nineteenth- to twentieth-century Granada, Spain

Sandra López-Lázaro[1,2], Miguel C. Botella[3], Inmaculada Alemán[3], Joan Viciano[4]

[1] Departamento de Antropología, Facultad de Ciencias Sociales, Universidad de Chile, Av. Ignacio Carrera Pinto 1045, 685033, Santiago, Chile

[2] Forensic Dentistry Lab, Centro de Investigación en Odontología Legal y Forense -CIO-, Facultad de Odontología, Universidad de La Frontera, Temuco, Chile

[3] Laboratory of Anthropology, Department of Legal Medicine, Toxicology and Physical Anthropology, School of Medicine, University of Granada, Granada, Spain

[4] Operative Unit of Anthropology, Department of Medicine and Ageing Sciences, 'G. d'Annunzio' University of Chieti–Pescara, Chieti, Italy

Correspondence
Joan Viciano
Operative Unit of Anthropology, Department of Medicine and Ageing Sciences, 'G. d'Annunzio' University of Chieti–Pescara, Piazza Trento e Trieste 1, 66100 Chieti, Italy.
E–mail: joan.viciano@unich.it

ORCID Sandra López-Lázaro: 0000-0002-5405-0105
ORCID Miguel C. Botella: 0000-0002-8132-9777
ORCID Inmaculada Alemán: 0000-0002-6392-9277
ORCID Joan Viciano: 0000-0001-7575-557X

Abstract

Objectives: This study explores the relation between dental enamel hypoplasia and socio-economic status and health conditions in the nineteenth- to twentieth-century Granada, Spain.

Materials and Methods: The Granada osteological collection of infants and young children was analyzed. Macroscopic enamel defects were examined and scored in 1,791 deciduous teeth of 177 individuals aged between 6 months of gestation and 85 months postnatal. Detailed information on the last address where the children's family lived, and the costs and type of coffin used in the burial, facilitated the estimation of the socio-economic status of the families.

Results: The prevalence of affected children was relatively high (19.8%) and there were no significant differences in the distribution of enamel defects between municipal districts of the city of Granada and the type of coffin. According to the age at onset of the physiological stressful events, 23.8% of children exhibited enamel hypoplasia during the prenatal period, 19.1% during the perinatal period and 57.1% in the postnatal period. Analyzing the chronological distribution, two peaks of occurrence of stressful events were observed in the decades of the late 1930s and the mid-1940s, and in the early 1970s.

Conclusions: The prevalence of enamel hypoplasia of the individuals analyzed does not seem to be associated with the socio-economic status of their families. However, the chronological distribution showed two peaks of high frequency related to different historical periods of economic and social crisis. The analysis of dental enamel hypoplasia provides an excellent index of developmental stress levels in the past.

Keywords: primary teeth; enamel hypoplasia; physiological stress; developmental disruption; socio-economic status

Acknowledgments

The authors are grateful to D. Jose Antonio Muñoz (Managing Director), Maribel Martín (coordinator of services) and all EMUCESA staff at the San Jose cemetery in Granada (Spain) for their invaluable assistance, as well as the Magistrate Judge (Court of First Instance no. 5) responsible for the Registry Office of Granada. The authors also thank the employees of the Laboratory of Physical Anthropology of the University of Granada who selflessly worked on the exhumation and preparation of the identified skeletal collection.

Declarations

Funding

This study did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Conflict of interest

All of the authors declare that they have no conflicts of interest.

Availability of data and material

The data that support the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available due to privacy or ethical restrictions.

Authors' contributions

Sandra López-Lazaro: Conceptualization; data curation; formal analysis; investigation; methodology; supervision; validation; visualization; writing-original draft; writing-review and editing. **Miguel C. Botella:** Conceptualization; writing-original draft; writing-review and editing. **Inmaculada Alemán:** Writing-original draft; writing-review and editing. **Joan Viciano:** Conceptualization; data curation; formal analysis; investigation; methodology; supervision; validation; visualization; writing-original draft; writing-review and editing.

1. Introduction

Dental enamel hypoplasia is an irreversible deficiency in enamel thickness that forms when an individual is exposed to physiological stress in early life during the deposition and mineralization phases of tooth development (Goodman and Rose 1990; Salanitri and Seow 2013). From a bioarcheological point of view, this pathology has been widely utilized as an indicator of growth disturbance during childhood, however, compared to the permanent dentition, the prevalence of enamel hypoplasia in deciduous dentition has not been well reported in the paleopathological literature, as its occurrence is highly variable depending on the population group, nutritional or socio-economic status of the child, birth weight, the method of examination and the type of classification system used (Goodman et al. 1987; Taji et al. 2000; Slayton et al. 2001; Lukacs et al. 2001; Lunardelli and Peres 2005; FitzGerald et al. 2006; Halcrow and Tayles 2008; Geber 2014).

Deciduous dentition begins to develop in the early stages of pregnancy and continues into the third year of postnatal life (Nelson and Ash Jr. 2010; Hovorakova et al. 2018). Because all mineralized tissues of deciduous teeth (i.e., enamel, dentin and cementum) have individual growth patterns, they can provide information on developmental disorders during prenatal and early postnatal life allowing the inference of infant and maternal–fetal physiological stress. The enamel is especially informative, the development of which varies from the central incisor (which begins to develop, on average, 144 days before birth, and is complete about 240 days after birth) to the second molar deciduous teeth (which begin to develop, on average, 118 days before birth, and are complete about 506 days after birth) (Birch 2012; Birch and Dean 2013).

Dental enamel hypoplasia develops during the secretory stage of formation, unlike other enamel defects, such as hypocalcifications or fluorotic enamel, which develop during the maturation stage (Xing et al. 2016). These enamel defects result from disruptions in the process of amelogenesis, due to a disturbance to ameloblasts during matrix secretion (Goodman and Rose 1990). A deficiency in the amount or thickness of enamel is the essential feature of enamel hypoplasias, and ranges from a slight variation in the prominence of individual perikymata, to large furrows involving many perikymata, to the absence of large enamel extensions (Hillson 2014). They have been classified as furrow-form, pit-form and plane-form types, depending on the volume of enamel affected for the cessation or diminution of ameloblast function (Hillson and Bond 1997; Hillson 2014).

The specific etiology of dental enamel hypoplasia is still unknown, so it is accepted as nonspecific physiological perturbations (Goodman and Rose 1990). Several studies have analyzed the association between living conditions and dental enamel defects, especially socio-economic, nutritional and health status in historic populations (Rathbun 1987; Lanphear 1990;

Blakey et al. 1994; Moggi-Cecchi et al. 1994; Saunders and Keenleyside 1999; Lukacs et al. 2001). Higher socio-economic status has been associated with the expectant mother having a lighter occupation and greater access to resources (e.g., foodstuffs, obstetric and pediatric care) (Amoroso et al. 2014). A lack of essential foodstuffs and malnutrition during childhood are strongly related to infections and infant mortality. Poor nutritional status decreases the protective mechanisms of the immune system, and these children are more vulnerable to infections (Solomons and Keusch 1981; Katona and Katona-Apte 2008). This situation may affect various aspects of child growth and development, including dental health during tooth formation (Goodman and Rose 1991). During pregnancy, fetal malnutrition resulting from the combined effects of maternal infections, parasitic infestations and the malnutrition of the expectant mother could also result in defects in the prenatal enamel of deciduous dentition (Enwonwu 1973; Taji et al. 2000).

One of the most important elements in the study of dental enamel defects is the timing at which they occur. There are several macroscopic methods to convert the locations of enamel defects to an individual's age when those defects were formed, for both permanent and deciduous dentition (Swärdstedt 1966; Goodman et al. 1980; Blakey and Armelagos 1985; Goodman and Rose 1990), based on the chronology of dental development in populations of different origin (Logan and Kronfeld 1933; Massler et al. 1941; Moorrees et al. 1963a, b). Several publications in the literature illustrate the considerable variation in crown formation times for each tooth (Sunderland et al. 1987; Liversidge et al. 1993; Goodman and Song 1999; Reid and Dean 2000). These macroscopic methods may not provide accurate age estimates for populations with a different chronology of dental development and crown height variation (Hodges and Wilkinson 1990; Goodman and Song 1999), and thus it is necessary to develop sample-specific methods to estimate the timing of enamel hypoplasia formation.

The aims of this study were: (i) to document the prevalence and distribution of dental enamel defects on deciduous dentition, (ii) to estimate the timing of the formation of these enamel defects, and (iii) to investigate the chronological distribution and socio-economic factors that may have been associated with this pathological condition in a nineteenth- to twentieth-century skeletal sample from Granada, Spain.

2. Materials and methods

2.1. Sample

This study was conducted on the Granada osteological collection of identified infants and young children (Alemán et al. 2012), which includes 230 identified individuals in an excellent state of preservation who were exhumed from the San José Municipal Cemetery of Granada (Spain). The osteological collection, composed of corpses that were unclaimed or abandoned beyond the legal period, is housed in the Laboratory of Anthropology at the University of Granada for research purposes. Reliable antemortem information was obtained from the burial records of the San José Municipal Cemetery and the death certificates in the Registry Office, which yielded detailed data on sex, dates of birth and death, and immediate and underlying causes of death. Other data available included the last address of the family in the city of Granada, and the type and cost of coffin used. The protocol for studying the osteological collection and its associated information was approved by the ethics committee of the University of Granada and complies strictly with national regulations (Law 14/2007, 3 July) on the protection of personal privacy and confidential treatment of personal data in biomedical research.

To be included in this sample, the individuals had to have retained at least three deciduous teeth. Individuals who lived outside the city of Granada, and teeth with severe dental wear on the incisal/occlusal surface (i.e., where more than a third of the crown is missing) were excluded from the study. After application of these criteria, 1,791 deciduous teeth belonging to 177 individuals (100 males, 75 females, two of unknown sex) aged between 6 months of gestation and 85 months postnatal (mean age of 10.89 months) formed the final study sample. Table 1 illustrates the distribution by age at death and by sex of the sample. Over half (58.6%) of the individuals were aged between 6 months of gestation and 3 months postnatal, which means that this sample largely represents fetuses in the last quarter of gestation and neonates in the first quarter year of life. Figure 1 illustrates the distribution of the sample by decade of birth and decade of death. The birth years of these individuals ranged from 1871 to 2000, and their death years ranged from 1871 to 2001. The great majority of these births (84.1%) and deaths (83.1%) were between 1940 and 1973, which means that this sample largely dates from the second third of the twentieth century.

2.2. Collection of qualitative and quantitative data

Macroscopic hypoplastic defects of the enamel were examined and scored in the deciduous dentition with the aid of a Motic SMZ-168 stereo zoom microscope (Motic China Group Co., Ltd.) at up to 10× magnification, under a Volpi Intralux 5000 illumination system (Volpi Manufacturing, New York, USA) with dual arm fiber optic light guide to direct the light to a specific point from two angles at once. Defects of enamel were documented in several ways, including: (i) recording each deciduous tooth that exhibited the presence or absence of enamel defects, using the Fédération Dentaire Internationale (FDI)/Defects of Dental Enamel standards (FDI [Fédération Dentaire Internationale] 1982; Clarkson 1989) when defects were observed. Only dental enamel hypoplasia (DEH) was analyzed as a dental enamel defect. DEH is defined as a quantitative defect involving the surface of enamel, and is associated with reduced thickness. The defective enamel may occur as shallow or deep pits, or rows of pits arranged horizontally, or as small or large, wide or narrow grooves (FDI [Fédération Dentaire Internationale] 1982; Clarkson 1989); (ii) descriptive location of enamel defects on the tooth crown (i.e., on entire crown surface, or on incisal/occlusal, medial or cervical thirds); (iii) metric location of enamel defects on the tooth crown (distance from the average of upper and lower limit of the DEH to the cemento–enamel junction), using a digital dental caliper (Masel Orthodontics Inc, USA), calibrated to the nearest 0.01 mm; and (iv) calculation of the chronological age of the stressful events according to the development of population-specific regression equations following the methodological description of Blakey and Armelagos (1985).

The results were tabulated in two ways: (i) tooth count prevalence (i.e., the frequency of teeth with DEH); and (ii) individual count prevalence (i.e., the number of individuals observed with DEH). These two specific ways of presenting the results will facilitate an accurate comparison with other populations, as well as the use of the data by other researchers.

Individuals were divided into eight groups according to the municipal district of the city of Granada in which they lived: Albaicín, Beiro, Centro, Chana, Genil, Norte, Ronda and Zaidín (Figure 2). Table 2 illustrates the distribution of the sample divided by sex and municipal districts. The individuals were divided into three groups according to the quality of the coffin in which they were buried: standard, medium and luxury coffins (a standard coffin is the most economic and simple coffin, made of low-quality unvarnished wood materials; a medium coffin is a solid hardwood coffin with more detailed finishes, such as the varnishing/lacquering of wood, and interior upholstery; a luxury coffin is a high-quality coffin made with noble woods and excellent finishes, such as metal religious decorations and high quality interior upholstery). Table 3 illustrates the distribution of the sample divided by sex and the quality of the coffin.

2.3. Timing of DEH formation

The study by Blakey and Armelagos (1985) provides useful information with which to construct regression equations for converting the location of an enamel developmental defect to an "age at formation" in deciduous teeth. The only necessary data is: (i) the ages at the first evidence of calcification and the end of crown formation; and (ii) total crown height. The equations can thus be formulated as:

> Age at DEH formation (in months) = Month at crown completion – [Distance in mm from DEH to cemento–enamel junction in mm / Crown development per month].

The crown development per month is calculated by dividing the average crown height of each tooth by the total period of months needed to complete formation of the crown.

In a first step, to determine the timing of enamel growth, data for the first evidence of calcification and completion of the crown was extracted for the different deciduous teeth were extracted from previous publications on the chronology of the development of deciduous dentition, based on the same osteological collection of identified infants and young children (Irurita et al. 2014a, b; Viciano et al. 2018); the dental data is therefore specific to our sample. In a second step, data for crown height (from cusp tips to cemento–enamel junction) in non-pathological and unworn teeth was obtained from 41 randomly selected individuals. Table 4 summarizes the data on crown height, the first evidence of calcification and crown completion for each deciduous tooth, and the calculated data for the total duration of enamel growth and the segment of enamel developed monthly. Table 5 summarizes the regression equations constructed from the data in Table 4, according to the methodological description of Blakey and Armelagos (1985).

When assigning dental age, the enamel defects that matched different tooth types were considered the result of the same stressful event if they were included in a three-month age range. The number of stressful events per individual was thus established on this basis.

2.4. Statistical analysis

All statistical analyses and graphs were performed using IBM SPSS Statistics for Windows, version 25 (IBM Corp., Armonk, NY, USA). Comparative studies were analyzed using chi-squared tests for the data on prevalence of DEH per tooth type. Because sample sizes were expected to be small, Fisher’s exact tests were used for the data on the prevalence of DEH per individual. The significant difference for all tests was determined at the confidence level of $P \leq 0.05$.

3. Results

The variation in hypoplastic enamel defects ranged from small pits to areas of missing enamel that exposed the dentin surface, and also varied greatly in size from 0.2 mm to 3 mm in diameter (Figure 3). No linear or grooved hypoplastic defects were observed in the deciduous dental sample.

3.1. Prevalence of DEH per tooth type

The prevalence of DEH for each tooth type is presented in Table 6. The prevalence of affected teeth was 4.3%; that is, of the 1,791 teeth that could be examined for DEH, 77 exhibited enamel defects. The teeth most affected were the canines (14.2%), followed by the lateral incisors (3.9%), first molars (2.5%) and second molars (2.3%). The least affected teeth were the central incisors (2.0%). Nevertheless, chi-squared tests showed that these differences were not significant ($P > 0.05$). The maxillary central incisor and first molar teeth tended to exhibit DEH more frequently than their mandibular isomers. In contrast, the maxillary lateral incisor, canine and second molar teeth were less frequently affected by DEH than mandibular isomers. None of these differences were significant according to chi-squared tests ($P > 0.05$).

Maxillary and mandibular central and lateral incisors and mandibular molars tended to exhibit DEH more frequently in females than males. In contrast, canines and maxillary first and second molars displayed the reverse pattern, in which males exhibited DEH more frequently than females. However, chi-squared tests showed that none of these differences were significant ($P > 0.05$).

Figure 4 shows that DEH were nonrandomly distributed within the crown of the teeth. With maxillary and mandibular teeth pooled, DEH were found more frequently on the incisal third in central incisors (62.5%). In the lateral incisors, DEH were found equally in incisal, medial and cervical thirds (33.3% for each region). In the canines, hypoplastic enamel defects were found more frequently on the medial third (58.3%). Among the molars, DEH were found more frequently on the medial third for first and second molars (72.7% and 57.1%, respectively). Overall, there is a trend of increased frequency of DEH in the middle third of the crown as one moves from the incisors toward the molar teeth. This trend was statistically significant ($\chi^2 = 24.649$, df = 12, $P = 0.017$).

3.2. Prevalence of DEH per individual

The prevalence of affected individuals was 19.8%; that is, of the 177 individuals that could be examined for DEH, 35 exhibited enamel defects. There were no statistically significant differences in DEH prevalence according to sex ($P > 0.05$).

Table 7 shows the distribution of DEH for each municipal district and the quality of the coffin. Considering the frequencies for municipal districts, 33.3% of individuals in Centro were affected by DEH, followed by Ronda (15.8%), Beiro and Zaidín (14.3%), Chana (9.1%) and Albaicín (5.3%). The individuals of Genil and Norte districts did not exhibit DEH. The differences between municipal districts were not significant (Fisher's exact test = 9.661, df = 7, *P* = 0.166; unknown municipal districts were excluded). There were also no significant differences between the sexes within each municipal district (*P* > 0.05). Table 8 shows the number of stressful events in each age range per municipal district (note that the incomplete formation of the crown in several teeth prevented measurement of DEH location from enamel defect to the cemento–enamel junction. It was therefore only possible to calculate the timing of DEH formation in 23 individuals [7 males, 16 females] for a total of 27 stressful episodes). The mean number of stressful events per individual (i.e., the ratio between the total number of DEH episodes and the number of individuals affected) showed that the municipal district of Centro presented a slightly higher number of stressful events per individual than the other districts (1.3 vs. 1.0).

27.8% of individuals with luxury quality coffins were affected by DEH, followed by medium (14.8%) and standard quality (9.8%). The quality of coffin was unknown for 37.8% of the individuals (Table 7). The differences between coffin groups were not significant (Fisher's exact test = 3.075, df = 2, *P* = 0.222; unknown qualities of coffins were excluded). The differences between males and females within each coffin group were not significant (*P* > 0.05). Table 9 shows the number of stressful events in each age range, where the luxury quality coffin presented a slightly higher number of stressful events per individual (1.3) than the medium (1.1) and standard qualities (1.0). Differences between sexes within the same quality of coffin were observed: for luxury quality, the number of stressful events per individual was higher in females (1.5) than in males (1.0), and vice versa for the medium quality. The standard quality of coffin showed no differences between the sexes

Table 10 shows the distribution of stressful events by sex and age range and illustrates that out of 23 individuals with DEH (with a total of 27 stressful episodes), 19 (6 males, 13 females) suffered a single stressful event, and 4 (1 male, 3 females) suffered two stressful events. When stratified by age of age of occurrence, for children who suffered a single stressful event, 26.3% of DEH occurred during the prenatal period (5 females), 10.5% during the perinatal period (i.e., from birth to the first 3 months of life; 1 male, 1 female), and 63.2% during the postnatal period (i.e., from the first 3 to 11 months of life; 5 males, 7 females). For children who suffered two stressful events, 25% of DEH occurred during the pre-perinatal period (i.e., first stressful event prenatal and second event perinatal; 1 female), 50% during the peri-postnatal period (i.e., first stressful event perinatal and second event postnatal; 2 females), and 25% during the postnatal period (i.e., both stressful events occurred postnatally; 1 male). Fisher's exact tests showed that the differences in DEH occurrence between males and females within each age group were not significant (*P* > 0.05). Figure 5 shows the distribution of individuals with DEH by decade of occurrence of these defects. Of the 23 individuals with DEH, the date of birth of two individuals was unknown, so the decade of occurrence of stressful events cannot be inferred. Thus, Figure 5 and the associated data refer only to the 21 individuals with a known date of birth. Individuals affected by DEH were distributed throughout all decades, from the 1870s to the 1970s; however, the occurrence peaked in the late 1930s and the mid-1940s and, decreased in the 1950s and 1960s, before slightly increasing in the early 1970s. For these individuals, 23.8% suffered physiological stress resulting in DEH during the prenatal period, 19.1% suffered stressful events during the perinatal period, and 57.1% suffered stressful events during the postnatal period (Note: the two individuals whose decade of occurrence of stressful events was unknown were excluded from this analysis).

4. Discussion

The overall prevalence of affected children with hypoplastic enamel defects in their deciduous dentition from the Granada osteological collection was 19.8%. This prevalence is higher than the overall typical prevalence found in children of modern developed countries and in well-fed children of undeveloped countries (usually less than 10%) (Hargreaves et al. 1989; Skinner and Hung 1989; Lovell and Whyte 1999; Casanova-Rosado et al. 2011). The prevalence is in line with that found in disadvantaged and malnourished children from developed and developing countries, where between 17.7% to 73.1% of children showed DEH in deciduous dentition (Sweeney et al. 1971; Enwonwu 1973; Infante and Gillespie 1974; Goodman et al. 1987; Kanchanakamol et al. 1996; Rugg-Gunn et al. 1998; Masumo et al. 2013). Nevertheless, it is necessary to highlight that the differences in the prevalence between the diverse studies vary depending upon: (i) the types of enamel defects evaluated; (ii) the classification systems employed; (iii) the methods used for observation and data collection; and (iv) the genetic, ancestry, medical and socio-economic status of the populations analyzed (Needleman et al. 1991).

The teeth most affected by DEH in the analyzed sample were the deciduous canines, presenting the highest frequencies of enamel defects in the middle third of the crown, and lowest frequencies in the incisal and cervical thirds. If the values of the mean crown heights of deciduous canines reported in Table 4 are used to produce an estimate of the age at which a specific percentage enamel crown is formed, using the exponential regression equations of Irurita et al. (2014b) for this specific population, it can be seen that the deciduous canines have completed approximately 50% of the crown at the time of birth. The low frequency of DEH in the incisal third of the crown may therefore reflect the prenatal enamel formation of this region of the tooth and the protective intrauterine environment during pregnancy. Enamel defects in the middle third of the crown may be attributed to the physiological stress related to physiological changes associated with birth. Enamel defects in the cervical third of the crown may represent the physiological stress of the less-buffered postnatal environment. Following the same reasoning as in the deciduous canine, analysis of the chronology of crown formation on the different deciduous teeth types explains that the prevalence of enamel defects in the incisal and middle thirds of incisors and occlusal third of molars may reflect physiological stress associated with severe instability of maternal homeostasis (e.g., compromised health status or nutritional imbalance); the cervical third of incisors and middle third of molars may be attributed to the stress associated with the birth process; the cervical third of molars may represent postnatal environmental challenges to the physiological homeostasis of the neonate. Variations in the location of DEH on the teeth crowns between individuals may be attributable to different developmental environments, and the influence of sexual hormones in the timing of crown formation.

The results showed that there were no statistically significant differences in DEH prevalence between the sexes. Although several studies found a higher prevalence of enamel defects in males due to their greater biological sensitivity to stress factors (El-Najjar et al. 1978; van Gerven et al. 1990; Guatelli-Steinberg and Lukacs 1999; Saunders and Keenleyside 1999; Palubeckaité et al. 2002) or higher prevalence in females due to cultural differences and daughter neglect (Goodman et al. 1987; May et al. 1993; Slaus 2000; King et al. 2005; Oyamada et al. 2012), this distinction between the sexes is not usually statistically significant (Goodman et al. 1980; Lanphear 1990; Duray 1996; Lovell and Whyte 1999; Taji et al. 2000; Lukacs et al. 2001). Despite the fact that several studies reveal differential access to basic resources such as food, shelter, and health care between male and female children, these conditions could only be explained if enamel defects occurred during the postnatal period (Guatelli-Steinberg and Lukacs 1999). The results showed that 59.3% of DEH events took place from 3 months to 11 months postnatal, but no significant differences were observed between the sexes. The data therefore suggests that cultural diversity or nutritional and health status do not seem to be factors associated with sexual dimorphism in DEH occurrence in the analyzed sample. The remaining 40.7% of individuals suffered physiological stress causing DEH in the prenatal and perinatal period (i.e., within the first 3 months after birth), and therefore several systemic factors may be associated with the hypoplastic enamel defects, such as the socio-economic status of their family, maternal conditions, prematurity, low birth weight, malnutrition, mineral deficiencies and infections during fetal life and neonatal period (Lunardelli and Peres 2006; Franco et al. 2007; Velló et al. 2010; Masumo et al. 2013). The results agree with those of Blakey and Armelagos (1985) and Goodman et al. (1987), who observed that enamel defects of deciduous teeth occurred during the last 3 months of gestation, and perinatally. The prevalence of DEH is usually high in the last months of the prenatal period, and remains high for the first months after birth as a consequence of the fetal and neonatal nutritional demands. Skinner and Hung (1989) also demonstrated the association of lower milk consumption postnatally with the baby’s health, and, subsequently, the presence of hypoplastic enamel defects. After this period of high prevalence of DEH in the last months of the prenatal period and the first months after birth, the frequency of DEH declines rapidly during the postnatal period, however, in the analyzed sample the frequency increased slightly from the sixth month postnatal, reaching its highest peak between the eighth and tenth months, to decline again rapidly. Skinner and Hung (1989) hypothesize that a high frequency of DEH during the postnatal period may be explained by the babys' ability to hand objects. This mechanism of manipulating objects begins as early as approximately 3 months after birth, when the baby can voluntarily hold and shake a toy. Immediately afterward, at around 5 months of age, the baby can nibble any hand-held object (White and Marks 1995). Moreover, since objects are normally introduced into the mouth from below, the non-coordinated motor movements in which these objects impact around the mouth would explain why DEH are more frequent in the mandibular teeth than in the maxillary teeth, regardless of the population studied (Jørgensen 1956; Skinner 1986).

In a broader sense, dental stress markers and oral pathological conditions are a valuable source of information on nutrition and the environment within which individuals grew and lived (Cook and Buikstra 1979). DEH is therefore used to analyze the health status of specific populations and for interpopulation comparisons (Goodman et al. 1980; Lanphear 1990; Palubeckaitė et al. 2002; Garcin et al. 2010). Several studies evaluate the prevalence of enamel hypoplasia in different geographic areas and communities (e.g., comparing urban and rural populations, comparing coastal and inland settlements) (Palubeckaité et al. 2002; Garcin et al. 2010). The distinctive feature of the present study is the evaluation of the prevalence of DEH within the city of Granada, thanks to the precise information about the exact addresses of the families in the analyzed sample. The results showed a higher prevalence in the downtown area (i.e., Centro district), followed by the districts of Ronda, Beiro, Zaidín and Chana. The evolution of the city of Granada was characterized by the few changes that its urban structure underwent until the nineteenth century, keeping its well-defined urban limits. There was an important point in the evolution and transformation of the city in the mid-twentieth century, thanks to significant migratory waves from rural to urban areas, which caused the city to almost double its population between 1950 and 1981 (Figure 6). These migratory waves favored the development of disadvantaged peripheral neighborhoods, which gave rise to the development of the Albaicín district and was the seed for the development of the current districts of Zaidín, Chana and Norte (Jiménez et al. 2009). Thus, for example, Ronda district was developed in the 1920s and Zaidín in the 1950s (Godoy 1974). At the end of the nineteenth century and the beginning of the twentieth century, due to the increase in population from rural areas and the remodeling in the city center to create wide streets following the model of other European cities, the district of Albaicín housed the poorest sectors of the populations because it was a more affordable area (Duque Calvache 2016). This situation caused the densification of the neighborhood and a deepening of social differences with the rest of the districts of the city (Duque Calvache 2016). The district of Centro, together with Albaicín, which still retains the narrow winding streets of its medieval Moorish past, dating back to the Nasrid Kingdom of Granada, is the oldest urban area in Granada, and therefore, this area has gathered the greatest number of individuals over a longer chronological period (Jiménez et al. 2009).

Several studies have attempted to confirm the relationship between socio-economic status and the prevalence of DEH. Individuals with higher socio-economic status generally have access to more resources, especially foodstuffs, have better hygiene and health conditions, and experience lighter workloads (Saunders and Keenleyside 1999); however, some studies suggest that unexpected prevalence of DEH is due to the multiple and varied effects that socio-economic factors have on stress levels (Rathbun 1987; Lanphear 1990; Blakey et al. 1994; Moggi-Cecchi et al. 1994). For example, King et al. (2005) and Cucina and İşcan (1997) found a high prevalence of DEH in children from middle-class families, and from families of high economic status, while Palubeckaitė et al. (2002), Miszkiewicz (2015) and Minozzi et al. (2020) found a higher prevalence of DEH in socially disadvantaged groups. These contradictory studies show that socio-economic status alone cannot explain population discrepancy in DEH prevalence, and suggest that socio-economic factors have an unequal and varied effect on stress levels (Goodman and Rose 1990). On the other hand, there are differences in the approaches used to assess the socio-economic status of the analyzed populations by the various studies. For example, some studies are based on historical and textual data (Lanphear 1990; Saunders and Keenleyside 1999), while other studies are based on the distinct places where the individuals were inhumed (e.g., buried in a cemetery or inside a church) (Cucina and İşcan 1997; Palubeckaitė et al. 2002; Miszkiewicz 2015; Minozzi et al. 2020), the type of burial (e.g., interred in a ceramic jar coffin or wooden coffin) (Nakayama 2016) or occupation (Amoroso et al. 2014). In the present study, the socio-economic status was established according to the quality of the coffin in which the individuals were buried. The results showed that individuals buried in a luxury coffin exhibited a higher frequency of enamel defects and suffered a higher number of stressful events than those buried in a medium or standard coffin. Despite the unequal and varying effects that socio-economic factors have on stress levels (as discussed above), the results allow exploration of the possibility that the type of coffin used in the burial does not uniquely reflect socio-economic status, and the choice of the coffin type for the burial may also have been influenced by religious feeling and other social factors. Many researchers have analyzed mortuary practices for patterns and trends related to social factors, such as socio-economic status, religion, gender and ancestry (Springate 2016). According to Bell (1990), while the quality of a coffin and its decoration is often related to what those responsible are able and willing to pay, the embellishment of a coffin does not necessarily correlate with the socio-economic status of the interred individual or its family. Moreover, the relatively low cost of the mass-produced coffin and its multiple and varied embellishments allowed any individual of lower socio-economic status to access specific types of coffins and finishes, enabling them to emulate individuals of a higher socio-economic status. Davidson (2004) analyzed burial costs in the early 1900s at Freedman's and Cedar Grove Cemeteries in Texas and Arkansas, respectively, showing that mortuary practices and burial costs were also influenced by demographic patterns. This study, for example, reflected that these costs were higher for women than for men, and much higher for adults than for children. In conclusion, cemeteries reflect the social structures of the living community that created and maintained them, depicting social factors such as socio-economic status, religion, family, gender, and demographic patterns (e.g., sex, age, and ancestry) (Davidson 2004; Springate 2016).

It is interesting to note that the 38.1% of individuals with DEH were concentered between the late 1930s and mid-1940s, and 14.3% of individuals in the early 1970s. From 1936 to 1939, Spain was engulfed in a Civil War with social and economic consequences that affected a large part of society. The pre-war period, the warlike conflict and the post-war period caused an increase in epidemics of infectious diseases for Spanish society. The resurgence of smallpox, diphtheria and exanthematous typhus during the immediate post-war period was one of many manifestations of hardships, along with the famine, that affected a large part of the population. Similarly, there was an increase in other diseases, such as typhoid fever (principally due to the poor hygiene conditions of water for human consumption) and tuberculosis, diseases endemic in Spain since before the war (del Cura and Huertas 2009). For example, Vargas-Frías et al. (2018) show that diarrhea and enteritis were the main cause of mortality during the period 1931-1945 in La Zubia, a town located at 6 km from Granada. The other hardships suffered by the population ranged from plain starvation to the various manifestations of the lack of essential food elements. This situation caused general malnutrition in the population due to a lack of essential foodstuffs (Arco Blanco 2006; del Cura and Huertas 2009). Hunger and poor diet greatly weakened the health of the poorest sectors of the rural and urban population, and increased the general mortality rate in the province of Granada after the end of the Civil War (Vargas-Frías et al. 2018). High rates of diseases associated with nutritional deficiencies have been reported particularly in 1936 and 1937, coinciding with the start of the Civil War, and in 1941 due to the start of the food rationing regime in Granada (Vargas-Frías et al. 2018). Although the period of food shortages was overcome around 1955, the situation in the early 1960s was that scarcity prevailed over surplus, characterized by pronounced nutritional imbalances (Díaz-Méndez and Gómez-Benito 2010). In addition to the post-war crisis, the city of Granada was tremendously affected by two natural disasters. In 1962, terrible floods took the lives of several people, thousands were left homeless and more than 7,000 earth-sheltered houses had to be abandoned and demolished in the neighborhood of Sacromonte (in the Albaicín district), affecting the poorest population (Ramos Espejo et al. 2002). In 1973, the torrential rains that were unleashed in the southeast of the Iberian Peninsula, apart from irreparable human losses, caused the disappearance of crops, seriously affecting the populations that settled on the banks of the rivers and boulevards and their proximities (Capel Molina 1974). Although it is not possible to infer that the high prevalence of DEH observed in the analyzed sample is the consequence of these specific historical and catastrophic natural events, it is plausible that these events contributed to its increase. In any case, the only thing that can be inferred is that the prevalence of DEH observed in the sample of Granada is a consequence of distinct historical periods of economic and social crisis, and/or hygiene collapse.

A large number of the inferences made in the present study are based on the estimation of the individual's age at the time of the formation of DEH as a consequence of physiological stress events. There are two main approaches to estimating this parameter on both permanent and deciduous dentitions: the decile-chart method, which is based on histological techniques (Reid and Dean 2000), and the linear distance-based regression method, which is based on the measured distance of DEH from the cemento-enamel junction (Goodman and Rose 1990); each method has its advantages and disadvantages. The decile-based chart method is increasingly used in studies to estimate the chronology of enamel defects (Cares Henriquez and Oxenham 2017, 2019; Gamble et al. 2017) because histologically derived data is more reliable and accurate. This method takes into account diverse parameters considered as sources of variation in the estimation of age at the formation of DEH (Goodman and Song 1999): (i) the non-linear growth of enamel; (ii) the hidden cuspal enamel; (iii) the population variations in crown initiation time by tooth type; and (iv) the population variations in crown height. The decile-based chart method corrects all these parameters, however, its major drawbacks are its high time-consumption and the associated subjectivity when this method is used in its graphic form, resulting in precision and replicability problems over other existing methods (Ritzman et al. 2008; Cares Henriquez and Oxenham 2019). To remove the subjectivity of the decile-based chart in its graphic form, Cares Henriquez and Oxenham (2019) recently proposed distance-based exponential regression equations; however, the proposed regression models are limited to permanent anterior teeth. On the other hand, the advantage of the linear distance-based regression method proposed by Goodman and Rose (1990) is that it provides an objective approach to estimating the chronology of DEH in comparison to the graphic form of the decile-chart. Moreover, it can easily calculate precise age estimations based on the distance of the enamel defect from the cemento–enamel junction by inputting the collected measurement into a regression formula, thus improving the replicability of the results (D'Anastasio et al. 2013). Its major drawbacks lie in the flawed assumptions that underlie the standards on which it is based (i.e., constant rate of enamel deposition, no consideration of hidden cuspal enamel, no consideration of crown initiation times and populational variations in crown height), thus reducing the accuracy of the estimation of age at formation of DEH (Ritzman et al. 2008; Cares Henriquez and Oxenham 2019). Because it is less time-consuming and due to the absence of reliable and accurate methods developed for all deciduous teeth, we used the linear distance-based regression method in the present study. In order to solve problems inherent to the method, we developed the formulae required for the estimation of age at the time of the formation of DEH, adapting the methodology of Blakey and Armelagos (1985) to the specific population of the Granada osteological collection. In this way, we assumed a constant velocity of tooth development, and the variations in crown initiation time by tooth type and the variations in crown height were corrected with population-specific data.

Finally, it is necessary to highlight a major limitation inherent to the study of stress indicators in bioarcheology, such as DEH, related to the so-called osteological paradox, or paleopathological paradox (Wood et al. 1992). The children who showed evidence of DEH were the survivors of one episode (or more) of physiological stress, however, children who died without any evidence of DEH do not necessarily represent healthy children, and could be those who died before DEH could develop. This situation is plausible, because, although DEH is a good indicator of physiological stress, it does not represent all stressors (Smith and Littleton 2019). A sudden death event will not be seen by analyzing the DEH, and therefore, it should not be assumed that the children without DEH from the Granada osteological collection were stress-free. Although DEH is a good indicator of stress at the individual level which provides valuable information on children’s development and disease history, the health-related interpretation of an entire population remains a challenge in bioarcheology (Wood et al. 1992; DeWitte and Stojanowski 2015). It is evident that all the children from the Granada sample were frail when they were alive, as they died prematurely, regardless of whether there was evidence of DEH. However, the discussion related to the osteological paradox is usually applied to adult samples when the affected individuals survive, so it cannot be assumed that children from the Granada sample without DEH were healthier than children with DEH, as none of them survived to adulthood. To study the possible effects of individual frailty and the effects of morbidity on mortality in the population, the results of this study should be related to other multiple evidence of physiological stress following a biocultural approach (Siek 2013; DeWitte and Stojanowski 2015): on the individual (e.g., other skeletal diseases) and in society (e.g., history and evolution of the city of Granada).

5. Concluding remarks

This study presents the results of macroscopic developmental defects from enamel analyses of deciduous dentition from nineteenth/twentieth-century Granada, Spain. The chronological distribution of the DEH frequencies showed that these were concentered in two time periods characterized by documented specific historical and catastrophic natural events that affected all the inhabitants of the city of Granada, independent of their sex, socio-economic status and the municipal district where individuals lived and/or grew up. These historical and natural events could be related to a general decline in health status as a consequence of social crisis, epidemic episodes and/or hygiene collapse. In summary, this study provides essential data for understanding the association between dental enamel hypoplasia prevalence and systemic stress in past and present societies.

References

Alemán I, Irurita J, Valencia AR, et al (2012) Brief communication: The Granada osteological collection of identified infants and young children. American Journal of Physical Anthropology 149:606–610. https://doi.org/10.1002/ajpa.22165

Amoroso A, Garcia SJ, Cardoso HF v. (2014) Age at death and linear enamel hypoplasias: testing the effects of childhood stress and adult socioeconomic circumstances in premature mortality. American Journal of Human Biology 26:461–468. https://doi.org/10.1002/ajhb.22547

Arco Blanco MÁ del (2006) «Morir de hambre». Autarquía, escasez y enfermedad en la España del primer franquismo. Pasado y Memoria 5:241–258. https://doi.org/10.14198/pasado2006.5.12

Bell EL (1990) The historical archaeology of mortuary behavior: coffin hardware from Uxbridge, Massachusetts. Historical Archaeology 24:54–78. https://doi.org/10.1007/BF03374137

Birch W (2012) Incremental growth of deciduous tooth enamel

Birch W, Dean MC (2013) A new method of calculating human deciduous enamel formation times. Bulletin of the International Association for Paleodontology 7:47–55

Blakey ML, Armelagos GJ (1985) Deciduous enamel defects in prehistoric Americans from Dickson Mounds: prenatal and postnatal stress. American Journal of Physical Anthropology 66:371–380. https://doi.org/10.1002/ajpa.1330660404

Blakey ML, Leslie TE, Reidy JP (1994) Frequency and chronological distribution of dental enamel hypoplasia in enslaved African Americans: A test of the weaning hypothesis. American Journal of Physical Anthropology 95:371–383. https://doi.org/10.1002/ajpa.1330950402

Capel Molina JJ (1974) Génesis de las inundaciones de octubre de 1973 en el sureste de la Península Ibérica. Cuadernos Geográficos de la Universidad de Granada 4:149–166

Cares Henriquez A, Oxenham MF (2017) An alternative objective microscopic method for the identification of linear enamel hypoplasia (LEH) in the absence of visible perikymata. Journal of Archaeological Science: Reports 14:76–84. https://doi.org/10.1016/j.jasrep.2017.05.040

Cares Henriquez A, Oxenham MF (2019) New distance-based exponential regression method and equations for estimating the chronology of linear enamel hypoplasia (LEH) defects on the anterior dentition. American Journal of Physical Anthropology 168:510–520. https://doi.org/10.1002/ajpa.23764

Casanova-Rosado AJ, Medina-Solís CE, Casanova-Rosado JF, et al (2011) Association between developmental enamel defects in the primary and permanent dentitions. European Journal of Paediatric Dentistry 12:155–158

Clarkson J (1989) Review of terminology, classifications, and indices of developmental defects of enamel. Advances in Dental Research 3:104–109. https://doi.org/10.1177/08959374890030020601

Cook DC, Buikstra JE (1979) Health and differential survival in prehistoric populations: prenatal dental defects. American Journal of Physical Anthropology 51:649–664. https://doi.org/10.1002/ajpa.1330510415

Cucina A, İşcan MY (1997) Assessment of enamel hypoplasia in a high status burial site. American Journal of Human Biology 9:213–222. https://doi.org/10.1002/(SICI)1520-6300(1997)9:2<213::AID-AJHB7>3.0.CO;2-0

D’Anastasio R, Cesana DT, Viciano J, et al (2013) The possible correlation between dental enamel hypoplasia and a historic natural disaster in the Roman population of Herculaneum (79 AD - central Italy). Anthropologischer Anzeiger 70:369–383. https://doi.org/10.1127/0003-5548/2013/0312

Davidson JM (2004) Mediating race and class through the death experience: power relations and resistance strategies of an African-American community, Dallas, Texas (1869-1907). Dissertation, University of Texas at Austin

del Cura I, Huertas R (2009) Public health and nutrition after the Spanish Civil War. An intervention by the Rockefeller Foundation. American Journal of Public Health 99:1772–1779. https://doi.org/10.2105/AJPH.2007.124875

DeWitte SN, Stojanowski CM (2015) The osteological paradox 20 years later: past perspectives, future directions. Journal of Archaeological Research 23:397–450. https://doi.org/10.1007/s10814-015-9084-1

Díaz-Méndez C, Gómez-Benito C (2010) Nutrition and the Mediterranean diet. A historical and sociological analysis of the concept of a “healthy diet” in Spanish society. Food Policy 35:437–447. https://doi.org/10.1016/J.FOODPOL.2010.04.005

Duque Calvache R (2016) Procesos de gentrificación en cascos antiguos: el Albaicín de Granada. Centro de Investigaciones Sociológicas, Madrid

Duray SM (1996) Dental indicators of stress and reduced age at death in prehistoric native Americans. American Journal of Physical Anthropology 99:275–286. https://doi.org/10.1002/(SICI)1096-8644(199602)99:2<275::AID-AJPA5>3.0.CO;2-Y

El-Najjar MY, DeSanti M v., Ozebek L (1978) Prevalence and possible etiology of dental enamel hypoplasia. American Journal of Physical Anthropology 48:185–192. https://doi.org/10.1002/ajpa.1330480210

Enwonwu CO (1973) Influence of socio-economic conditions on dental development in Nigerian children. Archives of Oral Biology 18:95–107. https://doi.org/10.1016/0003-9969(73)90024-1

FDI [Fédération Dentaire Internationale] (1982) An epidemiological index of developmental defects of dental enamel (DDE index). International Dental Journal 32:159–167

FitzGerald C, Saunders S, Bondioli L, Macchiarelli R (2006) Health of infants in an Imperial Roman skeletal sample: perspective from dental microstructure. American Journal of Physical Anthropology 130:179–189. https://doi.org/10.1002/ajpa.20275

Franco KMD, Line SRP, de Moura-Ribeiro MVL (2007) Prenatal and neonatal variables associated with enamel hypoplasia in deciduous teeth in low birth weight preterm infants. Journal of Applied Oral Science 15:518–523. https://doi.org/10.1590/S1678-77572007000600012

Gamble JA, Boldsen JL, Hoppa RD (2017) Stressing out in medieval Denmark: an investigation of dental enamel defects and age at death in two medieval Danish cemeteries. International Journal of Paleopathology 17:52–66. https://doi.org/10.1016/j.ijpp.2017.01.001

Garcin V, Velemínský P, Trefný P, et al (2010) Dental health and lifestyle in four early mediaeval juvenile populations: comparisons between urban and rural individuals, and between coastal and inland settlements. Homo 61:421–439. https://doi.org/10.1016/j.jchb.2010.06.004

Geber J (2014) Skeletal manifestations of stress in child victims of the Great Irish Famine (1845-1852): prevalence of enamel hypoplasia, Harris lines, and growth retardation. American Journal of Physical Anthropology 155:149–161. https://doi.org/10.1002/ajpa.22567

Godoy AC (1974) Sociedad y urbanismo en la Granada contemporánea (1808-1974). Cuadernos de Arte de la Universidad de Granada 11:345–358

Goodman AH, Allen LH, Hernandez GP, et al (1987) Prevalence and age at development of enamel hypoplasias in Mexican children. American Journal of Physical Anthropology 72:7–19. https://doi.org/10.1002/ajpa.1330720103

Goodman AH, Armelagos GJ, Rose JC (1980) Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. Human Biology 52:515–528

Goodman AH, Rose JC (1991) Dental enamel hypoplasias as indicators of nutritional status. In: Kelley M, Larsen C (eds) Advances in Dental Anthropology. Wiley-Liss, New York, pp 279–293

Goodman AH, Rose JC (1990) Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. American Journal of Physical Anthropology 33:59–110. https://doi.org/10.1002/ajpa.1330330506

Goodman AH, Song R-J (1999) Sources of variation in estimated ages at formation of linear enamel hypoplasias. In: Hoppa RD, FitzGerald CM (eds) Human growth in the past: studies from bones and teeth. Cambridge University Press, Cambridge, pp 210–240

Guatelli-Steinberg D, Lukacs JR (1999) Interpreting sex differences in enamel hypoplasia in human and non-human primates: developmental, environmental, and cultural considerations. American Journal of Physical Anthropology Suppl 29:73–126. https://doi.org/10.1002/(sici)1096-8644(1999)110:29+<73::aid-ajpa4>3.0.co;2-k

Halcrow SE, Tayles N (2008) Stress near the start of life? Localised enamel hypoplasia of the primary canine in late prehistoric mainland Southeast Asia. Journal of Archaeological Science 35:2215–2222. https://doi.org/10.1016/j.jas.2008.02.002

Hargreaves JA, Cleaton-Jones PE, Williams SD (1989) Hypocalcification and hypoplasia in permanent teeth of children from different ethnic groups in South Africa assessed with a new index. Advances in Dental Research 3:126–131. https://doi.org/10.1177/08959374890030021001

Hillson S (2014) Tooth development in human evolution and bioarchaeology. Cambridge University Press, Cambridge

Hillson S, Bond S (1997) Relationship of enamel hypoplasia to the pattern of tooth crown growth: a discussion. American Journal of Physical Anthropology 104:89–103. https://doi.org/10.1002/(SICI)1096-8644(199709)104:1<89::AID-AJPA6>3.0.CO;2-8

Hodges DC, Wilkinson RG (1990) Effect of tooth size on the ageing and chronological distribution of enamel hypoplastic defects. American Journal of Human Biology 2:553–560. https://doi.org/10.1002/ajhb.1310020511

Hovorakova M, Lesot H, Peterka M, Peterkova R (2018) Early development of the human dentition revisited. Journal of Anatomy 233:135–145. https://doi.org/10.1111/joa.12825

Infante PF, Gillespie GM (1974) An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. Archives of Oral Biology 19:1055–1061. https://doi.org/10.1016/0003-9969(74)90095-8

Irurita J, Alemán I, López-Lázaro S, et al (2014a) Chronology of the development of the deciduous dentition in Mediterranean population. Forensic Science International 240:95–103. https://doi.org/10.1016/j.forsciint.2014.04.014

Irurita J, Alemán I, Viciano J, et al (2014b) Evaluation of the maximum length of deciduous teeth for estimation of the age of infants and young children: Proposal of new regression formulas. International Journal of Legal Medicine 128:345–352. https://doi.org/10.1007/s00414-013-0903-y

Irurita Olivares J, Alemán Aguilera I, Viciano J, et al (2014) Evaluation of the maximum length of deciduous teeth for estimation of the age of infants and young children: proposal of new regression formulas. International Journal of Legal Medicine 128:345–352. https://doi.org/10.1007/s00414-013-0903-y

Jiménez CE, Calmaestra JAN, Clemente JD, Rego RAG (2009) Viejas y nuevas realidades urbanas. Identificación de zonas de habitalidad desfavorecida en la ciudad de Granada. Cuadernos Geográficos 45:83–105. https://doi.org/10.30827/CUADGEO.V45I0.758

Jørgensen KD (1956) The deciduous dentition: a descriptive and comparative anatomical study. Acta Odontologica Scandinavica 14:1–202

Kanchanakamol U, Tuongratanaphan S, Tuongratanaphan S, et al (1996) Prevalence of developmental enamel defects and dental caries in rural pre-school Thai children. Community Dental Health 13:204–207

Katona P, Katona-Apte J (2008) The interaction between nutrition and infection. Clinical Infectious Diseases 46:1582–1588. https://doi.org/10.1086/587658

King T, Humphrey LT, Hillson S (2005) Linear enamel hypoplasias as indicators of systemic physiological stress: evidence from two known age-at-death and sex populations from postmedieval London. American Journal of Physical Anthropology 128:547–559. https://doi.org/10.1002/ajpa.20232

Lanphear KM (1990) Frequency and distribution of enamel hypoplasias in a historic skeletal sample. American Journal of Physical Anthropology 81:35–43. https://doi.org/10.1002/ajpa.1330810106

Liversidge HM, Dean MC, Molleson TI (1993) Increasing human tooth length between birth and 5.4 years. American Journal of Physical Anthropology 90:307–313. https://doi.org/10.1002/ajpa.1330900305

Logan WHG, Kronfeld R (1933) Development of the human jaws and surrounding structures from birth to the age of fifteen years. Journal of the American Dental Association 20:379–428. https://doi.org/10.14219/jada.archive.1933.0080

Lovell NC, Whyte I (1999) Patterns of dental enamel defects at ancient Mendes, Egypt. American Journal of Physical Anthropology 110:69–80. https://doi.org/10.1002/(SICI)1096-8644(199909)110:1<69::AID-AJPA6>3.0.CO;2-U

Lukacs JR, Walimbe SR, Floyd B (2001) Epidemiology of enamel hypoplasia in deciduous teeth: Explaining variation in prevalence in Western India. American Journal of Human Biology 13:788–807. https://doi.org/10.1002/ajhb.1125

Lunardelli SE, Peres MA (2005) Prevalence and distribution of developmental enamel defects in the primary dentition of pre-school children. Brazilian Oral Research 19:144–149. https://doi.org/10.1590/s1806-83242005000200013

Lunardelli SE, Peres MA (2006) Breast-feeding and other mother-child factors associated with developmental enamel defects in the primary teeth of Brazilian children. Journal of Dentistry for Children 73:70–78

Massler M, Schour I, Poncher HG (1941) Developmental pattern of the child as reflected in the calcification pattern of the teeth. American Journal of Diseases of Children 62:33–67. https://doi.org/10.1001/archpedi.1941.02000130042004

Masumo R, Bårdsen A, Åstrøm AN (2013) Developmental defects of enamel in primary teeth and association with early life course events: a study of 6-36 month old children in Manyara, Tanzania. BMC Oral Health 13:21. https://doi.org/10.1186/1472-6831-13-21

May RL, Goodman AH, Meindl RS (1993) Response of bone and enamel formation to nutritional supplementation and morbidity among malnourished Guatemalan children. American Journal of Physical Anthropology 92:37–51. https://doi.org/10.1002/ajpa.1330920104

Minozzi S, Caldarini C, Pantano W, et al (2020) Enamel hypoplasia and health conditions through social status in the Roman Imperial Age (First to third centuries, Rome, Italy). International Journal of Osteoarchaeology 30:53–64. https://doi.org/10.1002/oa.2830

Miszkiewicz JJ (2015) Linear enamel hypoplasia and age-at-death at medieval (11th-16th centuries) St. Gregory's Priory and cemetery, Canterbury, UK. International Journal of Osteoarchaeology 25:79–87. https://doi.org/10.1002/oa.2265

Moggi-Cecchi J, Pacciani E, Pinto-Cisternas J (1994) Enamel hypoplasia and age at weaning in 19th century Florence, Italy. American Journal of Physical Anthropology 93:299–306. https://doi.org/10.1002/ajpa.1330930303

Moorrees CFA, Fanning EA, Hunt EE (1963a) Formation and resorption of three deciduous teeth in children. American Journal of Physical Anthropology 21:205–213. https://doi.org/10.1002/ajpa.1330210212

Moorrees CFA, Fanning EA, Hunt EE (1963b) Age variation of formation stages for ten permanent teeth. Journal of Dental Research 42:1490–1502. https://doi.org/10.1177/00220345630420062701

Nakayama N (2016) The relationship between linear enamel hypoplasia and social status in 18th to 19th century Edo, Japan. International Journal of Osteoarchaeology 26:1034–1044. https://doi.org/10.1002/oa.2515

Needleman HL, Leviton A, Allred E (1991) Macroscopic enamel defects of primary anterior teeth — types, prevalence, and distribution. Pediatric Dentistry 13:208–216

Nelson SJ, Ash Jr. MM (2010) Wheeler's dental anatomy, physiology and occlusion, 9th ed. Saunders, St. Louis

Oyamada J, Kitagawa Y, Kato K, et al (2012) Sex differences in linear enamel hypoplasia (LEH) in early modern Japan. Anthropological Science 120:97–101. https://doi.org/10.1537/ase.110715

Palubeckaitė Ž, Jankauskas R, Boldsen J (2002) Enamel hypoplasia in Danish and Lithuanian Late Medieval/Early Modern samples: a possible reflection of child morbidity and mortality patterns. International Journal of Osteoarchaeology 12:189–201. https://doi.org/10.1002/oa.607

Ramos Espejo A, Castro E, Romacho F, Juliá P (2002) Crónica de un sueño: memoria de la transición democrática en Granada (1973/1983). C&T editores, Granada

Rathbun TA (1987) Health and disease at a South Carolina plantation: 1840–1870. American Journal of Physical Anthropology 74:239–253. https://doi.org/10.1002/ajpa.1330740211

Reid DJ, Dean MC (2000) Brief communication: The timing of linear hypoplasias on human anterior teeth. American Journal of Physical Anthropology 113:135–139. https://doi.org/10.1002/1096-8644(200009)113:1<135::aid-ajpa13>3.3.co;2-1

Ritzman TB, Baker BJ, Schwartz GT (2008) A fine line: a comparison of methods for estimating ages of linear enamel hypoplasia formation. American Journal of Physical Anthropology 135:348–361. https://doi.org/10.1002/ajpa.20750

Rugg-Gunn AJ, Al-Mohammadi SM, Butler TJ (1998) Malnutrition and developmental defects of enamel in 2- to 6-year-old Saudi boys. Caries Research 32:181–192. https://doi.org/10.1159/000016451

Salanitri S, Seow WK (2013) Developmental enamel defects in the primary dentition: aetiology and clinical management. Australian Dental Journal 58:133–140. https://doi.org/10.1111/adj.12039

Saunders SR, Keenleyside A (1999) Enamel hypoplasia in a Canadian historic sample. American Journal of Human Biology 11:513–524. https://doi.org/10.1002/(SICI)1520-6300(1999)11:4<513::AID-AJHB11>3.0.CO;2-8

Siek T (2013) The osteological paradox and issues of interpretation in paleopathology. vis-à-vis: Explorations in Anthropology 13:92–101

Skinner MF (1986) An enigmatic hypoplastic defect of the deciduous canine. American Journal of Physical Anthropology 69:59–69. https://doi.org/10.1002/ajpa.1330690108

Skinner MF, Hung JTW (1989) Social and biological correlates of localized enamel hypoplasia of the human deciduous canine tooth. American Journal of Physical Anthropology 79:159–175. https://doi.org/10.1002/ajpa.1330790204

Slaus M (2000) Biocultural analysis of sex differences in mortality profiles and stress levels in the late medieval population from Nova Raca, Croatia. American Journal of Physical Anthropology 111:193–209

Slayton RL, Warren JJ, Kanellis MJ, et al (2001) Prevalence of enamel hypoplasia and isolated opacities in the primary dentition. Pediatric Dentistry 23:32–36

Smith CB, Littleton J (2019) Enamel defects at Roonka, South Australia: indicators of poor health or the osteological paradox? Australian Archaeology 85:139–150. https://doi.org/10.1080/03122417.2019.1644863

Solomons NW, Keusch GT (1981) Nutritional implications of parasitic infections. Nutrition Reviews 39:149–161. https://doi.org/10.1111/j.1753-4887.1981.tb06762.x

Springate ME (2016) Coffin hardware in nineteenth century America. Left Coast Press, Walnut Creek (CA)

Sunderland EP, Smith CJ, Sunderland R (1987) A histological study of the chronology of initial mineralization in the human deciduous dentition. Archives of Oral Biology 32:167–174. https://doi.org/10.1016/0003-9969(87)90130-0

Swärdstedt T (1966) Odontological aspects of a Medieval population from the province of Jamtland/Mid-Sweden. Tiden-Barnängen, Stockholm

Sweeney EA, Saffir AJ, de Leon R (1971) Linear hypoplasia of deciduous incisor teeth in malnourished children. American Journal of Clinical Nutrition 24:29–31. https://doi.org/10.1093/ajcn/24.1.29

Taji S, Hughes T, Rogers J, Townsend G (2000) Localised enamel hypoplasia of human deciduous canines: genotype or environment? Australian Dental Journal 45:83–90. https://doi.org/10.1111/j.1834-7819.2000.tb00246.x

van Gerven DP, Beck R, Hummert JR (1990) Patterns of enamel hypoplasia in two medieval populations from Nubia’s Batn el Hajar. American Journal of Physical Anthropology 82:413–420. https://doi.org/10.1002/ajpa.1330820403

Vargas-Frías L, Sáinz-Otero A, Marían-Paz AJ (2018) Mortalidad por enfermedades carenciales e infecciosas digestivas en La Zubia (Granada) (1931-1945). Temperamentum 14:e12207-undefined

Velló MA, Martínez-Costa C, Catalá M, et al (2010) Prenatal and neonatal risk factors for the development of enamel defects in low birth weight children. Oral Diseases 16:257–262. https://doi.org/10.1111/j.1601-0825.2009.01629.x

Viciano J, de Luca S, Irurita J, Alemán I (2018) Age estimation of infants through metric analysis of developing anterior deciduous teeth. Journal of Forensic Sciences 63:20–30. https://doi.org/10.1111/1556-4029.13505

White BL, Marks B (1995) The new first three years of life: the completely revised and updated edition of the parenting classic. Fireside, New York

Wood JW, Milner GR, Harpending HC, Weiss KM (1992) The osteological paradox: problems of inferring prehistoric health from skeletal samples. Current Anthropology 33:343–358

Xing S, Guatelli-Steinberg D, O’Hara M, et al (2016) Micro-CT Imaging and Analysis of Enamel Defects on the Early Late Pleistocene Xujiayao Juvenile. International Journal of Osteoarchaeology 26:935–946. https://doi.org/10.1002/oa.2504

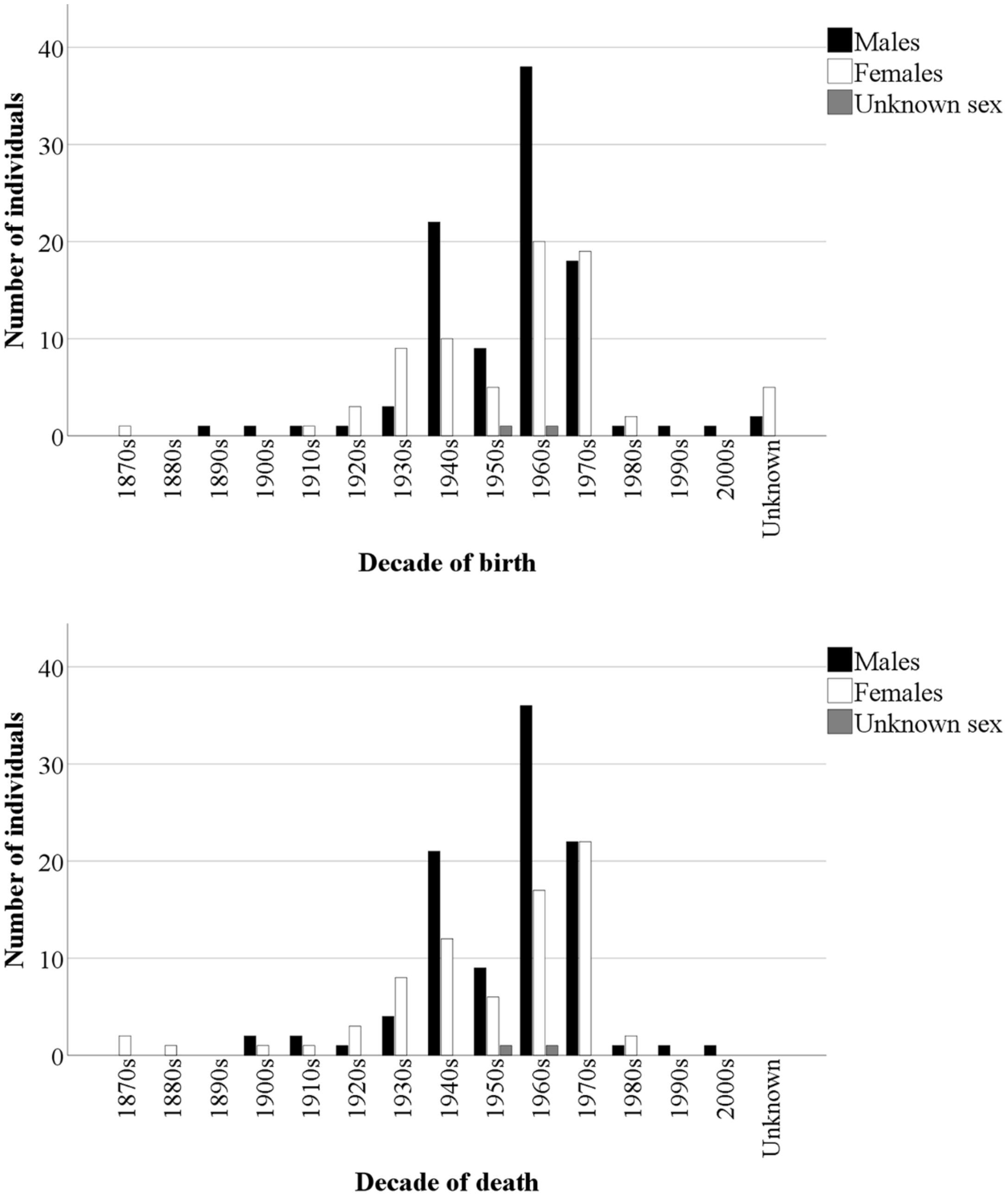

Fig. 1 Distribution of the sample by decades of birth and death.

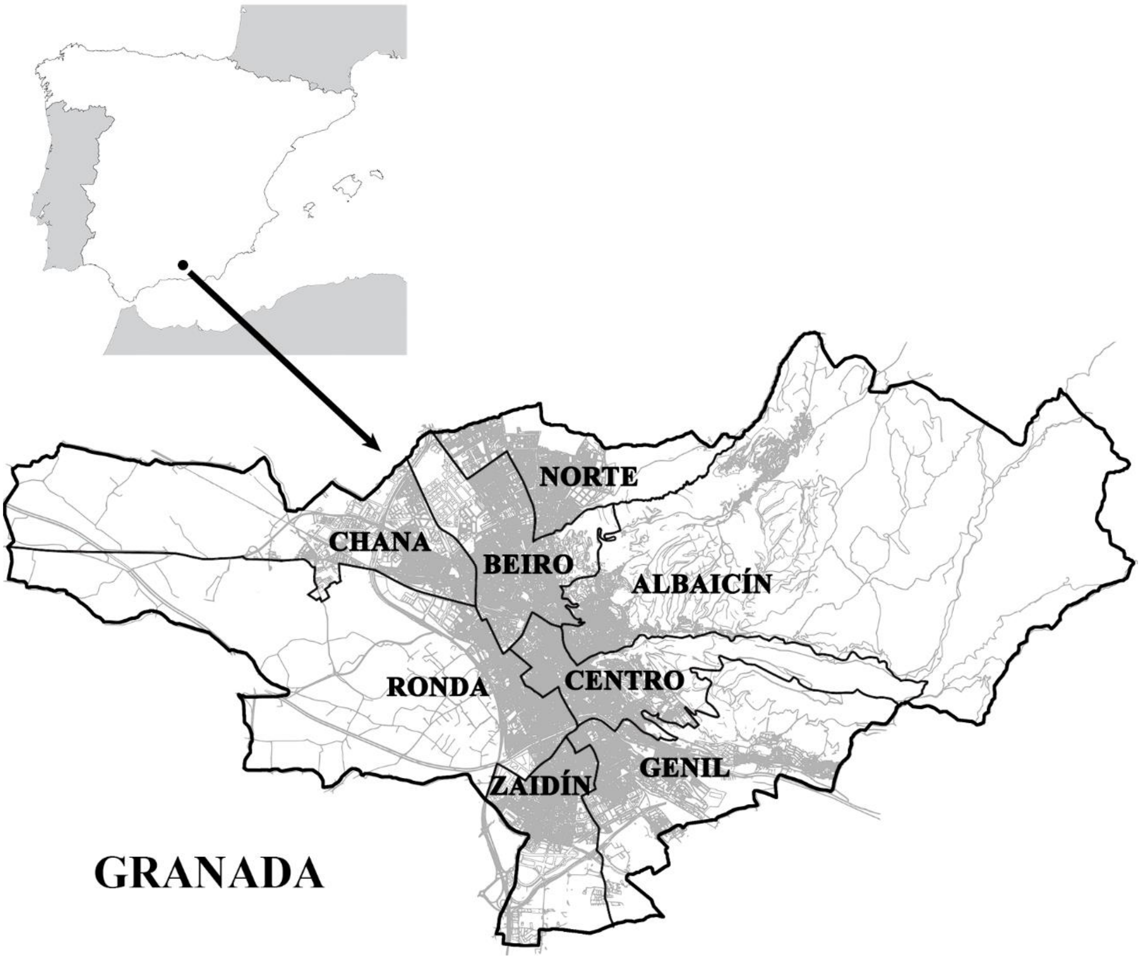

Fig. 2 Geographic location of the city of Granada (Spain) and its division in eight municipal districts.

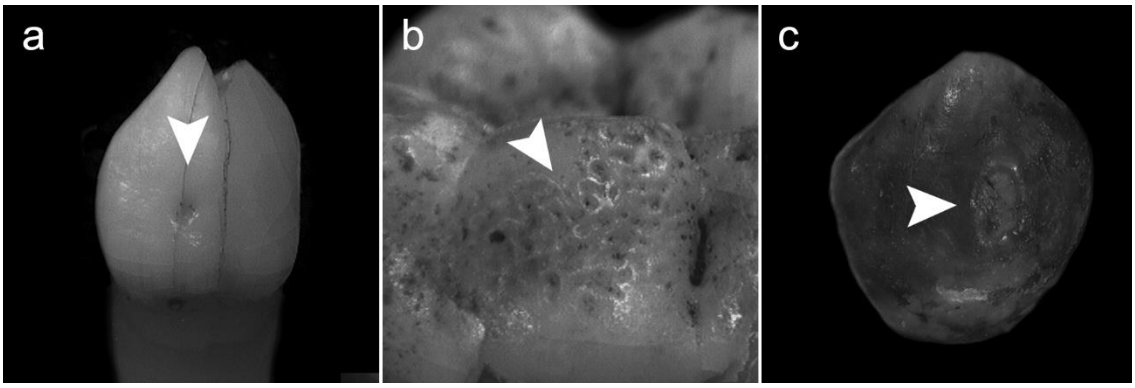

Fig. 3 Examples of dental enamel hypoplasia in the deciduous teeth of the Granada sample. (**a**) Mandibular right lateral incisor showing a pit in the middle third of the labial surface. (**b**) Mandibular right second molar showing uniform circular pitting enamel hypoplasia. (**c**) Maxillary left canine showing a localized area of missing enamel in the middle third of the labial surface.

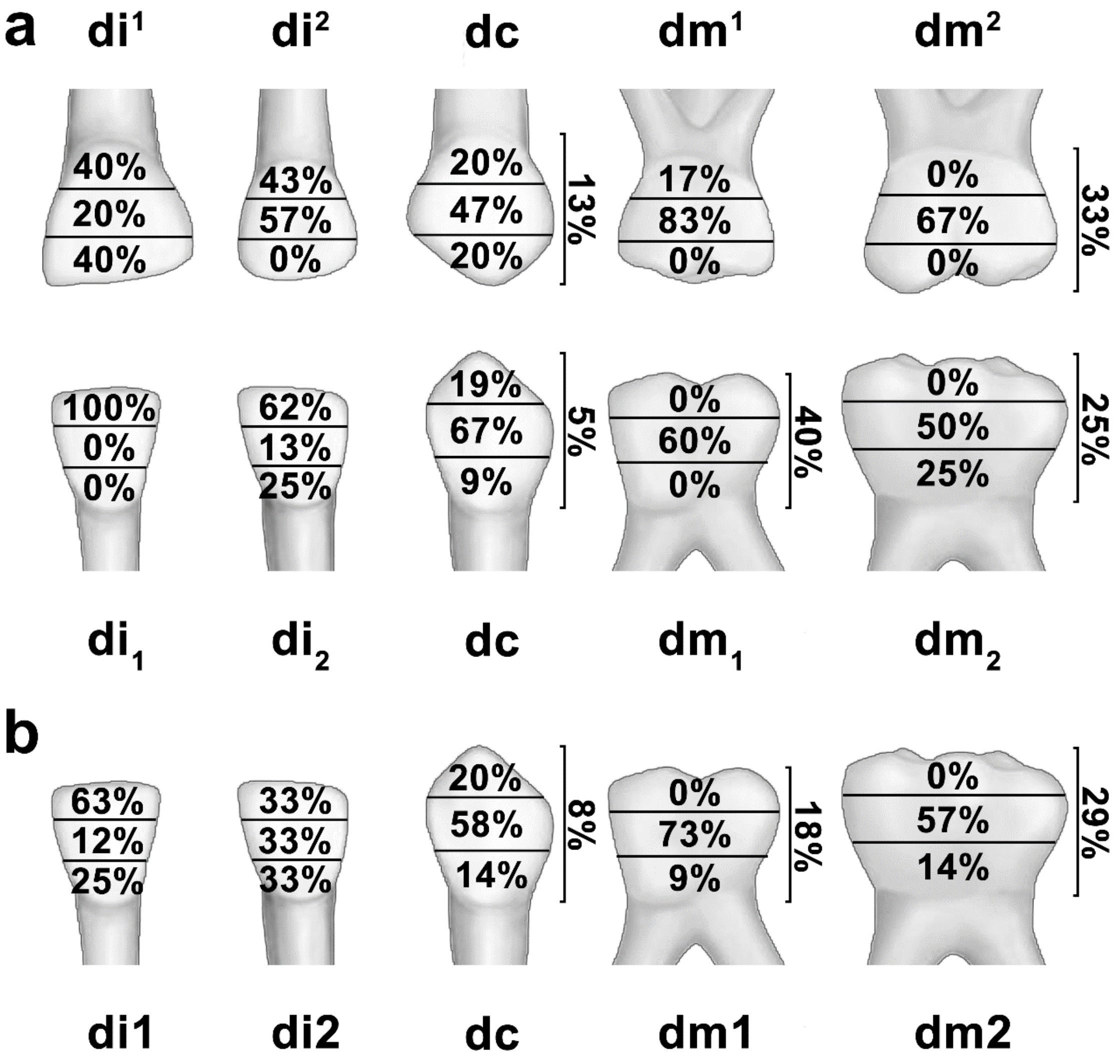

Fig. 4 Distribution of developmental enamel defects by incisal/occlusal, medial and cervical crown thirds and full crown by tooth type. (**a**) Distribution by maxillary and mandibular teeth. (**b**) Distribution with maxillary and mandibular teeth pooled. The vertical bar represents the percentage of enamel defects distributed on the entire crown surface.

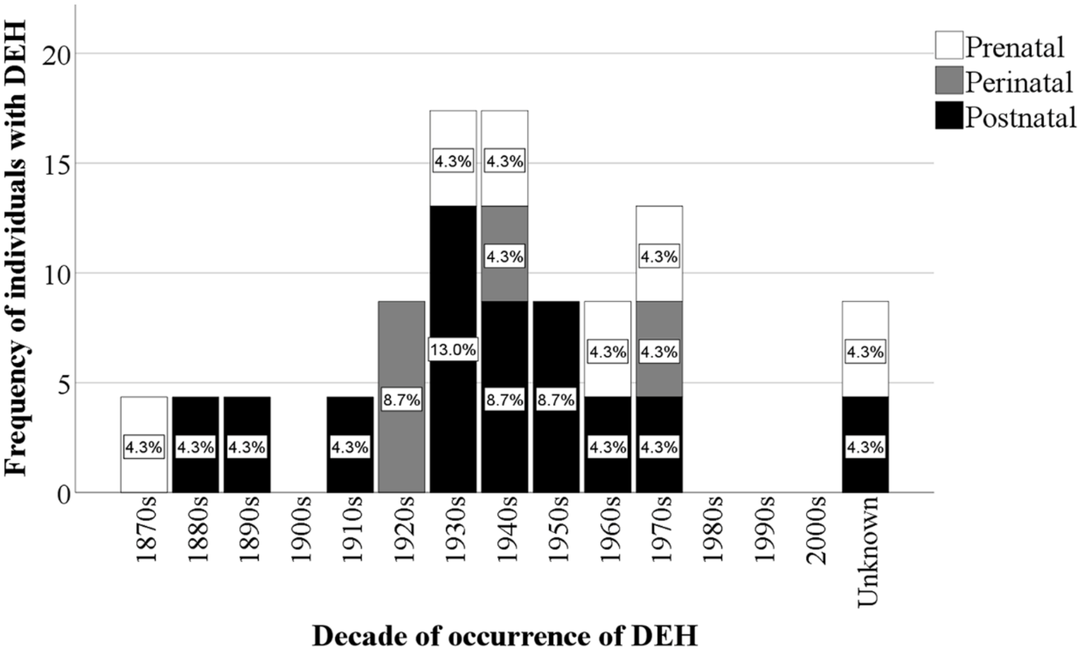

Fig. 5 Frequency of individuals with DEH by decade of occurrence of enamel defects.

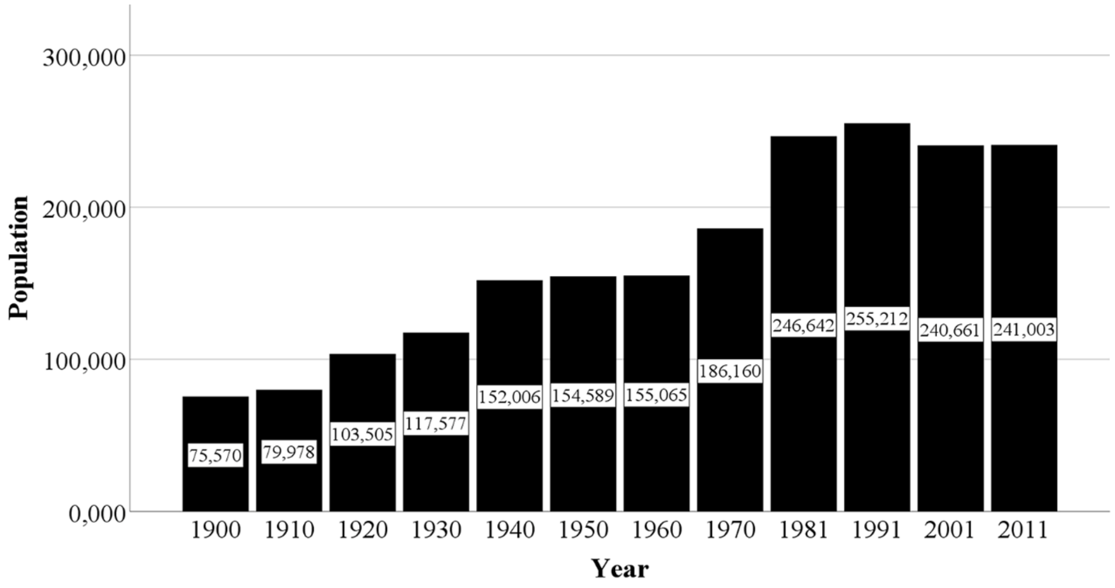

Fig. 6 Demographic evolution of Granada population from 1900 to 2011. Source: Instituto Nacional de Estadística (INE; Spanish National Statistics Institute). (https://www.ine.es/)

Table 1. Distribution of the sample by age at death and sex.

Age at death (in months)		Number of individuals by sex [*n* (%)] Males	Females	Unknown
Fetus	-3–-2.1	0 (0)	1 (1.3)	0 (0)
	-2–-1.1	1 (1.0)	4 (5.3)	0 (0)
	-1–Birth	1 (1.0)	1 (1.3)	0 (0)
	Unknown	10 (10.0)	9 (12.0)	2 (100.0)
Infant	Birth–0.9	13 (13.0)	12 (16.0)	0 (0)
	1–1.9	13 (13.0)	10 (13.3)	0 (0)
	2–2.9	9 (9.0)	6 (8.0)	0 (0)
	3–3.9	5 (5.0)	2 (2.7)	0 (0)
	4–4.9	5 (5.0)	1 (1.3)	0 (0)
	5–5.9	6 (6.0)	1 (1.3)	0 (0)
	6–6.9	2 (2.0)	2 (2.7)	0 (0)
	7–7.9	3 (3.0)	2 (2.7)	0 (0)
	8–8.9	2 (2.0)	0 (0)	0 (0)
	9–9.9	1 (1.0)	1 (1.3)	0 (0)
	10–10.9	3 (3.0)	0 (0)	0 (0)
	11–11.9	1 (1.0)	1 (1.3)	0 (0)
	12–12.9	1 (1.0)	1 (1.3)	0 (0)
	13–13.9	2 (2.0)	0 (0)	0 (0)
	14–14.9	3 (3.0)	0 (0)	0 (0)
	…	…	…	…
	16–16.9	0 (0)	1 (1.3)	0 (0)
	17–17.9	0 (0)	1 (1.3)	0 (0)
	18–18.9	1 (1.0)	0 (0)	0 (0)
	19–19.9	1 (1.0)	0 (0)	0 (0)
	20–20.9	1 (1.0)	0 (0)	0 (0)
	21–21.9	2 (2.0)	0 (0)	0 (0)
	22–22.9	0 (0)	2 (2.7)	0 (0)
	…	…	…	…
	25–25.9	0 (0)	1 (1.3)	0 (0)
	26–26.9	0 (0)	1 (1.3)	0 (0)
	27–27.9	0 (0)	1 (1.3)	0 (0)
	28–28.9	0 (0)	1 (1.3)	0 (0)
	…	…	…	…
	30–30.9	0 (0)	1 (1.3)	0 (0)
	…	…	…	…
	36–36.9	3 (3.0)	0 (0)	0 (0)
	37–37.9	1 (1.0)	1 (1.3)	0 (0)
	38–38.9	1 (1.0)	0 (0)	0 (0)
	…	…	…	…
	42–42.9	1 (1.0)	0 (0)	0 (0)
	…	…	…	…
	48–48.9	0 (0)	2 (2.7)	0 (0)
	…	…	…	…
	56–56.9	1 (1.0)	0 (0)	0 (0)
	…	…	…	…
	60–60.9	2 (2.0)	3 (4.0)	0 (0)
	…			
	72–72.9	1 (1.0)	1 (1.3)	0 (0)
	…	…	…	…
	85–85.9	1 (1.0)	0 (0)	0 (0)
	Unknown	3 (3.0)	5 (6.7)	0 (0)
	Total	100 (100.0)	75 (100.0)	2 (100.0)

Table 2. Distribution of the sample divided by sex and municipal districts.

	Number of individuals				Number of teeth			
Municipal district	Males	Females	Unknown	Total	Males	Females	Unknown	Total
Albaicín	11	8	0	19	123	75	0	198
Beiro	17	18	0	35	150	129	0	279
Centro	29	22	1	52	295	275	20	590
Chana	6	5	0	11	42	32	0	74
Genil	4	1	0	5	47	4	0	51
Norte	2	0	0	2	24	0	0	24
Ronda	13	6	0	19	135	72	0	207
Zaidín	6	8	0	14	53	95	0	148
Unknown	12	7	1	20	118	94	8	220
Total	100	75	2	177	987	776	28	1791

Table 3. Distribution of the sample divided by sex and quality of the coffin.

	Number of individuals				Number of teeth			
Quality of coffin	Males	Females	Unknown	Total	Males	Females	Unknown	Total
Standard	22	19	0	41	205	125	0	330
Medium	51	29	1	81	490	300	20	810
Luxury	10	8	0	18	116	109	0	225
Unknown	17	19	1	37	176	242	8	426
Total	100	75	2	177	987	776	28	1791

Table 4. Dimensions of crown height (crown completed) and periods of enamel development in deciduous dentition.

		Data collected			Data extracted†		Data calculated	
	Tooth type	Crown height (mm)			First evidence of calcification (prenatal months)	Crown completed (postnatal months)	Duration of enamel development (months)	Crown development per month (mm)
Dental arch		n	Mean	SD				
Maxilla	di1	41	6.08	0.41	4.93	3.12	7.19	0.85
	di2	32	5.92	0.36	5.54	5.28	8.74	0.68
	dc	15	7.16	0.40	6.60	12.00	14.4	0.50
	dm1	31	5.36	0.52	5.35	8.04	11.69	0.46
	dm2	8	5.62	0.22	5.69	16.68	19.99	0.28
Mandible	di1	36	5.54	0.52	5.48	4.44	7.96	0.70
	di2	31	5.90	0.48	5.90	6.48	9.58	0.62
	dc	15	7.07	0.46	6.53	15.36	17.83	0.40
	dm1	23	5.54	0.64	6.06	7.56	10.50	0.53
	dm2	6	5.97	0.24	6.06	13.56	16.50	0.36

†Data extracted from previous publications on the same osteological collection (Irurita et al. 2014a, b; Viciano et al. 2018).

Table 5. Specific regression equations constructed to calculate the age of the DEH formation in deciduous dentition for the Granada osteological collection of identified infants and young children.

Dental arch	Tooth type	Equation
Maxilla	di^1	Age at DEH formation (in months) = 3.12 – (DEH_{Ht}/0.85)
	di^2	Age at DEH formation (in months) = 5.28 – (DEH_{Ht}/0.68)
	dc	Age at DEH formation (in months) = 12.00 – (DEH_{Ht}/0.50)
	dm^1	Age at DEH formation (in months) = 8.04 – (DEH_{Ht}/0.46)
	dm^2	Age at DEH formation (in months) = 16.68 – (DEH_{Ht}/0.28)
Mandible	di_1	Age at DEH formation (in months) = 4.44 – (DEH_{Ht}/0.70)
	di_2	Age at DEH formation (in months) = 6.48 – (DEH_{Ht}/0.62)
	dc	Age at DEH formation (in months) = 15.36 – (DEH_{Ht}/0.40)
	dm_1	Age at DEH formation (in months) = 7.56 – (DEH_{Ht}/0.53)
	dm_2	Age at DEH formation (in months) = 13.56 – (DEH_{Ht}/0.36)

DEH_{Ht} = distance in mm between DEH and the cemento–enamel junction.

Table 6. Prevalence of dental enamel hypoplasia per tooth divided by sex and tooth type.

		Number of teeth [*n* (%)]											
		Maxillary teeth						Mandibular teeth					
Sex	Teeth	di^1	di^2	dc	dm^1	dm^2	Total	di_1	di_2	dc	dm_1	dm_2	Total
Males	Observed	128	115	73	124	89	529	105	95	68	108	82	458
	Affected	2 (1.6)	3 (2.6)	10 (13.7)	4 (3.2)	2 (2.2)	21 (4.0)	0 (0)	3 (3.2)	12 (17.6)	2 (1.9)	2 (2.4)	19 (4.1)
Females	Observed	95	84	54	106	67	406	75	82	54	95	64	370
	Affected	3 (3.2)	4 (4.8)	5 (9.3)	2 (1.9)	1 (1.5)	15 (3.7)	3 (4.0)	5 (6.1)	9 (16.7)	3 (3.2)	2 (3.1)	22 (6.0)
Unknown	Observed	3	4	2	3	2	14	2	4	2	4	2	14
	Affected	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Total	Observed	226	203	129	233	158	949	182	181	124	207	148	842
	Affected	5 (2.2)	7 (3.4)	15 (11.6)	6 (2.6)	3 (1.9)	36 (3.8)	3 (1.6)	8 (4.4)	21 (17.0)	5 (2.4)	4 (2.7)	41 (4.9)

Table 7. Prevalence of dental enamel hypoplasia per individual divided by municipal district and quality of coffin.

		Number of individuals [*n* (%)]														
		Municipal district										Quality of coffin				
Sex	Individuals	Albaicín	Beiro	Centro	Chana	Genil	Norte	Ronda	Zaidín	Unknown	Total	Standard	Medium	Luxury	Unknown	Total
Males	Observed	11	17	29	6	4	2	13	6	12	100	22	51	10	17	100
	Affected	1 (9.1)	3 (17.6)	7 (24.1)	0 (0)	0 (0)	0 (0)	1 (7.7)	1 (16.7)	2 (16.7)	15 (15.0)	3 (13.6)	6 (11.8)	3 (30.0)	3 (17.6)	15 (15.0)
Females	Observed	8	18	22	5	1	0	6	8	7	75	19	29	8	19	75
	Affected	0 (0)	2 (11.1)	10 (45.5)	1 (20.0)	0 (0)	0 (0)	2 (33.3)	1 (12.5)	4 (25.0)	20 (26.7)	1 (5.3)	6 (20.7)	2 (25.0)	11 (57.9)	20 (26.7)
Unknown	Observed	0	0	1	0	0	0	0	0	1	2	0	1	0	1	2
	Affected	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Total	Observed	19	35	52	11	5	2	19	14	20	177	41	81	18	37	177
	Affected	1 (5.3)	5 (14.3)	17 (32.7)	1 (9.1)	0 (0)	0 (0)	3 (15.8)	2 (14.3)	6 (3.3)	35 (19.8)	4 (9.8)	12 (14.8)	5 (27.8)	14 (37.8)	35 (19.8)

Table 8. Number of stressful events in 1-month-age ranges (sexes pooled) per municipal district.

Age range of stressful events (months)		Municipal district									
		Albaicín	Beiro	Centro	Chana	Genil	Norte	Ronda	Zaidín	Unknown	Total
Fetus	-2–-1.1	0	0	1	0	0	0	0	1	0	2
	-1–Birth	0	0	2	0	0	0	0	0	2	4
Infant	Birth–0.9	0	0	0	0	0	0	0	0	0	0
	1–1.9	0	0	1	1	0	0	0	0	0	2
	2–2.9	0	0	3	0	0	0	0	0	0	3
	3–3.9	0	0	1	0	0	0	0	0	0	1
	4–4.9	0	1	0	0	0	0	0	0	0	1
	5–5.9	0	0	0	0	0	0	0	0	0	0
	6–6.9	0	0	2	0	0	0	1	0	0	3
	7–7.9	0	0	1	0	0	0	0	0	1	2
	8–8.9	0	0	4	0	0	0	0	0	0	4
	9–9.9	0	0	3	0	0	0	0	0	1	4
	10–10.9	0	0	0	0	0	0	1~~0~~	0	0	1~~0~~
	11–11.9	0	0	0	0	0	0	0~~1~~	0	0	0~~1~~
Total events		0	1	18	1	0	0	2	1	4	27
Individuals affected		0	1	14	1	0	0	2	1	4	23
Mean number of events per individual		0	1	1.3	1	0	0	1	1	1	

Table 9. Number of stressful events in 1-month-age ranges in males and females per quality of coffin.

		Quality of coffin											
		Standard			Medium			Luxury			Unknown		
Age range of stressful events (months)		Males	Females	Total	Males	Females	Total	Males	Females	Total	Males	Females	Total
Fetus	-2–-1.1	0	0	0	0	2	2	0	0	0	0	0	0
	-1–Birth	0	0	0	0	1	1	0	0	0	0	3	3
Infant	Birth–0.9	0	0	0	0	0	0	0	0	0	0	0	0
	1–1.9	0	1	1	0	0	0	0	0	0	0	1	1
	2–2.9	0	0	0	0	0	0	1	1	2	0	1	1
	3–3.9	0	0	0	1	0	1	0	0	0	0	0	0
	4–4.9	0	0	0	0	1	1	0	0	0	0	0	0
	5–5.9	0	0	0	0	0	0	0	0	0	0	0	0
	6–6.9	1	0	1	1	0	1	0	0	0	0	1	1
	7–7.9	0	0	0	1	0	1	0	0	0	0	1	1
	8–8.9	0	0	0	0	0	0	0	2	2	0	2	2
	9–9.9	0	0	0	0	0	0	0	0	0	3	1	4
	10–10.9	0	0	0	0	1	1	0	0	0	0	0	0
	11–11.9	0	0	0	0	0	0	0	0	0	0	0	0
Total events		1	1	2	3	5	8	1	3	4	3	10	13
Individuals affected		1	1	2	2	5	7	1	2	3	3	8	11
Mean number of events per individual		1	1	1	1.5	1	1.1	1	1.5	1.3	1	1.3	1.2

Table 10. Distribution of stressful events in 1-month-age ranges by sex.

Age range of stressful events (months)		Males		Females		More details of those individuals that suffered two stressful events (age range in months):
		First event	Second event	First event	Second event	
Fetus	-2–-1.1	0	0	2	0	
	-1–Birth	0	0	4	0	*Individual #1: female, 72 months age at death*
Infant	Birth–0.9	0	0	0	0	First stressful event: -1–Birth
	1–1.9	0	0	1	1	Second stressful event: 1–1.9
	2–2.9	1	0	2	0	*Individual #2: female, 22 months age at death*
	3–3.9	1	0	0	0	First stressful event: 2–2.9
	4–4.9	0	0	1	0	Second stressful event: 8–8.9
	5–5.9	0	0	0	0	*Individual #3: female, 28 months age at death*
	6–6.9	1	1	1	0	First stressful event: 2–2.9
	7–7.9	1	0	1	0	Second stressful event: 8–8.9
	8–8.9	0	0	2	2	*Individual #4: male, 85 months age at death*
	9–9.9	3	0	1	0	First stressful event: 3–3.9
	10–10.9	0	0	1	0	Second stressful event: 6–6.9
	11–11.9	0	0	0	0	
Total events		7	1	16	3	