Patterns in dental enamel hypoplasia by sex and age at death in two archaeological populations

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Abstract

Aims: Levels of enamel hypoplasia in past populations are frequently used to study health. However, few studies have looked at patterning in the occurrence of different types of hypoplasia. In this pilot study, skeletal remains from an Iron Age tomb at Pella in Jordan were analysed for the presence of linear and pit enamel hypoplasia, to investigate enamel hypoplasia aetiology by comparison of the results obtained for adults and juveniles, and females and males.

Methods and Results: The proportion of individuals with enamel hypoplasia was determined for males and females and for adults and juveniles using the F.D.I. Developmental Defects of Enamel (DDE) Index. Although males and females had a similar percentage of individuals affected, females had a higher prevalence of enamel hypoplasia per tooth than males. Adults had a higher prevalence of enamel hypoplasia than juveniles. In particular, adults had a higher prevalence of linear enamel hypoplasias and pit enamel hypoplasia arrays, but a similar prevalence of single pit enamel hypoplasia when both the permanent and deciduous dentitions were considered. These differences were largely due to different patterns and frequencies of enamel hypoplasia in deciduous teeth compared to permanent teeth.

Conclusions: The different patterns of occurrence of the various forms of hypoplasia observed in this study imply that single pits may have a different aetiology to linear enamel hypoplasias and pit arrays. By investigating similar patterns in other archaeological populations, we may develop a better understanding of the specific causes of particular types of enamel hypoplasia, and may be able to more meaningfully interpret enamel hypoplasia data from past populations.

Keywords: palaeopathology, enamel defects, childhood health, aetiology, mortality

Introduction

The frequency of enamel hypoplasia in archaeological populations is used to study health in the past. Enamel hypoplasia frequencies are commonly compared to those in modern populations, to determine the relative childhood health status of a population (1, 2). However, the aetiology of enamel hypoplasia is multifactorial with many identified genetic and environmental factors contributing to its formation (3), rendering meaningful interpretation of their occurrence in archaeological populations problematic.

Epidemiological studies of enamel hypoplasia in modern populations have indicated the wide range of types of environmental factors related to enamel hypoplasia. Enamel hypoplasia occurrence has been linked to socioeconomic status (1, 4-6), malnutrition (7-11), infectious diseases including dental caries (7, 11-13), weaning (e.g. 14, 15), fluoride levels (e.g. 16, 17) and even premature birth (18, 19). Depending upon the severity of the stress, the extent to which the individual is exposed to this stress and the genetic susceptibility of the individual to enamel hypoplasia formation (20), the individual may not experience growth disruption, or may succumb to the stressor and die. Neither of these scenarios would produce an enamel hypoplasia, even though other individuals experiencing the same stressor may suffer from growth disruption, resulting in the formation of an enamel hypoplasia.

Some researchers have suggested that there is not a strong link between the duration of the stressful event and the absolute size of the enamel hypoplasia at the individual level and that the duration of the insult can be best determined by counting the perikymata within the defect (21, 22). However, examination of perikymata counts within hypoplastic defects have indicated that the average enamel defect width is a more accurate measure of the duration of physiological disturbance when determined at the population level (23).

Enamel hypoplasias can appear on the tooth in three different forms: linear, pit or planar. Linear enamel hypoplasias (LEH) are defined as grooves on the enamel surface. LEHs tend to run horizontally, parallel to the cementoenamel junction, and resemble exaggerated perikymata. In contrast, pit enamel hypoplasias are well-defined pits in the enamel surface. These can appear as a linear array, a non-linear array or single, isolated pits. In contrast, planar enamel hypoplasia results from the absence of whole sections of enamel from the tooth. The existence of these three forms of enamel hypoplasia is well established, but while the causes of LEH are relatively well understood, the aetiology of pit enamel hypoplastic defects is not yet fully known (21). LEH is assumed to be primarily due to generalized systemic stress, due to infectious disease, nutritional stress or a combination of the two, with planar enamel hypoplasia being an extreme case of LEH (24). In contrast, pit enamel hypoplasias have sometimes been linked with episodes of localised trauma (e.g. 25). However, the evidence for such a split in aetiology in human teeth is limited at present.

This paper reports the findings of a pilot study which sought to compare the occurrence of enamel hypoplasia between males and females and between adults and juveniles from an archaeological population, with a relatively high overall prevalence of hypoplasia (26) in order to investigate possible aetiological factors.

Methods

The skeletons examined in this study consist of the skeletal remains of Tomb 89 from the Early Iron Age (1100-900 BC) site of Pella in Jordan. Pella is located in the foothills of the Transjordanian Plateau (Fig. 1), close to the modern village of Tabaqat Fahl on the eastern side of the Jordan Valley (27), and was occupied from approximately 8000 BC until AD 1600

(28). At this time, Pella was a relatively small agricultural settlement, and its population is estimated at around 500 individuals (Bourke, pers. comm.). The population appears to have been relatively insular in the Early Iron Age, with a distinctive pottery style at the site that is unlike those of neighbouring settlements (29).

Fig. 1. The location of Pella within the Levant

Tomb 89 was excavated in 1987 by the University of Sydney School of Archaeology (Bourke, pers. comm.). The grave goods within the tomb suggest that the individuals buried within the tomb represent a range of social classes, although the absence of rich material culture suggests the absence of elites within the tomb. Analysis of the palaeopathology of the individuals from this tomb indicated that the population were generally healthy (30). However, the prevalence of enamel hypoplasia within this population is high, suggesting that general living conditions at the site were poor (26).

Although the skeletal remains within the tomb were disarticulated and often fragmentary, it was possible to identify the skeletal remains belonging to 78 individuals within the tomb. Because of taphonomic changes, it was not possible to determine in any detail the order of burial of the individuals within the tomb or the status of particular individuals within the tomb. The population analysed has a relatively low rate of dental caries and few cases of severe attrition.

All hypoplastic defects of the enamel observed were recorded, regardless of whether they were associated with hypoplastic lesions on other teeth of the dentition, to allow mapping of hypoplastic defects throughout the dentition. However, for the analysis of prevalences of

hypoplasia per individual, a multiple-tooth approach was used. While this approach is more complex than utilizing a single tooth type, it allows easier identification of enamel hypoplastic defects regardless of aetiology (31).

Only teeth that could be identified as belonging to a particular individual from the tomb were selected for study and all teeth that could be allocated to a tooth type were considered from each individual, including both permanent and deciduous teeth. While deciduous teeth were included in this study, it is recognised that as they form perinatally, any enamel hypoplastic defects present on these teeth will reflect maternal rather than childhood health. However, given the aim of this study to investigate patterns in hypoplastic defect occurrence to improve understanding of enamel hypoplasia aetiology in archaeological populations, this was felt to be a benefit.

Mud and soil adhering to the teeth were initially removed using tap water and cotton buds. Any calculus that was covering a large portion of the enamel surface was removed using a scaler, and retained for future analysis.

Enamel hypoplastic defects were recorded on the teeth using the method of Buikstra and Ubelaker (32), which utilizes the F.D.I. Developmental Defects of Enamel (DDE) Index (33). Although this index has received some criticism (34, 35), it remains widely used within biological anthropological research (24).

The teeth were observed wet at 16x magnification using a light microscope, with oblique lighting. The end of a scaler was used as a probe to aid in the identification of enamel hypoplastic defects, as recommended by Lukacs (36). An enamel hypoplastic defect was not

recorded unless it was both visible to the naked eye and identifiable by the probe (20). For linear enamel hypoplasia, defects were not recorded unless they ran across the majority of the width of the labial tooth surface. Because expression of enamel hypoplasia can vary between the left and right sides of the jaw, both left and right teeth of each tooth type were examined for enamel hypoplastic defect occurrence. To avoid double counting, each type of enamel hypoplastic defect was counted as being present in an individual tooth type if it was observed on at least one of the left and right antimeres of that tooth type.

The age at death of the individuals from the Early Iron Age tomb was determined using the eruption of the third molars as a marker of adult status. As these teeth erupt in early adulthood (37), they provide a useful skeletal indicator of adulthood. Where third molars were not recovered and where neither deciduous nor developing teeth were present, the individuals were assigned indeterminate status. All other individuals were assigned juvenile status. As the skeletal remains from Tomb 89 were often fragmentary, it was not always possible to associate a particular jaw with other parts of the skeleton which can be used to indicate age-at-death. Therefore, the ages-at-death used in this study are based on dental development. The age-at-death distribution for the juveniles within the population indicates that the adult and juvenile segments of the population are largely discrete (Fig. 2), as a result of the early age at death of the majority of the juvenile population. The population consisted of twenty-three adults, eleven juveniles and sixteen individuals whose age could not be ascertained, usually due to incomplete preservation of the cranial remains. While methods which could allow the splitting of the adult sample into young, middle and older adults exist (e.g. tooth wear), it was felt that the small sample size and preliminary nature of this study did not justify such an approach. Similarly, due to the small sample size of the population, the age at formation of enamel hypoplastic defects was not examined by sex or age-at-death in

the present study. The age at formation of enamel hypoplastic defects in the population as a whole has previously been reported in Griffin and Donlon (26).

Fig. 2. Age distribution of the juveniles from Tomb 89.

Sex was determined using morphological features supplemented by metric features for all individuals who were determined to be over 15 years of age. Due to the poor preservation and frequent lack of a clear association of the pelvic remains with the cranial remains used for the enamel hypoplasia analysis, the sex of most of the individuals was determined using only the cranium. Each individual was assigned a score for morphological features of the skull according to the method described by Buikstra and Ubelaker (32). Where the pelvis was present and clearly associated with the skull, the individual was also scored on morphological features of the pelvis, and these scores were combined with those from the cranium to produce a sex determination, according to the method of Buikstra and Ubelaker (32). The allocations of sex were supported by the use of the diagnoses described by Ferembach *et al.* (38) and by the measurement of the maximum diameter of the femoral head where present and associated with the cranial remains (39). The results from all three sets of measurements and observations were combined to determine the sex of the individual.

Of the individuals included in this study (i.e. those who had teeth available for study) for which sex could be determined, four were classed as male, six as probable male, four as female, and twelve as probable female. The population included in this analysis therefore consisted of ten males and sixteen females, and of twenty-three adults and eleven juveniles, resulting in the sample size per tooth type shown in Table 1.

Table 1. Sample size per tooth type of the population of Tomb 89

The possibility of an association between sex or age-at-death and the occurrence of each type of hypoplastic defect was analysed using Fisher's exact test for the data on prevalence by individual and using the χ^2 test for the data on prevalence by tooth. All analyses were conducted using the statistics package MedCalc 9.3.9.0. P values of less than 0.05 were taken to indicate statistical significance.

Results

Both females and males in Tomb 89 had a high frequency of enamel hypoplastic defects (Tables 2a and 2b), consistent with the high prevalence observed in the population as a whole (26). This high prevalence is presumably in part due to the protocol for recording of enamel hypoplastic defects used in the present study, which reported all hypoplastic defects observed regardless of their correspondence with defects on other teeth in the dentition. All females had some form of enamel hypoplastic defect (n = 16), while nine out of the ten males had enamel hypoplastic defects. Only pit and linear enamel hypoplastic defects were observed in the population of Tomb 89, with females having a higher prevalence of both types. However, the differences observed between males and females were comparatively small, and not statistically significant. In contrast, when the prevalences were considered by tooth, females had a higher occurrence of hypoplastic defects than males for linear pit hypoplasia arrays and for overall hypoplasia prevalence.

Table 2. Percentage of individuals from Pella with enamel hypoplastic defects by sex

A different pattern of enamel hypoplastic defect formation was observed between adults and juveniles (Tables 3a and 3b). While all individuals who lived to adulthood had at least one enamel hypoplastic defect (n = 23), only 73 % of those who died during childhood had at least one enamel hypoplastic defect (n = 11). Although the frequency of pit enamel hypoplasia is similar in the two groups, the prevalence of linear enamel hypoplasia is significantly lower in juveniles (46 %) than in adults (100 %) ($p<0.05$). Single pits are equally prevalent in adults and juveniles within the population. In contrast, both non-linear and linear arrays of pits are less common in juveniles than in adults, although none of these trends in pit hypoplasia occurrence were statistically significant. However, this trend is largely due to a different pattern of enamel hypoplastic defects in the deciduous dentition, and disappears when only permanent teeth are considered.

Table 3. Percentage of individuals from Pella with enamel hypoplastic defects by age

Examination of the patterns of occurrence of the various types of pit enamel hypoplasia across the dentition indicates that tooth type may influence the type of enamel hypoplasia formed (Table 4). While single pits were fairly evenly distributed throughout the dentition, the two types of pit arrays were more unevenly distributed. Linear arrays were more commonly found on the posterior teeth, while nonlinear arrays were more common in the anterior teeth. LEH were more commonly found on the anterior teeth, and were less common on molars. Due to the very small number of deciduous teeth within this sample, it was not possible to conduct a similar analysis on the deciduous dentition.

Table 4. Proportions of linear pit arrays, non-linear pit arrays, single pits and LEH in the teeth of the dentition.

Discussion

As few studies have attempted to investigate enamel hypoplasia aetiology through identification of patterns in hypoplasia occurrence, although this population is comparatively small, it provides a preliminary indication of the value of this approach. The few carious teeth in this population and the relatively few individuals with severe attrition mean that only limited amounts of enamel have been lost pre-mortem. While complete dentitions were available for only a few individuals, the high level of hypoplastic defect occurrence observed in this population (26) will make underenumeration of hypoplasia occurrence per individual minimal.

Goodman and Rose (20) recommended only reporting those linear enamel hypoplastic defects which are present on more than one tooth type at points on the tooth surface which represent the same point in dental development. However, as this study sought to investigate the patterning of a range of types of hypoplastic defects, it was decided to report all hypoplastic lesions observed. It is recognised that analysis of enamel hypoplastic defects by tooth type is preferable where possible, due to variations in the susceptibility of different tooth types to the formation of hypoplastic defects (14). However, because of the small sample size of the population, it was decided to report hypoplastic defect occurrence by individual rather than by tooth type. To partially compensate for this problem, enamel hypoplastic defect occurrence was also reported by tooth. Subsequent examination of larger, better preserved skeletal populations will be necessary to further investigate the potential of the approach piloted in the present study.

This study has provided little evidence for a variation in enamel hypoplasia prevalence between males and females in Tomb 89 at Pella by individual. While females have a significantly greater prevalence of enamel hypoplastic defects per tooth than males, as this is not reflected in the data for individuals, it is unclear whether this increased prevalence is due to sample bias as a result of the higher retention of teeth in females than in males. There are also no substantial variations in the occurrence of the different forms of hypoplastic defect between males and females. Accepting the limitations of sample size, the data on males and females suggests that within this population they experienced similar living conditions, in terms of diet and infectious disease exposure.

Examination of age at death and enamel hypoplasia occurrence provides the first indications of a variation in patterning between linear and pit hypoplastic defects. There is a significant difference in the occurrence of enamel hypoplastic defects in the two age groups, with juveniles being less likely to have hypoplastic teeth than those who lived to adulthood. This difference was found to be due to a lower prevalence of enamel hypoplastic defects in deciduous teeth, and when these were removed from the analysis no differences were observed between adults and juveniles in the enamel hypoplasia prevalence in permanent teeth. This may be due to the strong influence of maternal health on the development of deciduous teeth and not on permanent teeth. In addition, there may be an increased risk of infant mortality in mothers who experience stress in the pre-natal period, with most children who have been exposed to such stresses dying prior to the eruption of their deciduous teeth. Deciduous teeth were also more likely to have single pit hypoplastic defects than permanent teeth. Examination of populations with a greater proportion of juveniles might indicate whether this is a meaningful trend or simply an artefact of small sample size.

The difference in the prevalence of the various types of hypoplastic defect in deciduous and permanent teeth does suggest that LEH may have a different aetiology to pit enamel hypoplasia. While LEH has received much study, few studies have been conducted on human populations to elucidate the mechanism of formation of pit enamel hypoplasia. However, this topic has received attention from researchers working on animal populations. The findings of these studies may suggest a possible mechanism for the formation of different types of enamel hypoplasia in humans. In primates, the occurrence of pit enamel hypoplasia on the deciduous canine is believed to be induced by trauma to the developing tooth while it is still unerupted (25). The likelihood of such openings forming is increased by nutritional deficiencies that result in thinning of the bone of the tooth crypt. In the permanent teeth of domestic animals, pit enamel hypoplasia has been found to result from contact between the deciduous tooth root and the unerupted developing permanent tooth (40). Studies of domestic animals suggest that a spread of multiple small pits can be due to calcium deficiency (41). Other conditions which have been implicated in the formation of pit enamel hypoplasias include prematurity and poor maternal diet (42). It seems that the form of the pit enamel hypoplasia (an isolated pit or a diffuse spread of pits) may provide some hint as to which possible cause could be implicated in a particular case.

The variation in the prevalence of different types of hypoplastic defects observed in the present study between the permanent and deciduous dentitions suggests that single pits have a different aetiology to both LEH and pit enamel hypoplasia arrays. As LEH has been linked with nutritional deficiencies and systemic diseases, it seems likely that these factors will also be the causes of linear arrays of pits. Presumably non-linear arrays, which have received less attention in the past, are also caused by nutritional deficiencies or disease.

From comparison with the research on animals discussed above, single pits may be caused by trauma encountered during childhood or perinatally. This hypothesis is supported by the observation that they occur at a similar rate in adult and juvenile permanent teeth, reflecting the fact that these traumas were most likely non-fatal. However, single pits were more commonly found in the deciduous dentition than in the permanent dentition. This may be related to the different visibility of defects on the tooth surface, the smaller thickness of deciduous enamel and the shorter period of formation of deciduous enamel. For example, if a child experiences stress during the period of formation of the appositional enamel then any resulting regions with decreased enamel matrix deposition will not be visible on the enamel surface (21). Furthermore, the relationships observed here are unlikely to be universal, and given the very small sample size of deciduous teeth included in this study, examination of adult and juvenile remains from other archaeological populations is needed to clarify this relationship further.

The present study has also provided some evidence for patterning in the occurrence of the various types of hypoplastic defects within the mouth. This suggests that the type of hypoplastic defect that forms may be determined at least in part by the morphology of the affected tooth. This could be an artefact of the differing visibility of different types of defects on tooth surfaces with different shapes, with flat surfaces such as those of the anterior teeth displaying LEH, while due to the more complex, curvaceous surface of molars, defects on these teeth are more likely to appear as lines of pits. The tendency for single pit hypoplastic defects to be evenly spread throughout the dentition does not support the hypothesis that single pit hypoplastic defects are caused by trauma, as this is generally believed to be more commonly experienced by the anterior dentition. However, the small sample size of this study may be masking differences in the distribution of this type of enamel hypoplastic

defect. Contrary to the findings of previous researchers (e.g. 14), this study did not find a higher rate of hypoplastic defect occurrence on the anterior teeth overall, but instead observed a markedly higher rate of enamel hypoplastic defects on the canines and a lower rate on the first molars. It is possible that the different pattern seen here is due to the small sample size and the different methodology used in the present study, which would result in larger enamel hypoplastic defect counts than is observed in many previous studies. The analysis of larger populations, both archaeological and modern, with better preservation of the dentition is needed to clarify and confirm the distribution patterns reported here. This may help elucidate the aetiological factors determining any patterning observed.

Conclusions

The results of this pilot study suggest that there are differences in the distribution and prevalence of linear and pit hypoplastic defects within populations and within dentitions. Such patterning may reflect significant aetiological differences between the various forms of hypoplastic defect. While the data on males and females is inconclusive, the data on enamel hypoplastic defect occurrence in adults and juveniles has provided useful preliminary insights into enamel hypoplasia aetiology.

The occurrence of enamel hypoplastic defects appears to be higher in the permanent dentition than in the deciduous dentition. The types of enamel hypoplastic defect formed in the permanent and deciduous dentitions of those at Pella were also quite different. Deciduous teeth had a lower prevalence of LEH, and fewer linear pit arrays and non-linear pit arrays, but a higher prevalence of single pit defects to that observed in the permanent dentition. This may reflect the different aetiology of these groups of enamel hypoplastic defects, with studies on

animals suggesting that single pits are due to trauma episodes, while LEH and pit arrays are caused by systemic stresses such as malnutrition or disease. However, given the small sample size of this study, analysis of other populations will be necessary to investigate this possibility further. In addition, whether a systemic stressor causes a LEH, linear pit array or non-linear pit array appears to reflect the morphology of the affected tooth. These trends have not previously been detected in archaeological populations, and it would be interesting to see whether similar patterns are observed in other populations.

Although the results of this study show that the aetiology of enamel hypoplasia is indeed complex, the patterns identified may be of use in understanding the occurrence of enamel hypoplastic defects in past populations. Thus, by taking into account the types of enamel hypoplastic defect found in a population, it may be possible to provide a better interpretation of health in the past. Similar analyses of additional archaeological populations are needed to clarify the potential relationships observed in this study, and their impact on enamel hypoplasia aetiology.

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Text tables

Table 1. Total number of individuals from the population of Tomb 89 with each tooth type present

Tooth type	Total number
UM3	18
UM2	18
UM1	24
UP2	22
UP1	19
UC	21
UI2	21
UI1	22
LI1	10
LI2	11
LC	27
LP1	23
LP2	19
LM1	26
LM2	24
LM3	13
udm2	5
udm1	5
uc	3
lc	2
ldm1	3
ldm2	4

Table 2a. Percentage of individuals from Pella with enamel hypoplasia by sex

No. individuals with enamel hypoplasias		Male (n=10)	Female (n=16)	Fisher's Exact Test p value (one-tailed)
Any form of enamel hypoplasia		9	16	0.38
LEH		9	15	0.63
Pit enamel hypoplasias	All types	8	15	0.32
	Linear arrays	4	12	0.09
	Non-linear arrays	4	8	0.46
	Single pits	7	9	0.39

Table 2b. Percentage of teeth from Pella with enamel hypoplasia by sex

No. individuals with enamel hypoplasias		**Male (n=72)**	**Female (n=148)**	**χ^2 Test p value**
Any form of enamel hypoplasia		44	114	0.01
LEH		32	79	0.21
Pit enamel hypoplasias	All types	24	66	0.11
	Linear arrays	12	46	0.02
	Non-linear arrays	8	11	0.36
	Single pits	10	36	0.07

Table 3a. Percentage of individuals from Pella with enamel hypoplasia by age (P=permanent dentition only, P&D= both permanent and deciduous dentitions included)

No. individuals with enamel hypoplasias		**Adult (n=23)**	**Juvenile**		**Fisher's Exact Test p value (two-tailed)**	
		P	P (n=8)	P&D (n=11)	P	P&D
Any form of enamel hypoplasia		23	8	8	n/a	0.028
LEH		23	6	6	0.06	0.002
Pit enamel hypoplasias	All types	19	5	8	0.34	0.66
	Linear arrays	14	4	4	0.69	0.27
	Non-linear arrays	11	1	1	0.11	0.053
	Single pits	12	4	7	1.00	0.72

Table 3b. Percentage of teeth from Pella with enamel hypoplasia by age

No. individuals with enamel hypoplasias		**Adult (n=195)**	**Juvenile permanent (n=44)**	**Juvenile deciduous (n=11)**	**χ^2 Test p value (permanent teeth only)**
Any form of enamel hypoplasia		136	31	4	0.93
LEH		97	21	1	0.81
Pit enamel hypoplasias	All types	75	16	4	0.80
	Linear arrays	44	11	1	0.73
	Non-linear arrays	20	2	0	0.24
	Single pits	38	8	4	0.84

Table 4. Proportions of hypoplastic teeth with linear pit arrays, non-linear pit arrays, single pits and LEH on each tooth of the dentition.

Tooth type	**Total number of individuals with this tooth present**	**Total number of individuals with a hypoplastic tooth at this position**	**Linear pit enamel hypoplasia arrays**		**Non-linear pit enamel hypoplasia arrays**		**Single enamel pit hypoplasias**		**Linear enamel hypoplasia**	
			n	**%**	**n**	**%**	**n**	**%**	**n**	**%**
UM3	18	11	7	64	0	0	4	36	9	82
UM2	18	13	6	46	0	0	5	38	7	54
UM1	24	16	9	56	1	6	6	38	7	44
UP2	22	17	7	41	0	0	6	35	15	88
UP1	19	14	5	36	0	0	6	43	11	79
UC	21	18	10	56	5	28	4	22	14	78
UI2	21	14	1	7	2	14	3	21	10	71
UI1	22	11	4	36	2	18	4	36	10	91
LI1	10	0	0	0	0	0	0	0	0	0
LI2	11	0	0	0	0	0	0	0	0	0
LC	27	22	4	19	5	24	1	5	21	95
LP1	23	17	4	25	3	19	4	25	12	71
LP2	19	15	4	27	1	7	4	27	13	87
LM1	26	11	3	27	2	17	5	42	3	25
LM2	24	18	7	39	2	12	5	28	13	76
LM3	13	0	0	0	0	0	0	0	0	0
um2	3	3	0	0	0	0	3	100	1	0
um1	2	1	1	50	0	0	1	100	0	0
uc	2	0	0	0	0	0	0	0	0	0
lc	1	0	0	0	0	0	0	0	0	0
lm1	1	0	0	0	0	0	0	0	0	0
lm2	2	0	0	0	0	0	0	0	0	0

Figures

Fig. 1. The location of Pella within the Levant

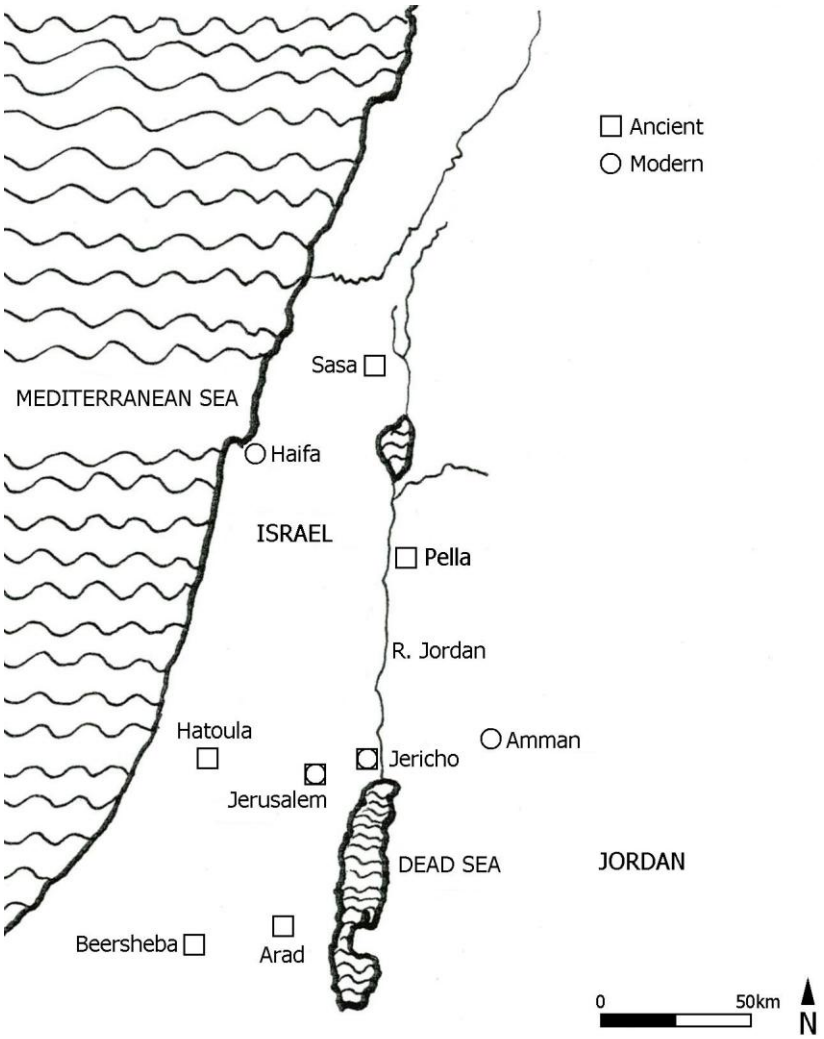

Fig. 2. Age distribution of the juveniles from Tomb 89