A paleopathological analysis of juveniles from Thebes:

Childhood health in Byzantine Greece

by

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A thesis
presented to the University of Waterloo
in fulfillment of the
thesis requirement for the degree of
Master of Arts
in
Public Issues Anthropology

Waterloo, Ontario, Canada, 2024

Author's declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Abstract

Limited publications address juvenile health in Byzantine Thebes, Greece. As vulnerable and abundant members of most populations, children are essential to understanding the health experiences of past populations. This thesis examines juvenile skeletal material recovered from a Byzantine period cemetery (5th-9th centuries CE) located on Ismenion Hill in Thebes, Greece. This site is located adjacent to the early Christian church of St. Luke the Evangelist. In antiquity, St. Luke was traditionally considered to be a physician. Previous analyses of the adult skeletal sample revealed a high prevalence of leprosy, cancer, and infection, leading researchers to hypothesize a relationship between the cemetery and the church of the physician saint. This thesis investigates this hypothesis with respect to the juveniles recovered at this site. Eleven graves were studied and a minimum of 59 juvenile individuals were identified. Perinates (*n*=15), infants (*n*=9), and young juveniles (*n*=25) make up the majority of the sample, followed by older juveniles (*n*=8) and adolescents (*n*=2). Overall, 16 (27%) juveniles present with dental and/or skeletal pathologies. Through the process of differential diagnosis, infection, metabolic conditions, and hematopoietic disorders were found to dominate the sample. To understand why children were brought to and buried at Ismenion Hill, cultural, social, and environmental factors shaping childhood health in late antiquity are addressed.

Key words: juvenile paleopathology, bioarchaeology, health, childhood, Byzantine, Greece

Acknowledgements

I would like to thank my research supervisor, Dr. Maria Liston, for her support and wisdom throughout my MA experience. Thank you for the opportunity to travel to Greece, learn from you, and for the opportunity to take part in such an exciting research project.

Thank you to my committee members, Dr. Maria Liston, Dr. Alexis Dolphin, and Dr. Bonnie Glencross for your time and words of encouragement. I would also like to thank Dr. Seçil Dağtaş, Dr. Adrienne Lo, Dr. Jennifer Liu, Miljana Kovacevic, Jennifer Doucet, and the faculty of the University of Waterloo Anthropology Department for your support throughout my MA, I am eternally grateful. To my cohort, Camila Font, Shannon Brandreth, Isabel Clasen, Wynne Manning, Robyn Wood, and Aparajita Bhattacharya, I am so proud of the research we completed.

Special thanks to the staff and researchers of American School of Classical Studies at Athens Malcolm H. Wiener Laboratory, and Dr. Jonathan Tomlinson and the Canadian Institute in Greece. Your kindness and support throughout my data collection period made my time in Athens truly enjoyable.

I am grateful to Dr. Kevin Daly and Dr. Stephanie Larson of Bucknell University, Dr. Alexandra Charami and Dr. Paraskevi Kalamara of the Greek Ministry of Culture and Sport, and the excavation teams that made the research of Ismenion Hill possible.

This thesis is supported by the Tri-Agency SSHRC Canadian Graduate Scholarship - Master's Award, the University of Waterloo Anthropology Department Iris Yuzdepski Memorial Graduate Award, and the University of Waterloo Graduate Studies and Postdoctoral Affairs President's Graduate Scholarship.

To my family and friends, thank you for your unwavering support and kindness throughout this research process.

Finally, I would like to thank the juveniles from Ismenion Hill, thank you for allowing me to share your story.

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List of abbreviations

AMTL	Antemortem tooth loss
ASCSA	American School of Classical Studies at Athens
BCE	Before common era
BIB	Bone-in-bone
c	Century
ca	Circa
CE	Common era
GMCS	Greek Ministry of Culture and Sport
HOA	Hypertrophic osteoarthropathy
ICH	Infantile cortical hyperostosis
LBW	Low birth weight
LEH	Linear enamel hypoplasia
MNI	Minimum number of individuals
Mo	Month(s)
SCA	Sickle cell anemia
Wk(s)	Week(s)
Yr(s)	Year(s)

Chapter one: Introduction to juvenile paleopathology and public issues anthropology

1.1 Juvenile bioarchaeology and paleopathology

Bioarchaeology is the study of skeletal remains in archaeological contexts to understand the lives of individuals in the past (Agarwal & Glencross, 2011; Lewis, 2009). Until recently, bioarchaeological research prioritized the study of adults, due to misconceptions of the interpretive value of studying children in the archaeological record. This misconception reflected institutional and academic biases that prioritized adult, androcentric analyses of the skeleton in the creation of methods and interpretation (Baxter, 2008; Halcrow & Tayles, 2008; Harrison, 2016; Lewis, 2009). Nevertheless, feminist movements in North America and Europe in the 1970's increased scholarly attention to gender-theory and disparities in power, representation, and identity between men and women in anthropological study. This encouraged an investigation of social roles and visibility for children, reflecting similar dichotomies in power and representation between men and women, and adults and children (Baxter, 2008). As a result, seminal works such as Lillehammer (1989), Sofaer Deverneski (1997), Kamp (2001), Perry (2006), Baxter (2008), and Lewis (2009) identified children as valuable topics of study in anthropology, advocating for their investigation with just as much rigour and dedication as adults. From this, the subspecialty of juvenile bioarchaeology emerged as a prominent and evolving interest for archaeologists, exploring how intersecting aspects of identity such as age, sex, and health impact childhood experience (Inglis & Halcrow, 2018; Lewis, 2009; Morrone, 2020; Sofaer Deverneski, 1997).

Recently, juvenile paleopathology, a sub-discipline concerned with skeletal evidence of illness and trauma in children, has also developed as a prominent field of interest in bioarchaeology (Lewis, 2009, 2017). However, numerous methodological and theoretical barriers limit the investigation of juvenile paleopathology. Child-specific methods, reference collections, and

osteological experts are limited, reflecting a long history of marginalization of juvenile-studies across the discipline (Lewis, 2009; Morrone, 2020). Regarding methodological limitations, differential burial practices that isolate juveniles from adults, poor preservation due to small size and the mineral composition of juvenile skeletal material, and limited experts in excavation contexts, historically have prevented comprehensive recovery and analysis of children in the past (Halcrow & Tayles, 2008; Perry, 2006). As such, not only does the identification of pathologies on the juvenile skeleton lack the extensive theoretical and applied attention given to adults, but the characteristics of juvenile skeletal material also make pathological investigations challenging. Growth throughout childhood allows bone to more readily heal and remodel, making the differentiation between pathological and growth-related new bone complicated. Additionally, acute or soft tissue illness or injury may not leave skeletal evidence, as pathologies take time to develop on bone. For young children, they may not have lived long enough for skeletal evidence of illness to appear, limiting interpretations of health experiences (Lewis, 2009, 2019). While these limitations complicate the assessment of juvenile health in the past, they present a need for the continued development of paleopathological methods, intersectional research, and historically, culturally, and socially relevant approaches that consider the complexities of childhood lived experience.

Identity refers to a series of characteristics that represent a population or individual such as age, sex, gender, or socioeconomic status (Baxter, 2008; Beauchesne & Agarwal, 2018; Roberts, 2011). Health and identity are intricately connected. Throughout the life-course, an individual's skeleton is influenced by environmental and cultural experiences, shaped by an individual's identity (Beauchesne & Agarwal, 2018; Inglis & Halcrow, 2018: Roberts, 2011). For bioarchaeologists, lived experience encompasses a wide range of social, cultural, and

environmental stressors that impact the integrity of the skeleton (Agarwal & Beauchesne, 2011; Beauchesne & Agarwal, 2018; Inglis & Halcrow, 2018). Bioarchaeologists utilize physiological indications of stressors on the skeleton to try to understand health and frailty in the past (Agarwal & Beauchesne, 2011; DeWitte & Stojanowski, 2015; Zedda *et al.,* 2021).

According to the World Health Organization (2023a) health is considered the "complete physical, mental and social well-being [of an individual] and not merely the absence of disease or infirmity." Frailty, on the other hand, refers to physiological stress experienced by an individual throughout their lifetime that promotes susceptibility to disease (DeWitte & Stojanowski, 2015; Zedda *et al.,* 2021). From a bioarchaeological perspective, observations of health and frailty are often inferred from the skeleton through the presence of lesions. Broadly, lesions refer to abnormal growth, destruction, shape, or size of bone (Ortner, 2012). However, the absence of skeletal lesions does not necessarily indicate good health (DeWitte & Stojanowski, 2015). Since illness-related lesions typically require living with a disease for an extended period, the presence of lesions suggests some resistance to a pathogen (Lewis, 2017). Conversely, the absence of lesions may indicate poor health as pathogens may have caused death rapidly (DeWitte & Stojanowski, 2015). Considering the complexities of juvenile skeletal analyses, a holistic approach to paleopathology that considers the environmental, social, and cultural contexts of children's lives in the past is necessary to attempt to understand health in the archaeological record (Beauchesne & Agarwal, 2018; DeWitte & Stojanowski, 2015; Lewis, 2009; Perry, 2006).

1.2 Public issues anthropology

In this thesis, a public issues approach to bioarchaeological study is considered. Public issues anthropology, defined as anthropological research that promotes and engages with critical discourse, publics, and contemporary concerns facing modern societies, is inherently concerned

with understanding health experiences (Armelagos, 2003; Borofsky & De Lauri, 2019; Stojanowski & Duncan, 2015). To that end, a public issues approach to bioarchaeology must engage with research questions that meet the interests of the modern public, while producing research that targets gaps in the field that may inhibit such investigative efforts (Borofsky & De Lauri, 2019; Stojanowski & Duncan, 2015). The public, broadly, refers to anyone who may engage with anthropological research or its outcomes (Borofsky & De Lauri, 2019; Scheper-Hughes, 2009). As such, this thesis engages with public issues bioarchaeology to explore how skeletal analyses of children's lives in the archaeological record can inform and mitigate challenges in assessing juvenile health experiences in the past and present (Borofsky & De Lauri, 2019; Lewis, 2009).

Children represent an abundant and vulnerable sector of most populations, and the health and death of children is a prominent public concern (Lewis, 2009; Lewis & Rutty, 2003). In their early years, children rely on the care of others to ensure proper nutrition, shelter, access to medical care, physical, and emotional support. This dependence makes children susceptible to infection and injury, reflecting trends of health, stress, and trauma experience in past and present populations (Lewis, 2009; Perry, 2006). As vulnerable individuals, children are subject to a variety of phenomena that may promote the formation of skeletal lesions, such as abuse, neglect, warfare, and labour (Lewis, 2009). Understanding skeletal evidence of disease or injury amongst cultural and environmental contexts of violence, care, or identity, allows researchers to begin to assess the complex nature of juvenile lived experience. Thus, presenting an opportunity to produce mitigative efforts, recommendations, and bringing attention to issues facing children today (Buzon, 2012; Lewis, 2009; Lewis & Rutty, 2003). To that end, the advancement of children's skeletal studies to understand health experience is inherently a public issue.

Forensic anthropologists engage with issues of childhood health and injury in a modern context, with the goal of identifying unknown individuals and reconstructing circumstances leading to death in matters pertaining to the law. Cases of child death often garner intense public response, concern, and calls for action and investigation, reinforcing children's skeletal studies as a prominent public issue (Lewis & Rutty, 2003). To aid in identification, forensic anthropologists establish a biological profile consisting of age, sex, ancestry, and stature on decomposed, poorly preserved, or skeletonized human remains (Lewis, 2009). Besides age, estimations of sex, ancestry, and stature, often cannot be completed in juvenile cases as the size and development of the juvenile skeleton prohibits the accuracy of these analyses (Lewis & Rutty, 2003). To that end, individuating characteristics, such as skeletal markers of pathology or injury, can be crucial to the identification of juvenile skeletal remains, necessitating the advancement of knowledge in the field (Cunha, 2006; Lewis, 2009).

Children found in forensic contexts often present skeletal evidence of frailty and stress (Steyn *et al.,* 2024). Evidence of poor dental hygiene, the distribution and prevalence of fractures, signs of repeated action (musculoskeletal stress markers), or malnutrition may allow forensic anthropologists to infer experiences of repeated stress, differential access to care, and possibly signs of abuse, neglect, or child labour (Lewis, 2009; Steyn *et al.,* 2024). To that end, this thesis explores juvenile skeletal and dental pathologies in an ancient Greek context, with the aim to contribute to the growing field of juvenile paleopathological studies in both forensic and bioarchaeological settings. Although the processes for which children find themselves in modern forensic contexts differ from the lives of children in ancient Greece, in most cases, the presentation of illness and injury on the skeleton remains the same, making the findings of this study applicable across temporal contexts (Lewis, 2009). Together, the study of childhood health in the past

(bioarchaeology) not only sheds light on an understudied and abundant portion of past societies, but it has the potential to directly support practitioners and researchers exploring childhood skeletal analyses in the present (forensic anthropology).

1.3 Proposed venue of publication

The proposed venue of publication for this thesis is *Hesperia* the journal of the American School of Classical Studies at Athens (ASCSA). *Hesperia* is a peer-reviewed journal published quarterly by ASCSA. *Hesperia* highlights ASCSA-affiliated research, on interdisciplinary topics relevant to the Greek world, aligning with the regional, temporal, and bioarchaeological approach of this thesis (ASCSA, 2023).

Chapter two: Paleopathological analysis of the juveniles at Ismenion Hill, Thebes, Greece

2.1 The site of Ismenion Hill

In 2011-2016, excavations at the archaeological site of Ismenion Hill in Thebes, Greece, revealed an early Byzantine cemetery. Excavations were completed under a permit by the Greek Archaeological Service of the Greek Ministry of Culture and Sport (GMCS) and the American School of Classical Studies at Athens (ASCSA), headed by A. Charami, P. Kalamara (GMCS), K. Daly and S. Larson (Bucknell University, ASCSA). The city of Thebes is located centrally in the region of Boeotia (see Figure 1) a fertile and densely populated region on the eastern mainland of Greece (Cartilage, 2020). Ismenion Hill, located outside of the ancient city walls, has revealed archaeological evidence dating as early as the Mycenean Bronze age (ca. 1600 BCE). Between 1906-1929, excavations led by A.D. Keramopoullous, revealed archaeological evidence of Mycenean chamber-tombs, followed by subsequent temples constructed between the 9th and 7th centuries BCE, supporting use as an extramural Sanctuary of Apollo Ismenios in the Greek Classical period (Cartledge, 2020; Larson, 2018). The recent ASCSA excavations identified continued use of Ismenion Hill into the Byzantine period with the presence of an early Byzantine and Christian period cemetery dated to the 5th-9th centuries CE (Arivantinos *et al.,* 2011; Charami *et al*., 2014). Dating of the cemetery was completed through ceramic analyses and ^{14}C radiocarbon dating of human skeletal material from one of the burials (Grave 4) by the Max Planck Institute for Evolutionary Anthropology in Germany (Bodling & Lindauer, 2022; Charami *et al*., 2012, 2013, 2014, 2015). Continued use of the site throughout the Byzantine period into the 13th century CE was identified though the archaeological evidence of *bothroi* disposal pits and the use of building materials from the ancient temples to expand the Theban city centre (Daly & Larson, 2017).

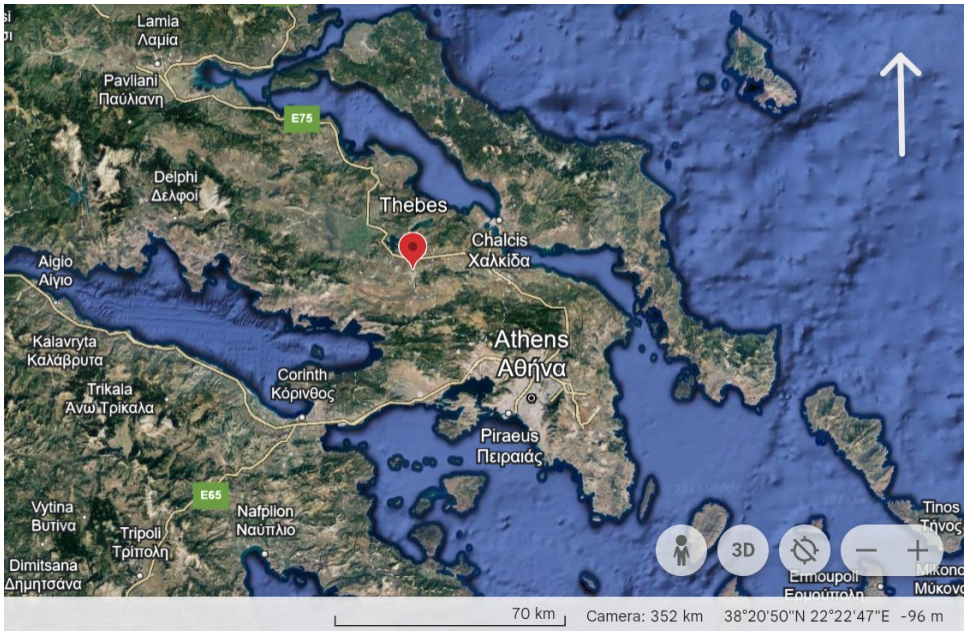


Figure 1: *Google Earth image of the eastern Greek mainland (2023).*

2.2 Previous hypotheses and current analyses

A total of 46 Byzantine period graves were excavated on Ismenion Hill, consisting of multiple burials, mass graves, and *bothroi.* Previous skeletal analyses by M. Liston identified a minimum of 184 individuals across the 46 graves. Although some of the grave cuttings turned out to have been excavated but unpublished earlier in the 20th century, all undisturbed individuals were buried in an east-west orientation, often indicative of Christian burial practices. Grave goods such as coins, bronze jewelry, and beads, were found in variable amounts across the graves, and their significance is still under investigation. Ceramics were consistently present and permitted ceramic dating for most of the graves (Charami *et al*., 2015).

Commingled multiple burials and disturbed graves were common at this site. The burial practice of partial exhumation, where individuals would be laid to rest and later the upper body and cranium were exhumed, resulted in multiple individuals that only presented with the lower

body and limbs (Liston, 2017). Exhumation and multiple burials are common in modern rural Greek Orthodoxy tradition, where individuals are exhumed 4-7 years after burial, and reinterred in an ossuary or repositioned within the same grave to allow for future inhumations (Danforth, 1982; Fox & Marklein, 2014; Liston, 2017). Similarly, it is reported in early Byzantine Christian practice that removal or repositioning of skeletal material was practiced to make space for future burials (Tritsaroli, 2017). Additionally, construction efforts within the Theban city-centre resulted in further disruption to the graves as stones and material were pulled from the site (Daly & Larson, 2017; Liston, 2017). Furthermore, the soil composition varied across the site but in general was silty with plant, stone, and artifact inclusions. Acidic root inclusions throughout the soil caused distinct circular erosions variably across the skeletal material (M. Liston, personal communication, June 7, 2023). Overall, the burial practices and postmortem environment resulted in highly fragmentary and commingled skeletal material.

Previous skeletal analyses revealed a high prevalence of pathologies across the adult sample (Liston 2017, 2018). Leprosy with a prevalence of 33.8% (338/1000), neoplastic diseases like metastatic cancer with a prevalence of 4.8% (48/1000), and bacterial infections such as brucellosis with a prevalence of 4.8% (48/1000), were identified (M. Liston, personal communication, January 2, 2024). Due to this, researchers have suggested a potential relationship between the cemetery and the nearby early Christian church of St. Luke the Evangelist, with foundations dated to the 5th century CE (see Figure 2) (Daly & Larson, 2017; Liston, 2017). Luke was identified as a physician in antiquity and Orthodox tradition believes that he was buried in this church in Thebes. This led researchers to hypothesize a relationship between the church of St. Luke and the cemetery as individuals may have been buried following pilgrimage to the church of the physician (Conlin, 1953; Daly & Larson, 2017; Livingstone, 2014; New American Standard

Bible, 1995, Colossians 4:14-16). The high prevalence of pathologies in the adult sample aligns with this hypothesis, and this research aims to investigate this hypothesis with respect to the juveniles from Ismenion Hill.

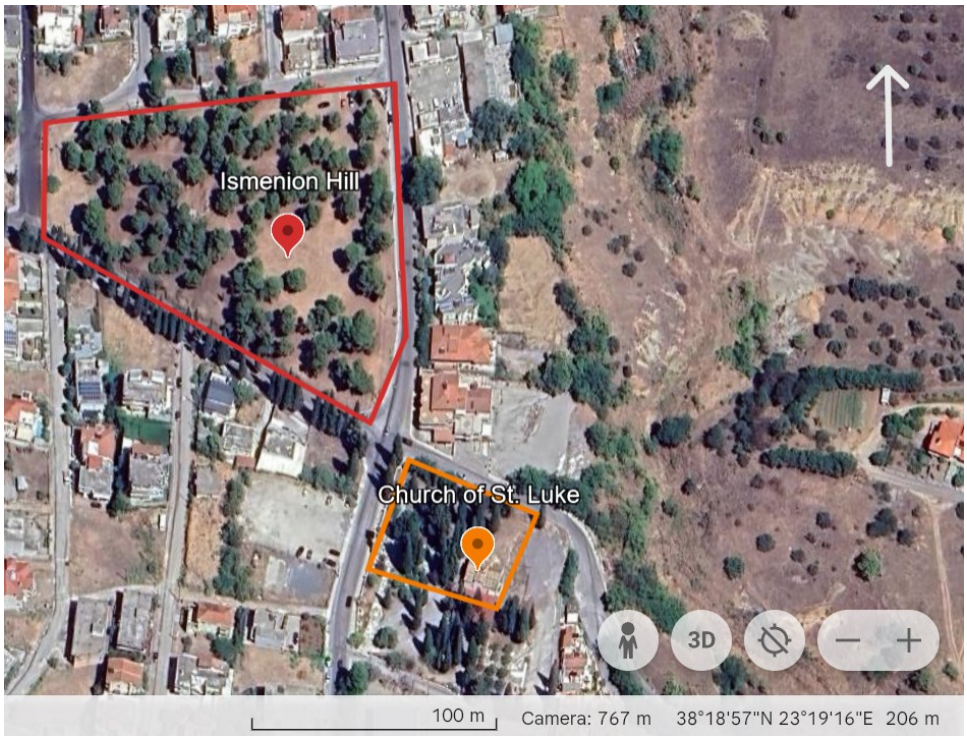


Figure 2: *Google Earth image of Ismenion Hill and the Church of St. Luke (2023).*

The objective of this thesis is to establish a demographic profile and to complete a paleopathological analysis to explore why children were brought to and buried at the site of Ismenion Hill. Eleven graves from the Ismenion Hill cemetery were examined in June and July of 2023. A *bothros* (Bothros #1), seven multiple burials (Graves 4, 5, 12, 25, 27, 38, 45), two mass graves (Graves 19, 20), and a burial just west of Ismenion Hill (Parking Lot Grave) were selected for analysis. Since the material recovered from Ismenion Hill is highly fragmentary, these graves were selected based upon their abundance of juvenile skeletal material determined by M. Liston, and their diverse locations across Ismenion Hill, allowing for a breadth of graves to be assessed within the data collection period. Bothros #1, and Graves 4, 5, 19, 20, 25, and 27 were located on

the east side of the temple, with Graves 4 and 5 encroaching the former Sanctuary foundations. Graves 38 and 45 were located on the south side of Ismenion Hill, outside the foundations of the temple. Grave 12 was located within the temple platform, in the south-west corner. Finally, the Parking Lot Grave was located north-west of Ismenion Hill, outside the main Sanctuary grounds next to the KTEL Bus Station. A map of the temple grounds, and the associated burials can be found in Appendix 1.

The 4th century CE marked the legalization of Christianity in late antiquity, and the widespread integration of Christian beliefs and institutions into communities across the Mediterranean. However, both Christianity and traditional Greco-Roman polytheistic religions were likely active during the use of the Byzantine period cemetery at Ismenion Hill (Miller, 2003; Vuolanto, 2020). The east-west orientation of the burials, and the proximity to the church of St. Luke, support a possible Christian affiliation of this cemetery (Bourbou, 2016; Daly & Larson, 2017). In other Christian cemeteries excavated in the Byzantine period, children are often found in high proportions compared to adults, and the presence of adults and children in the same graves is common, mirroring what was excavated at Ismenion Hill. This differs from traditional Greco-Roman practices where children are often buried separately from adults (Bourbou, 2016).

Regardless of Greco-Roman or Christian affiliation, religion and childhood health experiences were intricately linked in late antiquity. If a child was sick, prayer to the gods (Greco-Roman theology) or saints (Christianity) was common. Pilgrimage to temples of healing gods, like Asclepius, or to shrines of Christian saints, like St. Luke, were made seeking treatment or medical advice. Often, children would accompany their parents to temples or shrines (Dillon, 1997; Elsner & Rutherford, 2007). Pilgrims would be given blessings or objects like *ampullae* jugs filled with blessed water to protect their children from danger and illness (Marx, 1980; Vuolanto, 2020;

Ziegler, 2018). This reinforces the relationship between childhood health and religion for juveniles in late antiquity, supporting an investigation of the possible link between the church of St Luke and the graves at Ismenion Hill.

Methodology

2.3 Skeletal inventory

Juvenile skeletal material was identified based on overall size and morphology and was sorted by individual for each grave. Acknowledging the fragmentary condition of the sample, each individual was assessed for continuity between size, inventory (i.e., was there a correct number of skeletal elements from appropriate sides), and overall morphology. Then, repeated elements of the same side designation were individuated to ensure a minimum number of individuals (MNI) could be achieved. Considering the commingled nature of this sample, age estimation was also taken into consideration when establishing a MNI. Adjustments were made to group elements of similar ages, if there were no repeating elements contradicting their grouping. Written inventory forms by Buikstra & Ubelaker (1994) and visual inventory forms by Roksandic (2003) were used to score the presence or absence of elements and written notes detailed completeness. A final MNI of 59 juveniles was established across the eleven graves.

2.4 Sample demographics

This project identified juveniles as individuals yet to complete their skeletal growth. Typically, skeletal growth ceases around 18-20 years of chronological age (Lewis, 2009). Age-at-death was estimated on a case-by-case basis due to the fragmentary nature of the skeletal material. Standard osteological methods as outlined in Schaefer, Black, and Scheuer (2009) and

Cunningham and colleagues (2016) were utilized. Dental age was evaluated through the eruption and formation of deciduous and permanent dentition as outlined by AlQahtani, Hector, and Liversidge (2010). In cases where limited skeletal remains were complete, and dentition was unavailable, overall size and morphology of the skeletal material was compared to the ASCSA natural bone and cast reference skeletal material to provide an approximate age-at-death range. For a complete list of methods used, see Appendix 2.

Defining age categories in the past is challenging, as researcher-assigned age categories and social realities for past populations often do not align (Baxter, 2008; Perry, 2006). Bioarchaeological categories of age are determined through physiological stages of growth and development (Lewis, 2009; Roksandic & Armstrong, 2011). However, these categories reflect modern westernized ideologies of age and childhood identity, often failing to capture meaningful social understandings of age relevant to the population being studied (Lewis, 2009; Perry, 2006, Roksandic & Armstrong, 2011). For this reason, researchers argue for the use of socially relevant age categories to allow for more holistic interpretations of age-related experiences such as health and wellbeing in the past (Inglis & Halcrow, 2018; Kamp, 2001; Perry, 2006).

Documentary evidence from the Byzantine period recognized childhood in three stages: *infantia, pureitia,* and *adulescentes* (Nathan, 2020). *Infantia* or infancy was the title assigned to young children less than 7 years of age (Nathan, 2020; Vuolanto, 2020). Following this, juveniles would enter a period of childhood called *pureitia,* until reaching puberty around age 12 (girls) or 14 (boys) (Nathan, 2020; Tritsaroli & Valentin, 2008). Finally, *adulescentes* followed puberty. Adolescents were considered old enough to marry, bear children, and take on professions, but adulthood was not legally recognized until age 25 (Nathan, 2020; Tritsaroli & Valentin, 2008; Vuolanto, 2020). Reflecting these age categories, and integrating bioarchaeological categories of

growth and development, five age categories were selected for this thesis and are presented in Table 1 (Lewis, 2009; Nathan, 2020; Tritsaroli & Valentin, 2008).

Table 1: *Age-at-death categories used in this thesis.*

Byzantine period reference	Age category	Age-at-death
Infantia	Perinate	Less than 40 gestational weeks
	Infant	40 gestational weeks – 1 year
	Younger juvenile	1 – 7 years
Pureitia	Older juvenile	7 – 12 years
Adulescentes	Adolescent	12 – 20 years

Biological sex was not estimated in this thesis. Sex estimation of the juvenile skeleton is challenging, as the juvenile skeleton lacks evidence of sexual dimorphism in the pelvis or cranium (the elements most useful for accurate sex estimation) before puberty (Bourbou, 2020). These elements were not preserved amongst the adolescents and older juveniles of this study.

2.5 Paleopathological analysis

Paleopathological analysis of the juveniles at Ismenion Hill was completed through a series of macroscopic, microscopic, and radiographic observations. No destructive bone sampling was completed due to restrictions imposed by Greek law. Skeletal or dental pathologies were identified through visual analysis of skeletal elements and written notes detailed extent, location, and morphology of the suspected pathology (Ortner, 2012). Then, impacted skeletal elements were documented through photography and illustration. When appropriate, observations were enhanced using a digital microscope.[1] Additionally, some radiographs were completed, and previous radiographs of the bones were referenced. To investigate possible pathologies, differential

[1] DinoCapture 2.0 microscope software (Retrieved from: https://www.dino-lite.eu/en/software/general-software/dinocapture-windows)

diagnosis (DD) tables were used. DD tables allow researchers to systematically compare observations of skeletal lesions against expected symptoms of the various illnesses and injuries recorded in modern clinical literature, paleopathological texts, and anthropological references (Lewis, 2017; Ortner, 2003). DD is not exclusive to bioarchaeology, and is used in modern medical contexts, forensics, and other anthropological disciplines to systematically rule in or out possible causes of skeletal lesions and abnormalities (Klaus, 2017; Ortner, 2012). Differential diagnosis tables used in this thesis can be found in Appendices 3-5.

Results

2.6 Age-at-death

Younger juveniles made up 42% (*n*=25) of the skeletal sample, followed by perinates 25% (*n*=15), infants 15% (*n*=9), older juveniles 14% (*n*=8), and adolescents 3% (*n*=2). Results of the age-at-death estimations can be seen in Figure 3 and Appendix 2. In general, the age distribution of this sample reflects similar trends observed in late Roman and Byzantine period cemeteries throughout the Greco-Roman world (Bourbou, 2016; Lewis, 2010; Tritsaroli & Valentin, 2008). Lewis (2010) reports a late Roman juvenile cemetery assemblage (*n*=331 individuals with age-at-death estimated) where perinates (16.6%), infants <1 year (26.2%), and young juveniles <6.5 years (30.5%) also dominated the sample. Of a total 174 juvenile individuals studied by Bourbou (2016) across six 7th-12th centuries CE Byzantine period cemeteries on the island of Crete, perinates and infants less than 1 year made up 21.2% of the sample, while children 1-14.6 years, and adolescents 14.6-17 years, made up 56.8%, and 13.7% of the sample, respectively. Tritsaroli and Valentin (2008) report similar mortality patterns in two other Christian cemeteries from Thebes (*n*=10) and Xironomi (*n*=24) in middle-late Byzantine Boeotia (10th-14th c. CE), where young juveniles were the most frequently occurring age group. Figure 4 illustrates a clustering of individuals in the

young juvenile age-at-death category in the Ismenion Hill, Xironomi, and Thebes skeletal samples (Tritsaroli & Valentin, 2008).

While limited juvenile age-at-death data is published in an early Byzantine Boeotian context, a comparison of the sites studied by Tritsaroli and Valentin (2008) to the sites studied by Lewis (2010) and Bourbou (2016) in the late Roman and Byzantine period, allow for general conclusions to be drawn. In a typical archaeological sample, the highest percentage of juvenile deaths are expected in the perinate and infant age categories (Bourbou, 2020; Gowland & Chamberlain 2002; Lewis, 2009). This does not appear to be the case for Ismenion Hill, or the comparative sites from the Byzantine period (Bourbou, 2016; Tritsaroli & Valentin, 2008). Instead, the majority of deaths tend to cluster in the post-infancy and younger juvenile age categories, decreasing as juveniles enter older childhood and adolescence. This indicates that the post-infancy period was likely extremely risky for juveniles in the Byzantine period, presumably due to cultural, social, and environmental stressors (Bourbou, 2020).

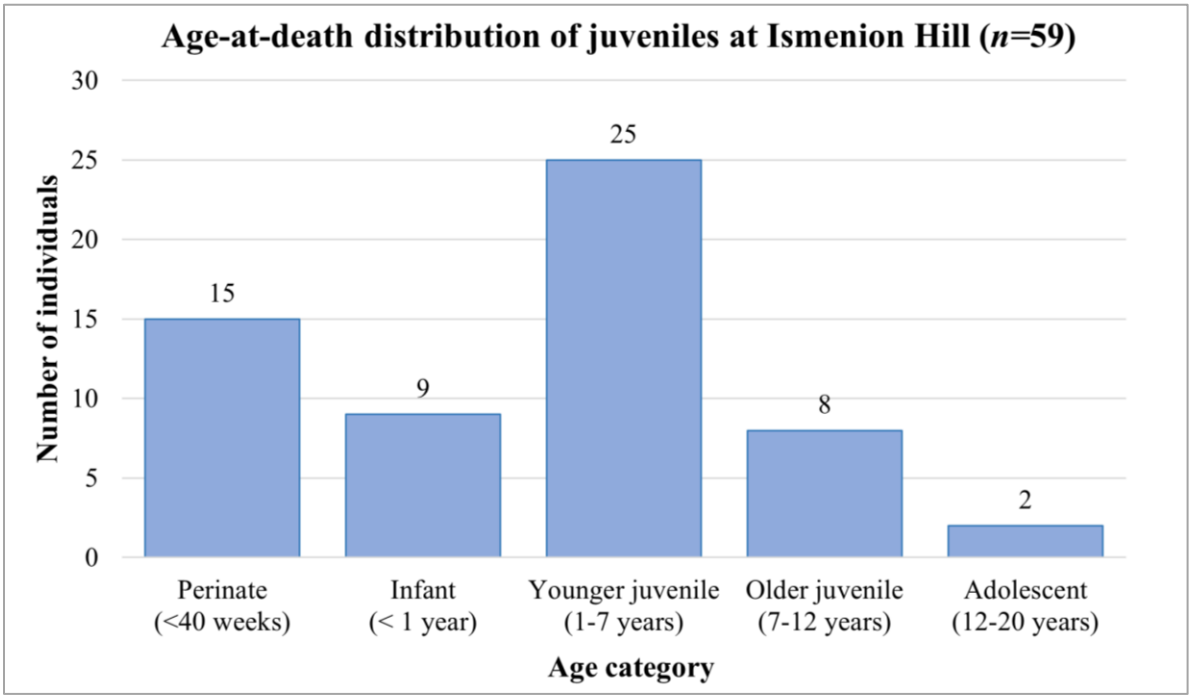


Figure 3: *Age-at-death distribution of juveniles at Ismenion Hill.*

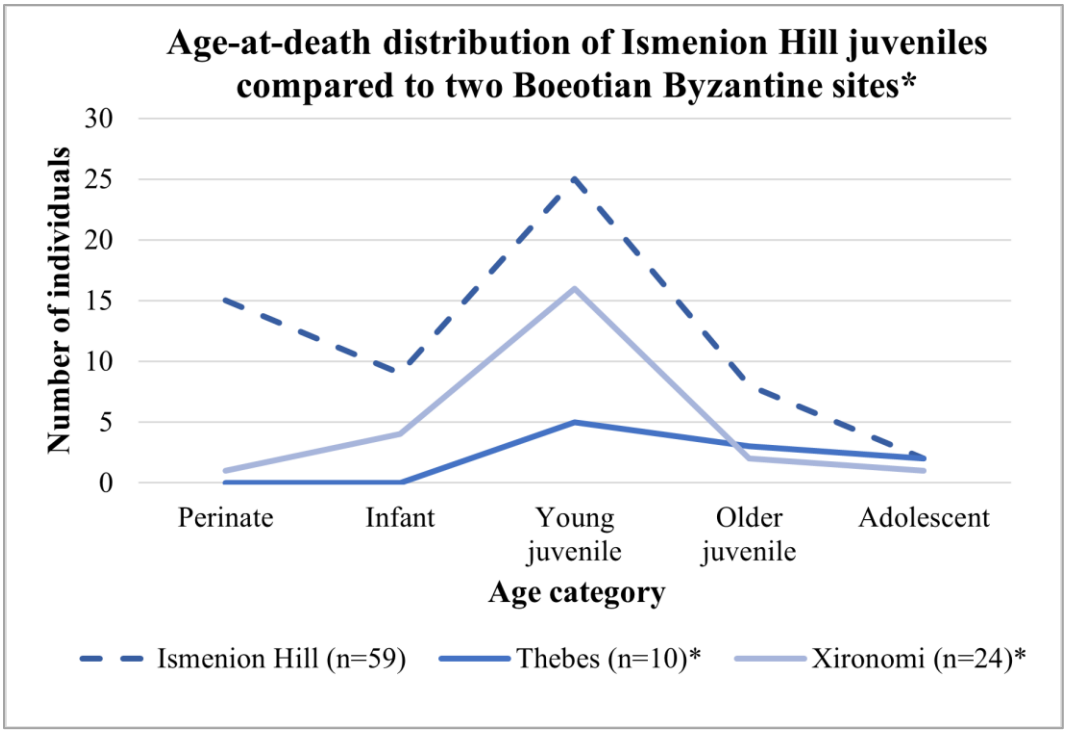


Figure 4: *Age-at-death distribution of the juveniles from Ismenion Hill (n=59), compared to two Byzantine period Boeotian cemetery sites (10th-14th c. CE) studied by *Tritsaroli and Valentin (2008) at Thebes (n=10) and Xironomi (n=24).*

2.7 Pathological overview

A total of 16 individuals (27%) presented with skeletal and/or dental evidence of pathology. Following pathological categories set out by Lewis (2017), skeletal observations fell into three main groups: metabolic and hematopoietic disorders (*n*=5, 31%), specific and non-specific infections (*n*=8, 50%), and dental pathologies (*n*=4, 25%). Four case examples of skeletal pathologies have been highlighted in the body of this thesis: Skeleton 1 from Grave 4 with a case of possible childhood leukemia, Skeleton 15 a perinate from Grave 19 with non-specific meningitis, and Skeletons 27 and 37 from Graves 25 and 38 respectively, an infant and perinate with similar presentations of infantile osteomyelitis. DD of the case studies can be found in Appendix 3, suspected metabolic and hematopoietic disorders are discussed in Appendix 4, DD of non-specific and specific infections are in Appendix 5, and Appendix 6 presents dental

pathologies. Table 2 identifies individuals by likely pathology type, and the corresponding Appendix for which DD analyses can be found.

Table 2: *Summary table of juveniles with pathologies by grave, skeletal number, age-at-death, pathology type, and reference Appendix, (n=16).*

Grave	Skeletal #	Age-at-death	Pathology type	Appendix #
4	1	Dental: 9.5-13.5 ± 0.5 yrs. Skeletal: 5-7 yrs.	Hematopoietic/ dental	3
5	5	Dental: 11.5 ± 0.5 yrs. Skeletal: 11-16 yrs.	Infection (specific)	5
5	6	Dental: 1.5 ± 3 mo. Skeletal: <1 yr.	Metabolic/ hematopoietic	4
19	14	Dental: 2.5-3.5 ± 0.5 yrs. Skeletal: 1-2 yrs.	Metabolic	4
19	15	Dental: 34 wks. - 1.5 ± 3 mo. Skeletal: 36-40 wks.	Infection (non-specific)	3
20	17	Dental 5.5-7.5 ± 0.5 yrs. Skeletal: 4-7 yrs.	Dental	6
20	20	Skeletal: 34 wks. – 6 mo.	Metabolic	4
25	27	Skeletal: 40 wks. - 2 yrs.	Infection (non-specific)	3
27	29	Skeletal: ~5 yrs.	Infection (non-specific)	5
38	36	Dental: 1.5-2.5 ± 0.5 yrs. Skeletal: < 2 yrs.	Infection (non-specific)	5
38	37	Skeletal: 24-32 wks.	Infection (non-specific)	3
45	38	Dental: >10.5 ± 0.5 yrs. Skeletal: 16-20 yrs.	Dental	6
45	41	Dental: 5.5-6.5 ± 0.5 yrs.	Infection (non-specific)	5
45	42	Dental: 38 wks-1.5 mo. ± 3 mo. Skeletal: 30-40 wks.	Metabolic	4
Bothros #1	45	Skeletal: ~5 yrs.	Infection (non-specific)	5
Parking Lot Grave	54	Dental: 10.5-11.5 ± 0.5 yrs. Skeletal: 10-12 yrs.	Dental	6

According to Ortner (2003), it is expected that in a given skeletal sample, approximately 15% of individuals will show some evidence of skeletal pathology. Typically, this involves three

main skeletal changes including infection, trauma, and age-related changes (i.e., arthritis) of the bones (Ortner, 2003). In this sample there is no evidence of skeletal trauma and age-related changes are not apparent (expected in an assessment of only juveniles) (Lewis, 2009). Considering that this is a subset of the juvenile, and overall, skeletal assemblage at Ismenion Hill, the observation of skeletal and/or dental pathologies in 27% of the juveniles studied is high (Ortner, 2003). Looking at the age distribution of the sample, three perinates (19%), three infants (19%), five younger juveniles (31%), four older juveniles (25%), and one adolescent (6%) presented with evidence of pathology. Juvenile skeletal and/or dental pathologies were observed in every grave, except for Grave 12.

Possible metabolic and hematopoietic conditions included two cases of scurvy, one of which likely co-occurred with anemia, and two possible cases of perinatal and infantile metabolic disorders due to maternal health. Also, one case of childhood leukemia was identified. Skeletal lesions associated with metabolic and hematopoietic disorders are useful indicators of health and frailty in a population, as they often reflect trends in food insecurity, environmental stress, and maternal health (Brickley & Ives, 2008; Hillson, 2019; Zedda *et al.,* 2021). In addition, non-specific infections, including periostitis (n=3), meningitis (n=1), and osteomyelitis (n=2), and one specific infection of juvenile leprosy was observed. Prevalence of infection is also a useful indication of the general health status of a population. Infections are often a product of unsanitary living conditions or overcrowding, and in juveniles can increase susceptibility to other morbidities (Lewis, 2017; Stathakopoulos, 2004; Zedda *et al.,* 2021). Furthermore, dental calculus (n=3), one instance of linear enamel hypoplasia (LEH), and one case of antemortem tooth loss (AMTL) was recorded. Conditions such as AMTL and dental calculus can be precursors to, or associated with, periodontal disease, which has been linked to increased mortality in archaeological populations

(Prowse, 2011; Zedda *et al.,* 2021). Moreover, LEH is associated with increased morbidity and mortality in older childhood and young adulthood (Keenleyside, 2008). To further explore the pathologies observed in this sample, a discussion of factors influencing childhood morbidity and mortality in Byzantine Thebes is provided.

Discussion

2.8 Factors impacting juvenile morbidity and mortality in the Byzantine period

Perinatal mortality

Endogenous and exogenous factors readily contributed to perinatal mortality in antiquity. Endogenous factors refer to maternal or genetic phenomena that cause death for fetuses (<28 gestational wks.) and perinates (perinate I: 28-37 gestational wks., perinate II: 37-42 gestational wks.) (Lewis, 2009; Morrone *et al.,* 2021). Typically, these factors include prematurity, low birth weight, congenital conditions, and trauma during the birth process (Lewis, 2009). A normal pregnancy is considered to last approximately 40 gestational weeks, and anything less than 37 gestational weeks (about 8.5 mo. of pregnancy) is considered premature (Lewis, 2009; Morrone *et al*., 2021; World Health Organization 2023b). It is generally accepted by bioarchaeologists that birth occurring less than 28 gestational weeks in antiquity was likely non-viable and premature birth between 28-37 weeks posed a significant risk to perinates (Lewis 2009; Liston *et al.,* 2018; Morrone *et al*., 2021).

At this site, eight individuals were estimated to be <37 gestational weeks (perinate I), and six were estimated to be >37 gestational weeks (perinate II) (see Figure 5). No skeletal material belonging to a fetus <28 gestational weeks was identified. Regression models charting the expected age-at-death distribution of natural perinatal mortality have been proposed by Gowland and Chamberlain (2002) for the Roman period and Butler and Alberman (1969) for the modern

era. These models suggest a clustering of perinate deaths around 38-40 gestational weeks, reflecting the challenges of the birthing process (Butler & Alberman, 1969; Gowland & Chamberlain, 2002). Typically, perinate deaths steadily increase from the fetal period onwards, as the chances for complications related to giving birth increase (Butler & Alberman, 1969; Gowland & Chamberlain, 2002; Liston *et al*., 2018). The perinates at Ismenion hill present a higher count of perinate I individuals than perinate II individuals, which does not reflect typical mortality models.

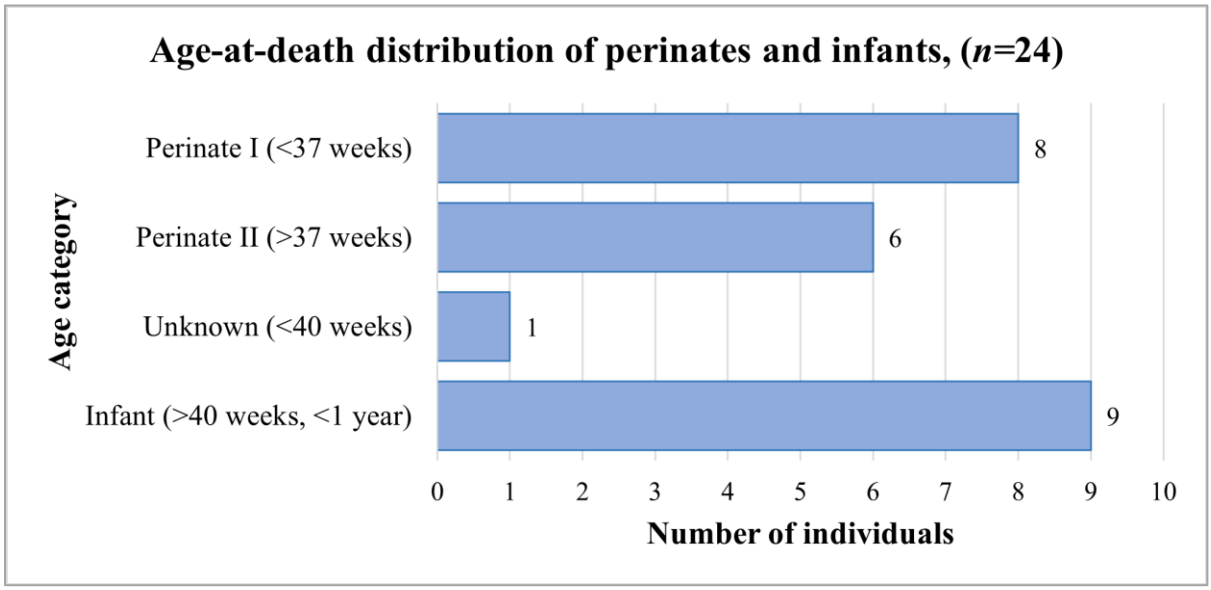


Figure 5: *Age-at-death distribution of perinates and infants at Ismenion Hill, age categories adapted from Lewis (2009) and Morrone and colleagues (2021).*

Limited literature discusses fetal and perinate mortality trends from early Byzantine period sites. However, a sample of 43 perinates and fetuses studied by Morrone and colleagues (2021) from middle-late Byzantine period Estonia (13^{th}-15^{th} c. CE) identified perinate I deaths to be slightly lower (44.2%) than perinate II deaths (46.5%). In addition, a middle Byzantine cemetery in Turkey (10^{th}-11^{th} c. CE) found fetal and perinate I deaths to be significantly lower (1.4%) than perinate II deaths (28%), in a sample of 141 juveniles (Demirel, 2023). While there

are multiple factors limiting the excavation and recovery of fetuses and perinates that could account for differences in this sample and others, the higher count of premature perinates at Ismenion Hill necessitates a closer look at factors influencing perinatal mortality in the Byzantine period (Lewis, 2009).

Preterm perinates have issues with latching, swallowing, feeding, and thermoregulating (Baumgart, 2008; Liston *et al.,* 2018; Mance, 2008). Their underdeveloped immune systems make them prone to respiratory distress syndrome, infection, hemorrhage, sepsis, intracranial bleeding, and meningitis (Halcrow *et al.*, 2018; Liston *et al.,* 2018; World Health Organization, 2023b). A systematic review of juvenile health by Perin and colleagues (2019) in the 21st century found that prematurity was not only extremely common but was the leading cause of death for children under 5 years of age globally. Infection (49.2%), birth complications (17.7%), respiratory infections (13.9%), intrapartum events such as preeclampsia or placental abruption (11.6%), and diarrhea (9.1%) were reported to be the main causes of death amongst premature newborns, highlighting the role of maternal health in perinatal mortality (Perin *et al.,* 2019). Similar concerns were raised by scholars in Greek antiquity, like Hippocrates (ca. 5th-4th c. BCE), where factors of maternal health and risky pregnancies were reported to increase the chance of prematurity and death for perinates (Malamitsi-Puchner, 2017). However, the risk associated with premature birth in late antiquity was likely significantly higher than that of the 21st century due to the lack of modern medical interventions, reinforcing the challenges of pregnancy for mothers and newborns in the Byzantine period (Bourbou, 2016; Liston *et al*., 2018; Malamitsi-Puchner, 2017).

It is important to note that young gestational age-at-death estimates do not always reflect prematurity, and instead could reflect general trends in population stress resulting in low-birth weight (LBW). LBW can be a result of multiple factors, but repeated LBW in a sample (identified

by multiple perinates measured to be young in gestational age), has been reported to be a result of shifts in cultural, environmental, or subsistence practices that make adequate maternal nutrition and health challenging (Blake, 2018). While the specific cause of mortality may not be apparent from the perinatal skeleton alone, it is likely that maternal health, the risks associated with prematurity, and LBW contributed to the deaths of the perinates at this site (Halcrow *et al*., 2018; Liston *et al.*, 2018).

As the perinates and infants at this site were too young to arrive to Ismenion Hill on their own, it is likely that mothers or families experiencing pregnancy-related complications or challenges caring for sickly premature infants took on pilgrimage to the church of St. Luke (Dillon 1997; Elsner & Rutherford, 1997). For Christian practicing families, baptism was seen as crucial to protecting the health and wellbeing of children. In late antiquity, baptism would take place on the 40th day of life, or as early as the 8th day if the child was sickly and would not likely survive until that time. If the child was generally healthy, baptism could often be put off until later childhood or even adulthood (Vuolanto, 2020). Documentary sources from the Byzantine period suggest that it was well known that prematurity was inherently risky and almost certainly resulted in death. As such, individuals may have specifically visited the church of St. Luke to allow for the rites of baptism to occur, acknowledging the likely impending death of their premature newborns (Bourbou, 2016; Dillon, 1997; Malamitsi-Puchner, 2017). While the practice of baptism shortly after birth for sickly perinates is known to occur in this period and was likely practiced by the Christian people of Thebes, the lack of documentary or archaeological evidence supporting the specific bringing of perinates to the church of St. Luke for baptism and subsequent burial at Ismenion Hill means that this interpretation requires further investigation.

Infant and juvenile mortality

If a perinate reaches full term and begins to enter infancy, mortality after birth is typically attributed to exogenous environmental or cultural factors such as diet and weaning practices, infection, inadequate sanitation, living conditions, or trauma (Bourbou, 2016; Lewis, 2009). These environmental factors are exacerbated as children age, as they enter increasingly risky environments and adopt more social responsibilities, typically through occupation or warfare (Lewis, 2009). Natural disasters, such as the 551 CE earthquake in Boeotia, disease outbreaks, like the ca. 541-747 CE Justinianic Plague that afflicted the Byzantine empire, and conflict from neighboring populations such as the Slavic invasions of Boeotia that began in the late 6th century CE are also factors that would have likely impacted the health status of juveniles and the general population of Boeotia in antiquity (Koder, 2020; Sarris, 2002; Stathakopoulos, 2004; Tritsaroli, 2017).

Subsistence crises were common in the Byzantine period. As a primarily agricultural society, natural disasters, weather, and climatic challenges such as drought, excessive rainfall, flooding, and vermin or pests could have been detrimental to crop production and maintenance, potentially resulting in food crises or famine (Brickley & Ives, 2008; Stathakopoulos, 2004; Tritsaroli, 2017). Moreover, reported warfare, conflict, and socioeconomic strife in the Byzantine period, support an increasing state of food insecurity in Boeotia (Stathakopoulos, 2004; Tritsaroli, 2017). In a similar vein, epidemics and disease outbreaks were common in late antiquity, as overcrowding of rural and urban centers in Boeotia and low public and personal sanitation standards posed a significant health risk to the public (Tritsaroli, 2017). Disease is often linked to famine, resulting in the likely presentation of both infectious and malnutrition-related skeletal changes (Roberts & Brickley, 2019). The potential impact of these factors on the juvenile skeletons

studied at Ismenion Hill can be divided into two main pathological categories, metabolic and hematopoietic disorders, and infection.

Metabolic disorders refer to conditions where the body does not obtain specific nutrients required to maintain skeletal or soft tissue health. This can result in malnutrition, frailty, and increase susceptibility to infection. Metabolic disorders are often identified by marrow hyperplasia (increased vascularity or porosity), osteopenia (loss of bone mass), and/or the formation of abnormal periosteal new bone growth (in response to hemorrhage) (Bourbou, 2020; Lewis, 2017). The most common metabolic disorders impacting children are scurvy, vitamin C deficiency (often caused by a lack of raw fruits or vegetables), and rickets, vitamin D deficiency (resulting from insufficient exposure to sunlight) (Brickley & Ives, 2008; Lewis, 2017). These conditions may result from cultural practices that dictate the appropriate foods for juveniles or require children to remain indoors (Brickley & Ives, 2008). The co-occurrence of metabolic deficiencies and hematopoietic blood-borne disorders is common (Lewis, 2017). Hematopoietic disorders, like iron deficiency anemia, vitamin B12 deficiency, sickle cell anemia, and leukemia occur when irregularities in the morphology or production of blood cells impact skeletal integrity, often resulting in marrow hyperplasia (Bourbou, 2020; Lewis, 2017; Walker *et al.,* 2009).

Considering the age-at-death distribution of this site, and the prevalence of metabolic and hematopoietic disorders in this sample, the impact of weaning on nutrition and skeletal health must be considered. It was a common practice in Greek late antiquity to provide newborn infants with *hydromel,* a mixture of water or goat's milk and honey for the first weeks of their lives (Bourbou *et al*., 2013). This was preferred by ancient Greek physicians over the colostrum, the initial vitamin and antibody rich milk produced by mothers, as it was perceived to be unhealthy. Ingesting potentially contaminated milk, water, or honey increases the risk of infection for infants and

newborns. For example, botulism, commonly caused by the fungal toxin *Clostridium botulinum* is found in honey and can result in difficulty eating, reduction in muscle tone, respiratory distress, and paralysis (Bourbou *et al*., 2013; Liston *et al*., 2018). Fungal and bacterial toxins transmitted via food have high mortality rates and are especially harmful to infants and newborns (Bourbou *et al*., 2013; Liston *et al*., 2018; Stathakopoulos, 2004).

In antiquity, children were often breastfed until 2-4 years of age, and solid foods began to be introduced around 6 months. Weaning typically involved the introduction of soft bread and *hydromel*, sheep or goat's milk, porridge, grains, and soft eggs, supplemented by breastmilk (Bourbou *et al*., 2013). These foods alone are not significantly rich in vitamins or nutrients, and the introduction of solid foods presented an opportunity for infection as children shifted away from the antibodies and nutrients provided to them by their mother's breastmilk (Bourbou *et al*., 2013; Brickley & Ives, 2008). In addition, any disruptions to this process such as maternal death or forced weaning would have increased possibility of infection, malnutrition, and ultimately mortality for these juveniles (Bourbou, 2016; Lewis, 2009). Grave 5, Skeleton 6, an infant less than 1 year of age with likely scurvy and anemia, and Grave 19, Skeleton 14, a young juvenile ~1-3 years of age with evidence of scurvy, present skeletal lesions consistent with inadequate nutrition and vitamin deficiency (see Appendix 4). The young age-at-death of these juveniles corresponds with the weaning period, supporting that the skeletal lesions observed could be a result of challenges acquiring adequate nutrition throughout the weaning process (Lewis, 2009, 2017).

Following weaning, the Byzantine period diet for older juveniles and adults consisted primarily of grains (wheat, barley, millet), olives and olive oil, and wine, with various legumes, lentils, fruits, vegetables, nuts, dairy, animal (goat, sheep, pig, cattle, wild birds) and marine proteins (anchovies, sardines) (Bourbou *et al.,* 2011). Regardless of socioeconomic class, olive oil,

olives, grains, lentils and chickpeas would have been staples for the Byzantine individual (Bourbou & Garvie-Lok, 2015). The Greek climate allowed for an abundance of fruits and vegetables to be available in antiquity, likely reducing the prevalence of vitamin deficiencies for those who could afford them (Bourbou *et al*., 2011). However, external factors like famine, epidemic disease, and natural disasters would have made adequate nutrition challenging.

As an additional consideration, in early Christianity protein consumption was reduced and dependent on perceived sex and social class. In general, meat was expensive and most accessible to those of the upper class. Moreover, women and girls were often told to restrict their protein intake. This was due to an association between fasting and sexual abstinence in Christian thought, where women and girls were expected to isolate from general society and eat very little to maintain the appearance of religious discipline and devotion (Bourbou & Garvie-Lok, 2015). While sex was not estimated in this sample and knowledge of socioeconomic class is unknown at this time, low intake in protein has been reported to have links with generalized osteopenia and bone loss, potentially impacting both maternal and juvenile health in this period (Brickley & Ives, 2008). In this sample, it is likely that maternal health played a role in the skeletal lesions observed in the infant and perinate from Grave 20, Skeleton 20 and Grave 45, Skeleton 42. Their young age-at-death suggests that a systemic metabolic condition was likely passed on from their mothers due to inadequate nutrition or vitamin deficiency throughout gestation (see Appendix 4) (Morrone *et al.,* 2021).

Sex, social class, and access to food products are important to consider when addressing the evidence of dental calculus and linear enamel hypoplasia at this site (see Appendix 6). Dental calculus is mineralized plaque formed by the build-up of bacteria on the dentition. It is not an indication of specific illness, but rather a general indicator of dental hygiene (Hillson, 2019). Three

juveniles presented with dental calculus (Grave 20, Skeleton 17, Grave 45, Skeleton 38, and Parking Lot Grave, Skeleton 54). If not managed, dental plaque can result in more serious pathologies like caries (cavities), periodontal disease, or antemortem tooth loss (Hillson, 2019; Prowse, 2011; Zedda *et al.,* 2021). Often the presence of calculus, without caries (resembling the individuals in this sample) indicate high protein, low carbohydrate diets (Keenleyside, 2008). This could suggest that the individuals with dental calculus had access to high protein foods through their social status (Bourbou *et al*., 2011; Keenleyside, 2008; Prowse, 2011). Linear enamel hypoplasia (LEH) was observed on the permanent incisors and canines of Skeleton 54, from the Parking Lot Grave. LEH presents as a disruption in the growth of the tooth enamel, causing a furrow in the typically smooth crown of the permanent dentition (Towle & Irish, 2020). The enamel of permanent incisors and canines are formed between years 1-7 in a juvenile's lifetime, suggesting a stressor within this period likely affected this juvenile (Hillson, 2019). There are many potential causes of LEH, including genetic disorders, deficiencies in vitamins A, C, or D, weaning practices, and illness (Pitsios & Zafiri, 2012). Therefore, LEH is typically considered a general non-specific indication of stress experienced during childhood and in this sample likely reflects food insecurity or disease experience in childhood (Hillson, 2019).

There are four main causes of infection: bacteria, viruses, eukaryotes, and prions. Typically, these organisms are transmitted through physical contact, the air, or through ingestion and are more frequently contracted in urban areas with higher population densities, barriers to adequate nutrition, or locations with poor sanitary practices. Infection can appear on the skeleton variably, but typically it appears in the form of lytic bone destruction, or new bone formation, and can be classified as specific or non-specific (Lewis, 2017; Roberts & Brickley, 2019). Specific infections refer to pathological lesions that are consistent with an identifiable disease or disease

process, such as leprosy or brucellosis. Non-specific infections refer to pathological lesions that cannot be associated with a specific etiology and often represent general levels of stress in a population (Lewis, 2017). In the juvenile sample studied in this thesis, non-specific infections were identified in seven juveniles, and one case of specific infection (possible leprosy) was identified.

A number of factors may have contributed to the infections observed in this sample. In utero, fetuses are protected by their mother's immune system. Shortly after birth, nutrients and antibodies passed from mother to child through breastmilk support a child's growth and development (Lewis, 2009). However, pathologies resulting from the birth process, such as tetanus or bacterial infection from cutting the umbilical cord, and respiratory infections like pneumonia or sepsis are possible. Moreover, infants and newborns are susceptible to localized injuries like burns or animal bites, both common causes of severe infection (Lewis, 2018; Liston *et al*., 2018). In addition, many infectious processes in late antiquity would have likely been reserved to the soft tissue (Liston *et al*., 2018). General unsanitary conditions resulting in contaminated food or water sources likely increased the prevalence of soft tissue gastrointestinal concerns such as dysentery, diarrheal, or intestinal disease, resulting in dehydration, malabsorption of nutrients, and ultimately increased mortality for infants and children (Brickley & Ives, 2008; Stathakopoulos, 2004). While a clear etiology cannot be determined for the seven cases on non-specific infection in this sample, any of the above factors could have contributed to the periostitis (Grave 27, Skeleton 29, Grave 45, Skeleton 41, and Bothros #1, Skeleton 45), meningitis (Grave 19, Skeleton 15), and osteomyelitis (Grave 25, Skeleton 27, and Grave 38, Skeleton 37) observed on these juveniles (see Appendices 3 and 5).

Previous aDNA research at the site has confirmed the presence of leprosy and typhoid fever (M. Liston, personal communication, October 7, 2023). Both leprosy and typhoid fever are

primarily soft tissue concerns, however, their prevalence in the adult individuals at this site necessitates their investigation in the context of the juveniles recovered (Lewis, 2017). M. Liston previously identified possible leprosy cases in all of the undisturbed graves, with definitive lesions found in six graves, two of which were studied in this thesis, Graves 4 and 45. Leprosy is an infectious disease caused by *Mycobacterium leprae* and is primarily transmitted through inhalation of droplets through the airway, or skin-to-skin contact. Leprosy is primarily a soft tissue disease, but prolonged infection can result in primary and secondary bone lesions (Lewis, 2002, 2017). Typically, only 3-5% of leprosy cases present bone involvement (Roberts & Brickley, 2019). Nevertheless, primary lesions include rhinomaxillary syndrome (pitting of the nasal cavity and palatine process of the maxilla), changes to the dentition, and osteomyelitis of the limbs, hands, and feet. Secondary bone lesions are typically a result of nerve damage (through sensory, motor, and autonomic neuropathy) and commonly present as remodeling and erosion of the phalanges, drop-foot, claw hand and foot deformities, navicular squeezing and dorsal barring, and diffuse new bone formation (Lewis 2017). One older juvenile, Grave 5, Skeleton 5, presented with evidence of secondary bone lesions associated with leprosy (see Appendix 5).

Children who contract leprosy typically do not present with lesions until older childhood, if they live that long. Juveniles 3-6 years of age are the most susceptible to acquiring leprosy, and it has been reported that children who experience symptoms of the infection in early life may spontaneously heal and relapse later in life (Lewis, 2002, 2017). Leprosy makes children more susceptible to other infectious processes, increasing chances of mortality prior to the formation of osteological symptoms (Lewis, 2017). The high prevalence of young juveniles at this site could be linked to the susceptibility of leprosy in this age group, and it is possible that several of the

juveniles at Ismenion Hill were impacted by leprosy without evidence of skeletal lesions. Further pathogen aDNA testing could investigate this possibility.

The social and religious stigma of a leprosy diagnosis in Byzantine Christian thought must be considered when assessing the potential link between the church of St. Luke and the Ismenion Hill cemetery. As a condition that primarily presents as grey-coloured skin lesions, leprosy was called "elephant disease" and was interpreted variably either as a punishment for sin, a moral lesson, or simply a trial set forward by God to test one's faith (Miller & Nesbitt, 2014; Santacroce *et al.,* 2021). Some Christian scholars took strongly negative views of leprosy diagnoses (i.e., Isidore of Pelusium ca. 4^{th}-5^{th} c. CE), suggesting that children acquired leprosy as a result of the sins of their mothers. Others, (i.e., John Chrysostom, Gregory of Nazianzus ca. 4^{th} c. CE) emphasized the importance of caring for those with leprosy, necessitating acts of charity, mercy, and support for those impacted by the disease (Miller & Nesbitt, 2014). While some efforts may have shifted towards supporting those with leprosy in the early Byzantine period, there was still significant fear and stigma held by the general public in antiquity. People with leprosy were often seen as outcasts, and the disease more often impacted those of lower socioeconomic status, compounding negative views of the disease amongst the public (Miller & Nesbitt, 2014; Santacroce *et al.,* 2021). The difference in opinions surrounding the causes of leprosy in the early Byzantine era makes understanding individual experiences of the disease challenging. However, charitable institutions, like churches, are reported to have provided support and care for individuals with leprosy (Miller & Nesbitt, 2014). This could explain the overwhelming evidence of individuals with leprosy at this site. The location of the church of St. Luke would have made the nearby Ismenion Hill a convenient location for interring those who did not survive the disease.

Pathogen aDNA testing, completed by the Max Planck Institute in 2021, revealed *Salmonella enterica* in a dental sample from an adult in Grave 19, one of the two mass graves at this site (M. Liston, personal communication, October 7, 2023). The burial context of Graves 19 and 20 suggest they were dug at the same time, and the individuals were rapidly interred over a short period of time, supporting their designation as mass graves (Chamari *et al*., 2014). Nine juveniles were identified in the graves, four of which showed evidence of dental (n=1) or skeletal pathologies (n=3). Typhoid fever is primarily a gastrointestinal disease without skeletal involvement, however, a juvenile experiencing metabolic or infectious concerns would undoubtedly be more susceptible to contracting typhoid fever (Roberts & Manchester, 2007).

Typhoid is considered to have been endemic in ancient Greece due to overcrowding, living conditions, and contaminated food and water sources from human or animal excrement (Papagrigorakis *et al*., 2006). Unsanitary conditions in modern populations result in approximately 110,000 typhoid-related deaths, and nine-million typhoid diagnoses per year, reinforcing its likely severity and prevalence in antiquity (World Health Organization, 2023c). Moreover, typhoid would have been common year-round in ancient Greece. Dry seasons with limited fresh water to maintain hygiene practices and wet seasons where contaminated water was more readily spread to wells and reservoirs would have increased transmission of the disease (Stathakopoulos, 2004). Typhoid incubates over two weeks before symptoms occur and prior to the use of modern antibiotics the mortality rate was 15% (Levine & Lepage, 2005). Symptoms such as fever, diarrhea, dehydration, rash, delirium, loss of appetite, and other gastrointestinal concerns often accompany typhoid fever (Papagrigorakis *et al*., 2006; Stathakopoulos, 2004).

Outbreaks and use of mass graves for victims of typhoid fever are reported elsewhere in ancient Greece, particularly in Athens from the 5th century BCE where dental aDNA testing

revealed *S. enterica* in a mass grave at the cemetery of Kerameikos (Papagrigorakis *et al.,* 2006). Considering the gastrointestinal symptoms of typhoid fever and the susceptibility of a young and underdeveloped immune system, typhoid fever cannot be ruled out as a cause of death for the juveniles recovered in Graves 19 and 20. It is likely typhoid fever played a role in the mortality of the broader skeletal assemblage at Ismenion Hill (Levine & Lepage, 2005; Liston *et al*., 2018).

While the pathological observations made in this thesis are based on the skeleton, infectious, metabolic, or hematopoietic disorders would likely not go unnoticed in a living juvenile in antiquity. Outward changes of the soft tissue such as the skin lesions associated with leprosy, differences in energy levels or physical ability associated with malnutrition and chronic infection, or the inability to hydrate or maintain food intake resulting from gastrointestinal diseases would take a significant toll on a juvenile (Brickley & Ives, 2008; Lewis, 2009, 2017). These changes may have encouraged parents to bring their children to the church of St. Luke, or older juveniles and adolescents may have visited independently, to seek out care, medical advice, or baptisms in light of their illness (Vuolanto, 2020).

2.9 Case study: Grave 4, Skeleton 1

Summary

Age-at-death: dental: 9.5-13.5 ± 0.5 yrs., skeletal: 5-7 yrs.

The thorax, upper and lower limbs, and vertebral column of this juvenile were mostly complete. The cranium presented with a partial maxilla, and some cranial vault and base fragments. Preservation was generally good but postmortem erosion and breakage was common on the long bones.

Observations

Grave 4, Skeleton 1 was estimated to be around 9-14 years of age through dental eruption and formation, and 5-7 years through skeletal measurements. The left maxilla presented with antemortem tooth loss (AMTL) at the location of the second permanent premolar, and first molar. A localized new bone reaction (~10 mm in diameter) was observed superior to the AMTL, suggestive of an abscess (Figure 6). Diffuse fine porosity was observed in the nasal aperture, the maxillary body, and on the palatine process of the maxilla. The left *pars lateralis* had extensive fine porosity on the ectocranial surface, focused around, but not on, the occipital condyle (Figure 7). Additionally, the left greater wing of the sphenoid presented with localized abnormal fine pitting on the superior aspect, ranging from 1-2 mm in diameter (Figure 8). Overall, the cranial elements exhibited general fine porosity on endo- and ectocranial aspects.

The postcranial skeleton exhibited similar porosity. The left scapula had large vascular foramina and diffuse pitting in the supraspinous area. Concavity and large pitting was observed on the bodies of the thoracic vertebrae (~2-5 mm in diameter) with fine porosity on the neural arches (Figure 9). The left and right femora and the left humerus had significant fine porosity at the proximal and distal metaphyses (Figure 10, 11). Additionally, radiographic imaging revealed

radiolucent lines adjacent to the metaphysis of the proximal tibia and the distal femora. The diaphyses of the metatarsals, pedal phalanges, the left and right talus, and the left calcaneus presented with abnormal fine porosity.

Differential diagnosis

The DD considered a number of metabolic, hematopoietic, and infectious pathologies associated with widespread bone loss and lytic activity, as recommended by Klaus (2016). Observations were unlikely to be a result of postmortem pseudo-pathologies, or normal growth. Significant osteoclastic resorption at these sites is not typical of regular growth and development (Klaus, 2016). Additionally, while postmortem damage and erosion were identified, those sites were distinctly lighter in colour and presented with areas of disturbed bone cortex, unlike the abnormal lesions on the unaffected cortex of this skeleton (Stodder, 2019).

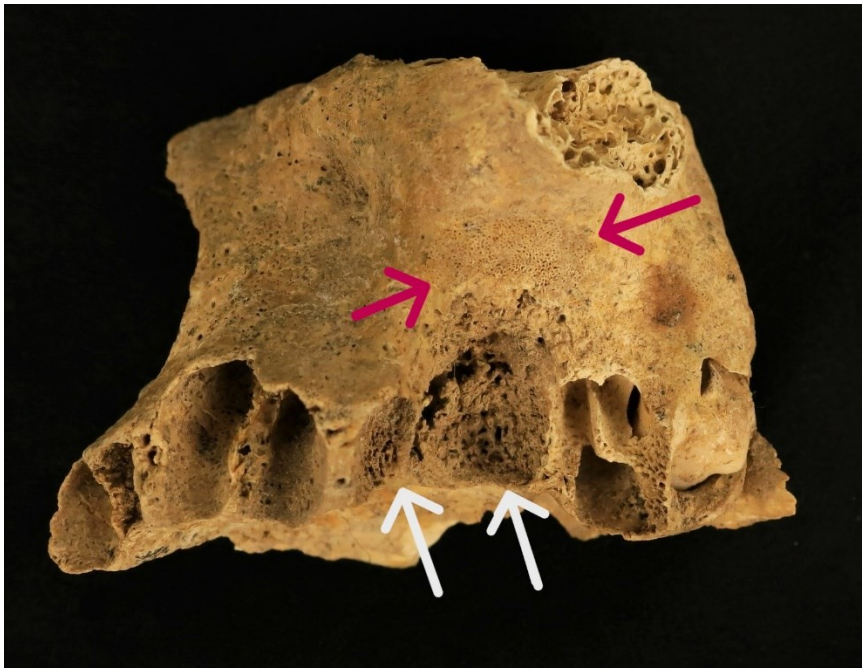


Figure 6: *Grave 4, Skeleton 1, left maxilla. White arrows denote AMTL, red arrows identify reactive woven bone suggestive of an abscess, not to scale.*

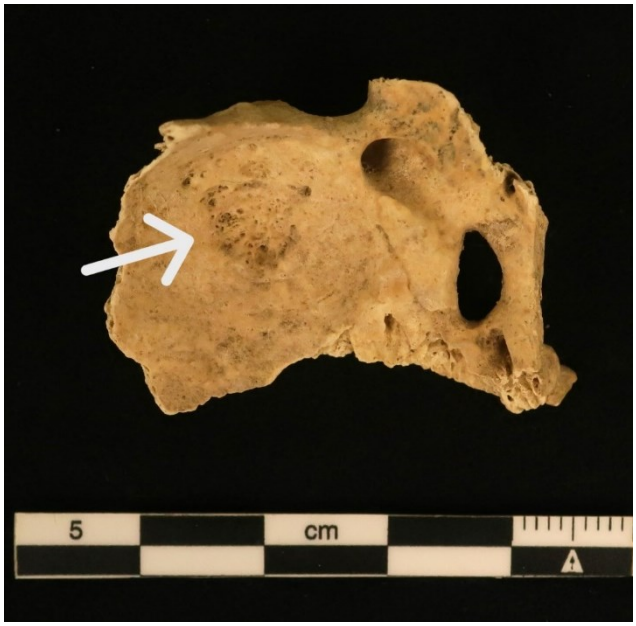
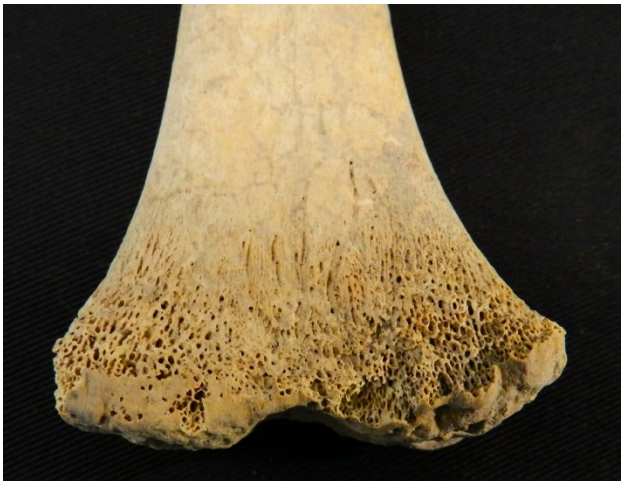


Figure 7: *Grave 4, Skeleton 1, left pars lateralis fragment, white arrows identify ectocranial porosity (left).*

Figure 8: *Grave 4, Skeleton 1, left greater wing of sphenoid, white arrow points to endocranial porosity (right).*

Figure 9: *Grave 4, Skeleton 1, thoracic vertebra presenting larger and fine porosity, and concavity of vertebral body, not to scale (photo by M. Liston, 2017).*



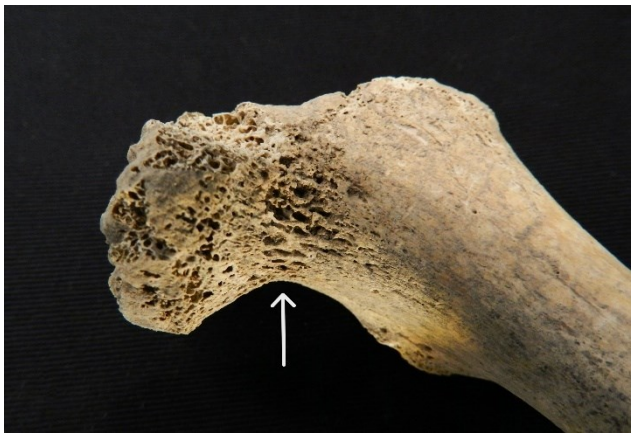


Figure 10: *Grave 4, Skeleton 1, distal metaphysis of left femur, note porosity, not to scale (left, photo by M. Liston, 2017).*

Figure 11: *Grave 4, Skeleton 1 proximal metaphysis of left femur, white arrow highlights porosity, not to scale (right, photo by M. Liston, 2017).*

Age-at-death

The significant difference between the dental and skeletal age-at-death estimates for Grave 4, Skeleton 1 may indicate that this juvenile experienced stress during their growth and development. Dental age estimates are considered a more reliable indication of age than skeletal age indicators, as the development of dentition is under tighter genetic control, and therefore more resistant to environmental stressors (Lewis, 2019). Growth of the skeleton is more readily impacted by external stressors, such as nutritional challenges, infection, and environmental changes, which may stunt skeletal development. This can result in an underestimation of the actual skeletal age-at-death of a juvenile, reflecting their stunted growth (Mays, 2018). Together, incongruity between the skeletal and dental age-at death suggests that this individual was likely entering adolescence, however, their skeletal growth was significantly stunted, suggesting a metabolic or infectious stressor impacted their skeletal development (Lewis, 2019).

Childhood leukemia

Leukemia is the most common cancer impacting modern children under 15 years of age (Cammarata-Scalisi *et al.,* 2020). Leukemia is a blood-born cancer resulting from the overproduction of white blood cells in the body. Leukemia can be acute or chronic, and is differentiated by the type of cells impacted, lymphocytes (blood cells that mature outside bone marrow) or myelocytes (blood cells that mature within bone marrow) (Hutter, 2010; Lewis, 2017). Clinically, acute lymphoblastic leukemia is the most common type of childhood leukemia, followed by acute myelogenous leukemia, and chronic myelogenous leukemia (Lewis, 2017; Sinigaglia *et al*., 2008). Archaeologically, few diagnoses of childhood leukemia have been reported (Lewis, 2017). Without medical intervention, acute leukemia can cause death within a few weeks, limiting skeletal evidence of the disease. However, prevalence of this disease in contemporary populations provides insight into its skeletal features (Lewis, 2017).

Clinical symptoms of childhood leukemia include bone and joint pain in the lower limbs, dental issues, bleeding of the gums, weakness, and fever (Lewis, 2017). Osteological features include diffuse osteopenia, osteoclastic resorption at the metaphyses, widespread porosity and pitting of the cranial and postcranial skeleton, compression of the vertebral bodies, widening of vascular foramina, radiographic translucent lines at the metaphysis (metaphyseal bands), and occasionally the production of new periosteal bone (Klaus, 2016; Lewis, 2017; Ortner, 2003, Rogalsky *et al*., 1986). With the exception of new periosteal bone, this individual's skeletal symptoms correlate well with the expected skeletal symptoms of childhood leukemia. The lack of new periosteal bone formation is not unusual. Kobayashi and colleagues (2005) suggest that new periosteal bone formation in cases of leukemia may be a result of healing of fractures caused by osteopenia, and Rogalsky and colleagues (1986) report periosteal bone formation as the least common symptom of childhood leukemia in a modern sample. To that end, diffuse osteopenia and

lytic osteoclastic lesions paired with the presentation of metaphyseal bands is consistent with a diagnosis of childhood leukemia, and other pathological possibilities fail to account for the suite of lesions observed (see Appendix 3 for a complete discussion of pathologies) (Lewis, 2017; Rogalsky *et al*., 1986).

The etiology of childhood leukemia is not well understood. It is not hereditary, but certain genes may predispose children to acquiring it (Buffler *et al.*, 2005). Modern research has linked childhood leukemia with exposure to significant levels of radiation, toxins, maternal exposure to chemicals, and possibly viral infections or vitamin deficiencies (Hutter, 2010; Klaus, 2016). It is thought that certain conditions such as Down Syndrome or immunodeficiency may also increase an individual's risk for acquiring leukemia in their lifetime (Hutter, 2010). Metabolic and infectious stressors impacting juveniles in late antiquity (discussed in Section 2.8) would have presented a number of precarious circumstances likely increasing morbidity and mortality for this juvenile. While it is not possible to discern the specific environmental or genetic factors that may have predisposed this juvenile to acquiring leukemia at this time, the presentation of comorbidities support a discussion of the health-related challenges this juvenile may have faced leading up to or around their time of death.

This juvenile presented with dental comorbidities. AMTL can be caused by caries, trauma, or overall poor dental hygiene, and is recognized skeletally by the absence of the tooth and the resorption of the alveolar bone in the tooth socket indicating that the tooth was lost before death (Ortner, 2003). A dental abscess is identified as a cavity of absent bone surrounding or in the area of a tooth root. Dental abscesses are typically a result of an inflammatory process brought on by caries, AMTL, or collections of plaque in the gum line. These processes collect bacteria resulting

in an accumulation of infection and pus, which in turn breaks down the surrounding bony tissue resulting in a cavity for the pus to evacuate (Roberts & Manchester, 2007).

In the case of Grave 4, Skeleton 1, incomplete resorption of the tooth socket suggests healing was actively taking place and the tooth-loss was recent. Abscesses, AMTL, and subsequent infection and inflammation of gums and alveolus can result in challenges eating, resulting in malnutrition and increased risk for infection (Prowse, 2011). Modern clinical literature has identified a link between AMTL and leukemia, suggesting a relationship between the pathological conditions in this individual. Acute childhood leukemia can manifest oral lesions such as spontaneous bleeding, ulcers, osteolytic lesions of the jaw, caries, and gum or dental pain (Cammarata-Scalisi *et al.,* 2020; Mathur *et al.*, 2012).

Overall, the incongruity between the skeletal and dental age-at-death estimates, the occurrence of AMTL, and the osteological symptoms consistent with a case of childhood leukemia, suggest that this juvenile experienced a significant amount of stress in their lifetime. It is not possible to distinguish the specific type of leukemia from macroscopic analysis, however, as the acute and more aggressive forms of childhood leukemia are the most common it is likely that this is the case for this juvenile. Chronology of the dental pathologies and the overall disruption of skeletal growth cannot be excluded as a potential result of the leukemia diagnosis, or a product of the environmental stressors that predisposed them to acquiring leukemia in the first place. It is possible that this juvenile and their family sought out medical attention. Grave 4 Skeleton 1's small size for age, the frailty associated with a childhood leukemia diagnosis, and dental comorbidities would have made it evident that this child was sick. Blessings, baptism, or care at the church of St. Luke in response to these symptoms, may have supported this child's burial at Ismenion Hill (Vuolanto, 2020).

2.10 Case study: Grave 19, Skeleton 15

Summary

Age-at-death: dental: 34 wks. – 1.5 ± 3 mo., skeletal: 36-40 wks.

The skeletal remains of this perinate were highly fragmentary due to postmortem breakage. However, most of the skeleton was recovered, including most of the cranium, thorax, upper, and lower limbs.

Observations

The left and right frontal squama presented with deep vascular grooves on the internal aspect of the bones (Figure 12, 13). The grooves are 1-2 mm in thickness and depth and localized at the area of the medial portion of the squama, and new periosteal bone was identified surrounding the grooves. New periosteal bone is observed in the left orbit. Thin linear deposits of bone are noted at the edges of the squama, likely growth-related. The occipital squama, *pars basilaris*, right sphenoid fragment, left and right temporals, left and right zygomatics, maxilla and mandible present with a porous and woven appearance on the endo- and ectocranial surfaces.

Differential diagnosis

The presence of woven bone on the cranial elements of a perinate is typically considered normal and associated with rapid growth and development (Ortner, 2003). Therefore, some observations of porosity and linear deposits of woven bone in the cranium of this individual are to be expected (Lewis, 2017). However, the lesions observed on the internal medial portion of the frontal squama of this perinate, and the presence of new bone in the orbit, support a pathological process rather than normal growth or pseudo-pathology (Lewis, 2017).

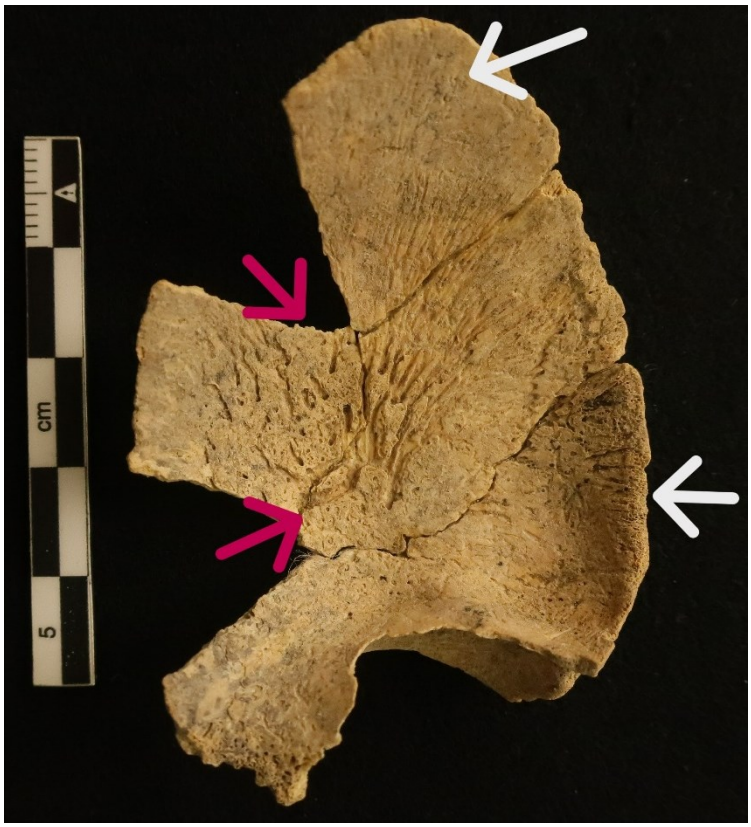


Figure 12: *Grave 19, Skeleton 15, left frontal bone, internal aspect, red arrow highlights endocranial lesions (left).*

Figure 13: *Grave 19, Skeleton 15, right frontal bone, internal aspect, red arrows highlight area of endocranial lesions, white arrows highlight normal growth (right).*

Endocranial lesions are recognized by a disorganized appearance, thick linear grooves and vascular impressions, and deposits of new bone on the internal surface of the cranium. Lesions can be focused or diffuse, resulting from new bone being laid down around meningeal vessels due to hemorrhage or disruption of venous drainage sites. The endosteal inner layer of the skull is composed of blood vessels that supply the bones of the cranium. This layer is part of the meninges, the vascular tissues that cover the brain, spinal cord, and inner surface of the cranium (Lewis, 2004). Subdural hematomas (pooling of blood between the layers of the meninges) or meningitis from the inflammation of the meninges have been reported as common causes of endocranial lesions in children (Lewis, 2004; Ortner, 2003). To that end, the cause of endocranial lesions is

typically unknown, but a review of the lesions by Lewis (2004) identifies infection, trauma, neoplasms, and metabolic deficiencies to be the most likely causes. For a complete discussion of the DD for Grave 19, Skeleton 15, see Appendix 3.

Infectious endocranial lesions

In the case of Skeleton 15, infectious processes resulting in endocranial lesions associated with meningitis are the most likely cause. Tuberculosis, typhoid fever, influenza, pneumonia, otitis media (ear infections), respiratory tract infections, measles, and whooping cough have been linked to meningitis in infants and children (Lewis, 2004; Ortner, 2003; Ripy, 1950). General bacterial infections from cutting the umbilical cord or from contaminated food and water, or bacteria spread from a localized injury is a common route of infection for infants (Lewis, 2004; Liston *et al*., 2018). *Haemophilus influenzae*, *Streptococcus* bacteria types, and *Escherichia coli* are the most likely infective agents in meningitis cases (Lewis, 2004; Liston *et al*., 2018). The majority of these pathogens cannot be confidently ruled out and the non-specific nature of endocranial lesions without other pathogen aDNA testing or skeletal indicators does not allow for the attribution of one disease over another (Ortner, 2003).

Children can survive for weeks with non-specific meningitis, allowing for the formation of new bone, however, a state of chronic infection can lead to significant complications and increase susceptibility to other fatal pathogens (Lewis, 2004; Liston *et al*., 2018). Considering the mass grave context of Grave 19, and the association with typhoid fever, this diagnostic avenue must be considered. Since typhoid fever is primarily gastrointestinal, it is likely not the root of the infection that caused the endocranial lesions observed, and instead it could be a secondary infection that contributed to the death of this perinate (Roberts & Manchester, 2007). Typically, newborns

benefit from the passive immunity passed on from mother to child, however, if not breastfeeding or already suffering from another infection, the acquisition of typhoid fever and the subsequent gastrointestinal symptoms would likely be fatal (Levine and Lepage 2005; Liston *et al*., 2018; Papagrigorakis *et al*., 2006).

Overall, the young age-at-death of this juvenile supports that they were possibly brought to the church of St. Luke for baptism or treatment (Vuolanto, 2020). However, as their death may have coincided with a typhoid outbreak, it is possible sickly individuals regardless of potential cause of death that died around this time, may have been interred together at Ismenion Hill in response to the overwhelming number of deaths in epidemic scenarios (Brzobohatá *et al.,* 2019). Acknowledging the proposed significance of the nearby church of St. Luke, this possibility cannot be ruled out. Pathogen aDNA testing could investigate if this juvenile presents with typhoid fever.

2.11 Case study: Grave 25, Skeleton 27 and Grave 38, Skeleton 37

Summary

Age-at-death Grave 25, Skeleton 27: skeletal: 40 wks. - 2 yrs.

Age-at-death Grave 38, Skeleton 37: skeletal: 24-32 wks.

Grave 38, skeleton 37 presented with limited skeletal material including ~10 cranial fragments, and four long bone fragments, all with significant postmortem breakage and erosion. Grave 25, skeleton 27 also presented with limited skeletal material including ~16 cranial fragments, some long bone fragments, ~15 vertebrae, and some hand and foot bones. This perinate and infant were grouped for analysis based on their similar age-at-death estimates and near identical pathological observations.

Observations

Grave 25, Skeleton 27 presents a bone-in-bone like appearance on their left and right femora (Figure 14, 15). An abnormal sheath of bone cortex encases the circumference of the original femur. There is a gap between the layers, with some spicules extending between the original femur and the outer sheath. Grave 38, Skeleton 37 presents a similar bone-in-bone like appearance on their femur (possibly left), where an outer cortex layer encases the original femur diaphysis (Figure 16). Some spicules project between the bone layers. As the outer layer approaches the edges of the fragment the space between layers decreases.

Figure 14: *Grave 25, Skeleton 27, left femur fragment, white arrows identify outer bone layer and red arrows identify cortex of original femur (left).*

Figure 15: *Grave 25, Skeleton 27, right femur fragment, white arrow identifies outer bone layer and red arrow identifies cortex of original femur (right).*

Figure 16: *Grave 38, Skeleton 37, femur fragment, white arrow identifies outer bone layer and red arrow identifies cortex of original femur.*

Differential diagnosis

The limited skeletal material recovered for these juveniles makes differential diagnosis challenging, however, the similar bone-in-bone (BIB) appearance of the femora and age allows for them to be grouped for discussion. In infants, BIB is rarely reported in paleopathological literature, however, modern clinical reports often identify BIB in infants through radiographs (Hancox, 1951; Williams *et al.,* 2004). BIB can be identified by the presence of a double-layered cortex, or the split in the cortex of bone, mirroring the pathological observations of Grave 25, Skeleton 27 and Grave 38, Skeleton 37. Commonly, BIB is accompanied by new periosteal bone growth and can be considered normal in rapidly growing infants (Williams *et al.,* 2004). Growth-related changes should be considered (Hancox, 1951). Infants and perinates experience significant growth-related remodeling of the long bones that gradually slows with increasing age. If a child is unhealthy or experiences trauma to the surrounding tissues of a bone, the balance between bone formation (at the periosteal surface of long bones) and bone removal (at the internal endosteal surface of long bones) can be affected. This results in a disruption to the remodeling of long bones

(Lewis, 2017). However, it can also be a result of abnormal bone-forming pathologies, trauma, infection, metabolic, or hematopoietic disorders (Williams *et al.,* 2004).

Trauma and bone-forming pathologies

Lewis (2017) identifies birth trauma as a potential factor disrupting long bone formation, however, in the case of these juveniles, no evidence of fracture or injury consistent with trauma was observed. However, the fragmentary nature of the femora recovered limits the confidence in this claim. Bone-forming neoplasms impacting the diaphysis of the femur were explored, however, relevant benign (i.e., osteoid osteoma, giant cell tumours) and metastatic tumours (Ewing's sarcoma) commonly impact older juveniles and adolescents, exceeding the young age-at-death estimates for Skeletons 27 and 37 (Lewis, 2017).

Metabolic, hematopoietic, and circulatory conditions such as scurvy, sickle cell anemia (SCA), hypertrophic osteoarthropathy (HOA), and infantile cortical hyperostosis (ICH) have been reported to cause extensive hemorrhage, new bone formation, or increase susceptibility to infection in juveniles potentially producing a BIB appearance (Lewis, 2009, 2017; Williams *et al*., 2004). However, the young age-at-death of this perinate and infant makes a scurvy diagnosis unlikely (Lewis, 2009). Additionally, while SCA can cause a BIB like appearance, it is typically observed in the vertebral bodies, not the femora (Williams *et al*., 2004). Moreover, HOA typically impacts the lower limb instead of the femur, and presents a "candle-wax" appearance, unlike the femora of this perinate and infant (Aufderheide & Rodríguez-Martín, 1998). Finally, ICH is diagnosed through the presence of widespread new bone formation on the long bones, clavicle, and mandible (Lewis, 2009). No new bone formation was observed on the tibiae or ulna of this infant and perinate, and they did not present with new bone formation on the mandible, typically considered

to be diagnostic of ICH (Caffey, 1956; Lewis, 2009; Ortner, 2003). As an isolated reaction to the femora, this tentatively rules out ICH for these juveniles (Lewis, 2009). For a complete DD see Appendix 3.

Infantile osteomyelitis

Osteomyelitis is an infection of the bone and bone marrow that is often non-specific in nature (Bourbou, 2020; Lewis, 2017). It presents as a sheath of new bone outside the existing cortex (an involucrum), and the formation of a sequestrum or gap between the layers due to tissue death (Ortner, 2003). New bone formation in response to infection can occur in as little as 1 week, and sequestrum and involucrum formation can occur within 2-3 weeks following infection (Lewis, 2017). In juveniles, hematogenous osteomyelitis (infection spread via the bloodstream) is common, and the tibia, femur, and knee are often impacted (Bourbou, 2020; Lewis, 2009). *Staphylococcus aureus*, *Streptococcus pyogenes*, *Haemophilus influenzae*, *Pseudomonas aeruginosa* and *Pasteurella multocida* are the most common bacteria in osteomyelitis cases (Lewis 2009, 2017).

Due to the limited skeletal material recovered and the complicated nature of non-specific infections, the etiology of this pathology cannot be confidently identified. However, localized injuries such as animal bites, burns, contaminated food or water, or from cutting the umbilical cord are all common routes of infection in infants (Lewis, 2009, 2017; Liston *et al*., 2018). According to Bourbou (2020) only one case of juvenile osteomyelitis has been reported in the Byzantine period, however, Trueta (1959) reports a 45% mortality rate in cases of infant and juvenile osteomyelitis in the pre-antibiotic era. To that end, the lack of modern antibiotics and medical treatment, unsanitary living conditions, and potential inadequate maternal or juvenile nutrition,

would have likely increased morbidity of osteomyelitis in the Byzantine period (Bourbou, 2020). The young age of this perinate and infant, susceptibility to infection, and the location and morphology of the symptoms observed are consistent with infantile osteomyelitis.

As Grave 38, Skeleton 37 likely represents a premature perinate, and Grave 25, Skeleton 27 a newborn infant, both juveniles may have been brought to the church of St. Luke for baptism. In both cases, their young-age-at-death, and knowledge of the challenges faced by premature perinates and newborns in antiquity would have encouraged baptism shortly after birth (Malamitsi-Puchner, 2017). Moreover, if the child presented outward signs of infection or frailty, care or healing blessings may have been sought out prior to their death and subsequent burial at Ismenion Hill (Vuolanto, 2020).

Conclusions

2.12 Conclusion

The 4th century CE marked the beginnings of early hospital-like institutions in the Byzantine world, mostly in the form of poor houses or hospices affiliated with local religious establishments (Miller, 2003). Increased population sizes, warfare, and general economic instability in the 4th century CE increased pressure on government officials and the newly legalized Christian church to care for the vulnerable sectors of Greece's population (Miller, 2003; Stathakopoulos, 2004). This was reinforced by Christian ideals of piety, charity, and care for those less fortunate (Miller, 2003). These views were starkly different from the ideals and practices of the late Roman period. Greco-Romans prior to the integration of Christianity were not unfamiliar with the idea of giving support. However, this typically took the form of donations to the state, votives or offerings to the gods, and through means of displaying personal or familial wealth, not charitable aid to the poor or sick (Brown, 2002). The focus on the state and financial matters was

mirrored in the treatment of children in the Greco-Roman period, as children were primarily born and raised to maintain the family name, property, and inheritance. The Christian belief in the presence of a soul encouraged a shift in thinking about the care of children and those typically cast out in Greco-Roman society, the homeless, poor, sick, orphaned, widowed, and the elderly (Miller, 2003; Nathan, 2020). As such, children came to be viewed as a gift, fully human, and more than a means of maintaining finances (Nathan, 2020; Perry, 2006).

It is likely that early Christian institutions like the church of St. Luke in Thebes upheld these values, especially considering Luke's reputation as a physician and healer, prioritizing the care and wellbeing of those less fortunate (Conlin, 1953; Marx, 1980). While in the 6th century CE Christian churches developed charitable institutions resembling early hospital-like clinics, and orphanages to support the growing needs of the Byzantine people, this site predates the formation of official hospitals in the later 11th-12th centuries CE (Magilton, 2008; Miller, 2003). In the late Byzantine, hospitals (usually associated with a local church) would provide inpatient care and individuals would spend their remaining days at the institution until their death and burial in affiliated cemeteries (Magilton, 2008). It is not clear if the Ismenion Hill cemetery is affiliated with the early church of St. Luke, but as the ancient foundations of the church (ca. 5th c. CE) correspond with the earliest Byzantine period burials (also 5th c. CE), it is possible they are contemporaneous. If there is a link between the cemetery and church, it is possible that this site represents an early form of a hospital-like institution with a designated burial ground for the sick.

Considering the social and religious context of this time period, it is possible that sick children were brought to the area of Ismenion Hill in Thebes for healing, baptism, or care at the church of St. Luke the Evangelist. Whether it be parents seeking baptism for their sickly perinates and infants, or care for juveniles suffering from nutritional or infectious conditions, the nearby

Ismenion Hill used as a cemetery for hundreds of years would have provided an ideal location for the burial of adults and children who did not survive the illnesses they sought to cure (Daly & Larson, 2017; Liston, 2017, 2018). At this time, osteological evidence alone cannot confidently support this claim and archaeological and documentary evidence is necessary to address this further.

To explore why children were brought to and buried at Ismenion Hill, and whether the hypothesis posed by previous researchers holds true for the juveniles, a paleopathological analysis, assessment of environmental and cultural factors influencing juvenile health, and an investigation of the religious and social climate of the early Byzantine period was provided. While Christianity may have influenced the circumstances for which children were brought to and buried at Ismenion Hill, it is important to acknowledge that health experiences were highly variable for children in antiquity (Nathan, 2020; Vuolanto, 2020). As presented in this thesis, infection, barriers to adequate nutrition, weaning, and environmental agents like famine and epidemic disease, all likely played a role in childhood health experience. Overall, the juveniles studied at Ismenion Hill, and their high prevalence of skeletal pathologies, reinforce that life for children in the early Byzantine period was challenging, mortality was high, and health was a product of social, cultural, and environmental factors.

2.13 Future directions

Moving forward, this research would benefit from an investigation of the remaining juveniles at Ismenion Hill. A complete analysis of the juvenile skeletal material will be critical to investigating the hypothesis posed by previous researchers and the conclusions of this thesis. Additional research would also benefit from a comparative analysis of the juveniles and adults at

this site to investigate if, or how, health-experiences differed for adults and juveniles buried at Ismenion Hill. Finally, studies should continue to integrate archaeological and documentary evidence of Byzantine Thebes as it becomes available to understand the significance of this site.

Future analyses would benefit from additional radiographic or microscopic imaging and chemical analyses, to provide a more nuanced understanding of the skeletal lesions observed at this site (Lewis, 2017; Mays, 2020). The use of nitrogen ($\delta^{15}N$) and carbon ($\delta^{13}C$) stable isotopes have proven useful in other juvenile studies in Byzantine Greece to explore diet and weaning practices, potentially aiding in the understanding of metabolic stressors experienced by the juveniles at Ismenion Hill (Bourbou *et al.,* 2011; Bourbou *et al.,* 2013). Further aDNA analyses, testing for *S. enterica* (typhoid fever) and *M. leprae* (leprosy) bacteria, or to estimate sex, would be extremely beneficial to explore the extent and prevalence of infectious pathologies and demographics of this site (Lewis, 2017). Together, these investigative efforts will aid in a more holistic understanding of health experiences impacting the juveniles buried at Ismenion Hill.

References

Agarwal, S. C., & Beauchesne, P. (2011). It is not carved in bone: Development and plasticity of the aged skeleton. In S. C. Agarwal & B. A. Glencross (Eds.), *Social bioarchaeology* (pp.312–332). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781444390537.ch11

Agarwal, S. C., & Glencross, B. A. (2011). Building a social bioarchaeology. In S. C. Agarwal & B. A. Glencross (Eds.), *Social bioarchaeology* (pp.1–11). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781444390537.ch1

AlQahtani, S. J., Hector, M. P., & Liversidge, H. M. (2010). Brief communication: The London atlas of human tooth development and eruption. *American Journal of Physical Anthropology*, *142*(3), 481–490. https://doi.org/10.1002/ajpa.21258

Andersen, J. G., & Manchester, K. (1988). Dorsal tarsal exostoses in leprosy: A palaeopathological and radiological study. *Journal of Archaeological Science*, *15*(1), 51–56. https://doi.org/10.1016/0305-4403(88)90018-0

Andersen, J. G., Manchester, K., & Ali, R. S. (1992). Diaphyseal remodelling in leprosy: A radiological and palaeopathological study. *International Journal of Osteoarchaeology*, *2*(3), 211–219. https://doi.org/10.1002/oa.1390020305

Arivantinos, V., Daly, K., and Larson, S. (2011). *Archaeological investigations in the area of the*

Ismenion and Herakleion Sanctuaries of Thebes. [Unpublished report]. American School of Classical Studies at Athens, Athens, Greece.

Armelagos, G. J. (2003). Bioarchaeology as anthropology. *Archaeological Papers of the American Anthropological Association*, *13*(1), 27–40. https://doi.org/10.1525/ap3a.2003.13.1.27

Aufderheide, A. C., & Rodríguez-Martín, C. (1998). *The Cambridge encyclopedia of human paleopathology*. Cambridge University Press.

Baumgart, S. (2008). Iatrogenic hyperthermia and hypothermia in the neonate. *Clinics in Perinatology*, *35*(1), 183–197. https://doi.org/10.1016/j.clp.2007.11.002

Baxter, J. E. (2008). The archaeology of childhood. *Annual Review of Anthropology*, *37*(1), 159–175. https://doi.org/10.1146/annurev.anthro.37.081407.085129

Beauchesne, P. & Agarwal, S.C. (2018). Introduction: Excavating childhood from the skeletal record. In Beauchesne, P. & Agarwal, S.C. (Eds.) *Children and childhood in bioarchaeology,* (pp.1-32). University Press of Florida.

Black, S., & Scheuer, L. (1996). Age changes in the clavicle: From the early neonatal period to skeletal maturity. *International Journal of Osteoarchaeology*, *6*(5), 425–434. https://doi.org/10.1002/(SICI)1099-1212(199612)6:5<425::AID-OA287>3.0.CO;2-U

Blake, K. A. S. (2018). The biology of the fetal period: Interpreting life from fetal skeletal remains. In S. Han, T. K. Betsinger, & A. B. Scott (Eds.), *The Anthropology of the Fetus: Biology, Culture and Society* (pp.34–58). Berghahn Books.

Bodling S, & Lindauer S. (2022). MHAAM_TBS_Neumann_18.11.2021. *Max Planck Institute for Evolutionary Anthropology.*

Borofsky, R., & De Lauri, A. (2019). Public anthropology in changing times. *Public Anthropologist*, *1*, 3–19. https://doi.org/10.1163/25891715-00201002

Bourbou, C. (2016). *Health and disease in Byzantine Crete (7th-12th centuries AD)*. Routledge.

Bourbou, C. (2020). "The greatest of treasures": Advances in the bioarchaeology of Byzantine children. In L. A. Beaumont, M. Dillon, & N. Harrington (Eds.), *Children in antiquity* (pp.594–607). Routledge. https://doi.org/10.4324/9781315542812-47

Bourbou, C., Fuller, B. T., Garvie-Lok, S. J., & Richards, M. P. (2011). Reconstructing the diets of Greek Byzantine populations (6th–15th centuries AD) using carbon and nitrogen stable isotope ratios. *American Journal of Physical Anthropology*, *146*(4), 569–581. https://doi.org/10.1002/ajpa.21601

Bourbou, C., Fuller, B. T., Garvie-Lok, S. J., & Richards, M. P. (2013). Nursing mothers and

feeding bottles: Reconstructing breastfeeding and weaning patterns in Greek Byzantine populations (6th–15th centuries AD) using carbon and nitrogen stable isotope ratios. *Journal of Archaeological Science*, *40*(11), 3903–3913. https://doi.org/10.1016/j.jas.2013.04.020

Bourbou, C., & Garvie-Lok, S. (2015). Bread, oil, wine, and milk: Feeding infants and adults in Byzantine Greece. *Hesperia Supplements*, *49*, 171–194.

Brickley, M., & Ives, R. (2008). *The bioarchaeology of metabolic bone disease*. Academic Press.

Brown, M., & Ortner, D. J. (2011). Childhood scurvy in a medieval burial from Mačvanska Mitrovica, Serbia. *International Journal of Osteoarchaeology*, *21*(2), 197–207. https://doi.org/10.1002/oa.1124

Brown, P. (2002). *Poverty and leadership in the later Roman Empire*. University Press of New England.

Brzobohatá, H., Frolík, J., & Zazvonilová, E. (2019). Bioarchaeology of Past Epidemic- and Famine-Related Mass Burials with Respect to Recent Findings from the Czech Republic. *Interdisciplinaria Archaeologica*, *10*(1), 79–87.

Buffler, P. A., Kwan, M. L., Reynolds, P., & Urayama, K. Y. (2005). Environmental and genetic

risk factors for childhood leukemia: Appraising the evidence. *Cancer Investigation*, *23*(1), 60–75. https://doi.org/10.1081/CNV-46402

Buikstra, J. & Ubelaker, D. (1994). *Standards for data collection from human skeletal remains*. Fayetteville: Arkansas Archaeological Survey.

Burnett, M. W., Bass, J. W. B., & Cook, B. A. (1998). Etiology of osteomyelitis complicating sickle cell disease. *Pediatrics*, *101*(2), 296–297. https://doi.org/10.1542/peds.101.2.296

Butler, N. R., & Alberman, E. D. (1969). *Perinatal problems; the second report of the 1958 British perinatal mortality survey, under the auspices of the National Birthday Trust Fund*. E. & S. Livingstone.

Buzon, M.R. (2012). The bioarchaeological approach to paleopathology. In A.L. Grauer (Ed.), *A companion to paleopathology* (pp.58-75). John Wiley & Sons, Ltd.

Caffey, J. (1956). Infantile cortical hyperostosis; A review of the clinical and radiographic features. *Proceedings of the Royal Society of Medicine*, *50*(5), 347–354. https://doi.org/10.1177/003591575705000516

Cammarata-Scalisi, F., Girardi, K., Strocchio, L., Merli, P., Bernardin, A. G., Galeotti, A.,

Magliarditi, F., Inserra, A., & Callea, M. (2020). Oral manifestations and complications in childhood acute myeloid leukemia. *Cancers*, *12*(6), 1634. https://doi.org/10.3390/cancers12061634

Cartledge, P. (2020). *Thebes: The forgotten city of ancient Greece*. Abrams, Inc.

Charami, A., Kalamara, P., Daly, K., & Larson, S. (2012). *Archaeological investigations in the area of the Ismenion and Herakleion Sanctuaries of Thebes.* [Unpublished report]. American School of Classical Studies at Athens, Athens, Greece.

Charami, A., Kalamara, P., Daly, K., & Larson, S. (2013). *Archaeological investigations in the area of the Ismenion and Herakleion Sanctuaries of Thebes.* [Unpublished report]. American School of Classical Studies at Athens, Athens, Greece.

Charami, A., Kalamara, P., Daly, K., & Larson, S. (2014). *Archaeological investigations in the area of the Ismenion and Herakleion Sanctuaries of Thebes.* [Unpublished report]. American School of Classical Studies at Athens, Athens, Greece.

Charami, A., Kalamara, P., Daly, K., & Larson, S. (2015). *Archaeological investigations in the area of the Ismenion and Herakleion Sanctuaries of Thebes.* [Unpublished report]. American School of Classical Studies at Athens, Athens, Greece.

Choudhary, G., Udayasankar, U., Saade, C., Winegar, B., Maroun, G., & Chokr, J. (2019). A

systematic approach in the diagnosis of paediatric skull lesions: What radiologists need to know. *Polish Journal of Radiology*, *84*, e92–e111. https://doi.org/10.5114/pjr.2019.83101

Colossians 4:14-16. (1995). In *New American Standard Bible*. Lockman.

Conlin, J. F. (1953). St. Luke, most dear physician. *New England Journal of Medicine*, *249*(19), 774–775. https://doi.org/10.1056/NEJM195311052491906

Cunha, E. (2006). Pathology as a factor of personal identity in forensic anthropology. In A. Schmitt, E. Cunha, & J. Pinheiro (Eds.), *Forensic anthropology and medicine* (pp.333–358). Humana Press. https://doi.org/10.1007/978-1-59745-099-7_14

Cunningham, C., Scheuer, L., & Black, S. (2016). *Developmental juvenile osteology* (2nd ed.). Academic Press.

Daly, K., & Larson, S. (2017, May 10). Excavations on the Ismenion Hill in Thebes, 2011-2016. [Video]. American School of Classical Studies at Athens, Athens, Greece. https://www.ascsa.edu.gr/news/newsDetails/excavations-on-the-ismenion-hill-in-thebes-2011-2016

Danforth, L. M. (1982). *The death rituals of rural Greece*. Princeton University Press.

Demirel, F. A. (2023). New insight into mid-Byzantine burial practices: Bioarchaeological

implications from the North Cemetery infant remains of the Lower City Church of Amorium (Afyonkarahisar, Turkey) (10th–11th centuries AD). *Anthropologischer Anzeiger*, *80*(4), 397–406. https://doi.org/10.1127/anthranz/2022/1626

De Silva, P. (2003). Physiological periostitis; a potential pitfall. *Archives of Disease in Childhood*, *88*(12), 1124–1125. https://doi.org/10.1136/adc.88.12.1124

DeWitte, S. N., & Stojanowski, C. M. (2015). The osteological paradox 20 years later: Past perspectives, future directions. *Journal of Archaeological Research*, *23*(4), 397–450. https://doi.org/10.1007/s10814-015-9084-1

Dillon, M. (1997). *Pilgrims and pilgrimage in ancient Greece*. Routledge. https://doi.org/10.4324/9780203352441

Elsner, J., & Rutherford, I. (Eds.). (2007). *Pilgrimage in Graeco-Roman and early Christian antiquity: Seeing the gods*. Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199237913.001.0001

Fain, O. (2005). Musculoskeletal manifestations of scurvy. *Joint Bone Spine*, *72*(2), 124–128. https://doi.org/10.1016/j.jbspin.2004.01.007

Fazekas, I. G., & Kósa, F. (1978). *Forensic fetal osteology*. Akadémiai Kiadó. https://books.google.ca/books?id=Z9FNAQAAIAAJ

Fox, S. C., & Marklein, K. E. (2014). Commingled human skeletal assemblages: Integrative techniques in determination of the MNI/MNE. In A. J. Osterholtz, K. M. Baustian, & D. L. Martin (Eds.), *Commingled and disarticulated human remains* (pp.193–212). Springer New York. https://doi.org/10.1007/978-1-4614-7560-6_3

González Martín, A., Robles Rodríguez, F. J., Cambra-Moo, O., Rascón Pérez, J., & Campo Martín, M. (2018). Comment upon "Basilar portion porosity: A pathological lesion possibly associated with infantile scurvy." *International Journal of Paleopathology*, *20*, 114–115. https://doi.org/10.1016/j.ijpp.2017.09.003

Google Earth. (2023). https://earth.google.com/

Gowland, R. L., & Chamberlain, A. T. (2002). A Bayesian approach to ageing perinatal skeletal material from archaeological sites: Implications for the evidence for infanticide in Roman-Britain. *Journal of Archaeological Science*, *29*(6), 677–685. https://doi.org/10.1006/jasc.2001.0776

Halcrow, S. E., & Tayles, N. (2008). The bioarchaeological investigation of childhood and social age: Problems and prospects. *Journal of Archaeological Method and Theory*, *15*(2), 190–215. https://doi.org/10.1007/s10816-008-9052-x

Halcrow, S. E., Tayles, N., & Elliott, G. E. (2018). The bioarchaeology of fetuses. In S. Han, T. K. Betsinger, & A. B. Scott (Eds.), *The anthropology of the fetus: biology, culture and society* (pp.83–111). Berghahn Books.

Hancox, N. M., Hay, J. D., Holden, W. S., Moss, P. D., & Whitehead, A. S. (1951). The radiological "double contour" effect in the long bones of newly born infants. *Archives of Disease in Childhood*, *26*(130), 543–548. https://doi.org/10.1136/adc.26.130.543

Harrison, F. V. (2016). Theorizing in ex-centric sites. *Anthropological Theory*, *16*(2–3), 160–176. https://doi.org/10.1177/1463499616652516

Health and well-being. (2023a). World Health Organization. https://www.who.int/data/gho/data/major-themes/health-and-well-being

Hesperia. (2023). American School of Classical Studies at Athens (ASCSA). https://www.ascsa.edu.gr/publications/hesperia

Hillson, S. (2019). Dental pathology. In M. A. Katzenberg & A. L. Grauer (Eds.), *Biological anthropology of the human skeleton* (3rd ed., pp.295–334). John Wiley & Sons, Ltd.

Hutter, J. (2010). Childhood leukemia. *Pediatrics in Review*, *31*(6).

Inglis, R.M., & Halcrow, S.E. (2018). The bioarchaeology of childhood: Theoretical

development in the field. In P. Beauchesne & S.C. Agarwal (Eds.), *Children and childhood in bioarchaeology* (pp.33-60).

Kamp, K.A. (2001). Where have all the children gone? *Journal of Archaeological Method and Theory,* 8(1):1-34. http://dx.doi.org/10.1023/A:1009562531188

Kaufman, M. H., Whitaker, D., & McTavish, J. (1997). Differential diagnosis of holes in the calvarium: Application of modern clinical data to palaeopathology. *Journal of Archaeological Science*, *24*(3), 193–218. https://doi.org/10.1006/jasc.1995.0104

Keenleyside, A. (2008). Dental pathology and diet at Apollonia, a Greek colony on the Black Sea. *International Journal of Osteoarchaeology*, *18*(3), 262–279. https://doi.org/10.1002/oa.934

Klaus, H. D. (2016). A probable case of acute childhood leukemia: Skeletal involvement, differential diagnosis, and the bioarchaeology of cancer in South America. *International Journal of Osteoarchaeology*, *26*(2), 348–358. https://doi.org/10.1002/oa.2411

Klaus, H. (2017). Paleopathological rigor and differential diagnosis: Case studies involving terminology, description, and diagnostic frameworks for scurvy in skeletal remains. *International Journal of Paleopathology*, *19*(2017), 96–110. https://doi.org/10.1016/j.ijpp.2015.10.002

Kobayashi, D., Satsuma, S., Kamegaya, M., Haga, N., Shimomura, S., Fujii, T., & Yoshiya, S. (2005). Musculoskeletal conditions of acute leukemia and malignant lymphoma in children. *Journal of Pediatric Orthopedics. Part B*, *14*(3), 156–161. https://doi.org/10.1097/01202412-200505000-00003

Koder, J. (2020). On the Slavic immigration in the Byzantine Balkans. In J. Preiser-Kapeller, L. Reinfandt, & Y. Stouraitis (Eds.), *Migration histories of the Medieval Afroeurasian Transition Zone: Aspects of mobility between Africa, Asia and Europe, 300-1500 C.E.* (pp.81–100). BRILL. https://doi.org/10.1163/9789004425613

Lagia, A., Eliopoulos, C., & Manolis, S. (2007). Thalassemia: Macroscopic and radiological study of a case. *International Journal of Osteoarchaeology*, *17*(3), 269–285. https://doi.org/10.1002/oa.881

Larson, S. (2018). Seventh-century material culture in Boiotia. In A. C. Loney & S. Scully (Eds.), *The Oxford handbook of Hesiod* (pp.31–42). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780190209032.013.33

Levine, M. M., & Lepage, P. (2005). Prevention of typhoid fever. In A. J. Pollard & A. Finn (Eds.), *Hot Topics in Infection and Immunity in Children* (pp.161–173). Springer.

Lewis, M. E. (2002). Infant and Childhood Leprosy: Clinical and palaeopathological

implications. In C. Roberts, M. Lewis, & K. Manchester (Eds.), *The past and present of leprosy: Archaeological, historical, paleopathological, and clinical approaches.* (pp. 163–170). Archaeopress.

Lewis, M. E. (2004). Endocranial lesions in non-adult skeletons: Understanding their aetiology. *International Journal of Osteoarchaeology*, *14*(2), 82–97. https://doi.org/10.1002/oa.713

Lewis, M. E. (2009). *The bioarchaeology of children: Perspectives from biological and forensic anthropology*. Cambridge University Press.

Lewis, M. E. (2010). Life and death in a civitas capital: Metabolic disease and trauma in the children from late Roman Dorchester, Dorset. *American Journal of Physical Anthropology*, *142*(3), 405–416. https://doi.org/10.1002/ajpa.21239

Lewis, M. E. (2017). *Paleopathology of children: Identification of pathological conditions in the human skeletal remains of non-adults*. Elsevier Academic Press.

Lewis, M. E. (2018). Fetal paleopathology: An impossible discipline? In S. Han, T. K. Betsinger, & A. B. Scott (Eds.), *The anthropology of the fetus: Biology, culture, and society* (pp.112–131). Berghahn Books. https://doi.org/10.2307/j.ctvw04h7z

Lewis, M. E. (2019). Children in bioarchaeology: methods and interpretations. In A. Katzenberg & A. Grauer (Eds.), *Biological anthropology of the human skeleton* (3rd ed., pp.119–

144). John Wiley & Sons, Ltd.

Lewis, M. E., & Rutty, G. N. (2003). The endangered child: The personal identification of children in forensic anthropology. *Science & Justice*, *43*(4), 201–209. https://doi.org/10.1016/S1355-0306(03)71777-8

Lillehammer, G. (1989). A child is born: The child's world in an archaeological perspective. *Norwegian Archaeological Review*, *22*(2), 89–105. https://doi.org/10.1080/00293652.1989.9965496

Liston, M. (2017, April 27). *Plagues, pestilence, and other dreadful diseases in Byzantine Thebes.* [Video]. American School of Classical Studies at Athens, Athens, Greece. https://www.ascsa.edu.gr/news/newsDetails/videocast-plagues-and-pestilence-in-thebes

Liston, M. (2018). *Inferring the presence of a leprosarium or hospital from the pathologies in the cemetery: Interpreting burials from Byzantine Thebes.* [Conference presentation]. International Congress of Classical Archaeology 2018, Cologne/Bonn, Germany.

Liston, M. A., Rotroff, S. I., & Snyder, L. M. (2018). *The Agora bone well*. American School of Classical Studies at Athens.

Livingstone, E. (2014). *The concise Oxford dictionary of the Christian church*. Oxford University Press.

Magilton, J. (2008). *"Lepers outside the gate:" Excavations at the cemetery of the Hospital of St James and St Mary Magdalene, Chichester, 1986-87 and 1993* (Vol. 158). Council for British Archaeology Research Reports. https://doi.org/10.5284/1081722

Malamitsi-Puchner, A. (2017). Preterm birth in ancient Greece: A synopsis. *The Journal of Maternal-Fetal & Neonatal Medicine*, *30*(2), 141–143. https://doi.org/10.3109/14767058.2016.1165201

Mance, M. J. (2008). Keeping infants warm: Challenges of hypothermia. *Advances in Neonatal Care*, *8*(1), 6. https://doi.org/10.1097/01.ANC.0000311011.33461.a5

Maresh, M. M. (1970). Measurements from roentgenograms. In R. McCammon (Ed.), *Human growth and development* (pp.157–200). CC Thomas.

Marx, W. G. (1980). Luke, the physician, re-examined. *The Expository Times*, *91*(6), 168–172. https://doi.org/10.1177/001452468009100603

Mathur, V., Dhillon, J., & Kalra, G. (2012). Oral health in children with leukemia. *Indian Journal of Palliative Care*, *18*(1), 12. https://doi.org/10.4103/0973-1075.97343

Mays, S. (2018). The study of growth in skeletal populations. In S. Crawford, D. M. Hadley, &

G. Shepherd (Eds.), *The Oxford handbook of the archaeology of childhood* (pp.71–89). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780199670697.013.4

Mays, S. (2020). A dual process model for paleopathological diagnosis. *International Journal of Paleopathology*, *31*, 89–96. https://doi.org/10.1016/j.ijpp.2020.10.001

Miller, T. (2003). *The orphans of Byzantium: Child welfare in the Christian empire*. The Catholic University of America Press.

Miller, T., & Nesbitt, J. (2014). *Walking corpses: Leprosy in Byzantium and the medieval west*. Cornell University Press.

Molleson, T., & Cox, M. (1993). *The Spitalfields project volume 2—The anthropology—The middling sort, research report 86*. Council for British Archaeology.

Moore, J., & Koon, H. E. C. (2018). Response to González et al.'s comment upon "Basilar portion porosity: A pathological lesion possibly associated with infantile scurvy." *International Journal of Paleopathology*, *20*, 116. https://doi.org/10.1016/j.ijpp.2017.09.004

Morrone, A. (2020). Giving a voice to the little ones: The bioarchaeology of children in the Baltics. *Archaeologia Lituana*, *21*(0), 97–116. https://doi.org/10.15388/ArchLit.2019.21.6

Morrone, A., Tõrv, M., Piombino-Mascali, D., Malve, M., Valk, H., & Oras, E. (2021). Hunger, disease, and subtle lesions: Insights into systemic metabolic disease in fetal and perinatal remains from 13th- to 15th-century Tartu, Estonia. *International Journal of Osteoarchaeology*, *31*(4), 534–555. https://doi.org/10.1002/oa.2970

Nathan, G. (2020). Looking for children in late antiquity. In L. A. Beaumont, M. Dillon, & N. Harrington (Eds.), *Children in antiquity* (pp.134–149). Routledge. https://doi.org/10.4324/9781315542812-11

Ortner, D. J. (2003). *Identification of pathological conditions in human skeletal remains* (2nd ed.). Academic Press.

Ortner, D. J. (2012). Differential diagnosis and issues in disease classification. In A.L. Grauer (Ed.), *A companion to paleopathology* (pp.250-266). John Wiley & Sons, Ltd.

Papagrigorakis, M. J., Yapijakis, C., Synodinos, P. N., & Baziotopoulou-Valavani, E. (2006). DNA examination of ancient dental pulp incriminates typhoid fever as a probable cause of the Plague of Athens. *International Journal of Infectious Diseases*, *10*(3), 206–214. https://doi.org/10.1016/j.ijid.2005.09.001

Perin, J., Mulick, A., Yeung, D., Villavicencio, F., Lopez, G., Strong, K. L., Prieto-Merino, D.,

Cousens, S., Black, R. E., & Liu, L. (2022). Global, regional, and national causes of under-5 mortality in 2000–19: An updated systematic analysis with implications for the Sustainable Development Goals. *The Lancet Child & Adolescent Health*, *6*(2), 106–115. https://doi.org/10.1016/S2352-4642(21)00311-4

Perry, M. A. (2006). Redefining childhood through bioarchaeology: Toward an archaeological and biological understanding of children in antiquity. *Archaeological Papers of the American Anthropological Association*, *15*(1), 89–111. https://doi.org/10.1525/ap3a.2005.15.89

Pitsios, T., & Zafiri, V. (2012). Frequency and distribution of enamel hypoplasia in ancient skulls from different eras and areas in Greece. *International Journal of Caring Sciences*, *5*(2).

Preterm birth. (2023b). World Health Organization. https://www.who.int/news-room/fact-sheets/detail/preterm-birth

Prowse, T. L. (2011). Diet and dental health through the life course in Roman Italy. In *Social bioarchaeology* (pp.410–437). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781444390537.ch15

Ripy, H. W. (1950). Typhoid fever in an infant complicated by typhoid meningitis. *The Journal of Pediatrics*, *36*(3), 376–380. https://doi.org/10.1016/S0022-3476(50)80109-9

Roberts, C. (2011). The Bioarchaeology of Leprosy and Tuberculosis. In S. C. Agarwal & B. A. Glencross (Eds.), *Social bioarchaeology* (pp.252–281). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781444390537.ch9

Roberts, C., & Brickley, M. (2019). Infectious and metabolic diseases: A synergistic relationship. In M. A. Kratzenberg & A. L. Grauer (Eds.), *Biological anthropology of the human skeleton* (3rd ed., pp.415–446). Wiley-Blackwell.

Roberts, C., & Manchester, K. (2007). *The archaeology of disease* (3rd ed.). Cornell University Press.

Rogalsky, R. J., Black, G. B., & Reed, M. H. (1986). Orthopaedic manifestations of leukemia in children. *The Journal of Bone and Joint Surgery*, *68-A*(4), 494–501.

Roksandic, M. (2003). New standardized visual forms for recording the presence of human skeletal elements in archaeological and forensic contexts. *Internet Archaeology, 13*. https://doi.org/10.11141/ia.13.3

Roksandic, M., & Armstrong, S. D. (2011). Using the life history model to set the stage(s) of growth and senescence in bioarchaeology and paleodemography. *American Journal of Physical Anthropology*, *145*(3), 337–347. https://doi.org/10.1002/ajpa.21508

Santacroce, L., Del Prete, R., Charitos, I. A., & Bottalico, L. (2021). Mycobacterium leprae: A historical study on the origins of leprosy and its social stigma. *Le Infezioni in Medicina*, *29*(4), 623–632. https://doi.org/10.53854/liim-2904-18

Sarris, P. (2002). The Justinianic plague: Origins and effects. *Continuity and Change*, *17*(2), 169–182. https://doi.org/10.1017/S0268416002004137

Scahill, D. (2018). Thebes excavations. [Map]. American School of Classical at Athens, Athens, Greece.

Schaefer, M., Scheuer, L., & Black, S. M. (2009). *Juvenile osteology: A laboratory and field manual*. Elsevier/Academic Press.

Scheper-Hughes, N. (2009). Making anthropology public. *Anthropology Today*, *25*(4), 2–3.

Scheuer, L., & MacLaughlin-Black, S. (1994). Age estimation from the pars basilaris of the fetal and juvenile occipital bone. *International Journal of Osteoarchaeology*, *4*(4), 377–380. https://doi.org/10.1002/oa.1390040412

Sinigaglia, R., Gigante, C., Bisinella, G., Varotto, S., Zanesco, L., & Turra, S. (2008). Musculoskeletal manifestations in pediatric acute leukemia. *Journal of Pediatric Orthopaedics*, *28*(1), 20–28. https://doi.org/10.1097/BPO.0b13e31815ff350

Snoddy, A. M. E., Buckley, H. R., Elliott, G. E., Standen, V. G., Arriaza, B. T., & Halcrow, S. E. (2018). Macroscopic features of scurvy in human skeletal remains: A literature synthesis and diagnostic guide. *American Journal of Physical Anthropology*, *167*(4), 876–895. https://doi.org/10.1002/ajpa.23699

Sofaer Derevenski, J. (1997). Engendering children, engendering archaeology. In J. Moore & E. Scott (Eds.), *Invisible people and processes: Writing gender and childhood into European archaeology* (pp.192-202). Leicester University Press.

Spekker, O., Tihanyi, B., Kis, L., Váradi, O. A., Donoghue, H. D., Minnikin, D. E., Szalontai, C., Vida, T., Pálfi, G., Marcsik, A., & Molnár, E. (2022). The two extremes of Hansen's disease—Different manifestations of leprosy and their biological consequences in an Avar Age (late 7th century CE) osteoarchaeological series of the Duna-Tisza Interfluve (Kiskundorozsma–Daruhalom-dűlő II, Hungary). *PLoS ONE*, *17*(6), e0265416. https://doi.org/10.1371/journal.pone.0265416

Stark, R. J. (2014). A proposed framework for the study of paleopathological cases of subadult scurvy. *International Journal of Paleopathology*, *5*, 18–26. https://doi.org/10.1016/j.ijpp.2014.01.005

Stathakopoulos, D. C. (2004). *Famine and pestilence in the late Roman and early Byzantine empire: A systematic survey of subsistence crises and epidemics*. Routledge.

Steyn, M., Brits, D., Botha, D., & Holland, S. (2024). Violence against children: A review of cases at a forensic anthropology unit, Johannesburg, South Africa. *Journal of Forensic and Legal Medicine*, *101*, 102623. https://doi.org/10.1016/j.jflm.2023.102623

Stodder, A. L. W. (2019). Taphonomy and the nature of archaeological assemblages. In M. A. Katzenberg & A. L. Grauer (Eds.), *Biological anthropology of the human skeleton* (3rd. ed., pp.73–115). John Wiley & Sons, Ltd.

Stojanowski, C. M., & Duncan, W. N. (2015). Engaging bodies in the public imagination: Bioarchaeology as social science, science, and humanities. *American Journal of Human Biology*, *27*(1), 51–60. https://doi.org/10.1002/ajhb.22522

Towle, I., & Irish, J. D. (2020). Recording and interpreting enamel hypoplasia in samples from archaeological and palaeoanthropological contexts. *Journal of Archaeological Science*, *114*, 105077. https://doi.org/10.1016/j.jas.2020.105077

Tritsaroli, P. (2017). Life and death at early Byzantine Akraiphnio, Greece: A biocultural approach. *Anthropologie*, *55*(3), 243–264.

Tritsaroli, P., & Valentin, F. (2008). Byzantine burials pratiques for children: Case studies based on a bioarchaeological approach to cemeteries from Greece. In F. Jener, S. Muriel, & C. Puyoles (Eds.), *Nasciturus, infans, puerulus vobis mater terra: La muerte en la infancia* (pp.93–116). Servei d'investigacions arqueologiques i Prehistoriques.

Trueta, J. (1959). The three types of acute haematogenous osteomyelitis: A clinical and vascular study. *The Journal of Bone and Joint Surgery*, *41*(4), 671–680.

Typhoid. (2023c). World Health Organization. https://www.who.int/news-room/fact-sheets/detail/typhoid

Vuolanto, V. (2020). Daily life of children in late antiquity. In L. A. Beaumont, M. Dillon, & N. Harrington (Eds.), *Children in antiquity* (pp.268–280). Routledge. https://doi.org/10.4324/9781315542812-22

Walker, P. L., Bathurst, R. R., Richman, R., Gjerdrum, T., & Andrushko, V. A. (2009). The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis. *American Journal of Physical Anthropology*, *139*(2), 109–125. https://doi.org/10.1002/ajpa.21031

Williams, H. J., Davies, A. M., & Chapman, S. (2004). Bone within a bone. *Clinical Radiology*, *59*(2), 132–144. https://doi.org/10.1016/S0009-9260(03)00337-4

Zedda, N., Bramanti, B., Gualdi-Russo, E., Ceraico, E., & Rinaldo, N. (2021). The biological index of frailty: A new index for the assessment of frailty in human skeletal remains. *American Journal of Physical Anthropology*, *176*(3), 459–473. https://doi.org/10.1002/ajpa.24394

Ziegler, T. A. (2018). The hospital in history, c. 3500 BCE–c. 500 CE. In T. A. Ziegler, *Medieval healthcare and the rise of charitable institutions* (pp.19–47). Springer International Publishing. https://doi.org/10.1007/978-3-030-02056-9_2

Appendix 1

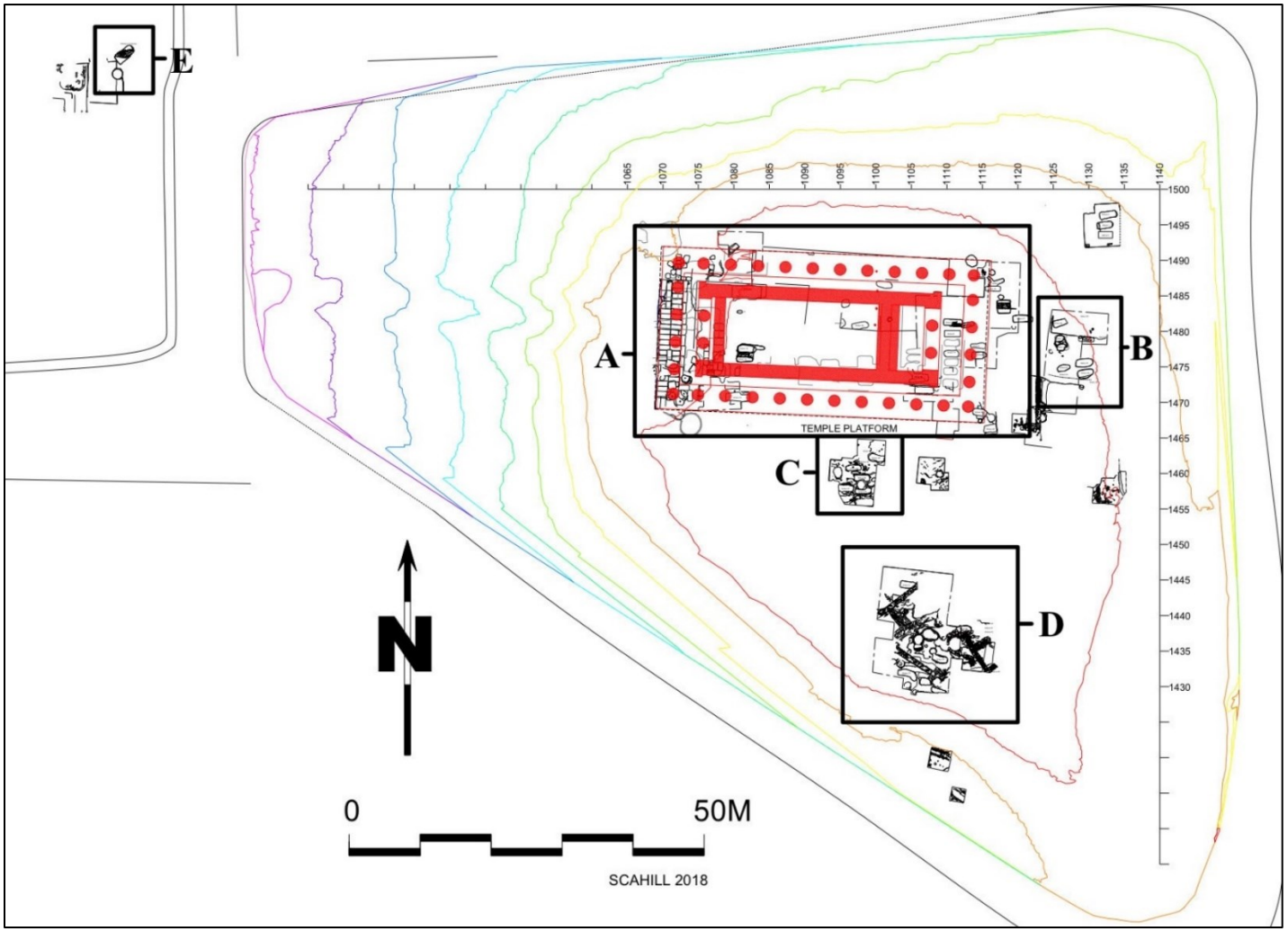


Figure 17: *Topographic map of Ismenion Hill. Note sections A-E. Drawing by D. Scahill (2018).*

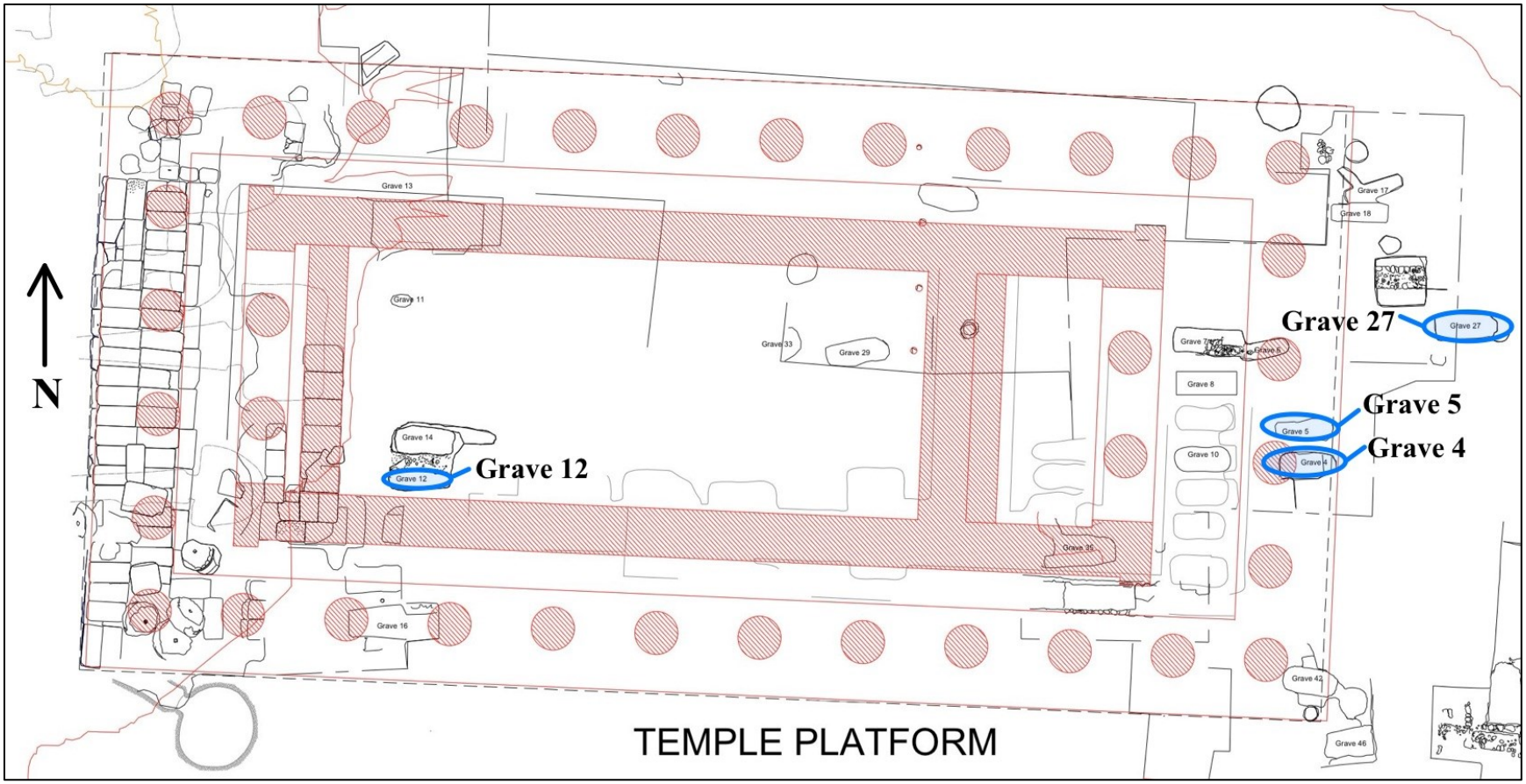
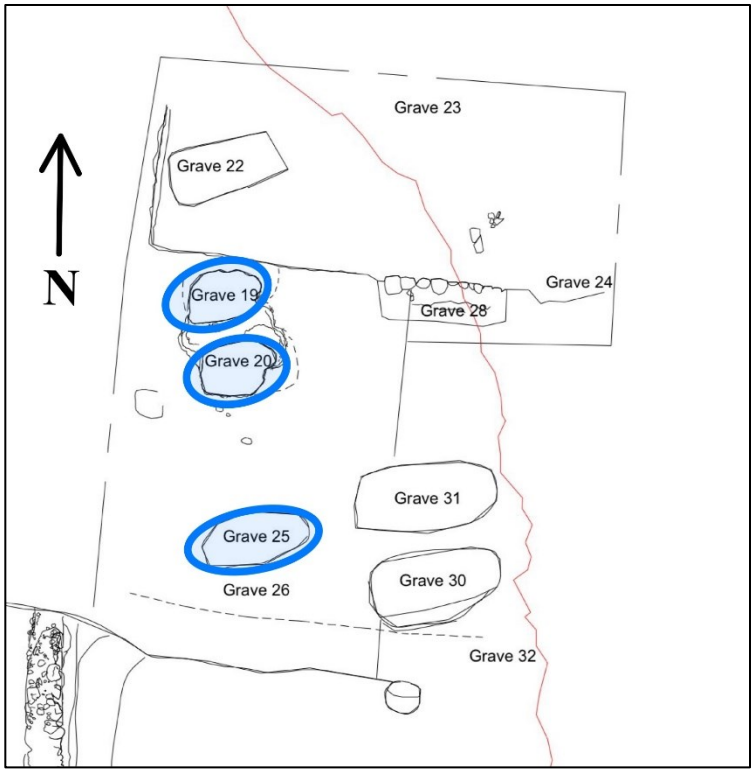


Figure 18: *Close up of section "A" topographic map in Figure 17, graves studied in this thesis highlighted in blue, not to scale.*



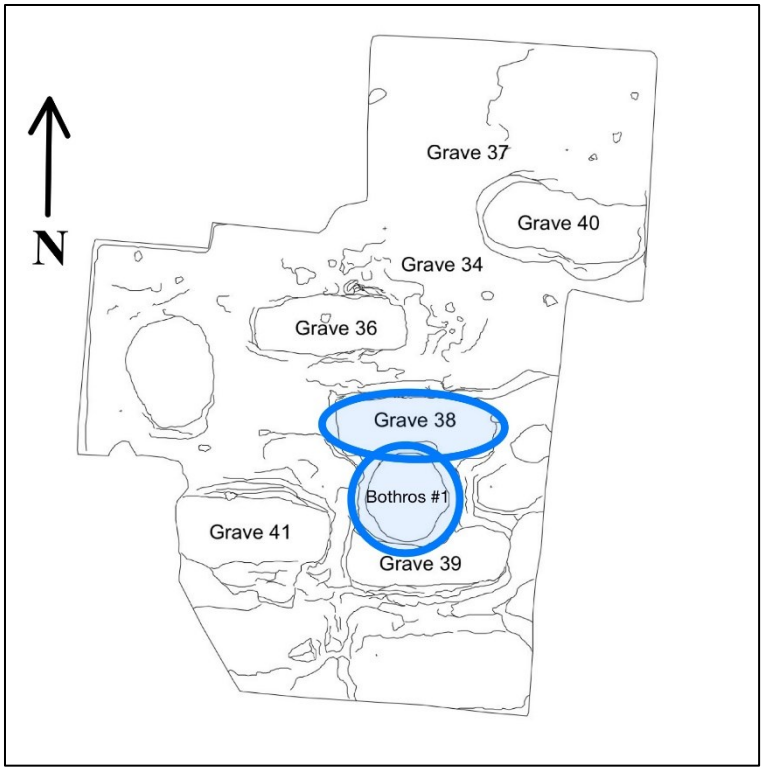
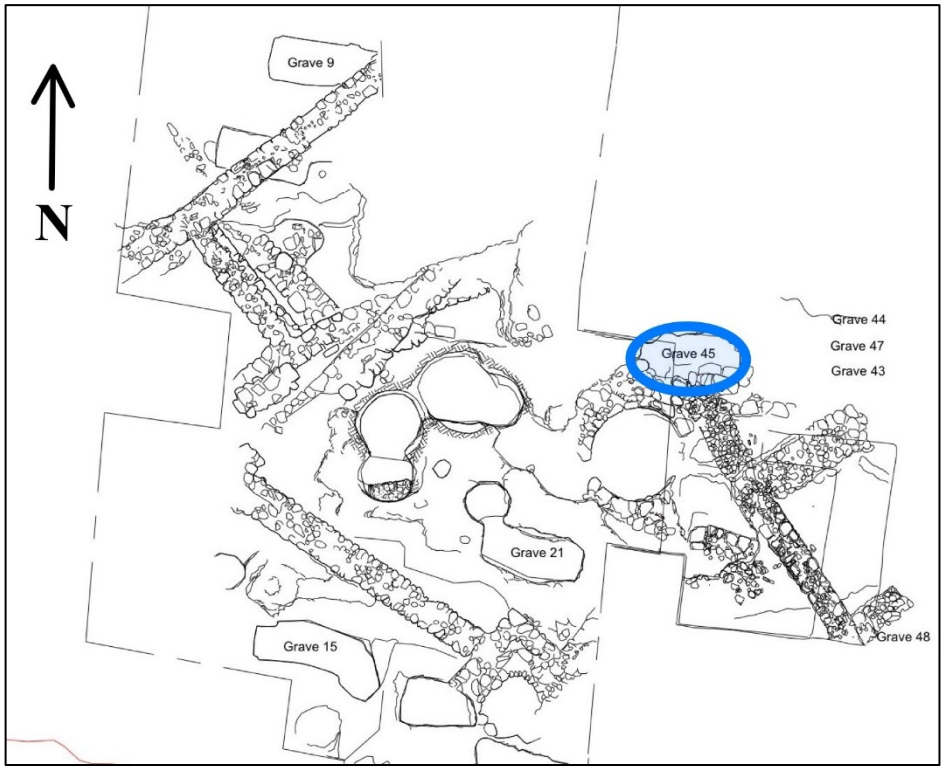


Figure 19: *Close up of section "B" from Figure 17, graves studied in blue, not to scale (left).*

Figure 20: *Close up of section "C" from Figure 17, graves studied in blue, not to scale (right).*



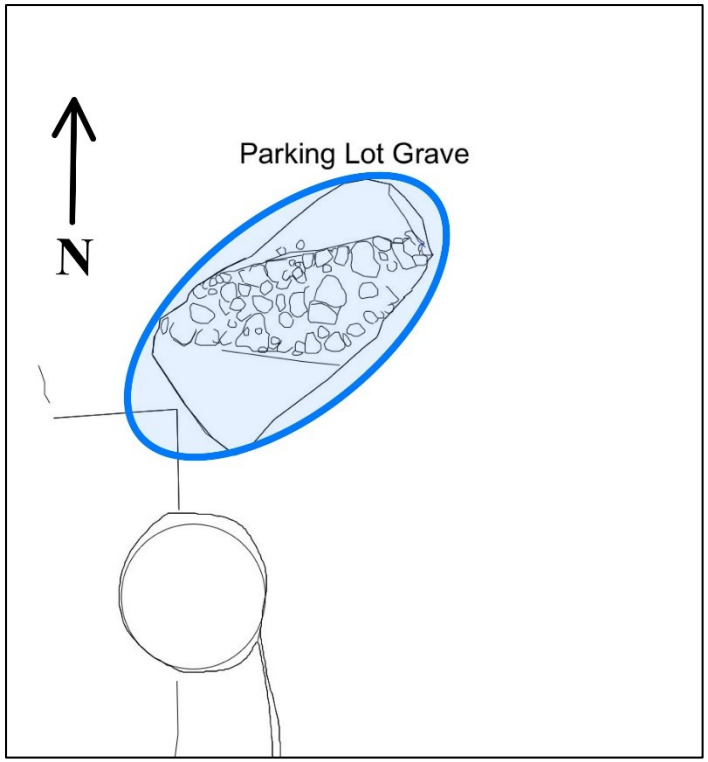


Figure 21: *Close up of section "D" from Figure 17, graves studied in blue, not to scale (left).*

Figure 22: *Close up of section "E" from Figure 17, graves studied in blue, not to scale (right).*

Appendix 2

Table 3: *Summary table of age-at-death estimations for the juveniles at Ismenion Hill, (n=59).*

Grave #	Skeletal #	Age-at-death estimation	Methods
4	1	Dental: 9.5-13.5 ± 0.5 yrs. Skeletal: 5-7 yrs.	**Dental eruption and formation:** 9.5-13.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** sacral vertebral bodies unfused <12 yrs. (Schafer *et al*., 2009) **Femur maximum diaphyseal length:** left element 277 mm, ~5-6.5 yrs. (Maresh, 1970) **Radius maximum diaphyseal length:** left element 138 mm, ~6-7 yrs. (Maresh, 1970)
4	2	Dental: 2.5-3.5 ± 0.5 yrs. Skeletal: 0.5-2 yrs.	**Dental eruption and formation:** 2.5-3.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** some thoracic neural arches fused ~1-2 yrs. (Schaefer *et al*., 2009) **Ulna maximum diaphyseal length:** left element 79 mm, ~0.5 yrs. (Maresh, 1970) ***Pars lateralis* measurements:** right element maximum length 35.3 mm, maximum width 25.8 mm, > 40 wks. (Fazekas & Kosa, 1978)
4	3	Skeletal: 34-38 gestational wks.	***Pars petrosa* measurements:** left element length 36.0 mm, width 15.0 mm, ~34-38 wks. minimum (eroded) (Fazekas & Kosa, 1978)
4	4	Skeletal: ~5 yrs.	**Comparative collection:** proximal femur fragment morphology and size similar to ASCSA cast of a "normal" growth 5-year-old (dental age)
5	5	Dental: 11.5 ± 0.5 yrs. Skeletal: 11-16 yrs.	**Dental eruption and formation:** 11.5 ±0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** partial union proximal epiphysis of distal pedal phalange digit 1, ~11-16 yrs. (Schaefer *et al.*, 2009)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
5	6	Dental: 1.5 ± 3 mo. Skeletal <1 yr.	**Dental eruption and formation:** 1.5 ± 3 mo. (AlQahtani *et al.,* 2010) **Fusion:** neural arches of vertebrae not fused to centrum <1 yr., mandible not fused < 1 yr. (Schaefer *et al.,* 2009) **Humerus maximum diaphysis length:** right element 71.7 mm, ~1.5 mo. (Maresh, 1970)
5	7	Skeletal: ≥26-30 gestational wks.	***Pars petrosa* measurements:** right length 20.2 mm, left length 20.7 mm, ~26-30 wks. minimum estimate (eroded) (Fazekas & Kosa, 1978)
5	8	Skeletal: ≥28-30 gestational wks.	***Pars petrosa* measurements:** right length 22.6 mm, left length 22.1 mm, ~28-30 wks. minimum estimate (eroded) (Fazekas & Kosa, 1978)
5	9	Skeletal: ≥26-30 gestational wks.	***Pars petrosa* measurements:** left length 21.7 mm, ~26-30 wks. minimum estimate (eroded) (Fazekas & Kosa, 1978)
5	10	Skeletal: ≥28-30 gestational wks.	***Pars petrosa* measurements:** left length 22.7 mm, ~28-30 wks. minimum estimate (eroded) (Fazekas & Kosa, 1978)
5	11	Dental: 4.5-5.5 ± 0.5 yrs. Skeletal: 5-7 yrs.	**Dental eruption and formation:** 4.5-5.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** union of cervical vertebrae and neural arches: >2-4 yrs. (Schaefer *et al*., 2009) **Maximum clavicle length**: right element 80.6 mm ~5-7 yrs. (Black & Scheuer, 1996)
12	12	Skeletal: 34-40 gestational wks.	***Pars petrosa* morphology:** semi-circular canal closed, likely > 7 gestational mo. (Schaefer *et al*., 2009) ***Pars petrosa* measurements:** right element maximum length 28.3 mm, maximum width 15.2 mm, ~34 wks. (Fazekas & Kosa, 1978) **Tibia maximum diaphyseal length:** right element 61 mm, ~38-40 gestational wks. minimum estimate (erosion) (Fazekas & Kosa, 1978)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
12	13	Dental: 1.5-3.5 ± 0.5 yrs. Skeletal: 1-2 yrs.	**Dental eruption and formation:** 1.5-3.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** some fused thoracic vertebral neural arches ~1-2 years (Schaefer *et al*., 2009) **Tibia maximum diaphyseal length:** right element 118 mm, 1.5 yrs. (Maresh, 1970) **Femur maximum diaphyseal length:** right element 128 mm, 6 mo. - 1 yr. (Maresh, 1970)
19	14	Dental: 2.5-3.5 ± 0.5 yrs. Skeletal: 1-2 yrs.	**Dental eruption and formation:** 2.5-3.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Maximum diaphyseal length humerus:** right element 114 mm, 1.5 yrs. (Maresh, 1970) **Maximum diaphyseal length tibia:** right element 115 mm, 1 yr. (Maresh, 1970)
19	15	Dental: 34 gestational wks. - 1.5 mo. ± 3 mo. postnatal Skeletal: 36-40 gestational wks.	**Dental eruption and formation:** 34 wks. - 1.5 ± 3 mo. (AlQahtani *et al.,* 2010) **Fusion:** mental symphysis unfused <1 yr. (Schaefer *et al.,* 2009) ***Pars petrosa* measurements:** left element, maximum length 36.6 mm, maximum width 14.6 mm (erosion), ~34-40 gestational wks. (Fazekas & Kosa, 1978) **Femoral measurements:** right element maximum diaphyseal length 71.9 mm, distal width 15.8 mm, 36-40 gestational wks. (Fazekas & Kosa, 1978) **Humeral measurements:** left element maximum diaphyseal length 61.4 mm, distal width 14.2 mm, 36-40 wks. (Fazekas & Kosa, 1978)
19	16	Dental: 2.5-6.5 ± 0.5 yrs.	**Dental eruption and formation:** 2.5-6.5 ± 0.5 yrs., loose dentition associated with juvenile (possibly commingled) (AlQahtani *et al.,* 2010)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
20	17	Dental: 5.5-7.5 ± 0.5 yrs. Skeletal: 4-7 yrs.	**Dental eruption and formation:** 5.5-7.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** partial union of left pubis and ischium ~5-11 yrs. (Schaefer *et al*., 2009) **Maximum length clavicle:** right element 85 mm, 5-7 yrs. (Black & Scheuer, 1996) **Maximum diaphyseal length tibia:** right element 193 mm, 4-5 yrs. (Maresh, 1970) **Maximum diaphyseal length radius:** right element 133 mm, 4.5-6 yrs. (Maresh, 1970) **Maximum diaphyseal length ulna:** right element 146 mm, 4.5-6 yrs. (Maresh, 1970)
20	18	Skeletal: 2-4 yrs.	**Fusion:** Cervical vertebrae 2 arches fused together >3 yrs., but not fused to body/dens <4 yrs. (Schaefer *et al*., 2009) **Femur maximum diaphyseal length:** left element 166 mm, 2 yrs. (Maresh, 1970) **Tibia maximum diaphyseal length*:*** left element 137 mm, 2 yrs. (Maresh, 1970)
20	19	Skeletal: 1-3 yrs.	**Clavicle maximum length:** left element 64 mm, 1-3 yrs. (Black & Scheuer, 1996) **Tibia maximum diaphyseal length:** left element 119 mm, 1.5 yrs. (Maresh, 1970) **Radius maximum diaphyseal length:** left element 90 mm, 1.5-2 yrs. (Maresh, 1970)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
20	20	Skeletal: 34 gestational wks. - 6 mo. postnatal	**Fusion:** mandibular symphysis unfused <1 yr. (Schaefer *et al*., 2009) ***Pars petrosa* measurement:** left element maximum length 36.6 mm, maximum width 16.3 mm, 34-40 gestational wks. minimum (erosion) (Fazekas & Kosa, 1978) **Clavicle maximum length:** left element 46 mm, 0-6 mo. (Black & Scheuer, 1996)
20	21	Skeletal: 1-2 yrs.	**Fusion:** some fusion of thoracic vertebral neural arches ~1-2 yrs. (Schaefer *et al.*, 2009)
20	22	Skeletal: ~1 yr.	**Comparative collection:** elements slightly greater in size than ASCSA comparative material of an infant 9 mo. - 1 yr. (dental age)
25	23	Skeletal: 1.5 yrs.	**Tibia maximum diaphyseal length:** right element 127 mm, 1.5 yrs. (Maresh, 1970) **Fibula maximum diaphyseal length:** 126 mm, 1.5 yrs. (Maresh, 1970)
25	24	Dental: 10.5 ± 3 mo. - 1.5 ± 0.5 yrs. Skeletal: 6 mo. -1 yr.	**Dental eruption and formation:** 10.5 ± 3 mo. - 1.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Femur maximum diaphyseal length:** left element 113 mm, 6 mo. (Maresh 1970) **Humerus maximum diaphyseal length:** 90 mm, 6 mo. (Maresh, 1970) **Clavicle maximum length:** right element 55 mm, 7-12 mo. (Black & Scheuer, 1996)
25	25	Skeletal: 2-2.5 yrs.	**Maximum fibula diaphysis length:** un-sided element 143 mm, 2-2.5 yrs. (Maresh, 1970)
25	26	Skeletal: 1-1.5 yrs.	**Maximum tibia diaphyseal length:** left element 117 mm, 1-1.5 yrs. (Maresh, 1970)
25	27	Skeletal: 40 wks. - 2 yrs.	**Fusion:** some fusion of thoracic vertebral neural arches ~1-2 yrs. (Schaefer *et al.*, 2009) **Rib one length:** right element 33.7 mm, >40 gestational wks. (Fazekas & Kosa, 1978)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
27	28	Dental: 1.5 ± 0.5 yrs. Skeletal: 1-3 yrs.	**Dental eruption and formation:** 1.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Humerus maximum diaphyseal length:** right element 98 mm, 6 mo. -1 yr. (Maresh, 1970) **Ilium measurements:** left element maximum length 68.6 mm, >3 yrs., maximum width 57.8 mm, 19 mo. - 3 yrs. (Molleson & Cox, 1993)
27	29	Skeletal: ~5 yrs.	**Comparative collection:** fragmentary elements similar in size and morphology to "normal" 5-year-old ASCSA cast (dental age)
27	30	Skeletal ~5 yrs.	**Comparative collection:** fragmentary elements slightly larger and more robust than "normal" 5-year-old ASCSA cast (dental age)
27	31	Dental: ~7.5-8.5 ± 0.5 yrs. Skeletal: <11-16 yrs.	**Dental eruption and formation:** loose but possibly associated, 7.5-8.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** head of metatarsal 3 unfused <11-16 yrs. (Schaefer *et al.,* 2009)
27	32	Dental: ~4.5 ± 3 mo. Skeletal: 3-6 mo.	**Dental eruption and formation**: loose but possibly associated 4.5 ± 3 mo. postnatal (AlQahtani *et al.,* 2010) **Radius maximum diaphyseal length:** right element 66 mm, 3-6 mo. (Maresh, 1970)
27	33	Skeletal: 36-38 gestational wks.	**Clavicle maximum length:** right element 38.8 mm, 36-38 gestational wks. (Fazekas & Kosa 1978) **Distal width humerus:** right element 14.5 mm, 36 gestational wks. (Fazekas & Kosa 1978)
38	34	Skeletal: 10-11 yrs.	**Femur maximum diaphyseal length:** right element 360 mm, 10-11 yrs. (Maresh, 1970)
38	35	Skeletal: ~5 yrs.	**Comparative collection:** tibia and fibula fragments are similar in size and morphology to ASCSA "normal" growth 5-year-old cast (dental age)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
38	36	Dental: 1.5-2.5 ± 0.5 yrs. Skeletal: < 2 yrs.	**Dental eruption and formation:** loose but possibly associated, 1.5-2.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** vertebral neural arches not fused <2 yrs. (Schaefer *et al*., 2009)
38	37	Skeletal: 24-32 gestational wks.	***Pars petrosa* measurements:** left element maximum length 19.5 mm, maximum width 13.2 mm, right element maximum length 20.0 mm, maximum width 13.4 mm, 24-32 gestational wks. (Fazekas & Kosa, 1978)
45	38	Dental: >10.5 ± 0.5 yrs. Skeletal: 16-20 yrs.	**Dental eruption:** >10.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** Coracoid fused >16 yrs., iliac crest partially fused 14-20 yrs. (Schaefer *et al*., 2009)
45	39	Skeletal: 11-16 yrs.	**Fusion:** distal pedal proximal epiphyses of phalanges fused >11 yrs., metatarsal heads 3-5 open < 16 yrs. (Schaefer *et al*., 2009)
45	40	Skeletal: <11-16 yrs.	**Fusion:** metatarsal head 4 unfused <16 yrs. (Schaefer *et al*., 2009), but smaller in size than skeleton 39, no other complete elements, cautious estimate
45	41	Dental: 5.5-6.5 ± 0.5 yrs.	**Dental eruption and formation:** 5.5-6.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010)
45	42	Dental: 38 gestational wks. - 1.5 ± 3 mo. postnatal Skeletal: 30-40 gestational wks.	**Dental eruption and formation:** 38 gestational wks. - 1.5 ± 3 mo. postnatal (AlQahtani *et al.,* 2010) ***Pars petrosa* measurements:** left element maximum length 31.2 mm, maximum width 14.6 mm, right element maximum length 31.0 mm, maximum width 14.8 mm, 30-36 gestational wks. (Fazekas & Kosa, 1978) **Tiba maximum diaphyseal length:** left element 67 mm, 40 gestational wks. (Fazekas & Kosa, 1978)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
45	43	Skeletal: 30-34 gestational wks.	***Pars petrosa* measurements:** left element maximum length 23.5 mm, maximum width 13.6 mm, right element length 22.8 mm, maximum width 13.5 mm, 30-34 gestational wks. (Fazekas & Kosa, 1978)
45	44	Dental: < 1 yr. skeletal: ~6 mo.	**Dental eruption:** <1 yr. (AlQahtani *et al.,* 2010) **Maximum tibial diaphyseal length:** left element 94 mm, 6 mo. (Maresh, 1970) **Maximum fibula diaphyseal length:** un-sided element 91 mm, 6 mo. (Maresh, 1970)
Bothros #1	45	Skeletal: ~5 yrs.	**Comparative collection:** tibial fragment similar in size and morphology to ASCSA "normal" growth 5-year-old cast (dental age)
Parking Lot Grave	46	Dental: 7.5-10.5 ± 3 mo. Skeletal: 5 mo. - 1yr 4 mo.	**Dental eruption and formation**: 7.5-10.5 ± 3 mo. (AlQahtani *et al.,* 2010) **Left ischium measurement:** maximum length 27.2 mm, maximum width 23.3 mm, > 40 gestational weeks (Fazekas & Kosa, 1978) ***Pars basilaris* measurement:** maximum width 17.8 mm, sagittal length 14.7 mm, width greater than length suggests > 5 mo., <1 yr. 4 mo. postnatal (Scheuer & MacLaughlin-Black, 1994)
Parking Lot Grave	47	Skeletal: < 1 yr.	**Metatarsal one maximum length**: 20.9 mm, > 40 wks. (Fazekas & Kosa, 1978) **Comparative collection:** left clavicle fragment similar in size and morphology to ASCSA 9 mo. – 1 yr. comparative material (dental age)
Parking Lot Grave	48	Skeletal: <1 yr.	**Distal width right femur**: 26.0 mm, > 40 wks. (Fazekas & Kosa, 1978) **Comparative collection:** femur similar in size and morphology to ASCSA 9 mo. - 1 yr. comparative material (dental age)
Parking Lot Grave	49	Skeletal: ~3-4 yrs.	**Clavicle maximum length:** left element 69.4 mm, 3-4 yrs. (Black & Scheuer, 1996)

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
Parking Lot Grave	50	Skeletal: ~1-5 yrs.	**Comparative collection:** elements greater in size than ASCSA comparative material of an infant 9 mo. - 1 yr. (dental age), but smaller than 5-year-old cast (dental age)
Parking Lot Grave	51	Dental: ~13.5 ± 0.5 yrs. Skeletal: 13-18 yrs.	**Dental formation:** dentition loose but if associated ~13.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** proximal humerus recently fused, >13 - 16 yrs., distal fibular epiphysis unfused < 18 yrs. (Schafer *et al*., 2009)
Parking Lot Grave	52	Skeletal: >1 yr.	**Comparative collection:** elements slightly greater in size than ASCSA comparative material of an infant 9 mo. - 1 yr. (dental age)
Parking Lot Grave	53	Skeletal: ~5 yrs.	**Comparative collection:** elements slightly greater in size than ASCSA comparative material of a 5-year-old "normal" growth cast (dental age)
Parking Lot Grave	54	Dental: 10.5-11.5 ± 0.5 yrs. Skeletal: 10-12 yrs.	**Dental eruption and formation:** 10.5-11.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** complete union of thoracic and cervical vertebrae >5 yrs., open metacarpal and metatarsal heads < 16 yrs. (Schaefer *et al.*, 2009) **Humerus maximum length:** left element 250.0 mm, 10-12 yrs. (Maresh, 1970)
Parking Lot Grave	55	Dental: 5.5-6.5 ± 0.5 yrs. Skeletal: 6-14 yrs.	**Dental eruption and formation:** 5.5-6.5 ± 0.5 yrs. (AlQahtani *et al.,* 2010) **Fusion:** posterior and anterior arch of cervical vertebrae 1 fused, > 6 yrs., metatarsal heads 2-5 unfused < 14 yrs. (Schaefer *et al.,* 2009)
Parking Lot Grave	56	Skeletal: fetal	***Pars petrosa* morphology**: heavily eroded, unable to assess

Table 3: *Summary table of age-at-death estimations*			
Grave #	**Skeletal #**	**Age-at-death estimation**	**Methods**
Parking Lot Grave	57	Skeletal: ~28-31 gestational wks.	***Pars petrosa* morphology:** semi-circular canal appears complete, ca. ~7 mo. (Schaefer *et al.*, 2009)
Parking Lot Grave	58	Skeletal: ~28-40 gestational wks.	***Pars petrosa* morphology:** some development/expansion of mastoid region, ca. ~7-9 mo. (Schaefer *et al.*, 2009)
Parking Lot Grave	59	Skeletal: ~28-31 gestational wks.	***Pars petrosa* morphology:** semi-circular canal appears complete, ca. ~7 mo. (Schaefer *et al*., 2009)

Appendix 3

Grave 4, Skeleton 1

Table 4: *Differential diagnosis of Grave 4, Skeleton 1.*

Grave 4	Skeleton 1	Leprosy	Rhinomaxillary syndrome	Tuberculosis	Brucellosis	Scurvy	Leukemia
Antemortem tooth loss (premolar/molar)	+					+*	+*
Fine diffuse porosity of the maxillary body	+	+	+			+	+
Fine porosity on palatine process of maxilla	+	+	+			+	+
Porosity superior aspect greater wing of sphenoid	+					+	+
Fine porosity ectocranial surface of *pars lateralis*	+					+	+
Pitting and increased vascularity supraspinous fossa	+					+	+
Fine porosity adjacent to long bone metaphysis	+					+	+
Radiographic translucent lines at metaphysis of long bones	+					+	+
Porosity and large pitting of vertebral bodies	+			+	+		+
Compression of the vertebral bodies	+			+	+		+
Porosity of vertebral neural arches	+						+
Fine porosity on metatarsals and pedal phalanges	+	+				+	+
Fine pitting cortex of tarsals	+	+					+

*Known comorbidities (Cammarata-Scalisi *et al.,* 2020; Fain, 2005).

Scurvy

A diagnosis of scurvy is typically made through significant periosteal new bone growth and increased porosity or osteopenia (Lewis, 2017; Snoddy *et al*., 2018). Vitamin C deficiency can result in antemortem tooth loss, due to weakening of the periodontal ligaments (Stark, 2014). It takes approximately 6 mo. of vitamin C deficiency for dental evidence to appear (Fain, 2005). Osteological evidence of vitamin C deficiency includes osteoporosis impacting the trabecular and cortex bone, bone cortex thinning, and periosteal new bone reactions (Fain, 2005). Porous foci surrounding long bone metaphyses are common due to chronic bleeding associated with scurvy, however, new bone formation is expected to begin once ascorbic acid is reintroduced (Stark, 2014). Often new subperiosteal bone growth is considered in the differential diagnosis of scurvy; however, the production of periosteal new bone growth indicates healing scurvy rather than active deficiency (Stark, 2014). Unlike this juvenile, scurvy does not typically present with vertebral porosity or compression of the vertebral bodies (Brickley & Ives, 2008).

Rickets

Vitamin D deficiency was considered. In older children and teens, rickets has been reported to be associated with genetic and kidney-related disorders. Cortical thinning is a common symptom of rickets, as observed in this juvenile. However, more diagnostic symptoms such as widening/fraying of metaphyses, anterior bowing of the tibia, and "plump bones", were not observed but would be expected in an older juvenile with chronic rickets (Lewis, 2017).

Anemia

Anemias often present with cranial porosity, as seen in this juvenile (Ortner, 2003). However, postcranial involvement is most common in genetic anemias, such as thalassemia (Ortner, 2003). Typically, thalassemia presents as osteopenia of the hands or feet, pitting in the cranial and flat bones, and lytic destruction of the spine and thorax. Like a case of thalassemia, porosity was observed on the occipital, and there was osteopenia of the vertebrae. However, to cause such significant pitting the thalassemia would have to be chronic, and without blood transfusions, it is unlikely this juvenile would have made it to this age in antiquity (Lagia, 2007; Ortner, 2003).

Tuberculosis

Tuberculosis is an infectious pathology that commonly presents as porous lytic foci on the vertebrae (Lewis, 2017). New bone formation on long bones is also expected. There can be involvement of facial bones, but typical cranial involvement is "punched out" in appearance and is focused on the occipital, unlike the fine porosity observed here (Lewis, 2017; Ortner, 2003). Childhood tuberculosis usually spares the neural arches and does not cause porosity of the metaphyses, so this can be ruled out (Klaus, 2016).

Brucellosis

Brucellosis presents large lytic lesions to the spine that are often multifocal. These large lesions can also be seen in the long bones or pelvis. In this case, the locations of porosity, and how fine it is, does not support a case of brucellosis (Ortner, 2003).

Leprosy and rhinomaxillary syndrome

Rhinomaxillary syndrome (RMS) can be identified through pitting on the palate, erosion and absorption of maxillary alveolar bone, the nasal spine, and the nasal aperture. This can be a symptom of juvenile leprosy (Lewis, 2002). Lewis (2002) notes that sensory neuropathy associated with leprosy can also result in osteoporosis of the feet which may present as increased porosity. While RMS was considered, leprosy does not account for the postcranial porosity observed on this juvenile (Lewis, 2017).

Grave 19, Skeleton 15

Table 5: *Differential diagnosis of Grave 19, Skeleton 15.*

Grave 19	Skeleton 15	Normal growth	Meningitis	Anemia	Scurvy	Tuberculosis	Trauma
Linear deposits of bone on endocranial surface of left/right frontal, at edges of bone	+	+			+	+	+
Disorganized, thick, capillary-like grooves and deposits of new bone endocranially, medial squama	+		+		+	+	+
New bone formation in orbit of frontal bone	+				+		
Porosity of occipital squama (endocranial) and *pars basilaris*	+	+	+	+		+	
Diffuse fine porosity to cranial vault bones (internal and external temporal/sphenoid/zygomatic/ maxilla/mandible)	+	+		+*	+*		

*Likely growth-related in this perinate

Anemia

Various anemias are linked to porotic hyperostosis (marrow hypertrophy from expansion of diploë in the cranial vault) and cribra orbitalia (porosity of the orbital roof). This is not observed here, instead the porosity is likely growth-related as it is continuous on endo- and ectocranial surfaces, and the endocranial lesions are vascular and capillary-like, not round, and pitted (Ortner, 2003; Walker *et al.*, 2009).

Neoplasms

Neoplasms affecting the juvenile cranium were considered. Malignant chordomas affect juveniles 0-20 years of age but are primarily found on the skull base, not the frontal bones. Non-Hodgkin's lymphoma can impact juveniles at any age but present a “moth-eaten” appearance in the skull, this is not observed on this perinate. Benign Langerhans Cell Histiocytosis may impact the frontal bone, but only in older juveniles typically aged 5-10 years (Lewis, 2017). The young age of this perinate and the morphology of the lesions does not support a neoplastic cause of these lesions (Lewis, 2004, 2017).

Trauma

There was no evidence of cranial trauma suggesting a subdural hematoma caused the endocranial lesions, however, the cranium was highly fragmentary, so this is cautiously ruled out (Lewis, 2017).

Metabolic disease

Considering both the presence of endocranial lesions and the formation of reactive bone found in the orbit, these lesions may be associated with infantile scurvy, but the young age-at-death of this juvenile must be considered (Brickley & Ives, 2008). At a maximum of 4 months postnatal (based on the dental estimate), this juvenile is quite young for a diagnosis of infantile scurvy. Typically, vitamin C deficiency peaks around 6 mo. - 2 yrs. of age, and recognition of scurvy in the skeleton typically requires the deficiency to be active for 4 -10 mo. (Lewis, 2009). There are several factors that would make a young infant more susceptible to scurvy including LBW, prematurity, or inability to breastfeed due to maternal death or other causes (Lewis, 2009). As it is not possible to tell if prematurity or LBW impacted the age-at-death estimate of this individual, suggesting they are older than the estimate provided, this diagnostic avenue is cautious and cannot be confidently excluded.

Grave 25, Skeleton 27 and Grave 38, Skeleton 37

Table 6: *Differential diagnosis of Grave 25, Skeleton 27 and Grave 38, Skeleton 37.*

Grave 25 & 38	Skeleton 27 Skeleton 37	Osteomyelitis	Hypertrophic osteoarthropathy	Infantile cortical hyperostosis	Scurvy*	Genetic anemia* (i.e., sickle cell anemia)	Trauma*
Bone-in-bone appearance of long bone	+	+	+	+	+	+	+
New periosteal bone formation on femoral diaphysis	+	+	+	+	+		+

*Condition can result in, or present like, osteomyelitis (Ortner, 2003; William *et al*., 2004).

Metabolic disease

Diffuse hemorrhage is common on the diaphysis of long bones in cases of scurvy, resulting in a sheath of new bone formation (Fain, 2005). However, in the case of Grave 25 Skeleton 27, there was no evidence of pathology on the other skeletal elements, suggesting a localized process to the femora, and the young age-at-death of these juveniles makes scurvy unlikely (Fain, 2005; Lewis, 2009).

Sickle cell anemia

Sickle cell anemia (SCA) is a disease commonly impacting individuals of African or Mediterranean descent. It has been found to protect children against malaria, a pathogen spread by mosquitoes, endemic to these regions. SCA can cause a BIB like appearance, but typically this is observed in the vertebral bodies (Williams *et al*., 2004). In addition, marrow hyperplasia and blood clots along the long bones, resulting in infarcts (limiting blood supply to bone and resulting in tissue death) are common in SCA and may resemble BIB (Aufderheide & Rodríguez-Martín, 1998; Lewis, 2017; Ortner, 2003). Researchers have identified a link between *Salmonella* bacteria and SCA, suggesting that juveniles with SCA more readily contract bone infections (Burnett *et al*., 1998). However, Ortner (2003) notes that infections or infarcts in SCA cases are typically only found in the vertebrae, hands, and feet, not the femora.

Hypertrophic osteoarthropathy

Hypertrophic osteoarthropathy (HOA), a circulatory disorder resulting in symmetrical new bone formation in the long bones, was considered (Aufderheide & Rodríguez-Martín, 1998). HOA presents as a dense lumpy accumulation of bone on the outside of the diaphysis, commonly referred to as a "candle-wax" appearance. Usually, HOA bone is thickest at the midshaft and tapers at the metaphysis, resembling the bone sheaths in these juveniles (Ortner, 2003). However, these juveniles lack a "candle-wax" appearance, and HOA typically impacts the lower limb instead of the femur (Aufderheide & Rodríguez-Martín, 1998).

Infantile cortical hyperostosis

Infantile cortical hyperostosis (ICH), also known as Caffey's disease, is a bone-forming pathology onset in infancy (Caffey, 1956). Typically, a diagnosis of ICH is determined through the presence of widespread new bone formation on the long bones, clavicle, and mandible. New bone formation on the ribs, scapula, hands, and feet is also common. The formation of thick new periosteal bone creates a sheath around the long bones, and can be symmetrical or asymmetrical, similar to what is observed in the femora of these juveniles. The etiology of ICH is unknown, but researchers speculate infectious, genetic, or traumatic origins (Caffey, 1956; Lewis, 2009). Onset in utero has been identified, but typically ICH is resolved around 5 mo. postnatal. No new bone formation was observed on the lower legs of Grave 38, Skeleton 37, and Grave 25, Skeleton 27. Also, they did not present with new bone formation on the mandible, ribs, hands, or feet (Caffey, 1956; Lewis, 2009; Ortner, 2003). This tentatively rules out ICH.

Tuberculosis and leprosy

Long bone lytic infections in tuberculosis diagnoses are not very common, but diffuse new bone formation has been recorded in the literature. Additionally, tuberculosis has been reported to be linked with hypertrophic osteoarthropathy. However, infections typically occur in the lower leg, forearm, hands, or feet, cautiously excluding tuberculosis for these juveniles (Lewis, 2017). Osteomyelitis can occur with leprosy (considering site context this diagnostic avenue was explored). However, this is typically seen in the hands and feet, so leprosy can cautiously be ruled out (Lewis, 2017).

Congenital anomalies

Developmental anomalies of the femur do not typically present with bone-in-bone appearance and therefore can be ruled out considering the localized nature of these observations (Aufderheide & Rodríguez-Martín, 1998).

Appendix 4

Metabolic and hematopoietic disorders

Table 7: *Summary table of possible metabolic and hematopoietic disorders of juveniles at Ismenion Hill (n=5).*

Grave #	Skeletal #	Pathology
4	1	Childhood leukemia
5	6	Infantile scurvy and anemia
19	14	Possible infantile scurvy
20	20	Possible metabolic disorder
45	42	Possible metabolic disorder

Grave 5, Skeleton 6

Age-at-death: dental: 1.5 ± 3 mo., skeletal: <1 yr.

The upper body of this infant was near complete, the lower body was absent (except for a pedal phalange). The upper limbs, clavicles, ribs, scapulae, some vertebrae, and cranial fragments were recovered. Preservation was compromised by postmortem erosion and breakage.

Table 8: *Observations and differential diagnosis of Grave 5, Skeleton 6.*

Grave 5	Skeleton 6	Normal growth	Physiological periostitis	Scurvy	Anemia	Infantile cortical hyperostosis	Trauma
Fine porosity in orbit	+			+	+		
New bone formation in orbit	+			+	+	+	
Abnormal porosity of maxilla (palate/body)	+			+			
Fine porosity of greater wing sphenoid	+	+		+			
Fine porosity of *pars basilaris*	+	+		+	+		+
New periosteal bone growth on scapular spine/body	+	+		+		+	
New periosteal bone growth on diaphysis of humeri	+		+	+		+	+

Pseudo-pathology, normal growth, and bone-forming pathologies

The porosity observed on this juvenile is unlikely to be a result of pseudo-pathology or postmortem processes (Stodder, 2019). The new bone is isolated to the diaphyses, suggesting it is not growth-related (Klaus, 2017; Lewis, 2017). It is challenging to identify and differentiate bone-forming pathologies in infants (Lewis, 2017). However, the lesions on this infant differ from typical infection-related new bone formation as non-specific infections are often isolated and not widespread. Trauma can also be excluded as there is no evidence of fracture or healing (Lewis, 2017). Physiological periostitis presents as symmetric periosteal bone reaction in preterm perinates and infants <6 mo. of age. The age-at-death estimation and the symmetrical appearance of new bone on the humeri of this infant are consistent with physiological periostitis (De Silva, 2003). Physiological periostitis is typically linked to rapid growth, this would be unusual in a fatally sick child (Lewis, 2017). Infantile cortical hyperostosis (ICH) can produce diffuse new reactive bone in young infants, aligning with the age-at-death estimate. However, ICH diagnoses typically include profuse new bone on the mandible and clavicle, not observed here (Lewis, 2009, 2018).

Infantile scurvy and anemia

Scurvy peaks in juveniles 6 mo. – 2 yrs. of age, aligning with the age-at-death estimate of this juvenile (Lewis, 2009). Observed porosity of the cranial vault, sphenoid, the scapula, and new bone formation in the orbits, the scapula, and the diaphysis of long bones, are consistent with a case of infantile scurvy (Brown & Ortner, 2011; Stark, 2014). According to Gonzalez and colleagues (2018) porosity of the *pars basilaris* is normal in early childhood, but other authors like Moore and Koon (2018) suggest that if other pathological symptoms (i.e., those associated with scurvy) are present, *pars basilaris* porosity may not be normal. Infantile scurvy, potentially brought on by an inability to breastfeed or weaning practices, accounts for most of the osteological symptoms of this juvenile (Lewis, 2009). However, the porosity in the orbits and the cranial bones could also be a result of anemia as they are commonly linked with cranial porosity (Walker *et al*., 2009). Typically, anemia can be genetic (i.e., thalassemia) or acquired (i.e., B12, iron, or folate deficiencies). Walker and colleagues (2009) argue that B12 and folic acid deficiencies are common in infants, likely as a result of maternal access to meat and vitamin rich foods when breastfeeding. Acknowledging the social and environmental climate of late antiquity, maternal access to protein may have been limited (Bourbou & Garvie-Lok, 2015). Overall, the diffuse porosity and new bone formation observed in this infant are consistent with infantile scurvy, with a co-occurrence of anemia (Lewis, 2017).

Grave 19, Skeleton 14

Age-at-death: dental: 2.5-3.5 ± 0.5 yrs., skeletal: 1-2 yrs.

Most of the cranium and both femora, the right tibia, and the right humerus were recovered for this infant. Preservation of the elements was good and postmortem erosion was limited on the available elements.

Table 9: *Observations and differential diagnosis of Grave 19, Skeleton 14.*

Grave 19	Skeleton 14	Infantile scurvy	Rickets	Non-specific infection	Trauma
Internal, medial aspect of foramen magnum (*pars lateralis*) presents with faint linear impressions, fan-like surrounding foramen	+			+	
Symmetrical new bone formation on coronoid and vertical ramus of mandible (internal aspect)	+	+		+	+
Periosteal new bone reaction on diaphysis of left and right femora	+	+	+	+	+
Periosteal new bone reaction on diaphysis of right humerus	+	+	+		
Periosteal new bone reaction on diaphysis of right tibia	+	+	+		

Pseudo-pathology and normal growth

The new bone formation at the diaphyses of the long bones, rather than the metaphyses (expected in cases of normal growth) supports an antemortem pathological process rather than pseudo-pathology (Lewis, 2017; Stodder, 2019).

Bone-forming diseases

Infantile cortical hyperostosis can result in diffuse bone formation across the infant skeleton, however, it typically appears in infants less than 6 mo. of age, making this infant slightly too old (Lewis, 2009). Hypertrophic osteoarthropathy (HOA) also presents as diffuse new bone formation across the skeleton. However, the primary type of this disease usually occurs in late childhood and the secondary type is quite rare in children. In addition, this infant lacks the “candle-wax” appearance of new bone consistent with HOA cases (Aufderheide & Rodríguez-Martín, 1998; Klaus, 2017; Ortner, 2003).

Trauma and infection

Trauma and non-specific infection are unlikely in this juvenile. There is no evidence of fracture or healing consistent with a traumatic experience (Lewis, 2017). In addition, the new bone reaction impacts multiple skeletal elements. This would be unusual in cases of non-specific infection. Due to the limited skeletal material recovered, tuberculosis, which can cause diffuse new bone formation on the lower arm and leg long bones, cannot be excluded confidently (Ortner, 2003).

Infantile scurvy and rickets

Infantile scurvy presents with abnormal porosity and new bone formation on the cranial and post-cranial elements of the skeleton, including the long bones, cranial vault, and mandible. This is consistent with the new bone formation observed on this juvenile (Klaus, 2017). New bone formation and porosity along the coronoid process of the ramus of the mandible, due to bleeding of a branch of the maxillary artery, is also common in cases of scurvy and consistent with this juvenile (Stark, 2014). Rickets can also result in diffuse new periosteal bone reactions, however, the long bones of this juvenile lack the typical "plump bones" appearance, fraying of metaphyses, and anterior bowing of the weight bearing bones, considered diagnostic of vitamin D deficiency (Ortner, 2003). However, rickets typically spares the greater wing of the sphenoid, and there were no lesions on the sphenoid of this infant (Klaus, 2017). Also, in young infants, rickets may not present the typical diagnostic long bone changes (Morrone *et al.,* 2021). Considering the new bone formation isolated to the diaphyses of the long bones and the mandible, and the age of this juvenile falling around typical weaning age for juveniles in antiquity, infantile scurvy, or less likely rickets, are potential contenders for the symptoms observed (Bourbou *et al.,* 2013; Klaus, 2017; Stark, 2014). Due to the limited skeletal material recovered, this diagnosis is said with caution.

The foramen magnum

The appearance of a "wrinkled" or undulating texture of the internal foramen magnum was observed. It was not the product of new bone formation or vascular impressions as expected in cases of meningitis or endocranial lesions (Lewis, 2004). Congenital anomalies, neoplasms, bone-forming disorders, infection, and hematopoietic concerns were explored but no confident explanation for this observation was reached (Lewis, 2017). It is entirely possible that this is an example of normal variation and future research should explore this further.

Grave 20, Skeleton 20 and Grave 45, Skeleton 42

Age-at-death, Grave 20, Skeleton 20: skeletal: 34 wks. – 6 mo.

Cranial fragments, the left and right ilium, some bones of the thorax and vertebral column, the left clavicle, and some long bone fragments were recovered. Preservation was poor, postmortem erosion and breakage was present across all skeletal elements.

Age-at-death Grave 45, Skeleton 42: dental 38 wks-1.5 mo. ± 3 mo., skeletal: 30-40 wks.

Most of Grave 45, Skeleton 42 was recovered, including the cranium, thorax, vertebral column, clavicles, scapulae, long bones of the upper and lower limbs, and the ilia. Preservation was variable across skeletal elements, and postmortem breakage resulted in significant fragmentation of the cranium.

Grave 20, Skeleton 20 and Grave 45, Skeleton 42 have been grouped for analysis due to their similar age-at-death, and the similar observations between the individuals. Due to the difference in quantity of material recovered for each juvenile, two differential diagnosis tables are provided but the analysis is grouped.

Table 10: *Observations and differential diagnosis of Grave 20, Skeleton 20.*

Grave 20	Skeleton 20	Normal growth	Infantile cortical hyperostosis	Physiological periostitis	Infection	Scurvy	Rickets
Woven fibrous bone on scapular spine, fine porosity to infraspinous area	+	+	+		+	+	
Radius diaphysis new reactive bone (does not extend to metaphysis)	+		+	+	+	+	+
Symmetrical new bone growth on posterior ilium	+	+	+	+	+	+	+

Table 11: *Observations and differential diagnosis of Grave 45, Skeleton 42.*

Grave 45	Skeleton 42	Scurvy	Rickets	Leukemia	Infection	Infantile cortical hyperostosis	Physiological periostitis
Abnormal new bone formation left orbit	+	+					
Supraspinous fossa new bone formation	+	+		+	+	+	+
Supraspinous fossa increased/enlarged vascular foramina	+	+		+			
New bone reaction on posterior ilia	+	+			+	+	+
New bone reaction anterior/medial aspect femora and tibiae diaphysis	+	+	+	+	+	+	+
New bone reaction on ulna, radii, humeri diaphysis	+	+	+	+	+	+	+

Normal growth, pseudo-pathology, and bone-forming diseases

New bone formation on the skeletons of perinates and infants is expected. Rapid growth in the postnatal period results in a woven appearance. However, growth-related woven bone is expected at the metaphyses, not isolated to the diaphyses as seen here (Lewis, 2017). The presence of antemortem new bone formation rules out postmortem pseudo-pathology (Klaus, 2017; Lewis, 2017). It is possible there is a mix of pathological and growth-related processes occurring (Lewis, 2017). In both juveniles, new bone formation is not present on the mandible, or clavicle, which would support a case of infantile cortical hyperostosis (Lewis, 2009). The lesions may be associated with growth-related physiological periostitis as they are occasionally symmetrical. In the case of Grave 20, Skeleton 20, the only paired element is the ilia, so physiological periostitis cannot be diagnosed with certainty (De Silva, 2003). However, in both cases it can be argued that a sickly child would not present with significant evidence of growth-related new bone, supporting that these observations are most likely pathological (Lewis, 2017).

Trauma

Birth trauma was considered as it can result in hemorrhage and new bone growth in perinates and infants. However, the widespread nature of these lesions does not support an isolated traumatic incident, and instead points towards a systemic disorder that impacted multiple skeletal elements (Lewis, 2017).

Infection

Infections like tuberculosis can present with new bone formation on the diaphyses of long bones in children. However, typically this is accompanied by lytic lesions of the spine and ribs, which appear unaffected in these juveniles. A non-specific infection could explain the presence of new periosteal bone growth on the elements recovered separately, however, widespread involvement of the entire skeleton suggests a broader systemic concern (Lewis, 2017). Nevertheless, young infants and perinates are especially susceptible to infection and the composition of their skeleton and immune system allows for pathogens to be more readily spread through the blood stream. As such, infection cannot be confidently ruled out, but the pathological symptoms are not diagnostic of any specific etiology (Lewis, 2017; Liston *et al.*, 2018). For a discussion on infectious processes impacting perinates and infants, see Section 2.8 of this thesis.

Metabolic disorders

The young age of these juveniles makes a diagnosis of infantile scurvy unlikely (Lewis, 2009). However, a recent study by Morrone and colleagues (2021) exploring evidence of systemic metabolic deficiencies, specifically vitamin C and D deficiency, in perinates and infants presented similar osteological findings. Morrone and colleagues (2021) observed widespread new periosteal bone formation on the long bones, on the posterior aspects of the ilia, and increased vascularity and porosity across most skeletal elements. They were able to differentiate these processes from growth-related changes based on the location of the lesions (not at the metaphyses/epiphyses) similar to Grave 20, Skeleton 20 and Grave 45, Skeleton 42. If impacted by scurvy or rickets, the young age of these individuals would suggest that their mothers would have had to been severely deficient in vitamin C or D to cause such significant lesions. Lesions appear similar in infantile scurvy and rickets, and perinates may not present with the traditional anterior bowing of the long bones or "plump bones" typically identified in older juvenile rickets cases (Morrone *et al.,* 2021; Ortner, 2003). A pregnant mother severely deficient in vitamin C or D is also likely severely deficient in other vitamins and nutrients, which can result in pregnancy-related complications such as spontaneous abortion or stillbirth. If the perinate is born viable, osteological symptoms are possible (Lewis, 2017; Morrone *et al.,* 2021). Considering these diagnostic avenues and the non-specific nature of the osteological symptoms observed, it is not possible to pinpoint a diagnosis. However, the fact that these lesions impact several skeletal elements suggests a systemic process, like a metabolic disorder, may have affected these juveniles. Likely this would be a product of maternal health and environmental stressors (Morrone *et al.,* 2021). Further research should utilize radiographic imaging and stable isotopes or pathogen aDNA to better understand the pathological processes impacting Grave 20, Skeleton 20, and Grave 45, Skeleton 42.

Appendix 5

Non-specific infection

Table 12: *Summary table of non-specific infections observed on Ismenion Hill juveniles, (n=7).*

Grave #	Skeletal #	Pathology	Observations
19	15	Non-specific meningitis	See Appendix 3
25	27	Infantile osteomyelitis	See Appendix 3
27	29	Non-specific infection, periostitis	Right tibia presents with isolated periosteal new bone reaction on anterior and medial aspect
38	36	Possible non-specific infection	See Table 13
38	37	Infantile osteomyelitis	See Appendix 3
45	41	Non-specific infection, periostitis	Left tibia presents with an isolated plaque of new periosteal bone on the anterior and medial aspect
Bothros #1	45	Non-specific infection, periostitis	Left tibia presents with periosteal new bone reaction on all aspects of the shaft, some postmortem erosion has eroded new bone reaction, revealing fine porosity on bone surface, but bone reaction is distinctly darker in colour

Non-specific infection

Non-specific infections are a result of unknown causes and typically include periostitis and osteomyelitis. Periostitis refers to asymmetrical plaques of new woven bone identified on top of the original bone cortex (Lewis, 2017). Osteomyelitis is discussed in Section 2.11. Usually, non-specific periostitis infections affect a single bone, and are common on the tibia (Lewis, 2009). This is the case with Grave 27, Skeleton 29, Grave 45, Skeleton 41, and Bothros #1, Skeleton 45. In the case of Bothros #1, Skeleton 45, a dark-stained periosteal reaction suggests a hematoma may have caused the periosteal reaction (Klaus, 2017). Infectious factors that may contribute to non-specific bone infections in antiquity are discussed in Section 2.8 of this thesis.

Grave 38, Skeleton 36
Age-at-death: dental: 1.5-2.5 ± 0.5 yrs., skeletal: < 2 yrs.
Limited skeletal material was recovered for this individual including a fragmentary left tibia, right and left fibula, bones of the hands and feet, and several cranial fragments. Preservation was poor and elements were heavily eroded.

Table 13: *Observations and differential diagnosis of Grave 38, Skeleton 36.*

Grave 38	Skeleton 36	Postmortem erosion	Non-specific infection	Metabolic/ hematopoietic
Disorganized internal grooves and pitting on cranial fragment	+	+	+	+
One large perforation ~1-2 mm in diameter extending inner-outer table of cranial fragment	+	+		
New periosteal bone reaction on anterior-medial diaphysis of tibia, separate from metaphysis	+		+	+
Calcaneus, fine porosity, and new reactive bone formation to cortex	+		+	+

Endocranial lesion and cranial foramina
Endocranial lesions can be caused by normal growth, cancers, infection, or hemorrhage (Lewis, 2018). While the grooves on this fragment appear disorganized, they are highly variable in depth and size, and there is no evidence of new bone growth, suggesting they are not likely the result of antemortem processes (Lewis, 2004).

Several pathological processes cause irregular foramina in the skull. Cranial changes associated with cephalohematomas and venous malformations are usually isolated to the outer table of the skull, not affecting the inner and outer table, like this juvenile. Parietal foramen or venous emissary foramina are benign normal variations in the parietal and cranial vault bones. These were considered but the small size of the foramen and the irregular sharp margins of the foramen suggest a postmortem rather than antemortem process (Choudhary *et al.*, 2019). Parietal fenestae, large oval defects with smooth edges, and venous connections also known as sinus pericranni resulting from abnormal communications between the veins that extend through the inner and outer table of the skull, are ultimately too large to be what is observed in this juvenile (Kaufman *et al*., 1997).

Pseudo-pathology

The cranial lesions and foramen observed seem to be a product of postmortem erosion, presenting a case of pseudo-pathology (Stodder, 2019). Microscopic analysis of the foramen that extends both the inner and outer table cannot confidently show that it was an antemortem processes that caused the foramen. The disorganized grooves on the inner table of the fragment resemble those seen on other cranial fragments of this juvenile, supporting a postmortem process.

Infection, trauma, and metabolic disorders

The isolated plaque of new reactive bone on the tibia and calcaneus resembles periostitis (Lewis, 2009). Typically, non-specific infections are isolated to one element, so it is possible a broader systemic concern is at play here (Lewis, 2017). However, no other skeletal elements present evidence of pathology, supporting an infection isolated to the lower limb and foot. Plaques of new periosteal bone can be the result of hemorrhage (usually caused by trauma or metabolic deficiencies) or infection (Lewis, 2009, 2017). Specific infections like tuberculosis have been noted to cause new bone formation on the long bones of juveniles, however, this is usually accompanied by lytic lesions on the spine, not observed here (Ortner, 2003). No evidence of trauma or fracture was observed on this juvenile (Lewis, 2017). Vitamin C or D deficiency can cause diffuse periosteal bone new bone in the weight bearing elements (Klaus, 2017). The age of this juvenile ~1-3 years, falls within typical weaning age in late antiquity potentially supporting a metabolic deficiency (Bourbou *et al.,* 2013; Lewis, 2017). However, there is no evidence of increased porosity, fraying of the metaphyses, or "plump bones" that would support a metabolic deficiency (Klaus, 2017). Considering the isolated plaque of new periosteal bone on the tibia and calcaneus, infection cannot be ruled out as a possible cause of these symptoms.

Specific infection

Grave 5, Skeleton 5
Age-at-death: dental: 11.5 ± 0.5 yrs., skeletal: 11-16 yrs.
Limited skeletal material was recovered for this juvenile, including cranial vault fragments, fragments of the right leg, and some left and right tarsals, metatarsals, and pedal phalanges.

Table 14: *Observations and differential diagnosis of Grave 5, Skeleton 5.*

Grave 5	Skeleton 5	Leprosy	Scurvy	Rickets	Osteopenia
Dorsal bar on navicular and cuboid	+	+			
Fine porosity observed in tarsal bone cortex	+	+	+	+	+
Trabecular thinning of the calcaneus	+	+	+	+	+
Porosity around ligamentous (talonavicular, dorsal tarsometatarsal, talocalcaneal) and muscle (fibularis) attachment sites of the tarsals	+	+	+		+
"Knife-edge" diaphyseal remodeling of Metatarsal 4, mediolateral compression	+	+			

Leprosy
Secondary bone lesions of leprosy include drop foot (arch collapse), navicular squeezing and dorsal barring, "knife-edge" remodeling of metatarsals, and generalized osteoporosis from disuse of hands/feet and limbs (Lewis, 2017). This resembles the remodeling of metatarsal 4, dorsal barring, evidence of trabecular thinning on the calcaneus, and potential arch collapse identified by porosity at various ligamentous attachment sites throughout the tarsals of Grave 5, Skeleton 5.

Leprosy can result in neuropathy of the nerves innervating the muscles of the foot, impacting the stability of the longitudinal arch, resulting in flat foot. This stresses the dorsal ligaments (talonavicular, cuneonavicular, and cubonavicular) at their sites of attachment increasing porosity of the bone cortex. Chronic stress at these sites results in a buildup of bone and in extreme cases exostoses (Andersen & Manchester, 1988). Pitting and bone growth at ligamentous attachment sites reflect the fine porosity and dorsal barring seen in Grave 5, Skeleton 5 (Spekker *et al.,* 2022).

The presence of a sharp, thin, edge on the plantar surface of a metatarsal diaphysis is referred to as "knife-edge" diaphyseal remodeling. Andersen and colleagues (1992) suggest that this type of remodeling does not present with evidence of irregular bone deposition or evidence of infection, and instead the bone will not appear to have any abnormalities on the surface, and outer layers of bone will have resorbed and reshaped the element. This reflects what was observed in this juvenile. This type of thinning is often a precursor to more severe resorption or erosion of elements in cases of leprosy. Bilaterality is common, and often a result of neuropathies related to leprosy (Andersen *et al.*, 1992).

As more diagnostic maxillary and dental changes associated with leprosy could not be observed due to preservation, a confident diagnosis is not possible. However, considering the high prevalence of leprosy at this site, the older age of this juvenile (making the presence of secondary bone lesions more likely as they had more time to develop), and the abnormal changes to the bones of the feet consistent with secondary bone lesions in cases of leprosy, a leprosy diagnosis cannot be excluded (Lewis, 2017).

Scurvy, rickets, and osteopenia

Vitamin C deficiency can result in diffuse osteopenia, trabecular thinning, and porosity at ligamentous or muscle attachment sites (Brickley & Ives, 2008; Stark, 2014). Rickets is also reported to cause cortical thinning similar to what is observed in the tarsals of this juvenile. However, other diagnostic features of rickets like fraying of the long bone metaphyses and anterior bowing of the tibia, were not observed (Lewis, 2017). General osteopenia can be a symptom of metabolic deficiencies or infection (Brickley & Ives, 2008). There is also research suggesting a link between osteopenia and leprosy, and vitamin D deficiency and leprosy, but this research avenue is understudied (Roberts & Brickley, 2019). However, a diagnosis of vitamin C deficiency, rickets, or osteopenia does not account for the navicular dorsal bar (although flat foot can be a benign comorbidity) or the "knife-edge" remodeling observed (Andersen & Manchester, 1988; Lewis, 2017; Stark, 2014). Nevertheless, metabolic stressors would have made this juvenile more susceptible to acquiring leprosy, and could be comorbidities (Roberts & Brickley, 2019).

Pseudo-pathology

The porosity observed on the post-cranial skeleton of this juvenile likely occurred antemortem as it is the same colour as the surrounding bone cortex and is focused at the site of ligamentous attachments (Spekker *et al.,* 2022; Stodder, 2019).

Appendix 6

Dental pathologies

Table 15: *Summary table of dental pathologies observed on Ismenion Hill juveniles (n=4).*

Grave #	Skeletal #	Pathology	Observations
4	1	Antemortem tooth loss, abscess	See Appendix 3
20	17	Calculus	Mandible: calculus observed on lingual aspect of permanent central incisors Maxilla: calculus observed on lingual and labial aspects of permanent central incisors
45	38	Calculus	Mandible: left permanent premolar two presents with dental calculus on buccal surface near alveolus
Parking Lot Grave	54	Calculus, linear enamel hypoplasia	Mandible: evidence of furrows in the enamel and calculus concentrated near occlusal edge (labial aspect) of permanent left mandibular permanent lateral incisor and right canine Maxilla: permanent left canine presents with evidence of furrows/ridges in the crown enamel, calculus observed on occlusal edge of dentition (lingual/labial), and labial aspects of crown