

Developmental Defects in Ancient Context –
Causations of Cleft Palate in the Athenian Agora

by

Alisha Adams

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

Waterloo, Ontario, Canada, 2016

© Alisha Adams 2016

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

This research looks at the infant cleft palates recently identified in the Athenian Agora. This assemblage provided the opportunity to expand the ways which bioarchaeology may study developmental defects which affect the skeleton. A biological, historical, and archaeological study was undertaken in order to analyze cleft palate in the archaeological record, and to understand and identify possible causation factors in the ancient environment. Based on this research, the prevalence of cleft palate in the Athenian Agora, estimated from modern perinatal infant mortality rates and cleft palate prevalence showed that the cleft palates in the Athenian Agora may have been the result of syndromic etiologies. The shape of the palate as well as deviations along the line of the cleft are discussed as possible support for a syndromic etiology of the infant cleft palates.

Acknowledgements

I would like to express my deep gratitude to Dr. Maria Liston, my research supervisor, for her patient guidance, encouragement, advice, assistance in writing this thesis, and for allowing me to accompany her to Greece to study the infant cleft palates in the Athenian Agora. I would also like to thank Dr. Bonnie Glencross and Dr. Russell Adams, my thesis committee members, for their advice and useful critiques of this research work.

My grateful thanks are also extended to: Prof. John McK. Camp of the excavations in the Athenian Agora; Dr. Panagiotis (Takis) Karkanis at the Wiener Laboratory at the American School of Classical Studies in Athens for his research assistance; and to Prof. Kevin Daly and Prof. Stephanie Larson, Bucknell University, and the Thebes Synergasia Excavation Project Summer 2015 team for their welcoming support and opportunity to experience an excavation and concurrent laboratory work.

I would also like to thank my University of Waterloo Public Issues Anthropology graduate cohort, Samantha Lawrence, Danielle Nadeau, Jennifer Rickert, and especially Ava Giachino, for their never ending support and encouragement throughout the research and writing process. Special thanks go to my long-time friend Kyle Campbell, who despite a great amount of distance, have kept in contact with me and supported me without question, and Steven Ruttan, for listening to my rambling thoughts and being the shoulder I have leaned on throughout this process.

Finally, I wish to thank my parents and siblings for their support and encouragement throughout my studies, with a special acknowledgement extended to my sister, Roberta Adams, for her advice, critique, and formatting knowledge.

Table of Contents

Author’s Declaration	ii
Abstract	iii
Acknowledgements	iv
Table of Contents.....	v
List of Figures	vi
List of Tables.....	vii
Chapter 1: Public Issues Anthropology	1
1.1 A Public Issue.....	1
1.2 Publication Venue	2
Chapter 2: Developmental Defects in Ancient Context	3
2.1 Introduction.....	3
2.2 Bioarchaeological Theory.....	3
2.3 Cleft Palate in the Archaeological Record	4
2.4 Embryology.....	4
2.5 Epidemiology.....	5
2.6 Environmental Risk Factors	6
2.7 The Ancient Environment.....	7
2.8 Cleft Palate and the Athenian Agora	13
2.9 Differential Diagnosis.....	14
2.10 Conclusion	23
Bibliography	24
Appendix: Published Archaeological Examples of Cleft Lip and Cleft Palate	32

List of Figures

- Figure 1: The nine maxillae exhibiting cleft palate from the Athenian Agora. Maxillae which were identified as having a questionable extent of clefting are marked with an asterisk. Photo Credits: Dr. M. Liston and Alisha Adams..... 13
- Figure 2: (a) A right maxilla exhibiting cleft palate compared to an intact left maxilla (b) A left maxilla exhibiting cleft palate. Analyzing the shape of the cleft has the potential to determine a possible syndromic etiology. Photo Credit: Dr. M. Liston. 20
- Figure 3: Four Maxillae which show an “uneven” growth at the midline. Photo Credit: Dr. M. Liston. 20
- Figure 4: R2, a “normal” cleft palate, compared against an intact maxillae, and R2’s corresponding magnification of the palatal shelf, showing a normal porosity. Photo Credits: Dr. M. Liston and Alisha Adams 21
- Figure 5: Maxillae R3 and L13 and their corresponding magnifications of the palatal shelf, showing enlarged porosity. Photo Credits: Dr. M. Liston and Alisha Adams..... 21
- Figure 6: Maxillae L3 and L12 and their corresponding magnifications of the palatal shelf, showing small to normal porosity. Photo Credits: Dr. M. Liston and Alisha Adams. 22

List of Tables

Table 1: Important nutritional compounds for development and their common sources. All sources from the Dietitians of Canada 2015.	10
Table 2: Perinatal and infant mortality rates of various regions, and cleft lip and palate and cleft palate prevalence. All Regional Data and Mortality Rates from WHO 2006, unless otherwise indicated.....	16
Table 3: Regional Perinatal and Infant Mortality Rates and Calculated Estimate Ancient Cleft Palate Prevalence. Perinatal Mortality Rates from WHO 2006, unless otherwise indicated.....	17
Table 4: Global Regional Cleft Lip and Palate and Cleft Palate Prevalence and Calculated Estimated Athenian Agora Cleft Palate Prevalence. Cleft Lip and Palate and Cleft Palate Prevalence averaged from Mossey and Little 2002.....	18

Chapter 1: Public Issues Anthropology

1.1 A Public Issue

Developmental defects are a cause for public concern. They affect over 300,000 infants annually worldwide, with one in thirty-three infants being born with a developmental defect (Center for Disease Control (CDC) 2015, World Health Organization (WHO) 2015a). Cleft lip with and without cleft palate is one of the most common developmental defects in the world affecting 1.7 in 1000 infants (WHO 2015a, CDC 2015). Despite advances in the prevention and care of developmental defects, they remain an issue in all societies. Not only do these defects impact the lives of those children, in their mobility, morbidity, and mortality, there is also an increasingly significant financial burden for those affected (Mossey et al 2009, CDC 2015).

It is estimated that the average household with a child affected by a cranial or neural tube defect in the United States spends approximately \$706,000 in medical and living costs (CDC 2015). This widespread impact and growing financial stress can be demonstrated by oral clefts. Management of cleft lip, with or without cleft palate, does not end in corrective surgery. Multidisciplinary care, such as speech therapy and orthodontia, is required for those affected into adulthood. This leads to lifelong financial, emotional, and time investments and commitments for families (Mossey et al 2009). Geographical areas which see the highest prevalence of cleft lip and palate are often less developed or are developing countries and communities. These areas have reported having difficulty managing children with defects due to the cost, availability of care, and training for defect correcting surgeries (CDC 2015). This is just one of the reasons why research is focused on prevention.

The most common way science looks at prevention of cleft lip and palate, and developmental defects in general, is to study causal factors. Many avenues of causation are being studied however it is becoming clear that cleft lip and palate has no easy solution for prevention. Research can only say for certain that malnutrition, alcohol, and parental age can exacerbate the risk of a fetus having developmental defect (Weiss 2014). Even folate, which is often portrayed as the single most important factor in the prevention of developmental defects, is only the main causal factor in a percentage of cases (Czeizel 2002, Munger 2002). It is therefore vitally important that research continues to look at the expression of developmental defects globally, in an effort to discover etiological pathways and to end future suffering.

The need for focused research on developmental defect causation is apparent; however means of studying birth defects can be difficult. Animal testing provokes many ethical concerns, especially when there is a legal precedent that animal testing does not accurately predict a human reaction (Ndreu 2006). Epidemiological studies can also be inconsistent due to lack of data for a country, a short history of recording, and inaccuracy of language (Brent 2004, Mossey et al 2009, Mossey and Little 2002, Christensen 2002). Therefore there is a need to find new ways of studying developmental defects.

Developmental defects are by no means a modern phenomenon. They exist in virtually every society, and have occurred throughout history, and have likely been a factor in the prehistoric past as well. Bioarchaeology may study those defects that affect the skeleton, however it also

presents a two-fold opportunity to use past historical examples of developmental defects as a secondary method of studying routes of causation, and to explore and expand the ways which bioarchaeology studies birth defects beyond case studies.

Not only can cleft palates be studied in an ancient context, we can examine the prenatal environment, and contribute to modern causation studies by identifying known causation factors in the past. Additionally it opens up new paths for consideration of causation, as some ancient environments mirror those areas which experience the highest occurrences of developmental defects.

1.2 Publication Venue

The International Journal of Paleopathology is the venue of choice for publication as this journal encourages interdisciplinary communication and is the peer reviewed journal for the Paleopathology Association. This venue is appropriate because they accept both historical and prehistorical research studies of a disease or community. They accept studies of unique diseases, and on work which is unique for its temporal or geographical context. This thesis work suits the journal's research goals as it involves a multidisciplinary approach on the study of cleft palates in ancient Greece.

Chapter 2: Developmental Defects in Ancient Context

2.1 Introduction

Developmental defects may develop before birth, during labour, or only be apparent as the child grows (Barnes 1994). Bioarchaeology is limited to studying those defects that affect the skeleton; however we are not limited to studying questions of how these defects appear in the archaeological record. Cultural patterns, environmental factors, kin relationships, and their co-existence with other anomalies can be used as markers for investigation.

Cleft lip and palate is listed among the most common developmental defects in the world, affecting 1.7 infants in 1000 (WHO 2015a, CDC 2015). Cleft palate and cleft lip are serious conditions, as they impair the ability to create suction, resulting in an inability to breastfeed. As the child grows, speech and hearing issues can also arise (Roberts 2005, Saal 2002, WHO 2015a, CDC 2015). The causes of orofclefts, clefts of the oral cavity and lip, are multi-factorial and polygenetic (Mossey et al 2009, Mossey et al 2007, Arakeri et al 2010). Diet, environment, and genetics all contribute to orofcleft etiologies. In an effort to discover possible etiological pathways and to end future suffering, it is important that research continue to look at the expression of cleft lip and palate globally. Cleft lip and palate in the archaeological record presents additional opportunities to study causation, and to expand the bioarchaeological study of birth defects. This study requires further integration with clinical categorizations of cleft palate based on causation, though this may be difficult as the categorization of cleft palate and cleft lip is based on soft tissue as well as the bone. Bioarchaeology is able to assist in clinical categorization of cleft palate by emphasizing the skeletal features.

2.2 Bioarchaeological Theory

Case studies and differential diagnoses are the standard approaches that bioarchaeologists use when examining birth defects (Weiss 2014, Roberts 2005). While most reports present a summary of modern causations, few look at what causal factors may have existed in the original environment. We can analyze not only the osteological presentation, but also the cultural and biological factors behind these defects. This premise is inspired by the work of Ethne Barnes (1994:5) who stated that, “detecting developmental defects within a prehistoric skeletal population permits interpretive projections for the occurrence of major defects, biological affinities, and both cultural and environmental influences”.

Examining the cultural, historical, and biological aspects of birth defects in the archaeological record contributes to the integration of biological and social aspects of human life. The concept that human biology and society affect one another is not new in anthropology (Goodman and Leatherman 1998; Agarwal and Glencross 2011). The examination of how inequality interacts with human biology is at the core of biocultural synthesis. Current research focuses on greater contextualization of skeletal remains with archaeological, historical and ethnographic resources, along with the skeletal analysis (Agarwal and Glencross 2011). This contextualization includes the study of birth defects in a bioarchaeological context.

2.3 Cleft Palate in the Archaeological Record

Despite being one of the most common birth defects currently, we have few examples of cleft palate archaeologically. My research has located 23 archaeological instances of cleft lip or cleft palate mentioned within published reports (MacCurdy 1923, Derry 1938, Brooks and Hohenthal 1963, Berndorfer 1962, Alexandersen 1967, Gladykowska-Rzeczka 1989, Brothwell 1981, Ortner 2003, Sandison 1980, Anderson 1994, Miller and Merbs 1993, Phillips and Sivilich 2006) (See Appendix). Cases have been identified in North and South America, Egypt, Denmark, England, Australia and the South Pacific. The majority of the individuals were adults, aged 20-50 years old. The youngest example was an eight to ten year old child with cleft lip and palate from Peru (Ortner 2003).

Due to its association with fatal conditions and inability to breast-feed, high infant and juvenile mortality associated with cleft palate would be expected in ancient society, but few affected skeletons are found in the archaeological record. The lack of juvenile remains may result from the fragility of infant bones and pathological maxillae, but also to issues in infant archaeology, such as past excavation techniques overlooking infant bones, preservation issues, and differential burial practices for infants (Roberts 2005, Lewis 2009, Barnes 1994). The recently identified infant cleft palates excavated during 1937 and 1938 of a well in the Athenian Agora are important for the archaeological record as they are the first infant cleft palates that have been recovered, and mark the largest assemblage of this birth defect.

The well is located approximately 40 meters north of the temple to Hephaestus, close to the center of ancient Athens, but isolated in an alley between the Stoa of Zeus and the arsenal. Through dating of pottery, it is estimated that the well accumulated the various bones and artifacts for 15 years during the late Hellenistic period, from 165 to 150 BCE. A minimum of 457 infants, an adult male, an approximately 8 year old child, and the remains of over 150 dogs were recovered from the well (Liston and Rotroff 2013). At least 40% of the infants in the well were demonstrated to have died from natural causes, though infanticide or exposure was possible for some. The median age at death for the infants in the well was one week post-partum (Königsberg and Liston 2013, Liston and Rotroff 2013). Although 457 infants are present in the well, only 164 maxilla halves were recovered or preserved, and nine maxilla halves were identified as exhibiting cleft palate. Although the number is too small for robust statistical analysis, the sample is larger than any other known at any time period.

2.4 Embryology

In order to begin this study, it is important to understand the embryology of cleft palate. The least severe type of clefting is classified as the bifid uvula, with increasingly severe clefting moving anteriorly through the soft and hard palates, in the opposite direction of fusion. Cleft palate occurs when the two halves of the hard palate (known as the “secondary palate” during development) fail to grow or fuse, causing a gap in the roof of the mouth. The mid-palatal suture is diverted or nonexistent in cleft palate, and the resulting line of fusion shows the diversion from the mid-line (Sperber 2002). Clefting of the premaxilla (also known as the dental arch and “primary palate” in development) may occur with cleft palate, though they have different embryological processes. If cleft palate involves the premaxilla, clefting will occur between the lateral incisors and

the canine teeth, at the division between the primary palate and secondary palate (Sperber 2002, Saal 2002). In archaeological remains only the involvement of the palate and premaxilla can be observed. It is assumed that the lip was also involved when the premaxilla is cleft, though archaeologically, there is no preservation of soft tissue.

Cleft palate forms in the secondary palate after the development of the primary palate and lip. The secondary palate is formed from the two lateral palatal processes, which project downwards in the mouth cavity due to the raised position of the fetal tongue, and the inferior orientation of the head against the heart prominence (Sperber 2002). During the eighth week of development, the head lifts and mouth opens, lowering the fetal tongue, and allowing the lateral palatal prominences to rise and fuse (Sperber 2002). Cleft palate can occur if the lateral palatine processes fail to rise, or fail to fuse with each other, the primary palate or nasal septum (Sperber 2002). Fusion starts in the hard palate and moves posteriorly to the soft palate along a thick layer of epithelial cells once the edges of the palatine processes touch. If the epithelial cells fail to disintegrate, fusion and ossification will not occur properly, causing a cleft (Sperber 2002). The genetics and the teratogens¹ affecting these actions are poorly understood, though some studies suggest this process is particularly sensitive to teratogens (Sperber 2002, Arakeri et al 2010, Mossey et al 2009)².

2.5 Epidemiology

Cleft lip and palate is estimated to affect 1 in 700-1000 births, and cleft palate alone is estimated to affect 1 in 2000³ (Mossey and Little 2002). The prevalence of cleft lip with or without cleft palate varies according to ethnicity, sex, and socioeconomic factors. Native Americans, Japanese, and Chinese have the highest prevalence in the world; males are more often affected at a ratio of 2:1, and low socioeconomic populations have a higher risk than those individuals of similar ethnicity in a higher socioeconomic position (Saal 2002, Sperber 2002). Cleft palate alone is less prevalent, females are more likely to be affected by cleft palate at a ratio of 3:2, and no one ethnicity predominates another for prevalence (Saal 2002).

Not all oroclefts have the same etiology. Syndromic clefting is caused by genetic conditions and is usually accompanied by multiple other anomalies. Non-syndromic clefting is caused from teratogenic exposures, and has one or no “major” anomalies present with the cleft, or two or less “minor” anomalies (Saal 2002)⁴. Overall, cleft palate is syndromic in 41-55%⁵ of oroclefting cases.

¹ Any substance or agent which can disrupt development.

² Many teratogens which have been studied do not have a strong correlation with cleft palate alone, if any, suggesting that these would be teratogens that have not been identified yet. See “Environmental Risk Factors”.

³ Epidemiological knowledge can be inconsistent and obscured due to how statistics are collected, the accuracy of the categorization of types of clefts (discerning of syndromic and nonsyndromic clefting, and separation of cleft lip and palate and cleft palate alone), and the lack and/or short history of records in many countries. This is furthered by casual studies that examine issues regarding the control and non-control groups utilized, research methods (interviews, surveys, and/or birth records), consistency in measuring exposure levels, and how they affect different types of clefts. For these reasons, prevalence statistics and the risk factor associated with a certain element may differ according to which source is utilized (Brent 2004, Munger 2002, Hayes 2002, Mossey 2002, Christensen 2002, Arakeri et al 2010, Massey 2009).

⁴ Major anomalies are those which are functionally or cosmetically significant, and requiring medical intervention, where as minor anomalies are ones which do not require attention. These are subjective definitions though, and differ between

Cleft lip and palate is syndromic in 25%⁶ of oroclefting cases (Saal 2002). To determine external causations of cleft palate, we must look at those cases of nonsyndromic clefting alone. On a skeleton, we are limited to identifying anomalies on the bone, making it difficult to classify them as major or minor.

Nonsyndromic cleft lip and palate shows several patterns of recurrence. The more severe the condition (ie: bilateral cleft lip and palate), the more likely recurrence will occur within siblings (Saal 2002). The sex, ethnicity, and degree of relationship to other affected familial individuals also affect the prevalence within a family (Saal 2002). Cleft palate alone does not share these tendencies. The prevalence of non-syndromic cleft palate is relatively steady across populations. The risk of recurrence remains the same for future offspring, even if one child is affected (Saal 2002).

2.6 Environmental Risk Factors

A critical review of the epidemiological and experimental data suggests that environmental risk factors and maternal exposure are important in the development of oroclefts, though no universal cause has been identified (Mossey et al 2009, Mossey et al 2007, Arakeri et al 2010, Hayes 2002). Molina-Solana (2013) found that tobacco, alcohol, folic acid intake, stressful events, low blood zinc levels, and fever during pregnancy were high risk factors contributing to oroclefting. Maternal cigarette smoking was shown to have a weak association to oral clefting. Increased alcohol intake was found to have significant increased risk for cleft lip and palate, though not with cleft palate alone (Hayes 2002). Other teratogenic studies looked at the relationship between oroclefting and caffeine, epilepsy, anti-convulsing drugs, benzodiazepines, corticosteroids, and various organic solvents and pesticides (Hayes 2002). Overall, studies suggest that external teratogens appear to affect the occurrence of cleft lip and palate more than the occurrence of cleft palate, suggesting cleft lip and palate is more environmentally sensitive (Hayes 2002).

The influence of maternal nutrition in the development of oroclefts is a complex interaction of nutrition and genes, though the genetic interaction is unclear. The co-relation of nutritional factors and oroclefts has been understudied since the discovery of folate and its effect on birth defects. Folate has been the focus of maternal nutritional studies (Czeizel 2002, Munger 2002), and is notable for its success in prevention of neural tube defects. Folate's ability to combat oroclefting is seen as secondary to its affect on the neural tube, due to their shared developmental origins (Czeizel 2002, Munger 2002).

Closure of the neural tube in weeks 3-4 of development signals the creation of neural crest cells which give rise to the cranial and orofacial region (Gilbert 2010, Sperber 2002). It has been suggested that the failure of the neural tube to close affects the neural crest cells and their descendants (Weingartner et al 2007). Due to this close linkage, many cranial and neural tube defects share a list of non-genetic origins, including folate levels, smoking, and alcohol consumption

publications. There is disagreement on how to classify "number" of anomalies, different presentations of "major" anomalies which may seem minor or vice versa, and if the degree of affliction (ie: bifid uvula vs. bilateral cleft lip and palate) should be considered too (Saal 2002).

⁵ The instances of syndromic cleft palate have also been identified as occurring in 10-50% (Christensen 2002), as well as 25% (Mossey 2002) of cases.

⁶ The instances of syndromic cleft lip and palate was also found to be 2-15% (Christensen 2002), and 12% (Mossey 2002).

(Weingartner et al 2007, Czeizel 2002, Munger 2002). However, there are few instances of co-occurrences of neural tube defects and oroclefting, indicating they interact differently with the same teratogens and nutritional factors (Munger 2002)⁷.

Other nutritional studies show that deficiency of vitamin B6, and excess of vitamin A may be causal factors. Smaller studies have looked into the deficiency of riboflavin, retinoic acid, zinc, vitamin B12, pantothenic acid, and alkaloid plant toxins from arid environments (e.g. *Nicotiana* [tobacco] and *Punica* [pomegranate]) (Czeizel 2002, Munger 2002, Hayes 2002). The role of maternal nutrition does not stop at the ingestion. Arakeri et al (2010) proposed that celiac disease, which can cause the mal-absorption of folic acid and other nutrients, may be a missing link in causal studies.

2.7 The Ancient Environment

The causative factors identified by modern science provide a template for studying causation of cleft palate in ancient Greece. The specific factors which caused the cleft palates in the Athenian Agora may not be completely understood, however factors that may have existed in the ancient environment causing birth defects can be examined.

Heavy metals and other harmful elements have not been the focus of cleft palate research, but their involvement in other developmental defects, such as neural tube defects, may be analogous, and therefore play some role in cleft palate's development. While there is currently no evidence to suggest that potentially harmful elements are innately present in the soil or water in Athens or Greece, this does not prove that the environment is free of these contaminants. This type of environmental testing is not generally a priority for archaeologists, and if performed, is not publicly available from the Greek government (personal communication, Panagiotis Karkanias, July 2015). However, it is known that industrial pollution was produced within the ancient environment, and that industrial sites, such as mines, were heavily polluted.

Laurion, located south of Athens, was mined for silver bearing lead ore beginning ca. 1500 BCE, until the mines became unproductive by 326 BCE (Hughes 2014). The environmental cost of the mine was tremendous, and made the land "unwholesome" (Xenophon, *Memorabilia*, 3.6.12)⁸. Deforestation was rampant in the area to produce fuel for fires, and lead from the mine impacted health greatly. Lead poisoning hastened the death of mine workers and most died within ten years of starting work (Hughes 2014). Vitruvius noted the effect of smelting and lead fumes on workers (*On Architecture* 8.6.11)⁹. Strabo (*Geography*, 12.3.40)¹⁰, and Lucretius (*On the Nature of Things*,

⁷ Munger (2002) also suggests published research in this matter is lacking.

⁸ Xenophon, *Memorabilia*, 3.6.12: "As for the silver mines ... I would think that you have not gone there, and thus cannot say why now less revenue than before comes from them. ...for by Zeus, the region is said to be unwholesome" (quoted in Humphrey et al 1998)

⁹ Vitruvius, *On Architecture*, 8.6.11: "We can take example by the workers in lead who have complexions affected by pallor. For when, in casting, the lead receoeved the current of air, the fumes from it occupy the members of the body, and burning them thereupon, rob the limbs of the virtues of the blood." (quotes in Hughes 2014)

¹⁰ Stabo, *Geography*, 12.3.40: "Mt. Sandaracurgium is hollowed out in consequence of the mining done there, since the workmen have excavated great cavities beneath it. The mine used to be worked by publicans, who used as miners the slaves sold in the market because of their crimes; for, in addition to the painfulness of the work, they say that the air in the mines is both deadly and hard to endure on account of the grievous odor of the ore, so that the workmen are doomed to a

6.808-815)¹¹ also remarked on the poor ventilation at mines, and dangers of fumes and smoke. Approximately 80,000 to 100,000 metric tons¹² of pollution was present in the air from ancient mining and smelting activities (Nriagu 1996, Hughes 2014). Pollution from runoff and other activities was leached into the surrounding ecosystem, affecting plants and animals (Hughes 2014). The aftermath of ancient copper smelting activity in Jordan causes issues today, thousands of years later. Copper is present in the soil and domestic areas, but bioaccumulation of metals is also seen in the plant and animal life. The same movement of toxins in the food chain would have likely been present in industrial areas of ancient Greece, and passed onto those who ate it through plants, animals, and animal by-products (Grattan et al 2003, Pyatt et al 2000, Pyatt et al 1999). The affect of bioaccumulation and biomagnification should be kept in mind when considering those food sources and habitations which were in close proximity to toxic areas. For example, the plains just north of the Laurion mines were an important source of food for ancient Athens.

Although there is no way of knowing how far midwives travelled to deposit infants in the well, the area around the Agora can be considered for possible detrimental environmental factors which would have affected a developing fetus. Lead was present in the city, in the joints of aqueducts, lead pipes (Vitruvius, *On Architecture*, 8.6.9-11)¹³, white pigments (Theophrastus, *On Stones* 56)¹⁴, and from acidic food being cooked in silver and lead vessels. Temples and shrines surrounded the Agora but there is also archaeological evidence that buildings had been used for industrial work between the Areopagus and Hill of the Nymphs (Morgan 2010). This industrial work included marble working (dust), bronze casting (smoke and metals), and dyeing (smell and water pollution) (Morgan 2010). Smell and air quality pollution was noted in the city from tanning¹⁵, dyeing¹⁶, smelting, and casting (Hughes 2014, Humphrey et al 1998); however, smelting

quick death. What is more, the mine is often left idle because of the unprofitableness of it, since the workmen are not only more than two hundred in number, but are continually spent by disease and death.” (quoted in Humphrey et al 1998)

¹¹ Lucretius, *On the Nature of Things*, 6.808-815: “What foul smells Scaptensula [a mining town in Thrace] breathes out from below! What harm comes when the gold mines let loose their fumes! What an appearance they give men, and what pallor! Don’t you see or hear in how short time they usually die, and how the source of life fails the men whom the great force of compulsion binds to such work?” (quoted in Humphrey et al 1998)

¹² During the Roman Empire, 80,000 to 100,000 metric tons/year of lead were found to be produced, almost four times greater than background lead content. A similar rise in lead pollution was seen in the 11th century, with the greatest growth occurring in the 16th century, and peak in the 1970s (Nriagu 1996).

¹³ Vitruvius: *On Architecture*, 8.6.9-11: [On why terracotta pipes are preferred] “Lead seems to make water harmful for this reason, that it generates lead carbonate, and this substance is said to be harmful to the human body. So if what is generated by it is harmful, it cannot be doubted that it is itself not healthful. Lead workers can provide us with an example, since their complexions are affected by a dead pallor. For when a blast of air is used in casting lead, the fumes from it infiltrate the parts of the body and, subsequently burning them up, it deprives the limbs of the virtue of their blood. And so it seems that water should in no way be carried in lead pipes if we wish to keep it healthful. Our everyday dining can show that the flavour of water from terracotta pipes is better, for everybody, even when they have tables piled high with silver dishes, nevertheless uses pottery to preserve the taste.” (quoted in Humphrey et al 1998).

¹⁴ Theophrastus: *On Stones*, 56: “Lead about the size of a brick is put in jars above vinegar. When it acquires a thick coat, which it usually does in ten days, the jars are opened and a type of mould is scraped from the lead; and the lead is placed in the same way again and again until it is all consumed. The part that has been scraped off is pounded in a mortar and continually filtered off. The white lead is the material finally left at the bottom.” (Quoted in Humphrey et al 1998).

¹⁵ Artemidorus, *Interpretation of Dreams*, 1.51; 2.20: The tannery is an irritant to everyone. Since the tanner has to handle animal corpses, he has to live far out of town, and the vile odour points him out even when hiding...The vultures are companions to the potters and the tanners since they live far from towns and the latter handle dead bodies. (quoted in Humphrey et al 1998)

may not have been present near the centre of the city during the Hellenistic period, due to the smell, noise, and heat. Olynthus, in northern Greece, shows this type of city planning, separating domestic and industrial activity (Morgan 2010, Cahill 2005), but Athens does not appear to have the same planning.

There was little differentiation between domestic and industrial activities around the Agora, and some buildings contained evidence for both habitation and industrial activity (Morgan 2010, Tsakirgis 2005). The well containing the infants was originally dug to provide water to a bronze casting workshop, close to other homes (Liston unpublished). The lack of differentiated areas was the result of the continual habitation of the Agora since the Neolithic period (Morgan 2010), allowing industrial wastes to be present in the home. Even if the waste had not been produced in the area for some time, residual pollution in the ground would have affected plants, animals, and future of the occupants of the site.

Smoking has been shown to be weakly associated with oroclefting; however, tobacco was not introduced to Europe for almost 1400 years after the Hellenistic period. However, smoke from the burning of other substances, such as incense, was involved within rituals in ancient Greece (Dannaway 2010). This may have led to the issues of smoke inhalation, and altering elements within those substances may have adversely affected a fetus.

Smoke pollution from fires was also an issue in ancient cities. Heating of bath houses, kilns and workshops would produce smoke and dust within the city (Hughes 2014). Heating, lighting, and cooking would all produce smoke and dust within the household. There is little evidence that the design of the ancient Athenian household accommodated smoke ventilation (Tsakirgis 2007). The elements being burned (wood and charcoal) may have not caused issues upon inhalation; however, the inhalation of smoke could cause respiratory distress or exacerbate respiratory infection. This is important as maternal stress and illness, specifically fever, has been identified as a oroclefting risk factor.¹⁷

Although the smoke and industrial waste present in the city was noted, ancient medical writers appeared to have no concept of “public health.” An ancient doctor would have focused on the individual rather than the entirety of the community. An individual’s reaction to an environment was thought to be the cause of illness, rather than the environment itself (Nutton 2000). It is therefore unlikely that a medical doctor would have identified the environment as a cause of birth defects, but instead would have focused on the mother’s actions.

Alcohol is known to cause cleft palate and other defects. Wine was a staple in the Greek and Roman diet; however the alcoholic strength of ancient wine is unknown. Hesiod (*Works and Days*, 609-14)¹⁸ describes leaving the grapes to dry out for ten days and nights, making a sweet wine, high

¹⁶ Strabo, *Geography*, 16.2.23: [Describing the island of Tyre, famous for production of Tyrian purple dye] Although the great number of dye-works makes the city unpleasant to live in, nevertheless it makes the city rich since its people are so enterprising. (quoted in Humphrey et al 1998)

¹⁷ The chance of infection would have also been quite high in the ancient world, due to many factors, such as poor hygiene, sanitation, and malaria, which all also lead to fever.

¹⁸ Hesiod, *Works and Days*, 609-614: Show [the grape clusters] to the sun ten days and ten nights: then cover them over for five, and on the sixth day draw off into vessels the gifts of joyful Dionysus.

in alcohol, before it was diluted with water (Donahue 2015, Cook et al 2007). Galen and Eubulus wrote of the ill effects of wine, which worsened with increased consumption (Leibowitz 1967, Cook et al 2007). Alcohol was recognised as causing deformity in an infant; however, this was due to paternal drunkenness at conception, rather than maternal consumption throughout pregnancy (Calhoun and Warren 2007, Abel 1999).

It was believed that a maternal source of deformity was by her psyche and her thoughts at conception (e.g. what she was looking at) rather than her actions throughout pregnancy (Soranus, *Gynecology* 1.10, 1.12). It is unlikely that a woman would have stopped drinking wine when she was pregnant; however, it is unknown if the daily consumption of ancient wine was enough to be harmful, and if the alcoholic content of ancient wine was enough to harm a fetus. A mother's consumption of certain foods throughout pregnancy was thought to affect the fetus; however those foods were feared for causing miscarriage, rather than defects (Soranus *Gynecology* 1.46).¹⁹

Nutritional deficiencies were present throughout the ancient world, associated with socioeconomic status and the availability of food in the city. Table 1 shows the nutritional factors which have been studied for their effect on oroclefts and their most common food sources. With these food sources in mind, we can examine the ancient diet to attempt to locate any areas of questionable quality.

Table 1: Important nutritional compounds for development and their common sources. All sources from the Dietitians of Canada 2015.

Nutritional Compound	Sources
Folate	Dark green vegetables (spinach, broccoli) asparagus, artichoke, legumes (beans, chickpeas, lentils), liver
Zinc	Oysters, crab, liver, beef, seeds, beans, peas and lentils
Vitamin B6	Prunes, liver, kidney, pork, chicken, chickpeas, lentils , pistachios
Vitamin A	Liver, fish, dark green, yellow, orange and red vegetables
Riboflavin	Milk and dairy products, mushrooms, meat, liver, fish, almonds
Vitamin B12	Milk and dairy products, meat, fish, yeast
Pantothenic Acid	Chicken, beef, liver, kidney, yeast, egg, broccoli, whole grains

Cereals, mainly barley and wheat, were the staples of the Mediterranean diet (Braun 1995, Donahue 2015). Barley was the most common cereal for daily consumption and use in sacrifice and ritual (Braun 1995). Barley was also easier to grow than wheat as it required less water, but wheat

¹⁹ Soranus recommended that a mother avoid heavy, greasy, and pungent foods, as they were most likely to disturb the attachment of the fetus to the womb, and potentially cause a miscarriage

was grown during the rainy winter months and imported from abroad (Braun 1995, Donahue 2015)²⁰. Other common crops included: kidney beans, peas, broad beans, lentils, chickpeas, figs, apples, pears, plums, apricots, pomegranate, lemons, cabbage, asparagus, artichokes, cucumbers, garlic, onions, and leeks (Hughes 2014).

Olives were a main tree crop, due to their multi-purpose usage, but also the ability of the trees to grow in almost anywhere and provide a reliable harvest (Donahue 2015). Figs were an important fruit in ancient society, not just for their sweetness and use in desserts, but they also had more calories per unit than any other crop, and a reliable production (Strabo, *Geography*, 14.1.27, Donahue 2015). Beans were overlooked by the elite, but utilized by the poor as a “poor man’s meat” and a substitution for grain. Although versatile and providing lacking nutrients like proteins, beans were associated with a wide variety of disorders, least of which was flatulence, but also weakness, jaundice, haemoglobinuria²¹, and death (Donahue 2015).

Meat was incorporated into stews and soups, with vegetables, and heavy seasoning (Bottero 1995). Nearly all consumable meat came from ritually slaughtered animals in public feasts, festivals, political functions, and ceremonies within the home. It is estimated that a citizen, an adult male, would receive two kilograms of beef in a year from these events (Gallant 1991). Goats and pigs, along with vegetables, legumes, wine, fish and fowl, would also be disbursed (Gallant 1991). Hunting mainly existed as sport for the elite, and the poor practising it in a supplementary manner. Hunting may have been limited due to the large scale deforestation, destroying habitats. Fish and other marine animal bones were also found in rubbish dumps, indicating another source of meat in some regions (Gallant 1991). Despite its seeming availability, meat largely remained a luxury item and was consumed less than vegetables and grains, and even less by the poor (Donahue 2015).

Livestock, like goats, cattle and sheep, could be used as a beast of burden and a milk producer. Their value as a sacrifice came last. Keeping livestock was seen as more profitable than agriculture, though more expensive overall (Howe, 2014a,b). Cattle were seen as the most valuable and profitable²², serving as a status symbol, but they were the most expensive animal to keep (Howe 2014a,b). Goats and sheep were likewise prized, but cheaper to keep as they could graze on less valuable plants and utilize less land (Howe, 2014a,b). In ancient Greece, the value of these animals alive may have increased, as no year round meat market existed outside of sacrificial needs (Howe 2014b).

Milk was used not only as a beverage, but added to recipes (Solomon 1995, Bottero 1995). Milk derivatives like dry, salted cheese could be kept longer in hotter climates (Dar 1995), providing additional protein and fats into the diet. It is likely that not everyone was in a position to consistently maintain milk-producing livestock, nor was everyone able to consume milk products.

²⁰ In the sixth century BCE, Solon enacted a law in which brides were required to bring barley roasters to their weddings, which speaks to the importance of this grain. It was not until the rise of the Roman Empire that emmer wheat would surpass barley as the main staple cereal, despite the increased workload that it entailed (Braun 1995, Donahue 2015).

²¹ A condition which causes abnormally high levels of haemoglobin in the urine, making it purple.

²² Cato, *De Agricultura* 54-5: There is nothing more profitable than to take care of cattle. (quoted in Howe 2014b)

Ancient poets were aware of lactose intolerance, or at least that eating milk products had unpleasant repercussions (Craik 1995).²³

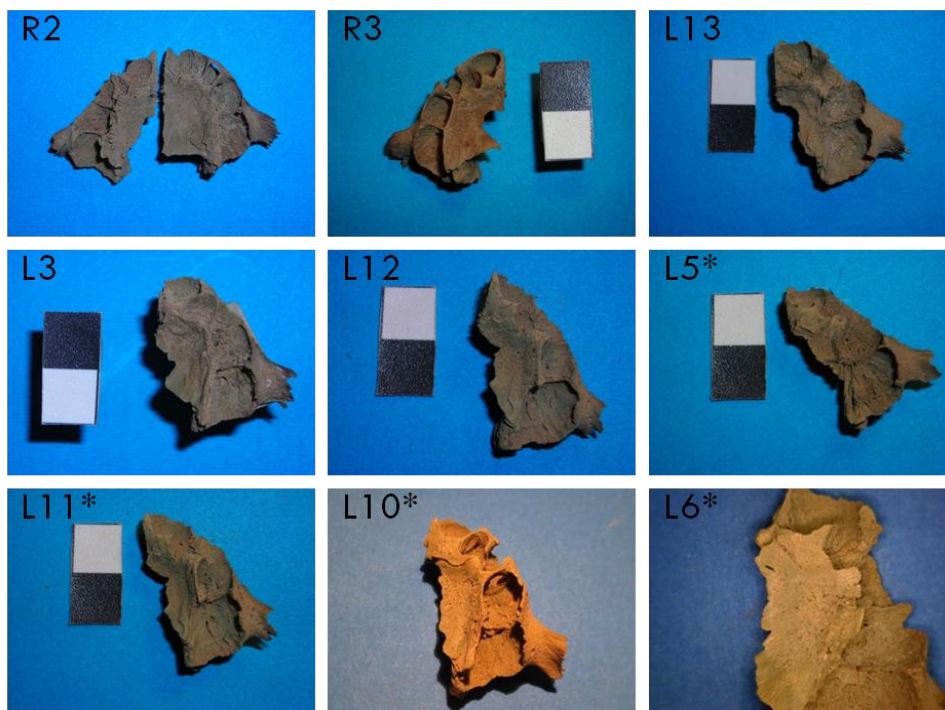
It is important to consider the diet of the everyday person, rather than relying descriptions of banquets to discern the quality of diet. J. Lawrence Angel (1975) described the early Hellenistic period (300 BCE) as having a balanced and adequate diet. Waterlow (1989) also found that the Greek and Roman diet would have been “adequate”, based on grain rations; however, he warns that unequal distribution of rations disrupt that conclusion. There is little to no quantitative evidence for the amount or quality of food that was given out or available. The ancient diet was sufficient as populations were maintained, however the inequalities in the availability of food and the variety and quality of foods can be considered as a possible source of birth defects in the ancient world.

²³ Hippocrates, *On Ancient Medicine* 20: Cheese does not harm all people alike, and there are some people who can eat as much of it as they like without the slightest adverse effects; indeed it is a wonderfully nourishing food for the people it agrees with. But others suffer dreadfully... (quoted in Craik 1995)

2.8 Cleft Palate and the Athenian Agora

Within the Agora well, 164 maxilla halves were excavated, 89 right sides and 75 left sides. Nine maxillae bones, seven left-sides and two right-sides, were found to exhibit cleft palate (Figure 1). In addition, two maxillae were reported to show evidence of clefting on premaxilla (Liston unpublished). In June 2015, I visually examined the maxillae at the Athenian Agora with use of a “Dino-Lite” digital microscope. I identified four of the nine maxillae, all left sided bones, as having the extent and shape of the clefting as questionable, due to some post-mortem damage on the palate margin. The remaining five maxillae (two right sided, and three left sided bones) exhibited incomplete growth along the majority of the palatal shelf. I was not able to confidently identify clefting of the premaxilla with the tools I had available²⁴.

Figure 1: The nine maxillae exhibiting cleft palate from the Athenian Agora. Maxillae which were identified as having a questionable extent of clefting are marked with an asterisk. Photo Credits: Dr. M. Liston and Alisha Adams



The margin of the cleft was determined to be the product of growth due to the curved and rounded nature of the palatal line, rather than the sharp, irregular surfaces of broken bones. Taphonomic influences were ruled out as causing the cleft palates as there was no evidence of environmental weathering, animal activity, extensive post-mortem breakage, that the bones were previously buried in a different location, or exposed above ground before being placed into the well (Liston unpublished).

²⁴ Three maxillae were marked as having a questionable extent of clefting in Liston and Rotroff 2013. I believe my numbers are different as I did not have a high powered microscope to examine the cleft margins thoroughly.

2.9 Differential Diagnosis

Perinatal trauma was ruled out as the origin of the clefts, since the remaining maxillary bone was not damaged. It is difficult to image an injury which would have broken the palate and not the remainder of the maxilla in a significant manner. Likewise, the rounded, grown edges would not have been the result of healing in the perinatal period. Infection was also ruled out as the origin of the clefts for several reasons: other areas of the maxillae were not seen to be affected; *in utero*, an infant would be unlikely to survive or maintain an infection with osteoclastic activity, and a severe infection would have resulted in an early termination; if an infection was acquired after birth, it is unlikely that centralized bone destruction would have taken place in the perinatal period; and though cranial bones in the collection were shown to have osteoblastic activity due to infection (Liston unpublished), we do not see the same reaction on the maxillae.

Unfortunately, precise ageing could not be determined for the maxillae. Due to the pathological nature of the bones, the growth of the bone could not be compared to aging standards of normal palates (Scheur and Black 2000). Additionally, no traditional bones used for perinatal aging could be paired with the maxillae. This is due to the fact that the bones were not identified as human in their original excavation, and their positional contexts were lost. The affected maxillae were also unable to be paired with one another or the remaining 155 maxillae from the well. Due to the fact that at least another 264 maxillae were not recovered from the well, we cannot confidently say that these palates could not be matched to another infant from the well or if they were either unilaterally or bilaterally affected. If this assemblage of palates follows modern trends, we may suggest that they were possibly unilateral cleft palates.

An interesting aspect of this assemblage is that only two of nine maxillae could be said to show attributes affecting the premaxilla. This means that seven of the nine palates show evidence of cleft palate alone. Cleft palate alone occurs in 1 in 2000 births, where cleft lip and palate presents in 1 in 1000 births. We can assume that the prevalence would have been the same in ancient society, as there is no reason to think causal factors would have been different for these defects (Turkel 1989, Roberts 2005). We would expect to see a higher ratio of the palates affecting both the premaxilla and the palate; however this is not the case. Although this sample is small and only includes infants that died and were placed in the well, this may still be significant.

In order to calculate if this is a significant number of cleft palates in comparison to modern society, an estimation of the total number of births (still and live births) must be known. Unfortunately we have no way of knowing exactly how many births occurred at this time, however we know that at least 457 infants died and were placed in the well. From the number of deaths, we may be able to estimate the number of births, and to suggest prevalence for cleft palate in Late Hellenistic Athens.

The infant mortality rate is the number of deaths within a year per 1000 births. The analysis of age shows that the 457 infants died in the perinatal period. The perinatal mortality rate is the number of deaths within 7 days of birth per 1000 births. No perinatal mortality rate has yet to be published for ancient society. We may look at historical statistics for comparison, although perinatal and infant mortality rates have dropped dramatically over the past 50 years due to

perinatal care, improved sanitation, and emergency care (WHO 2006). Therefore, mortality rates from the past can be assumed to have been greater than they are today.

In the late Roman Empire, the infant mortality rate was estimated to be 300 in 1000 (Golden 2015²⁵, Frier 1982) (Table 2). Though this is a Roman statistic, we may consider it a relatively comparable society to Hellenistic Athens. Even within the past 300 years, the infant mortality rate has stayed in the hundreds, as seen by the infant mortality rate in London between the years of 1675 and 1849, which averaged 262 deaths per 1000 births (Galley and Shelton 2001). We may suggest that the infant mortality in Hellenistic Greece would have also stayed in this same range of 200-300 deaths per 1000 births. With this range in mind, we can find a comparably high infant mortality rate in modern society. The highest in the world is Sierra Leone at 117.4/1000. It has a high perinatal mortality statistic as well, 90/1000. We would see a similarly high perinatal mortality statistic in ancient society, as we still see 75% of all infant death occurring in the perinatal period, and a high percentage of newborn death occurs in low to middle income countries (WHO 2011).

²⁵ Golden implied this was an ancient Greek statistic, but his source, Frier 1982, is utilizing Roman tax records from the third century AD.

Table 2: Perinatal and infant mortality rates of various regions, and cleft lip and palate and cleft palate prevalence. All Regional Data and Mortality Rates from WHO 2006, unless otherwise indicated.

Region	Perinatal Mortality (per 1000 births)	Infant Mortality (per 1000 births)
Late Roman Empire (300 AD)	-	300 ²⁶
1675 - 1849 London	-	262 ²⁷
More Developed Countries (2006)	10	-
Less Developed Countries (2006)	50	-
Least Developed Countries (2006)	61	-
Global (2006)	47	53 ²⁸
Europe (2006)	13	18.5 ²⁹
Asia (2006)	50	64.1 ³⁰
Africa (2006)	62	93.7 ³¹
Sierra Leone (2006)	90	117.4 ³²

²⁶ Golden 2015

²⁷ Galley and Shelton 2001

²⁸ WHO 2015b

²⁹ WHO 2015b

³⁰ WHO 2015b

³¹ WHO 2015b

³² WHO 2013

Table 3: Regional Perinatal and Infant Mortality Rates and Calculated Estimate Ancient Cleft Palate Prevalence. Perinatal Mortality Rates from WHO 2006, unless otherwise indicated.

Region	Perinatal Mortality (per 1000 births)	Infant Mortality (per 1000 births)	Estimated Agora Well prevalence of Cleft Palate
Less Developed Countries (2006)	50	-	1 in 1,306
Least Developed Countries (2006)	61	-	1 in 1070
Global (2006)	47	53 ³³	1 in 1389
Africa (2006)	62	93.7 ³⁴	1 in 1053
Sierra Leone (2006)	90	117.4 ³⁵	1 in 725
			Average: 1 in 1109

Equation 1: Sample Calculation of Estimated Number of Births

$$15\left(90 \text{ Deaths}/1000 \text{ Births}\right) = 457 \frac{\text{deaths}}{15 \text{ years}} / X \frac{\text{births}}{15 \text{ years}}$$

$$X \frac{b}{15\text{yr}} = \frac{457 \frac{d}{15\text{yr}} \times (15\text{yr}) 1000 \frac{b}{\text{yr}}}{(15\text{yr}) 90 \frac{d}{\text{yr}}}$$

$$X = \frac{457,0000 \frac{b}{15\text{yr}}}{47}$$

$$X = 5078 \frac{b}{15\text{yr}}$$

³³ WHO 2015b

³⁴ WHO 2015b

³⁵ WHO 2013

Equation 2: Sample Calculation of Estimated Cleft Palate Prevalence

$$\left(7 \frac{CP}{15yr} / 5078 \frac{b}{15yr}\right) \div 15years = 0.47CP/338b$$

$$0.47CP/338b = 1CP/725b$$

Estimated Agora Well Cleft Palate Prevalence = 1 in 725

Table 4: Global Regional Cleft Lip and Palate and Cleft Palate Prevalence and Calculated Estimated Athenian Agora Cleft Palate Prevalence. Cleft Lip and Palate and Cleft Palate Prevalence averaged from Mossey and Little 2002.

Region	Cleft Lip and Palate Prevalence (per 1000 births)	Cleft Palate Prevalence (per 1000 births)	Estimated Agora Well prevalence of Cleft Palate (per 1000 births)
Global	0.51 to 1.99 (average 1.7)	0.21 to 0.655 (average 0.5)	0.72 to 1.37 Average 0.9
Europe	0.67 to 1.46	0.36 to 0.97	
Asia	0.97 to 2.13	0.15 to 0.73	
Africa	0.2 to 1.63	0.02 to 0.4	
USA	0.29 - 1.99	0.22 to 1.11	
India	0.77 to 1.32	0.32 to 0.48	
Far East	0.97 to 2.13	0.15 to 0.73	

Using known perinatal mortality rates, an estimated prevalence of cleft palate can be calculated for each region (see Table 3. See Equations 1 and 2 for sample calculations). The average estimated prevalence for the well is 1 in 1109 (or 1.8 in 2000 or 0.9 in 1000) If the modern prevalence is 1 in 2000 (0.5 in 1000), this is nearly double the expected rate, and above many areas of the world (Table 4). The prevalence in the Agora becomes even more notable when you consider the highest modern prevalence rate, 1.37 in 1000, was calculated from Sierra Leone’s perinatal mortality statistic. This prevalence rate far above the global averages, but is also the rate calculated from an area which most closely resembles the infant mortality rate of ancient society. The ancient infant mortality rate was almost three times higher than today, and if ancient perinatal infant mortality rate was even half of the infant mortality rate, we would see greater prevalence estimated for the well (i.e. if Ancient Rome’s perinatal mortality rate was 150/1000, this would estimate a prevalence of 4.6 in 2000). Therefore, we can take a very tentative working assumption that the

prevalence of cleft palate in the well may be indicative of a change in causation producing a much higher prevalence in the ancient city.

What would account for this? There is nothing to suggest that ancient Greeks would have been exposed to drastically different factors than today to raise the prevalence rate. Similar environments, such as India and Africa, show lower prevalence rates than those found in the well. While 50% of cleft palates are thought to be caused by environmental and teratogenic effects, the remaining fifty percent are thought to be caused by syndromes, either directly or secondary to other conditions.

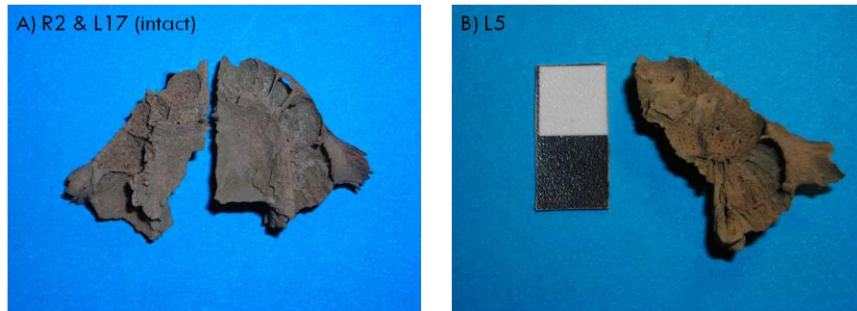
Therefore, syndromic clefting must be considered. The prevalence of cleft palate in the well is unlikely given modern epidemiology; even modern areas that have environmental factors associated with cleft palate maintain a constant cleft palate prevalence rate. This differs only when syndromes are causal factors, such as families with genetic dispositions to clefting, like Van der Woude's syndrome (Saal 2002)³⁶. Even though we cannot identify if the lip was involved, the existence of a cleft lip, no cleft premaxilla, and a cleft palate is known to be the result of a syndrome (Saal 2002). Therefore, it is possible that some of the cleft palates within the well were the result of syndromes, rather than the environment.

The shape of the cleft may further indicate the etiology. Pierre Robin's sequence is a set of physical anomalies which seen together in many syndromic conditions. This involves the tongue gathering at the back of the throat, glossoptosis, and breathing problems (Saal 2002). On the skeleton, a micrognathic and retrognathic mandible is present, and will often be accompanied by a "U" shaped cleft palate.³⁷ The irregular development of the mandible does not allow the fetal tongue to depress at the right time, therefore blocking the palatine processes from rising, creating a cleft palate around the tongue (Saal 2002). Clinically, the shape of the palate should indicate if Robin's sequence is present. If a cleft has formed around the fetal tongue, we should see a "U" shape to the cleft, in contrast to the subtle "V" shape of a 'naturally' occurring palate (Saal 2002). However, the "U" and "V" shape is defined by a combination of the bone and soft tissue, and it is unclear if this pattern is present in the bone alone. I find it difficult to judge the shape in the maxillae we have due to this (Figure 2). We also only have one side of each of the maxillae, and breakage along the line of cleft obscures the shape of the palate. I was not able to identify clear "U" and "V" shapes in these maxillae for these reasons.

³⁶ A prevalence rate for syndromic cleft palate is not available due to the fact that syndromes causing clefting occur at different frequencies, and there are several hundred different syndromes which are known to cause oroclefting, such as Van der Woude's syndrome, Strickler's Syndrome, Pierre Robin's Sequence, holoprosencephaly, and Catel-Manzke syndrome.

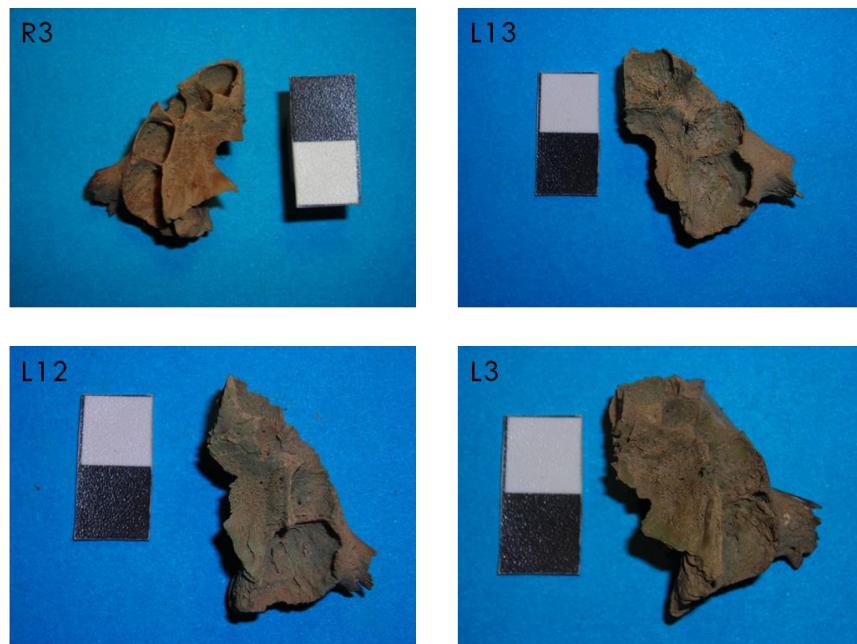
³⁷ This sequence is most common in Stickler Syndrome (Saal 2002).

Figure 2: (a) A right maxilla exhibiting cleft palate compared to an intact left maxilla (b) A left maxilla exhibiting cleft palate. Analyzing the shape of the cleft has the potential to determine a possible syndromic etiology. Photo Credit: Dr. M. Liston.



Even though the shape of palate may not indicate the etiology of the cleft palates, looking at deviance in the line of the cleft may. In the maxillae collection, four maxillae showed evidence of significant “uneven” growth at the line of the cleft (Figure 3). Though a cleft palate by definition exhibits abnormal growth, significant inward growth and distinctive curves can be seen on palates R3 and L13. L3 and L12 also show an inward and outward growth of the line of the cleft, though not to the extent of R3 and L13; however, post-mortem breakage obscures the full cleft profile on some.

Figure 3: Four Maxillae which show an “uneven” growth at the midline. Photo Credit: Dr. M. Liston.



Cysts and oral synechia are known to cause or be associated with cleft palate, and may or may not be the result of syndromes. They may also affect the growth of the palate. One of the most common perinatal cysts, Bohn’s nodules, usually develops in the alveolus (Sperber 2002). Nasopalatine duct cysts occur at the midline in the anterior portion of the palate, but are not common in children (Sperber 2002, Cecchetti et al. 2012). Without further soft tissue evidence, the cause of a midline cyst in an infant is difficult to identify. Greggs et al (1983) identified several clefts as the result of cysts; however, the skulls exhibited multiple cysts, and evidence of infection on

multiple bones. As they also occurred in adults, it is not clear if the infant maxillae in the well may have similar origins.

Oral synechia are soft tissue adhesions which develop from the palate and can insert into the tongue, alveolar ridges, and floor of the mouth. They occasionally occur with cleft palate and can insert at the midline, however it is still unclear if synchia create a cleft in the palate; are caused due to errors in the epithelium of a cleft palate; or if synchia and cleft palate have separate etiologies caused by the same factor (Sybil and Sagtani 2013). A modern case study reported on five family members with cleft palate and synechia, strongly suggesting a genetic cause (Sybil and Sagtani 2013). By examining the palates further, we may be able to identify if their growth was affected by either of these factors.

Figure 4: R2, a “normal” cleft palate, compared against an intact maxillae, and R2’s corresponding magnification of the palatal shelf, showing a normal porosity. Photo Credits: Dr. M. Liston and Alisha Adams

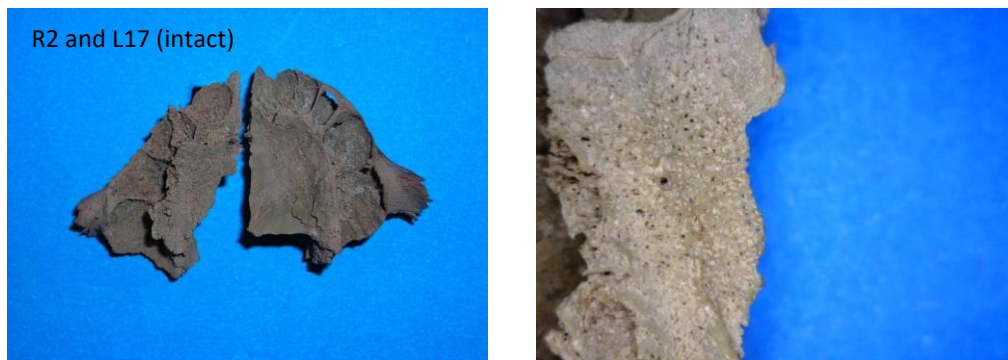
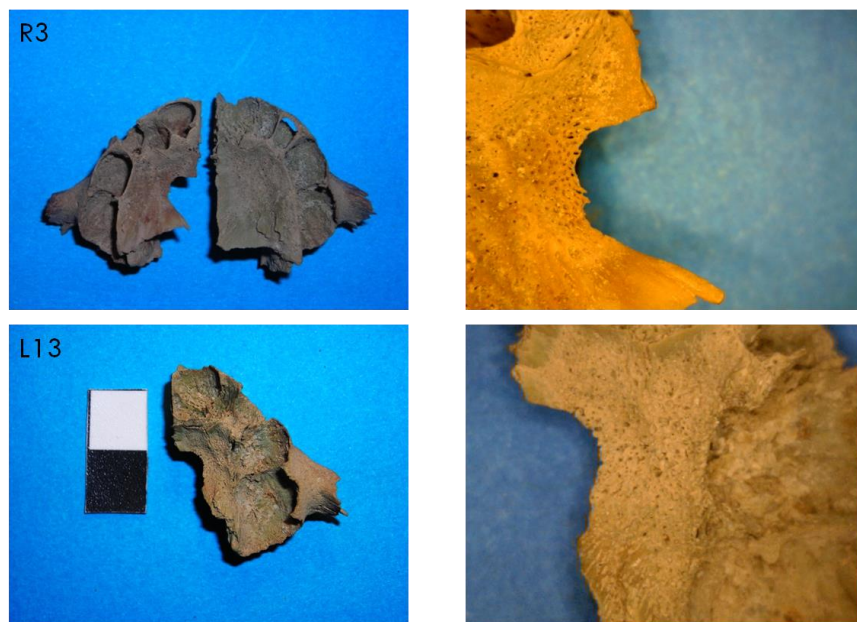
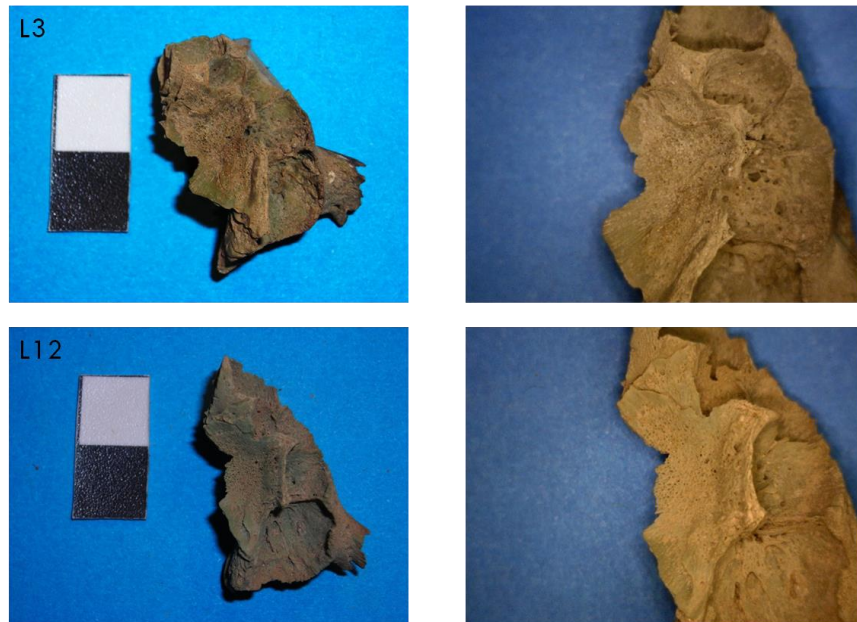


Figure 5: Maxillae R3 and L13 and their corresponding magnifications of the palatal shelf, showing enlarged porosity. Photo Credits: Dr. M. Liston and Alisha Adams



In Figure 5, the posterior portion of the palate of R3 and L13, where the hard palate meets the soft palate, has grown “forward”, towards the anterior of the mouth. This is more prominent in R3. They each show greater porosity at the apex of the curvature than the normal cleft, Figure 4. This may be a symptom a physical impediment of some sort at the midline, like a cyst.

Figure 6: Maxillae L3 and L12 and their corresponding magnifications of the palatal shelf, showing small to normal porosity. Photo Credits: Dr. M. Liston and Alisha Adams.



In comparison, L3 and L12 (Figure 6), have porosity similar to that of the normal cleft Figure 4. They also lack the definitive obvious curvature when compared Figure 5. This inward and outward growth of the palate may just be a symptom of irregular growth of the cleft palate, rather than a physical impediment.

It is reasonable to suggest that syndromic etiologies may have been a possible origin for the cleft palates in the Athenian Agora. While a concentration of birth defects in one area over a period of may indicate a familial or “homogenous population” (Roberts 2005), this assemblage is small and the origins too variable to confirm a homogenous population. Due to the fact that the infants came from an unknown area of the city, it is also not possible to say if it was familial syndrome which caused some of the palates, if any of the infants were related, or if it was the same syndrome at all. A syndrome may not always produce the same pattern of defects on each individual affected, and the evidence which would be visible on the bone would be highly dependent on the syndrome in question (Roberts 2005, Saal 2002).

2.10 Conclusion

Developmental defects in the archaeological record provide the opportunity to study the pathological presentation as well as the etiology of a defect. Case studies often fail to examine defects within the environmental and social context. I was able to illuminate several factors that may have contributed to the occurrence of cleft palate in the infants from the Athenian Agora by integrating a multi-field approach to studying birth defects. By understanding the environmental risk factors present in the ancient environment, I identified the possibility that the occurrence of cleft palates in the Agora was unusually high, and was able to suggest that their origin may have been the result of syndromes.

I hope this work will encourage the continued study of infant bones, and particularly, the recognition that incomplete palates should be examined carefully to distinguish breakage and growth patterns. I also hope my work can encourage skeletal biologists to carefully examine assemblages of birth defects in an effort to understand ancient causation.

Bibliography

Abel, Ernest L.

1999. "Was Fetal Alcohol Syndrome Recognized by the Greeks and Romans?" *Alcohol and Alcoholism*. 34(6): 868-872.

Agarwal, Sabrina C., and Bonnie A. Glencross.

2011. *Social Bioarchaeology*. Malden, MA: Wiley-Blackwell.

Alexandersen V.

1967. "The pathology of the jaws and temporomandibular joint." In D. Brothwell and A.T. Sandison, eds. *Diseases in Antiquity*, 551-595. Springfield: CC Thomas.

Anderson, T.

1994. "Medieval Example of Cleft Lip and Palate from St. Gregory's Priory, Canterbury." *Cleft Palate-Craniofacial Journal* 31(6):466-472.

Andersen, J. A. and K. Manchester

1992. "The Rhinomaxillary Syndrome in Leprosy: a Clinical, Radiological, and Paleopathological Study." *International Journal of Osteoarchaeology*. 2:121-129.

Angel, J. Lawrence.

1975. "Paleoecology, paleodemography and health." In Steven Polgar, ed. *Population, Ecology, and Social Evolution. International Congress of Anthropological and Ethnological Sciences*, 167-190. Chicago: Mouton Publishers, The Hague.

Arakei, G., V. Arali, and P. Brennan.

2010. "Cleft lip and palate: An adverse pregnancy outcome due to undiagnosed maternal and paternal coeliac disease." *Medical Hypotheses* 75:93-98.

Berndorfer, A.

1962. "A 500 year old skull with cleft lip." *British Journal of Plastic Surgery*. 15:123-128.

Bottero, Jean.

1995. "The Most Ancient Recipes of All." In John Wilkins, David Harvey, and Mike Dobson, eds. *Food in Antiquity*, 248-255. Exeter: University of Exeter Press.

Braun, Thomas.

1995. "Barley Cakes and Emmer Breads." In John Wilkins, David Harvey, and Mike Dobson, eds. *Food in Antiquity*, 25-27. Exeter: University of Exeter Press.

Brent, Robert L.

2004. "Environmental Causes of Human Congenital Malformations: The Pediatrician's Role in Dealing with These Complex Clinical Problems Caused by a Multiplicity of Environmental and Genetic Factors." *Pediatrics* 113(3):957-968.

Brooks S. and W. Hohenthal.

1963. "Archaeological defective palate in crania from California." *American Journal of Physical Anthropology* 21:25-32.

Brothwell, D. R.

1981. *Digging up Bones*. London. British Museum of Natural History, London.

Cahill, Nicholas.

2005. "Household Industry in Greece and Anatolia." In Bradley A. Ault, and Lisa C. Nevett, eds. *Ancient Greek Houses and Households: Chronological, Regional, and Social Diversity*, 54-66. Philadelphia: University of Pennsylvania Press.

Calhoun, Faye, and Kenneth Warren.

2007. "Fetal Alcohol Syndrome: Historical Perspectives." *Neuroscience and Biobehavioural Reviews*. 31:168-171.

CDC

2015, June 22. "Folic Acid: Birth Defects COUNT".
<http://www.cdc.gov/ncbddd/folicacid/global.html>, accessed November 30, 2015.

Cecchetti, F., L. Ottria, F. Bartulli, N.E. Bramanti, and C. Arcuri.

2012. "Prevalence, distribution, and differential diagnosis of nasopalatine duct cysts." *Oral & Implantology*. Apr-Sept 2012:5(2-3):47-43.

Christensen, Kaare.

2002. "Methodological Issues in Epidemiological Studies of Oral Clefts." In D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 101-107. New York: Oxford University Press.

Cook, Christopher, Helene Tarbet, and David Ball.

2007. "Classically Intoxicated." *British Medical Journal* 335(7633): 1302-1304.

Craik, Elizabeth

1995. "Hippocratic Diaita." In John Wilkins, David Harvey, and Mike Dobson, eds. *Food in Antiquity*, 343-350. Exeter: University of Exeter Press.

Czeizel, Andrew E.

2002. "Prevention of Oral Clefts through the use of folic acid and multivitamin supplements: Evidence and Gaps." In D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 443-457. New York: Oxford University Press.

Dannaway, Frederick R.

2010. "Strange Fires, Weird Smokes and Psychoactive Combustibles: Entheogens and Incense in Ancient Traditions." *Journal of Psychoactive Drugs* 42(4): 485-497.

Dar, Shimon.

1995. "Food and Archaeology in Romano-Byzantine Palestine." In John Wilkins, David Harvey, and Mike Dobson, eds. *Food in Antiquity*, 326-335. Exeter: University of Exeter Press.

Davidson, James.

1995. "Opsophagia: Revolutionary Eating at Athens." In John Wilkins, David Harvey, and Mike Dobson, eds. *Food in Antiquity*, 204-213. Exeter: University of Exeter Press.

Derry, D. E.

1938. "Two skulls with absence of the premaxilla." *Journal of Anatomy*. 72: 295-298

Dietitians of Canada.

2015. "Nutrition A-Z" <http://www.dietitians.ca/Your-Health/Nutrition-A-Z/>, accessed October 31, 2015.

Dietienne, Marcel, and Jean-Pierre Vernant.

1989. *The Cuisine of Sacrifice among the Greeks*. Chicago: The University of Chicago Press.

Donahue, John F.

2015. *Food and Drink in Antiquity: Readings from the Graeco-Roman World – A Source Book*. London: Bloomsbury Academic.

Frier, Bruce.

1982. "Roman Life Expectancy: Ulpian's Evidence." *Harvard Studies in Classical Philology*. 86: 213-251.

Gallant, Thomas W.

1991. *Risk and Survival in Ancient Greece: Reconstructing the Rural Domestic Economy*. Cambridge: Polity Press.

Galley, C. and N. Shelton.

2001. "Bridging the Gap: Determining long term changes in infant mortality in pre-registration England and Wales." *Population Studies* 55: 65-77.

Gladykowska-Rzeczyka, J.

1989. "Congenital Anomalies of early polish man." In L. Capasso ed. *Advances in Paleopathology*, 111-116 Journal of Paleopathology Monograph No.1. Chieti: Marino Solfanelli.

Gilbert, Scott

2010. *Developmental Biology*. 9th ed. Stamford: Sinauer Associates Inc.

Golden, Mark.

2015. *Children and Childhood in Classical Athens*. Baltimore: John Hopkins University Press.

Goodman, Alan H., and Thomas, L. Leatherman.

1998. *Building a New Bicultural Synthesis: Political-Economic Perspectives on Human Biology*. Ann Arbor: The University of Michigan Press.

Grattan, J.P., S. I. Huxley, and F. B. Pyatt.

2003. "Modern Bedouin Exposures to Copper Contamination: an imperial legacy?" *Ecotoxicology and Environmental Safety* 55: 108-115.

Gregg, J., M. Allison, S. Clifford, E. Gerszten and W. Klippel.

1983. "Ancient Inborn Facial Clefts and Nonodontogenic Fissural Cysts." *Plains Anthropologist* 28(102, Part 1): 293-304.

Hayes, Catherine.

2002. "Environmental Risk Factors and Oral Clefts." In D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 159-169. New York: Oxford University Press.

Howe, Timothy.

2014a. "Domestication and Breeding of Livestock: Horses, Mules, Asses, Cattle, Sheep, Goats and Swine." In: Gordon Lindsay Campbell ed. *The Oxford Handbook of Animals in Classical Thought and Life*, 99-108 Oxford: Oxford University Press.

2014b. "Value Economics: Animals, Wealth, and the Market." In: Gordon Lindsay Campbell ed. *The Oxford Handbook of Animals in Classical Thought and Life*, 136-155 Oxford: Oxford University Press.

Hughes, J. D.

2014. *Environmental Problems of the Greeks and Romans: Ecology in the Ancient Mediterranean*. Second ed. Baltimore: Johns Hopkins University Press.

Humphrey, John W., John P. Olsen, and Andrew N. Sherwood.

1998. *Greek and Roman Technology: A Source Book: Annotated translations of Greek and Latin texts and documents*. New York: Routledge.

Konigsberg, L.W., and M.A. Liston.

2013. "Paleodemography and perinatal mortality from the Agora well, Athens Greece." *American Journal of Physical Anthropology*. 150(56): 171-171.

Leibowitz, J. O.

1967. "Studies in the History of alcoholism – II. Acute Alcoholism in Ancient Greek and Roman Medicine." *British Journal of Addiction*. 62: 83-86.

Lewis, Mary E.

2009. *Bioarchaeology of Children: Perspectives from Biological and Forensic Anthropology*. Cambridge: Cambridge University Press.

Liston, Maria and Susan Rotroff.

2013 "Babies in the Well: Archaeological Evidence for Newborn Disposal in Hellenistic Greece." In Judith Evans Grubbs and Tim G. Parkins, eds. *The Oxford Handbook of Childhood and Education in the Classical World*, 62-82. New York: Oxford University Press.

Liston, Maria.

Unpublished. *The Bone Well*. Forthcoming manuscript, Department of Anthropology, University of Waterloo.

MacCurdy, G. C.

1923. "Human skeletal remains from the highlands of Peru." *American Journal of Physical Anthropology* 6: 217-330.

Morgan, Janett.

2010. *The Classical House*. Exeter: Bristol Phoenix Press.

Miller, E., and C. F. Merbs.

1993. "A case of cleft palate in an Anazi burial from Colorado." *Paleopathology Association Newsletter*, 87.

Molina-Solana, R., et al.

2013 "Current Concepts on the Effect of Environmental Factors on Cleft Lip and Palate." *International Journal of Oral and Maxillofacial Surgery* 42(2): 177-184.

Mossey P., J. A. Davies, and J. Little.

2007 "Prevention of orofacial clefts: does pregnancy planning have a role?" *Cleft Palate Craniofacial Journal* 44: 244-250.

Mossey, P. and J. Little.

2002. "Epidemiology of Oral Clefts: An International Perspective." In: D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 227-156. New York: Oxford University Press.

Mossey, P., J. Little, R. Munger, M. Dixon, and W. Shaw.

2009. "Cleft Lip and Palate." *Lancet* 374: 1773-1785.

Munger, Ronald.

2002. "Maternal Nutrition and Oral Clefts." In D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 170-192. New York: Oxford University Press

Ndreu, D.

2006. "Keeping bad science out of the courtroom: why post-Daubert courts are correct in excluding opinions based on animal studies from birth defect cases". *Gold Gate University Law Review* 36: 459-488.

Nriagu, Jerome O.

1996. "A History of Global Metal Pollution." *Science*. 272 (5259): 223-224.

Nutton, Vivian.

2000. "Medical thoughts on Urban Pollution." In Valerie M. Hope, and Marshall Eireann, eds. *Death and Disease in the Ancient City*, 65-73. New York: Routledge.

Ortner, Donald J.

2003. *Identification of Pathological Conditions in Human Skeletal Remains*. Second Edition. New York: Academic Press.

Phillips, S.M. and M. Sivilich

2006. "Cleft Palate: A case study of disability and survival in prehistoric North America." *International Journal of Osteoarchaeology* 16:528-535.

Pyatt, F.B., G.W. Barker, P. Birch, D.D. Gilbertson, J.P. Grattan, and D.J. Mattingly.

1999. "King Solomon's Miners – Starvation and Bioaccumulation? An Environmental Archaeological Investigation in Southern Jordan." *Ecotoxicology and Environmental Safety* 43:3-5-308.

Pyatt, F.B., G. Gilmore, J.P. Grattan, C.O. Hunt, and S. McLaren.

2000. "An Imperial Legacy? An Exploration of the Environmental Impact of Ancient Metal Mining and Smelting in Southern Jordan." *Journal of Archaeological Science* 27:771-778.

Roberts, Charlotte A.

2005. *The Archaeology of Disease*. Keith Manchester, ed. 3rd ed. Ithaca, NY: Cornell University Press.

Saal, Howard M.

2002. "Classification and description of nonsyndromic clefts." In D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 47-52. New York: Oxford University Press.

Sandison, A. T.

1980. "Notes on some skeletal changes in pre-European contact Australian aborigines." *Journal of Human Evolution* 9: 45-47.

Scheuer, L. and S. Black.

2000. *Developmental Juvenile Osteology*. London: Academic Press Limited.

Solomon, Jon.

1995. "The Apician Sauce – *Ius Apicianum*." In John Wilkins, David Harvey, and Mike Dobson, eds. *Food in Antiquity*, 115-131. Exeter: University of Exeter Press.

Soranus of Ephesus.

1991. *Gynecology*. Translated by T. Owsei. Baltimore: John Hopkins University Press.

Sperber, Geoffrey H.

2002. "Formation of the primary palate" and "Formation of the secondary palate." In D. F. Wyszynski, ed. *Cleft Lip and Palate: From Origin to Treatment*, 5-24. New York: Oxford University Press.

Sybil, D., and A. Sagtani.

2013. "Cleft palate lateral synechia syndrome." *National Journal of Maxillofacial Surgery* 4(1): 87-89.

Tsakirgis, Barbara.

2005. "Living and Working Around the Athenian Agora." In Bradley A. Ault, and Lisa C. Nevett, eds. *Ancient Greek Houses and Households: Chronological, Regional, and Social Diversity*, 67-82. Philadelphia: University of Pennsylvania Press

2007. "Fire and Smoke: hearths braziers and chimneys in the Greek House." In: "Special Issue: Building Communities: House, Settlement and Society in the Aegean and Beyond". *British School at Athens Studies* 15: 225-231.

Turkel, Spencer Jay.

1989. "Congenital Abnormalities in Skeletal Populations." In M. Yaşar İşcan and Kenneth A. R. Kennedy, eds. *Reconstructing Life from the Skeleton*, 109-128. New York: Alan R. Liss.

Waterlow, J. C.

1989. "Diet of the Classical Period of Greece and Rome." *European Journal of Clinical Nutrition*. 43(2): 3-12.

Weiss, E.

2014. *Paleopathology in Perspective: Bone Health and Disease through Time*. Maryland: Rowman & Littlefield.

Weingartner, J., K. Lotz, J. Fanghänel, T. Gedrange, V. Bienengräber, P. Proff.

2007. "Induction and Prevention of Cleft Lip, Alveolus and Palate and neural tube defects with a special consideration of B Vitamins and the methylation cycle." *Journal of Orofacial Orthopedics* 68:266-277.

World Health Organization (WHO).

2006. "Neonatal and Perinatal Morality Statistics: Country, Regional, and Global Estimates." http://apps.who.int/iris/bitstream/10665/43444/1/9241563206_eng.pdf, accessed November 2, 2015.

2011. "Newborn Death and Illness: Millennium Development Goal (MDG) 4." http://www.who.int/pmnch/media/press_materials/fs/fs_newborndhealth_illness/en/, accessed Nov. 20, 2015.

2013. "Sierra Leone : Neonatal and Child Health Profile." http://www.who.int/maternal_child_adolescent/epidemiology/profiles/neonatal_child/sle.pdf?ua=1, accessed October 31, 2015.

2015a. "Congenital Anomalies." <http://www.who.int/mediacentre/factsheets/fs370/en/>, accessed October 31, 2015.

2015b. "Global Health Observatory Data Repository: Child Mortality Levels." <http://apps.who.int/gho/data/view.main.CM1300R?lang=en>, accessed October 31, 2015.

Appendix: Published Archaeological Examples of Cleft Lip and Cleft Palate

Authors	Location	Time Period	Sex and Age	Pathological Description	Notes
MacCurdy 1923	Huisband, in the highlands of Peru near Cuzco	Pre-Columbian America	Adult Male	A small cleft on a board palate.	
Derry 1938	Egyptian site, east bank of the Nile.	700 BC (25th Egyptian Dynasty)	Adult Female	Absence of the premaxilla, hard palate is reduced in size. Mandibular prognathism.	
	Unknown location (assumed Egyptian)	Unknown	Adult Female	Premaxilla absent, and underdevelopment of the maxilla. Mandibular prognathism.	
Brooks and Hohenthal 1963	Newark site on the southeastern shore of San Francisco Bay (Museum of Anthropology collection, University of California, Berkley)	Late Middle Horizon Period – 2340 BP. (4000-2000 BC)	Adult male, 22-25	Cleft of the maxilla and palate, but atypical porosity on forehead and face. Possibly from injury.	Specimen 9859
	Newark site on the southeastern shore of San Francisco Bay (Museum of Anthropology collection, University of California, Berkley)	Late Middle Horizon Period – 2340 BP (4000-2000 BC)	Adult male, 30-40	Maxilla and palate have cleft defects.	Specimen 8474
	Sacramento County in California	2000 - 4000 BP.	Adult Female, 25-28	Unilateral hare lip. Porosity may be indicative of a reactive response to injury and/or infection.	Skull 22117
Berndorfer 1962	Southern Hungary	15th C. AD	Adult female, 25-30	Slight cleft lip and deformity of the right pyriform aperture	Later diagnosed as leprosy in Andersen and Manchester 1992
Alexanderse -n 1967	Unknown (Collection of the Laboratory of Physical Anthropology, Copenhagen)	Unknown	Adult female	Cleft Palate	No. 506/B 1923
Gladkowska-Rzeczyka, 1980, 1989:	Two instances of neolithic cleft lip and palate	Neolithic	Two adults	Cleft Lip and Palate	
Brothwell 1981	Cambridgeshire, England	6th to 7th C. AD	Child	Cleft palate is incomplete, and may have involved soft tissue.	Anglosaxon

Orther 2003	Unknown (Nubian Collection at the British Museum of Natural History)	Unknown	Adult female	Large bilateral cleft palate, involving the bilateral portions of the central and posterior portions of the hard palate.	BMNH 210 72/291
	Archaeological site in Kentucky, United States	Unknown	Adult Male	Bilateral cleft palate, aplasia of the vomer and conchae, hypoplasia of the left premaxilla, distortion of the left nares, and the central and lateral incisors never developed.	NMNH 243208. Diagnosed as a midpalatine cyst in Greggs et al. 1983
	Southwestern Colorado (National Museum of Natural History Collection at the Smithsonian Institution)	Prehistoric	Adult female, 18-20	Small cleft in the central portion of the maxillary suture.	NMNH 316482
	Pachamac, Peru (National Museum of Natural History Collection at the Smithsonian Institution)	Unknown	Child, 8-10 years old	Cleft on the left side of the maxilla, extending into the nasal cavity.	NMNH 293252
	South Pacific Island (Welcome Museum of Pathology, Royal College of Surgeons of England, London.)	Unknown	Unknown	Cleft of the left side of the palate and anterior maxilla.	WM A1.3
Sandison 1980	Unknown (Murray Black Collection of the Department of Anatomy of the University of Melbourne)	Unknown	Child, 12 years old	Hare lip and cleft palate	
	Unknown (Department of Anaomy of the University of Sydney)	Unknown	Child	Cleft Palate	
	Unknown (Australian Institute of Anatomy in Canberra)	Unknown	Two children	Cleft Palate	
Anderson 1994	Cantebury, England	11th-12th century	Adult Male, 40-50 year old.	Cleft Lip and Palate	SK 1134
Miller and Merbs 1993	Arizona, United States	12th century	Adult Female, 30-40 years old	Cleft Palate, and dental anomalies.	Anasazi
Phillips and Sivilich 2006	Grider Site, Late Woodland context, Pike County, Indiana, United States	500-1000 AD	Adult Male, 20-30s	Cleft Palate	