ORBITAL LESIONS IN HIGHLAND AND LOWLAND PERU

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by

Christina Sophia Alonso

San Francisco, California

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CERTIFICATION OF APPROVAL

I certify that I have read Title of Thesis by Christina Sophia Alonso, and that in my opinion this work meets the criteria for approving a thesis submitted in partial fulfillment of the requests for the degree: Master of Arts in Anthropology at San Francisco State University.

_____

Dr. Cynthia Wilczak,
Professor of Anthropology

_____

Dr. Mark Griffin,
Professor of Anthropology

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Christina Sophia Alonso

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Cribra orbitalia, or porosity of the bony orbit, has one of the most documented pathological changes. Until recently, most manifestations of orbital porosity were attributed to iron-deficiency anemia, however, Walker et al. (2009) convincingly argued that the megaloblastic anemias are the most likely cause for hyperostotic lesions. Porosity of the orbits can also result from scurvy, rickets, and inflammations of the orbit and surrounding soft tissue (Schultz 2001, Wapler et al. 2004, Ortner et al. 1999). During the research at the PAHMA, it was seen that there was no significant difference at the .05 level for most of the sites in terms of age/sex and classification of orbital lesions, or when testing for hyperostotic lesions between highland and lowland sites. There was a statistical significance when testing between adults with lesions and children with lesions. There are a variety of orbital lesions in Peruvian individuals; porosity, inflammation, and hyperostotic lesions. Each of these lesions can be caused by numerous conditions, and sometimes co-occur. Due to the co-occurrence of these lesions types, orbital lesions should be scored more vigorously using detailed methods in order to further distinguish differences between populations.

I certify that the Abstract is a correct representation of the content of this thesis.

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Chair, Thesis Committee Date

PREFACE AND/OR ACKNOWLEDGMENTS

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICIES

Appendix Page

CHAPTER 1 – INTRODUCTION

Paleopathology is the study of disease within ancient bones. This discipline has advanced since its inception in the mid 19th century. Each phase contributed to the overall understanding of a complex and dynamic field. New methodology and etiological inferences are constantly occurring and it is important to periodically re-evaluate and retest previous methodology in order to continue advancing the field and challenging previous assumptions and research.

One of the major focuses of paleopathology is orbital lesions due to the high prevalence of these skeletal manifestations in populations from numerous archaeological sites all over the world and the distribution through time. Orbital lesions are skeletal manifestations in the bony orbit that can be caused by many diseases such as various types of deficiency anemia, rickets, scurvy, and chronic inflammation. They can manifest in the bone as porosity, hyperostotic lesions, and increased vascularization. However, these impressions left in the bone can be caused by normal variation as well, the severity of the lesions can help researchers to determine if the lesions is pathological or within the realm of normal variation.

Besides chronic malnutrition being associated with these lesions, numerous other etiological reasons have been postulated for their manifestations. Previous researchers

have associated hyperostotic orbital lesions as being associated with iron deficiency anemia,

Differential diagnosis is a major focal point in paleopathology within the current literature. Many diseases can create similar manifestations on bone, such as the orbital lesions associated with both cribra orbitalia and scurvy (Marks and Hamilton 2007:217). These similar lesions necessitate a differential diagnosis in order to properly understand the nature of disease itself and its accurate diagnosis. Differential diagnosis views all possible causes for the lesions, pathognomic (diagnostic) features, compiles a detailed summary of the lesion and its physical form, and then chooses a most likely diagnosis.

Over the last hundred years, the understanding of cribra orbitalia has changed drastically. Hrdlicka described this type of lesion as a childhood condition, and thought it was a reaction to toxic substances in the body (1914). He believed it started with increased vascularity of the orbits, and then deposition of porous bone. At the peak of the disease, it was similar to, "a low growth of coral" (Hrdlicka 1914:58), but healing could drastically affect the macroscopic look of the lesion.

In 1966, J. Lawrence Angel associated porotic hyperostosis with types of iron deficiency anemia, and related them to falciparum malaria. It was postulated that these types of malaria were very common in children and were related to weaning and lactation as well as subsistence strategies.

In the 1980's-1990's Patty Stuart-Macadam delved into both the etiology and manifestations of cribra orbitalia and porotic hyperostosis (1985, 1987a, 1987b, 1991,

1992). Stuart-Macadam believed that iron-deficiency anemia in childhood and infancy caused the severe manifestations seen in children and not adults.

This study will examine orbital lesions in Peru using the new methodology set by Wilczak and Jeney (2008). By using this new classification system, the goal is to view orbital lesions in more detail in terms of manifestation and etiology without using just the one common explanation of cribra orbitalia. Since most researchers in the past have associated all orbital lesions with cribra, prevalence rates have been unusually high. By separating out true hyperostotic lesions from healing cribrotic lesions, inflammatory lesions, and porous lesions, more information can be gleaned about access to resources, sanitation practices, and other health stresses.

Throughout this thesis, three major questions are posed: (1) is there a difference between the prevalence of orbital lesions in highland and lowland individuals, (2) is there in fact a differences between the ages of individuals who show lesions within the bony orbit, and (3) is there a difference in orbital lesions between the sexes? Two additional questions were posed during the research phase of this thesis due to the abundance of literature indicating the possibility of geographical patterning in certain types of lesions. These two questions are; (1) is there a difference between the prevalence rates between highland and lowland Peru in terms of cribrotic lesions, and (2) is there a difference in rates of inflammatory lesions. Finally, this thesis seeks to show if the results from this thesis corroborate what other researchers have found in their studies.

In the past, researchers focusing on orbital lesions have been scoring all lesions types as cribrotic lesions, and not separating out lesions that do not have the diagnostic criteria of hypertrophic marrow expansion. Starting with Hrdlicka (1914), J. Lawrence Angel (1966), Patty Stuart-Macadam (1985, 1987a, 1987b, 1991, 1992), and Walker and coauthors (2004)/Walper and coauthors ( ), there has been no standard for orbital lesions classification that seeks to separate these lesions by their manifestation and correctly classify cribrotic lesions.

The data was collected during the summer of 2011 at the Phoebe A Hearst Museum of Anthropology. The data was collected and entered into a spreadsheet using SPSS version 20. No invasive or destructive actions were taken. Chi square tests, and Fisher's Exact tests were used to see if there were differences between the groups, age and sex, and types of orbital lesions present. All of the research questions could be answered using these tests.

The skeletal samples used for this thesis consisted of males and females from a wide variety of ages from four Peruvian sites encompassing both highland and lowland Peru. There were a total of 82 individuals from Ancon, 118 from Cuzco, 23 from Marca Huamachuco, and 79 from Tate-Chulpaca. These skeletal samples were collected by Max Uhle in the early part of the 1900's and sent to Phoebe A. Hearst for curation at the PAHMA, formerly known and the Lowie Museum of Anthropology. Unfortunately, the prevailing practices at the time were to collect pathological specimens and skulls. This can bias the results of this study. Additionally, two of the four sites did not have dates for

occupancy, Marca Huamachuco and Tate-Chulpaca. Ancon dated to BC 110-1476 AD, and Cuzco dated to 1300-1500 AD (Anton 1989).

The Wilczak and Jeney (2008) method uses a classification system that scores both anterior and posterior orbits seperately using a scaled system of scoring for categories such as porosity, inflammation, diploic expansion, and percent of the orbit affected. The non-invasive method views the macroscopic changes in bone, and the scoring system contributes to the overall classification of the type of orbital lesions. The main categories of orbital lesions, based on the scoring system, condenses orbital lesions into cribrotic lesions (healing, and active), inflammatory lesions, and porous lesions.

This thesis seeks to re-evaluate the prevalence of cribrotic lesions in both highland and lowland Peru, by using a scoring system set up by Wilczak and Jeney (2008). These orbital lesions can be classified into major types such as: inflammation, porosity, cribrotic lesions, and healing cribrotic lesions. Older research has overestimated the prevalence of cribrotic lesions due to the lack of or confusion with etiological understanding. This thesis will only allow for a diagnosis of cribra orbitalia if the lesion demonstrates the diagnostic criteria associated with true cribra, marrow hypertrophy within the orbits.

There are numerous caveats associated with this thesis, most of them are based on the collection techniques of archaeologists in the early 20th century. Due to the face that only skulls were collected, reliable age and sex estimations are challenging to create. Post cranial methods to estimate age are far more accurate than cranial methods, and

unfortunately the lack of teeth, and the amount of non-articulating mandibles made occlusal wear aging impractical for this thesis. For sex estimation, cranial methods are useful, but still not as accurate as post cranial sex estimation using the pelvis.

The shear amount of data collected during this study is impossible to analyze in a single masters thesis, which included data on primary, secondary, and tertiary lesion classification, numeric coded values based on porosity/vascularity/diploic expansion. Instead this thesis will focus on the primary lesion classifications between highland and lowland Peru, and the causes of these lesions.

CHAPTER 2 – LITERATURE REVIEW

2.1 History of Paleopathology

Paleopathology comes from the root words palaios, meaning ancient, and pathos for suffering. Over the course of the discipline, there have been four major stages that correspond with the changing literature and advancement in the field. These phases include the antecedent, genesis, interbellum, and new paleopathology (Rodriguez-Martin 1989a,1990, Rodriguez-Martin and Ramírez 1991).

The antecedent phase began in the mid 19th century with Esper, Scheuchzer, Goldfuss and numerous others (Aufderheide and Rodríquez-Martin 1998). Most diseases were viewed in fossilized animal skeletons instead of human skeletal remains. Ubelaker (1982) described the birth of paleopathology as the moment when Johann Friederich Esper diagnosed an osteosarcoma in cave bear femur. Phillip Franz von Walther noted the importance of inheritance related to etiology. P.C. Schmerling began to notice the problem that some diseases manifest in similar ways and this became a pivotal part in recognizing the importance of differential diagnosis. This phase was characterized by some microscopic analysis, but was mainly very generalized, and had little precision due to the mainly descriptive analyses completed for curiosity and not for scientific purposes.

The genesis phase, mid 19th century to World War 1, began with the shift from looking at animal remains to viewing human remains (Ortner 2003, Aufderheide and

Rodríquez-Martin 1998). This period coincided with the rise of archaeology and focus was given to individual cases of pathological human bones that were extraordinary. Rudolph Virchow became known as the father of paleopathology and began viewing pathological conditions in terms of their medical and diagnostic characteristics. In America, William F. Whitney published the first paleopathology manual titled, Notes on the Anomalies, Injuries and Diseases of the Bones of the Native Races of North America. The genesis phase was also the instrumental time for three major osteological collections to be created and curated which led to future research: U.S. Army Medical Museum (National Museum of Health and Medicine), National Museum of Natural History (Smithsonian Institution), and The Museé de L'Homme.

During the Interbellum Phase, between World War 1 and World War 2, the field became more systematic and scientific (Ortner 2003, Aufderheide and Rodríguez-Martin 1998). During this phase, Mark Armand Ruffer was instrumental in popularizing the term "paleopathology" after numerous publications on osteoarthritis, congenital conditions, and tuberculosis. The term paleopathology had been used earlier in the 1890's by Shufeldt, and was already in the Funk and Wagnall's Standard Dictionary in the mid 1890's, before the popularization by Ruffers. In America, Ruffers editor, Roy Lee Moodie published, Paleopathology. An Introduction to the Study of Ancient Evidences of Disease. Ales Hrdlicka, a Czechoslovakian anthropologist, created the division of Physical Anthropology at the Smithsonian Institution. One of Hrdlicka's contemporaries,

Earnest Hooton, began the process of collecting specimens with a known etiology for comparative research.

During the final phase, leading up until the current era, Buikstra, Cook, and Ortner began synthesizing older literature and putting more emphasis on the etiological processes of diseases. Angel (1981) split this stage of the discipline into four areas: (1) limiting diagnosis by looking for trait patterning, (2) the relationship between society and health, (3) demography, (4) normal ontological growth. Differential diagnosis, consideration of the osteological paradox, paleoepidemiology, and hypothesis testing became commonplace.

In 1991, Ortner discussed steps for the future of paleopathology. The six stages that he proposed were: (1) defining the area of interest, (2) creation of new methodology, (3) a body of descriptive data related to the field, (4) classification systems, (5) new hypotheses for testing the significance of findings, and (6) comparing the data to other data in similar fields.

Paleopathology studies numerous things including: metabolic disease, infectious diseases, trauma, degenerative join disease, congenital malformations, endocrine disorders and neoplastic diseases (Ortner 1991). The study of paleopathology, and especially metabolic diseases, have been instrumental in looking at health and wellness patterns in both modern and prehistoric populations. Metabolic diseases are caused by nutritional deficiencies, whether because of a lack of dietary availability or the inability of the body to properly process and synthesize essential compounds. Cribra orbitalia, a

metabolic disorder, is one of the most frequently noted pathological conditions associated with archaeological sites, and therefore has received a lot of attention (Walker et al. 2009). This type of orbital lesion has been associated almost exclusively with anemia since the 1960's, but this association has been challenged in recent research (Angel 1966, Walker et al 2009, Walper et al 2004). It is a pathological condition that manifests in the bony orbit as diploic expansion, when the interior portion of the cranium pushes outwards creating a coral-like appearance (Walper et al 2004). In previous literature, cribra orbitalia has been diagnosed as porosity within the orbit, which does not account for the numerous other pathological conditions that can create porous orbital lesions. The ability to differentially classify these lesions and advance the understanding of the etiology leads to new understandings of health in the past.

Since one of the most often discussed pathology within the literature and at archaeological sites is cribra orbitalia, the attention has given rise to numerous articles and publications all trying to understand it. Not only does this orbital lesion occur all over the world, it occurs in many time periods, and this simple fact alone makes cribra a very popular lesion to study. Not only can it provide information about diet and resource stress, but it can shed light on sanitation practices and overall well-being of past populations. Over the last hundred plus years, scholars have gone back and forth on the etiology of cribrotic lesions, their manifestations, and better ways to classify the lesions.

2.2 History of Cribra Orbitalia

Ales Hrdlicka was one of the first anthropologists to examine pathologies in Peruvian skeletal materials (1914). In 1914, the Smithsonian Miscellaneous Collections document titled, Anthropological Work in Peru in 1913 with Notes on the Pathology of the Ancient Peruvians was published (Hrdlicka 1914). He explored many of the regions in Peru such as Ancon, Pachacamac, Occujage, Chicama Valley, Chan Chan, and the Moche Valley. His initial trip in 1910 was to look at the relationships between the coastal and mountain populations, much like the research that is being completed now. In this trip, Hrdlicka did not excavate any sites, but looked at the skeletal material that were exposed due to grave robbery, viewing around 4,800 skulls with post cranial remains as well. The skeletal material from Ancon, its corresponding archaeology and burials, have been studied more often than any other burial site in Peru, partially because of the abundance of material culture.

Hrdlicka explored many of the regions in this thesis sample including, Ancon (1914). He noted while studying these skulls that people residing in the mountain regions showed no signs of symmetric osteoporosis, which was his termonology that encompassed both cribra orbitalia and porotic hyperostosis (1914). Hrdlicka described this pathology as a condition that manifested during infancy and affected solely the cranium (Hrdlicka 1914). In extreme cases it was considered to look like, "a low growth of coral" (Hrdlicka 1913:58). Hrdlicka believed that the pathology was often fatal in the initial stages, indicating the possibility that children were more affected than adults. He also noted porosity on the greater wing of the sphenoid as part of this symmetrical

osteoporosis, but this lesion is now considered pathognomic for scurvy which also creates porosity within the eye orbits (Ortner 2003).

Between the early 1900's, and the 1960's there was not a substantial amount of new research on the etiology or manifestations of these cribrotic lesions. Many people accepted the hypotheses of Hrdlicka, up until Patty Stuart Macadam and J. Lawrence Angel.

1960's

In 1966, J. Lawrence Angel became integral in associating porotic hyperostosis with types of iron deficiency anemia, especially thalassemia and sicklemia. Before this time, other researchers had mentioned the correlation but had not brought the concept to the forefront of paleopathology. Angel postulated these specific types of anemia to be related to the distribution of falciparum malaria. In areas of the world where the malaria parasite was atypical, such as coastal Peru, parasite load and iron deficiency anemia were considered to be the casual factors behind this pathology. Geographical patterning was seen to influence the pathology as well with marshy areas creating higher prevalence of lesions and dry/rocky areas showing less skeletal manifestations of the disease. In the sample of five populations residing near marshes, there was evidence of more severe lesions than those who resided in arid environments. The original name of symmetrical osteoporosis was changed to cribra orbitalia when it was found to affect only the orbital roof, and porotic hyperostosis when it affected the vault.

Based on the initial diagnosis of falciparum malaria, Angel saw correlations between the overall ecology of the area of residence, development and advancements in farming and agriculture in relation to the abnormal genes that could have been a selective factor for the lesions. New World lesions were most likely caused by iron deficiency anemia due to a regimented diet lacking in nutritional value, and/or nutritional depletion due to lactation. In the Old World, as malaria frequencies increased, lesions increased, and the change of subsistence strategy from hunting/gathering to agriculture would have put people near wet areas that were breeding grounds for malarial parasites.

Another study published in 1967 by Vanier viewed the skulls of children with bony manifestations of iron deficiency anemia. A number of the individuals who were between 6 and 7 years did not show clinical signs of anemia even though there were hypertrophic lesions; this could have been one of the key moments for changing the "iron deficiency anemia" hypothesis. These changes were instead explained by suggesting anemia was a childhood condition that could continue into adulthood.

1990's

In the 1980's-1990's Patty Stuart-Macadam published a series of papers delving into both the etiology and manifestations of cribra orbitalia and porotic hyperostosis (1985, 1987a, 1987b, 1991, 1992). In the original articles, Stuart-Macadam related both these lesions to iron-deficiency anemia in childhood and infancy due to the severe manifestations seen in children and not adults macroscopically, microscopically and radiologically. Sandford and coauthors (1983) supported this concept by comparing iron

levels in the hair follicles of both adults and children showing that adults had no significant difference in iron content regardless of lesions, and children showed a higher depletion of iron when there was skeletal manifestation. Stuart-Macadam (1985) postulated that the reasons for this phenomena was that the iron deficiency issues were severely affecting the children and the adults were somehow buffered or protected from the most severe manifestations.

One of the key factors explaining the manifestations of this disease was the placement and genesis of red blood cells. Weatherall and Clegg (1981), Lewis (1981), and Ascenzi (1976) began to notice the hematopoeitic marrow placement in children versus adults. In children this marrow is located throughout the body, and over time yellow fatty marrow begins to appear in the long bone shafts. During adulthood, age 20, the hematopoeitic marrow is only found within the vertebrae, sternum, clavicle, scapulae, ribs, and bones of the pelvis and skull. The large production centers of marrow and red blood cells could indicate why children manifest the lesions more easily than adults because the juvenile bone has less room for marrow expansion within the cavity and one of the hematopoeitic centers is located within the skull. Adults have much more open space within the bones available to absorb the pressure of expanding marrow thus possibly not expressing the lesions as drastically.

Davidson and coauthors (1975) and Witts (1966) noted that these lesions manifest less in men than in younger women and children who have greater needs for iron than males. In cases where there was the typical “hair-on-end” hypertrophic lesions, Stuart-

Macadam (1987b) found that these vault lesions were only found in conjunction with cribrotic lesions.

Walker (1986) began to study porotic hyperostosis and cribra orbitalia in California Native American populations from the Channel Islands. Through his study he found that anemias linked to iron deficiencies were not a proper explanation of hyperostotic lesions. Previously linked to maize-based diets, local residents of the Channel Islands did not have access to such food and still displayed numerous cases of hyperostotic lesions. Their diet was rich in proteins, both terrestrial and marine, which are all high in iron levels and more easily absorbed by the body than plant based irons (Roberts and Manchester 2005). Instead, Walker (1986) related these lesion to contaminated water sources, parasite load, and diet.

Stuart-Macadam began discussing pathogen load in 1992 to widen the research on cribra and porotic hyperostosis. In previous literature, dietary bioavailability of iron was considered to be the main impact on these types of lesions, but Wadsworth (1975) challenged that. Wadsworth began to notice that diet did not necessarily correlate with iron deficiency anemia as previously thought. By 1992, it was seen that bioavailability of dietary iron was only one small component to the overall health and hypertrophic lesions that create both cribra and porotic hyperostosis. Arthur and Isbister (1987) believed that even a complete lack of dietary iron would not immediately cause these severe lesions. As people begin ingesting less iron, a greater percentage of iron would be absorbed as a preventative measure. It was beginning to be thought that decreased iron in the body

could act as a protectant against parasites (Weinberg 1974, 1977, 1978, 1984, 1990). Weinberg noted that those individuals who have stronger iron withholding systems can reduce the risk of infection because the constant paucity of iron slows down bacterial growth.

Looking at pathogen load was difficult for these thousands of year old sites. One of the only ways to view amount and types of parasites located within an area was to sample to coprolites, or fossilized fecal matter. Reinhard (1990) examined coprolites in Southwest Anazasi sites and found numerous samples had pinworms within them. Since there was a large number of individuals at the site manifesting hyperostotic vault lesions, Reihard felt that there must have been a strong correlation between the presence of these worms and the lesions.

Since sanitation and chronic disease can affect the body and possibly aggravate/instigate these types of hyperostotic lesions, sanitation in the past becomes an area of high interest. Chronic infections of the stomach can deplete the body of vital nutrients leading to the possibility of cribrotic lesions. Ubelaker (1990) used data from Ecuador to show that skeletal remains from coastal sites have a much higher prevalence of porotic hyperostosis than those inland.Dunns' (1972) research on Malayan aborigines viewed health in terms of a sanitary score related to overcrowding, land space, mobility, subsistence type, house type (pile or raised), types of animals present, and the parasite availability in relation to temperature. It was seen by Dunn that the sanitary score and parasite load were correlated. The higher the sanitation scores (ie the cleanlier the areas),

the lower the parasite load and vica versa. By looking into past populations ways of living, it is possible to interpret if their living conditions could have affected the prevalence rate of hyperostotic lesions. In future excavations, researchers could be viewing not only the skeletal remains for signs of disease, but viewing the landscape around them as a conduit for disease.

2000's

In the early 2000's more in-depth and microscopic tests were being used to determine etiology and manifestations of lesions. Wapler and coauthors (2004) acknowledged that other researchers had found cases of cribra orbitalia without histological signs of anemia. The thin-ground sections of bone used showed other types of pathology such as rickets and inflammation. This indicated that anemia was an over simplified etiology for a very complex pathological condition. If it is possible to have cribrotic orbital lesions without iron deficiency anemia, other causes must be examined and tested.

In Schultz (2001), a study completed on paleohistopathology viewed both cribrotic orbital and vault lesions using light microscopy. The benefit to this type of study is that the light microscopy can differentiate true lesions from taphonomy. This increased the reliability of diagnoses, and allows for a microscopic view of lesions. Even though macroscopic analysis is still valid and useful, the micro-analysis coupled with macroscopic analysis allows for more accurate results.

Schultz (2001) viewed orbital and vault lesions to differentiate them from taphonomic processes such as roots, and post mortem damage, as well as scurvy, rickets, inflammatory lesions, and hematomas caused by trauma. Since children's skulls are thinner than adults, trauma resulting in a hematoma is a likely cause for lesions of the vault due to the natural porotic and vascular nature of healing. The visual appearance of hyperostotic bone and woven bone deposition can appear almost identical which means that a more in-depth analysis must be used for proper classification and/or diagnosis.

Porotic hyperostosis follows two stages: (1) the external lamina thins out allowing the cancellous bone to become visible thus creating porosity, (2) the atrophy of the cancellous bone due to pressure (Schultz 2001). Due to the high number of etiologies that look the same visually and without microscopic analysis, macroscopic analysis should not try and diagnose, but instead, classify lesions based on appearance.

Wapler et al (2004) used microscopy with polarized light to view and examine the structure of the bone within the orbit. For instance, in marrow hypertrophy, the marrow spaces widen and pressure can open the external lamina of the bone, and when the marrow spaces are widened and the lamina is opened, the trabeculae are seen at right angles to the it. Many different types of anemia including thalassemia, and deficiency anemia cause these types of reactions. Within the study, Wapler (2004) noted that other conditions such as inflammation, post mortem erosion, and undetermined causes can create orbital lesions resembling cribra orbitalia. In the sample only 43.5% of the cases showed the diagnostic characteristics of anemia yet still manifested orbital lesions. This

indicated that orbital lesions were not necessarily only associated with anemia. Wapler (2004) noted that only chronic severe anemia can create the change in bone that leads to the orbital lesions we consider typical of conditions such as cribra orbitalia, and porotic hyperostosis.

Anemia is considered not a disease in itself, but a pathological symptom (Walker 2009). It means, "without blood", and can be further defined as a deficiency in hemoglobin or red blood cells. In a normal, healthy individuals, the red blood cell production within bone marrow equals the destruction rate of the cells. Three main causes of anemia include: (1) loss of blood, (2) problems with erythropoiesis, and (3) increased destruction of red blood cells (hemolysis). These conditions are often found to co-occur within individuals that suffer from anemia. There are two categories of anemia: genetic, such as thalassemia and sicklemia, and acquired anemia caused by blood loss or deficient nutrition.

Porotic hyperostosis is a sign of megaloblastic anemia because the porosity and projecting diploe is linked to the expansion of the marrow of the skull as a systematic response to a lack of both red blood cells and hemoglobin. Marrow hypertrophy results from increased red blood cell production, and only anemias that can both stimulate and maintain increased levels of erythropoiesis can be the causes of the lesions that contain marrow hypertrophy, cribra orbitalia and porotic hyperostosis (Walker 2009). Walker and coauthors (2009) reappraised the iron-deficiency anemia hypothesis and found that only anemias that cause premature death of red blood cells (RBC) and increase RBC

production (erythropieses) can be used to explain porotic hyperostosis and cribra orbitalia. These types of anemia consist of megaloblastic anemia and hemolytic anemia.

In the process of red blood cell homeostasis, the nutrients required consist of amino acids, iron, vitamins A, B12, B6, and folic acid (Walker 2009). Not only does the iron levels in the body assist with transporting oxygen through the body, it assists in collagen synthesis and immune boosting activities (Roberts and Manchester 1995).

When iron absorption or intake is hampered, the production of red blood cells becomes deficient and anemia is a possible outcome (Walker 2009). The body responds to this lack of iron and/or nutrients in a hierarchical process. Marrow hypertrophy of the cranial vault and remodeling occur only after other less extreme measures of restoring homeostasis have failed to remedy the deficiency. The normal mature red blood cell lifespan of 120 days, with a maturation period of seven days, is disturbed during anemic states. When the hemoglobin levels fall, the body becomes deprived of oxygen and this hypoxic condition causes release of erythropoietin, which can increase red blood cell production and maturation. If, and when this response becomes inadequate, the centers for hemopoietic marrow are signaled to increase production of red blood cells. In the vault bones, the outer table becomes compromised and ultimately resorbed when the diploe expands. The resorption is what causes the appearance of the lesions that are commonly seen.

Anemia induced lesions, as they were once referred to as, were seen most often in children. Waldron and coauthors (1978) focused on the biological possibilities for this.

During parturition, iron depletion in the mother does not necessarily affect the child. The iron levels within the body are proportionate to individual body weight. For the first few months of life, a child will have a "normochromic normocytic anemia" which is the slowing down of erythropoeisis due to the lungs becoming the source of oxygen and not the placenta (1978:14-15). This reduction and increased destruction of red blood cells becomes the major hemoglobin source. Children who are premature and/or underweight for their age can experience iron deficiency anemias called "anemias of prematurity" (Waldron 1978 citing Schulman 1959).

In childhood and shortly after, the red blood cell production is centered within the cranial vault bones and the medullary cavities of long bones, during adulthood, these red blood cell centers are located within the spongy bone in the sternal, costal, and vertebral regions (Walker 2009). Viewing the shift in red blood cell production centers, it could explain the higher prevalence of hypertrophic lesions in children than in adults (Stuart-Macadam 1985, Kent 1986, Stuart-Macadam 1992). Most active lesions are seen in children, and adults show signs of healed or healing lesions (Walker et al 2009, Stuart-Macadam 1985, Walker 1985, 1986).

Acquired anemias have long been thought to influence the body and create hyperostotic lesions. Unfortunately, in the paleopathology literature, there is no distinction between the three clinical types of acquired anemias. Sullivan (2005) discussed three major models of these types of anemia. They included iron deficiency anemia influenced by the poor absorption and/or intake of iron, anemias from chronic

conditions where the low iron content inhibits bacterial and microbial actions, and megaloblastic anemias related to low vitamin B and folic acid levels.

Acquired iron deficiency anemia is due to the low absorption rate and intake of iron, this has been the most popular answer to hyperostotic lesions found in archaeological remains. This has been seen to be untrue due to the numerous foods that are high in iron content: meat, shellfish, and animal proteins (Sullivan 2007). Iron from animal protein is more efficient for absorption than iron found in plant products, and the addition of vitamin C consumption to the diet increases iron absorption whereas an increase of tannins can reduce absorption rates of iron. In cases of chronic diseases, the iron withholding in the body caused by said infections can halt or slow bacteria problems within the body. Diet has been long thought to affect iron stores in the body. During the digestion of food, dietary iron is absorbed into the body via the duodeum (Waldron 1978 citing Davis 1970). Meat based iron is more readily absorbed than plant based iron, but they can help over all iron retention if eaten together. During chronic infection, iron is not left long enough in the intestines to properly absorb into the body reducing the iron levels.

In 2007, Brenton and Paine from St. Johns University and Texas Tech University viewed skeletal manifestations of niacin and tryptophan depletion. Pellagra, or niacin deficiency, has been linked to mostly maize based diets, which are lacking in niacin (a type of vitamin B), and the amino acid tryptophan. This disease can cause diarrhea, vomiting, facial lesions, and inflammation of the mouth. Pellagra can induce some forms

of anemia because of the chronic malnutrition that accompanies constant intestinal problems. As niacin levels decrease, tryptophan levels must increase which means more iron is necessary. In a sample of known individuals from the Raymond Dart Collection, the 14 known pellagrins showed lesions that were seen in both iron deficiency anemia and scurvy. There is no way as of the present to distinguish pellagra from either scurvy or iron deficiency anemia, especially since pellagra can induce some forms of anemia and co-occurrence would be hard to differentially diagnose.

2.3 Differential Diagnosis

Many different diseases can cause lesions that mimic the look of cribra orbitalia, which is why it is important to use differential diagnosis. Since it was once defined and described as any porosity within the eye orbit, noticing the differences and subtle nuances between these diseases can be challenging. A lot of metabolic diseases can manifest lesions that look very similar to cribra orbitalia indicating the possibility of a similar root cause and mechanism for such types of lesions.

Scurvy

Scurvy and rickets have been two diseases that have been known to show some of the same skeletal manifestations as cribra orbitalia such as porous orbital lesions. Scurvy is the absence of, or subpar amount of vitamin C (ascorbic acid) (Ortner 1997). It is a disease that is mostly found in humans, because most animals that need ascorbic acid can synthesize their own, whereas humans cannot. Vitamin C is necessary for the formation of connective tissues in the human body, collagen and cement material that becomes a

binding agent for the endothelial material within blood vessels. When there are problems with the cement material in the blood vessels, the individual becomes susceptible to hemorrhage from normal range of motion and muscle activities. Since children have such fast growing bodies and the remodeling of bone, tissue, and blood vessels, the presence of scurvy at a young age will create poorly formed connective tissues and blood vessels. This causes the periosteum to be loosely attached to the bone underneath which allows for subperiosteal hemorrhages to lift it away from the underlying bone and stimulate proliferative bone growth. In order to remove the abnormal blood the body uses cellular and vascular processes associated with inflammation. If both bleeding and inflammation occur at the same time for weeks on end, bony reactions can occur since active hyperamenia can create bone destruction and passive hyperanemia can cause bone formation.

Ortner (1997) has created a diagnostic pattern of both porous and hypertrophic lesions that occur on the skull in cases of scurvy. These lesions include porous bone, described as being the result of inflammation, and it is noted as being separate from porous bone that is caused by marrow hyperplasia, as in the case of porotic hyperostosis.

Ortner attributes these types of bilateral lesions to two major causes: (1) inflammation and bleeding from both abnormally formed blood vessels due to scurvy and mechanical trauma from muscle contraction, and (2) the branches of the maxillary artery and its anatomical position (1997). Barlow (1883), has described the cranial bosses as being enlarged, and the zygomatic and occiput being thickened, but it was unknown if he

was describing the changes in bone of soft tissues. Hemorrhagic processes often affect the eye, and the inferior surface of the orbital plate on the frontal. This can create inflammation that stimulates the deposition of porous bone, and hypertrophic lesions which have also been attributed to cribra orbitalia, and other forms of anemia.

In scorbutic bone (vitamin C deficient), pathognomic porous lesions are present on the greater wing of the sphenoid, and in most cases are bilateral (Ortner 1997). The hemorrhagic processes that occur in relation to the greater wing of the sphenoid are directly related to both the anatomy and the vascularity of the temporalis muscle (used in chewing). This fan shaped muscle has deep temporal arteries that when inflamed would most likely cause hemorrhages. Other lesions indicative of scurvy include increased porosity in the maxilla and the palate. The deep temporal arteries are implicated in the palatal lesions because of their pathways. The maxillary artery and its branches could be implicated in the distribution of the lesions associated with scurvy. In cases where porosity is seen on the greater wing of the sphenoid, there is also the tendency for hypertrophic lesions of the orbits, and maxillary/palatal lesions. Post-cranial lesions are common on the scapula as well because of the proximity of the arteries to the bone similar to what is seen in the temporalis muscle and its blood supply.

Other authors such as Brickely and Ives (2006) viewed scorbutic orbital lesions as well as the diagnostic criteria set forth by Ortner (1997). Within their sample, they noted highly vascularized orbital lesions around the anterolateral corner or the anterior orbit in both scorbutic and non scorbutic infants. By using SEM (scanning electron

microscope), Brickley and coauthor were able to show that the bone deposition was occurring rapidly and was most likely in a response to some sort of trauma and/or inflammatory process (2006). Older infants showed spiculated bone growth within the orbits. Ortner and Ericksen (1997) postulated that there could be a higher chance of hemorrhage due to the improperly formed blood vessels in individuals with scurvy.

One of the most important conclusions stated by Ortner and coauthors (2001) was that there are three nutritional deficiencies that can affect the skeleton; rickets, scurvy, and deficiency anemias. Within the sample viewed by these authors, one case out of 22 total individuals expressed signs of anemia as well as scurvy. Since these diseases can occur at the same times it is necessary to view the full complement of pathological signs to properly diagnose and classify lesions.

Rickets and Subperiosteal Hematoma

Rickets, and its manifestations on the bone have also been documented by Ortner and Mays (1998) and Ortner (2003). Rickets in its most basic definition is a vitamin D deficiency. It has multiple causes, which relate to the failure to mineralize osteoid, the protein precursor to bone. Vitamin D inadequacy can be caused by a deficiency in the diet, problems and/or failure in absorbing the vitamin, kidney problems resulting in conversion of vitamin D and/or kidneys not retaining phosphate for bone mineral production, and a lack of ultraviolet light exposure needed for conversion.

The causes of vitamin D deficiency all produce the same end result, inadequate mineralization of the osteoid leading to weak bones that deform when subjected to weight

bearing activities and muscle tension. The areas of the human skeleton that are at risk for unmineralized osteoids are locations where there is a high turnover of bone tissue and rapid growth, such as the knee. Rickets can cause porotic lesions of the skull and postcranial elements, especially those close to growth plates. In addition, rickets that presents itself in pre-walking infants will not affect the legs as there is no weight bearing activity placed on them. In these cases where rickets predates walking, the arms will show evidence of the disease, and in cases where it presents after walking can produce both arm and leg deformities.

In Ortner and Mays (1998) evaluation of the Wharram-Percy collection they found 10 features in bone common in rickets: (1) orbital porosity, (2) vault porosity, (3) mandibular ramus deformation possibly due to the motion of chewing, (4) arm deformation, (5) leg deformation, (6) flared rib ends, (7) porous and irregular rib end morphology, (8) long bone growth plate abnormalities, (9) metaphyseal cortex being irregularity and porosity, and (10) thickened long bones. The orbital lesions were seen as being porous with no indications of marrow hypertrophy or hyperostotic lesions. Both rickets and scurvy have been known to be co-occurrent, which makes diagnosis of the disease difficult especially when both scurvy and rickets produce orbital lesions that can mimic, and in some cases are caused by anemia.

Subperiosteal hematomas can create orbital lesions as well, those that may present as porosity (Walker et al. 2009). These lesions can be caused from numerous pathologies including, but not limited to, scurvy, traumatic injury, and rickets. During healing, the

clot becomes a vascular plaque that can mimic the look of cribra orbitalia. These lesions are seen mostly in children because the periosteum attached to the orbital roof is not as strongly attached as in adults, and is highly vascular in children.

Inflammation

Constant pressure within the orbit causes atrophy of the surrounding bone. In cases of the eye, it is possible that the lacrimal gland, or ophthalmic infections can contribute to lesions found within the orbit. Wapler et al (2004) noted that increased vascularization of the orbital lamina could be indicative of periostitis and/or osteitis. Other inflammations of the head can have orbital involvement as well such as sinusitis, abscesses/oral disease, skin infections, and nasopharyngeal infections. Orbital infections, possibly dacryoadenitis and conjunctivitis, can cause inflammation that may originally affect the periosteum and then transmit to the bone. It has now been acknowledged that a considerable number of cases of cribra orbitalia are caused by inflammatory responses within the orbit.

Postmortem Damage and Erosion

At times, postmortem alterations can be mistaken for a pathological lesion. Wapler (2004) found that an accurate distinction between antemortem and postmortem changes and destruction of bone was not possible using macroscopic methods. In a sample from the Sudan, sand and wind contributed to as much as 20% of the supposed orbital lesions in the sample. This postmortem alteration to bone could increase the prevalence of cribra orbitalia within the archaeological record. The thin orbital lamina

within the orbit is susceptible to postmortem alterations. The thinner the cortex, the more pronounced the alterations become. Normal variation within humans can occasionally mean that there is no diploe in the orbital roof (Susse 1961, Lang 1975, Lang and Bruckner 1981). In individuals with thin orbital lamina and diploe, porosity would be more likely to appear postmortem. Careful macroscopic examination of the bone along with a better understanding of etiology can help to distinguish a true lesion from post mortem damage.

The study of cribra orbitalia has been advanced not only by acknowledging the numerous diseases that can cause hypertrophic and porous orbital lesions, but by realizing that geography can play a role in it as well. Part of how geography affects skeletal health is due to the differences in resources available depending on climate, water supply, and temperature. That being said, it has been postulated for a long time that people in lowland areas show a higher prevalence of cribra orbitalia than those in highland areas. For a long while this was thought to be related to high altitude hypoxia and the bloods reaction to atmospheric levels. Hrdlicka (1916) noted that when he was in Peru, the highland people had no lesions consistent with what he was calling cribra orbitalia. The understanding of the lesion in highland populations depends on a working understanding of how blood oxygen functions at high levels.

2.4 Blood Hemoglobin and Iron Content at High and Low Altitudes

In 1985, it was estimated that around 17 million people resided at altitudes over 3000m in the Andes of South America (Tufts et al 1985), and in 2009, approximately 9

million people, 30% of the population, resided in the Andean region of Peru (Gonzales et al. 2009). This high altitude hypoxic and hypobaric environment which requires the body to adapt certain ventilatory and hematological responses (Tufts et al 1985). At low altitudes, hemoglobin levels that are considered normal are not sufficient at high altitudes. Adaptive responses from the body increase the red blood cell counts and hemoglobin concentrations. Sometimes, the increase in hemoglobin results in high-altitude polycythemia. While the increase of hemoglobin and hematocrit in high altitude cases are known, normal ranges are not well documented creating problems diagnosing both anemia and polycythemia.

Tufts and coauthors (1985) conducted a hematological survey of individuals living in La Paz, Bolivia. Most of the current and historic Andean populations live within a range of 3600 and 4000 meters, including the ones in La Paz at 3700 meters. In the Bolivian sample, only 1% of the adult male individuals showed overt anemia, but 6% showed signs of polycythemia, which could be related to arterial oxygen desaturation while resting. The iron levels of men in the sample were considered to be similar to those found in healthy individuals living at lower altitudes. When all of the iron-deficient individuals were removed from the sample, the subsample of people did not display traits of anemia. This demonstrates that most of the anemia within the sample was a product of iron-deficiency. Functional anemia has been seen to be population specific depending on normal hemoglobin concentrations for given populations and not necessarily a product of absolute hemoglobin concentration within the blood.

During pregnancy, hemoglobin values drop slightly from the first trimester to the last (Gonzales 2009). When pregnant women at high altitudes were studied in Peru, it was seen that hemoglobin concentrations were higher at high altitudes than low altitudes with increased plasma viscosity, and increased blood viscosity. These women also were seen to have a higher rate of parity, greater than or equal to 4 births (P=0.001), and higher rates of preterm or stillbirths (P=0.001). Independent of hemoglobin levels within the blood between high and low altitudes, women at high altitudes were seen to have an increased risk of reproductive outcomes (stillbirths, preterm births, and births small for gestational age).

During the pregnancy at high altitude study, it was determined that no changed definition of anemia is needed for high altitudes (less than 9 g/dl). At this level there were increased risks during pregnancy for both high and low altitude women. On the contrary, hemoglobin content about 14.5 g/dl also can cause pregnancy problems by increasing the viscosity of the blood, which could possible inhibit blood flow to placenta and restrict intrauterine growth. Previous studies show that populations at high altitude are not lacking in iron (Cook et al 2005, Reynafarje 1987), which contradicts previous research by Tufts and coauthors (1985).

Other research states that in relation to body iron measurements, the hemoglobin concentration must fall below 20 g/L or 4 mg iron/kg of body weight to constitute anemia (Cooks et al 2005). Positive values of body iron show the amount of iron that is being stored within iron-replete people, and negative values show deficits in the tissue iron in

iron deficient people. A test completed on 800 Bolivian mothers and one of their children in altitudes ranging between 156 m and 3750 m, looked at the measurements of both serum ferritin and transferrin receptors to view iron status. There was a significant difference between the 4 altitudinal intervals (P=.02), but there was no altitudinal trend indicated at the 0-1000 m, 1000-2000 m, 2000-3000, or 3000-4000 meter levels. They also noted a decrease in iron within the mothers that was a faster decline than in the children after birth.

During this study, the hemoglobin and body iron measurements created differing estimates of iron deficiency (Cooks et al 2005). Women at these high altitudes were able to enhance the iron absorption to gather three-quarters of the additional iron needed for red blood cell expansion. Studies on small animals have shown that the iron regulatory peptide, hepcidin, could have a positive effect between hypoxia and iron absorption independent of the erythropoiesis changes and iron status. The higher sTfR concentrations (serum transferrin receptors), seen in women living at about 3000 m, are most likely related to their diminished iron than enhanced erythropoiesis. Half of the children under one year of age had tissue iron deficiency with a decline in the oneyear old children (Cooks et al 2005). This could be due to the loss of iron from the mother that buffers the child during the first few months of life. An increase in sTfR could be useful in terms of being able to pinpoint concurrent iron deficiency and chronic disease, but it may be due to enhanced erythropoiesis at altitudes higher than 3000 m. The most important product of this study was the realization that iron status of children under five

is highly correlated with their mothers iron status during that same time possibly due to diet and the iron bioavailability of the diet.

Throughout the history of Peruvian civilization, there have been changes in diet and changes in accessible resources. Peru has been excavated and researched for over 100 years, and new data has come forth explaining the shifts in subsistence and culture. When subsistence strategies change, it could be due to a number of things including climate change, movement of individual groups, and over usage of certain resources. The time periods in Peru and the advent of major faming techniques could shed light into the resource access and the bioavailability of nutritional necessities.

2.5 Peruvian Archaeology and Time Periods

Multiple chronologies have been created for Peru, starting with the original chronology by Uhle (Menzel 1977). Max Uhle is known to most as the father of Peruvian archaeology (Menzel 1977). He excavated numerous sites in Peru, including all of the sites included in my sample. The excavations began in 1895. He was able to help distinguish four main periods in Peruvian history including the Initial period, the Early, Middle and Late Horizons.

These horizons, by Uhle, have been deemed inaccurate and have been corrected by Willey (1991). Willey described Peruvian culture starting as early as 4000 BC, when the people were mostly sedentary due to their reliance on marine resources that were easily accessible. The late Preceramic began around 2200 BC and is the time when the first public use buildings appeared on the coast and highlands. The Initial Period, 1800 -

1200/800 BC marks the beginning of farming on the coast, and the constriction of ceremonials mounds and large scale public buildings. The Early Intermediate Period (EIP), 200 BC- 550 AD was the beginning of regional diversity and the white-on-red pottery style for the North and Central coasts (Willey 1991). The Tiahuanaco-Huari style begins in AD 550 and continues till 1000 AD. Within the larger horizon there are four subphases (Willey 1991). The Late Intermediate Period (LIP), 1000 to 1475 AD, began the diffusion of bronze through Peru (Willey 1991).

2.6 Peruvian Geography and Climate

The geography and climate in Peru can be vastly different depending on location. Peru has coastal areas, arid desserts, and highland mountain regions such as the Andes. Peru is known for its wildlife partially because of the Peru Current, which sweeps up the coast of Peru starting 900 feet below the ocean surface and brining nutrients to the ocean waters surrounding Peru (Sabloff 1994). The Peruvian current is also responsible for creating the arid desserts that are near the coastal areas. The wind currents that pass over Peruvian current are forced to release their moisture before they reach the land, and then only regain moisture once they have reached the mountains where rainfall is common. This creates the arid strip that separates the coastal and mountain regions.

The Andean areas of Peru, including the mountains, valleys, and high grasslands are characterized by wet conditions (Sabloff 1994). The yunga, or warm valley, is a drier region that is located between 1600 and 7500 feet. It is characterized by a deep canyons, forests, and vegetation such as columnar cacti and algarroba trees. All indigenous crops

flourish in this type of climate. Between 7200 and 11000 feet are the cloud forests that are constantly nourished via the moist winds from within the Amazon basin.

The areas between 9200 and 11500 feet are known as the quecha (Sabloff 1994). This area has been modified by humans for thousands of years by agriculture and tree clearing. The rainfall is seasonal, and the people depend on the river for terrace style farming and agriculture. This region is not as fertile as the yunga, but still most crops can grow, especially potatoes.

The grassland, or puna, are between 11500 and 13000 feet (Sabloff 1994). They lie in the transitional zone called a suni. Most of the domesticated animals in Peru are found here including, guinea pig, llamas and alpaca. Lake Titicaca is the source of water for some of the most densely populated areas in Peru and Bolivia. These agropastoralists grow many quecha crops and focus on domestication and breeding of llamas and alpacas. Lastly, the area between 1300, and 3299 feet is known as the selva alta, or high tropical forests. These forests extend into the Atlantic coast of Brazil. It also is the greatest diversity of both plant and animal life in the Western Hemisphere.

2.7 A History of Cribra Orbitalia in Peru

Hrdlicka noted while studying skulls that the people residing in the mountain territories showed no signs of symmetric osteoporosis (cribra orbitalia/porotic hyperostosis) (Hrdlicka 1914:56). He described this type of lesion as a condition that manifests during infancy and affects the cranium. Even in 1913, he thought it was a reaction to a toxin and not a nutritional or degenerative pathology. He stated that it was

often fatal before the disease peaked, but some could survive it. The roof or the eye orbits or the frontal squama between the frontal and coronal sutures was affected. Hrdlicka believed that it started with increased vascularity of the orbits, followed by the deposition of porous bone. In extreme cases it was considered to look like "a low growth of coral" (Hrdlicka 1914:58). He also noted porosity on the greater wing of the sphenoid as part of this symmetrical osteoporosis (hyperostosis), which we now know is pathognomic for scurvy orbits (Ortner 2009). When healing had occurred, the areas affected were seen to be thickened, and the growths disappeared.

Examining the skulls, Hrdlicka (1914) noted that the coastal groups, such as Ancon, were brachycephals (round headed) and normal in stature while the mountain groups had oblong heads. When the cemetery was excavated, it was interpreted that the people of Ancon were most likely fishermen. Hrdlicka noted numerous cases of what he referred to as "cribra orbitalia" in the skulls of the people buried in Ancon.

In Alto Salaverry, at the south end of the Moche Valley on the north coast of Peru, a partial juvenile skeleton was found with cribra orbitalia, which was attributed to iron deficiency anemia (Trinkaus 1977). Based on the architecture near the burial site and the midden characteristics, the burial was dated to the Cotton Preceramic period, approximately 1,700 to 2,000 BC. The child was estimated to be between 10 and 11 years of age, and the fragmentary skeleton was encrusted with salt, which also caused postmortem alterations to the bone.

During a visual inspection of the orbital roof, it was seen to have pitting on the bone which Trinkaus attributed to iron deficiency anemia (Trinkaus 1977). The inner and outer tables of the skull did not exhibit porosity, but there was supposed expansion of diploe on the cranial bones. The facial and postcranial skeleton did not show signs of other pathology. Trinkaus noted that heavy reliance on marine foods high in iron, such as mollusks, squash, and other domesticated plants would have supplied a substantial amount of dietary iron, which called into question his original assumption. Based on the amount of dietary iron available in the surrounding areas, alternative explanations for the cause of the anemia include parasite infection, and severe blood loss.

Viewing the published figures of this specimen, it is difficult to tell if the orbital lesions are a result of anemia, a different pathology, or if it is postmortem damage. Trinkaus noted during the initial viewing of the individual that the salt had damaged the bone, and with the quality of the photos in the older journal articles it becomes more difficult to diagnose individuals without seeing the actual specimen and knowing the conditions of the remains.

Ortner and Ericksen (1997) examined an individual from the Smithsonian estimated to be approximately 12 years old from a mortuary site in Peru. This individual from Pachacamac may be from between AD 1000 and 1530, but the dating in uncertain. For this individual, there were bilateral lesions on the greater wing of the sphenoid, and very porous lesions in both orbits. These porous lesions could have easily been mistaken for cribrotic lesions had the researchers not considered a complete differential diagnosis.

Figure 1: Orbital Lesions associated with scurvy (cat #3174)

Ortner and coauthors (1999) viewed another collection from Peru that was also curated and stored within the Smithsonian. Their sample was coastal and highland populations including Pachacamac, Chillon Valley, and Huarochiri, among others. The skeletal remains were collected by Hrdlicka at the turn of the 19th century, and because of a lack of provenience, the dating is imprecise. Subadults are the most at risk population for scorbutic lesions because of the rate of active growth. In the 38 cases of scurvy found in the sites from Peru, 37 of them also presented orbital lesions. Figure 1

above shows orbital lesions that were found in an individual with scurvy from the research for this theis, the specimen is located at the PAHMA. Most scorbutic orbital lesions were consistent with porosity (97%), and hyperostotic orbital lesions occurred on 61% of the scorbutic individuals. Within this sample, there was no evidence of rickets of marrow hypertrophy that can be associated with a diagnosis of anemia, even though these deficiency pathologies can occur at the same time. Ortner and coauthors (2001) have noted some challenges to diagnosing scurvy in the Peruvian samples based on a lack of postcranial remains. During Ortners' examination in the 1990's of scurvy in Peru, none on the individuals had postcranial remains, similar to the samples examined for this thesis. This has the possibility create a higher prevalence of scurvy within this population since the postcranial bones cannot be viewed.

Blom and coauthors (2005) looked at anemia and mortality rates of children along the coast of Peru. Hypertrophic lesions can occur with types of megaloblastic anemia, vitamin B deficiencies, and niacin deficiencies as well (cite), so it was important to take a closer look at the geographical patterning and dietary bioavailability. They used Hrdlicka's (1914) original diagnoses of porotic hyperostosis and noted that he diagnosed a high number of individuals with the pathology along the coastal regions of Peru. Since this pathology has been believed to be associated with anemia, it was important to reassess the conclusions now that new research has shown that multiple pathologies create orbital lesions similar to what is seen with anemia.

Blom and coauthors (2005) looked at 1,465 individuals; 512 from the Peruvian collection at the Field Museum of Natural History in Chicago and 953 from Moquegua, Peru. The goal of the research was to examine the pattern of lesions by region, and look at factors that would influence the prevalence of the disease. The time periods associated with these specimens are the Early Intermediate Period (200 BC- AD 600), the Middle Horizon (Ad 600-1000), and the Later Intermediate Period (AD 1000-1476). The sites associated with the study were located across Peru including the North, Central portion, and the South. Each of the sites had different dietary patters, some mostly maize based, others more marine based, and some are mostly terrestrial animals.

One site in the study, Chen Chen, was a large producer of maize, and maize based products, but it was most likely for export (Blom et al 2005). The dental attrition in the area is not significant, and when maize is a predominant part of a diet, the teeth show evidence of significant attrition and carious lesions.

Blom and coauthors found that 23.1% (n=402) of adults in the sample exhibited signs of cribra orbitalia (Blom 2005). In children, the prevalence was 81.8% (n=66). Blom et al. did find an associated between cribra orbitalia and porotic hyperostosis which is found on the outer posterior portion of the cranium. The individuals from the South had fewer lesions that those in the Central and South Central areas. The conclusion of the research was that children in the Central coast were less likely to survive with anemia than those children living in the South or South Central areas (Blom 2005). The prevalence of the lesions also increased with time when time was looked at as a factor.

Blom and coauthors (2005) also found that a mostly maize or marine based diet was not associated with higher rates of cribra orbitalia, but that mixed diets (marine and maize) had higher prevalence rates of cribra orbitalia. Populations from highland Peru, Machu Picchu have also been studied isotopically by Turner and Armelagos (2012). They concluded that individuals displaying hyperostotic lesions lived in more arid environments, and that diet did not play an integral role in the manifestation of such lesions. The place of residence may have played a greater role in the distribution of these lesions than diet.

Through this research, the expected findings are that children and adults will have significantly different prevalence rates of cribra orbitalia, and that coastal populations will have higher prevalence of cribra orbitalia as compared to the highland populations. Walker and coauthors (2009) supported that childhood anemia would most likely be the cause for severe lesions because most active porotic lesions with no remodeling were found in juvenile and adolescent remains. Adults were found to have signs of remodeled lesions without the severity of the childhood lesions (Stuart-Macadam 1985; Walker 1985, 1986).

In terms of geographical patterning associated with cribra orbitalia, it has been shown that coastal populations are more likely to manifest orbital lesions than the highland groups. Walker et al. (2009) noted that parasites and chronic sickness can also cause anemia by depleting B12. It has been shown that pre-Columbian mountain populations in Peru showed no orbital lesions when they were examined in the early

1900's (Hrdlicka 1914, Blom et al 2005). Blom and coauthors found in the 2005 study that individuals were more likely to have childhood anemia in arid environments. They also were able to show that children buried in the lower altitudes, possible raised in lowland areas, who were closer to the coast, and had a diet heavy in marine resources were less likely to die from childhood anemia. Since there have been numerous documented cases of cribrotic lesions in coastal areas, this indicates that the previous ideas of iron deficiency anemia associated with hyperostotic lesions is not necessarily correct. Since these populations had access to such iron rich foods, iron deficiency anemia is not the proper explanation for these lesions. Something that could explain it is the increased parasite load associated with shellfish and seafood causing chronic depletion of bodily resources such as vitamin B. Wilczak (personal communication) and others have noticed higher prevalence of inflammatory lesions in coastal populations, and this research may provide more evidence to support that theory.

CHAPTER 3 – MATERIALS

The new advancements in the field of paleopathology allow biological anthropologists to obtain a better understanding of past health concerns and the processes that create pathological conditions. Reassessment of previous methods that have been challenged is important to the field of paleopathology because it allows for a more complete and accurate view of health in the past.

3.1 PAHMA Collection

The collection at the Phoebe Hearst Museum has not been viewed for orbital lesions since the collection of the remains in the early 1900's. These collections from highland and coastal Peru allow for comparative studies of orbital lesions across Peru and across different geographical areas. Since cribra orbitalia and other types of orbital lesions can be caused by dietary deficiencies, chronic infections/inflammation, and other causes, a look at multiple sites allows for more complete interpretations and a better understanding of the range of these lesions.

In the case of certain types of anemia, cribra orbitalia is classified by diploic expansion. As outlined by Walper et al., 2009 the trabeculae, spongy bone, grow at right angles to the orbital laminae. In the case of periostitis and osteitis, there is increased vascularity, and possible bone resorption. Vitamin C deficiency is associated with

hemorrhagic processes and can also create porous bone and ossified subperiosteal hemorrhages in the eye orbit.

Blom and coauthors (2005) have looked at anemia and mortality rates of children along the coast of Peru. They used Hrdlicka's (1914) original diagnoses of porotic hyperostosis and noted that there was a high number of individuals diagnosed with the pathology along the coastal regions of Peru. Blom and coauthors did find an association between cribra orbitalia and porotic hyperostosis and postulate that both cribra and porotic hyperostosis are related pathologies in terms of their etiology (2005). Part of this study was to look at heterogeneity in frailty of people in terms of the osteological paradox (Wright and Yoder 2003). The conclusion of the research was that children in the Central coast were less likely to survive with anemia than those children living in the South or South Central areas (Blom et al. 2005), and the prevalence of the lesions also increased with time. Blom and coauthors (2005) also found that a mostly maize or marine based diet was not associated with higher rates of cribra orbitalia, but that mixed diets (marine and maize) had higher frequencies of individuals with cribra orbitalia.

Table 1: Samples From Each Site

Site	Total N	Total Usable N	Location	Time Period
Ancon	100	82	Lowland	BC 1100-1476 AD
Tate Chulpaca	91	79	Lowland	No Dates
Marca Huamachuco	25	23	Highland	No Dates
Cuzco	131	118	Highland	1300-1500 AD

The samples that I will be examining were excavated by Max Uhle starting in 1895, and ending in 1905, these sites are broken down by the total sample size for the site, the usable sample numbers, location and time period in Table 1. The specimens come from Marca Huamachuco, Tate Chulpaca, Ancon, and Cuzco. All four of the sites were curated at the University of California, Berkeley at the Phoebe Hurst Museum, formerly the Lowie Museum.

UC Berkeley was chosen because the collection has not been viewed for orbital lesions since it was sent over from Peru by Uhle. The major caveat associated with the collection at the PAHMA is that the skeletal samples are represented by only crania. During excavations in the early 1900‘s it was commonplace to collect only skulls. This creates challenges for this thesis in terms of the estimation of age and sex.

Uhle and Hrdlicka were both in Peru around similar times and collected remains from similar locations. Hrdlicka published data of his skeletal sample, but Uhle did not. Since this collection is contemporaneous with many other Peruvian collections, and has a significant number of individuals within the collection, this sample becomes a good comparative sample to test new methods.

The map (Figure ----) shows the location of the four sites that will be analyzed in terms of lowland or highland populations, access to major rivers, and how far inland each of the sites is.

Figure : Map (Base map ESRI 2013 courtesy of Basin Research Associates Inc.)

3.2 Samples by Site

Ancon

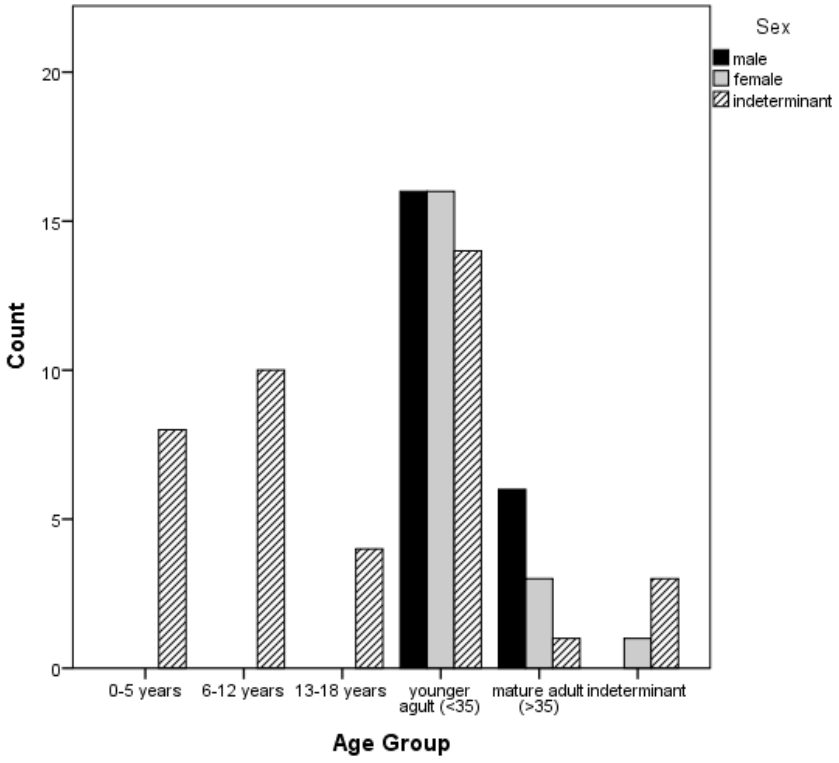

Figure 3:Distribution of individuals in Ancon

Ancon is a coastal lowland site located next to the bay. From this site, there was a total of 100 skeletons, of which 82 could be scored for orbital lesions. Figure 3 shows the composition of the site consists of 22 males and 20 females, and 40 indeterminate sex individuals (22 under the age of 18, and 18 adults). There are seven children between the ages of 0 and 5, 11 children between the ages of six and 12, and 4 children between the ages of 13 and 18. For the adults, there are 46 under age 35, and 10 over the age of 35.

There are also an extra four individuals that could not be aged at all, because they were represented skeletally by elements that had no diagnostic criteria for age.

The necropolis of Ancon was one of Uhle's most famous excavations (Menzel 1977). The site is an isolated bay that is around 35 kilometers (22 miles) north of Lima on the central coast of Peru between the Chillon and Chancay rivers. This was the first site in ancient Peru that was excavated in a systematic fashion and was one of the largest scale excavations. Ancon was excavated in 1904 by Uhle and some other German archaeologists including Aphons Stubel. In 1942, more excavations were done by Gordon R. Willey, and Marshall T. Newman. They excavated in the southeastern section of the site, and uncovered 20 tombs (Menzel 1977). Excavations continued nonstop from 1945 to 1954 and later. The 1945 excavations were led by the Museo Nacional de Antropologia y Arqueologia of Lima. In 1950, Marino Gonzales Moreno began his own excavations. Reiss and Stubel were the only ones to publish extensively on the necropolis at Ancon (Menzel 1977). Located above the bustling town of Lima, this town sits between two rivers: the Chillon and the Chancay. This systematic excavation was the first of its kind undertaken in Peru. Over most of the 19th century, excavations took place here and became some of the most famous ones.

The bay of Ancon is shaped much like a horseshoe with beach that slopes into more arid desert conditions (Menzel 1977). The southern portion of the bay of Ancon was the hub of marine resources for the people residing there. Archaeological evidence suggests that the inhabitants were building their homes nearest to the shellfish resources

in the southern portion of the bay. The water within the bay is "brackish" and well water was more than likely used for the prehistoric inhabitants much as they are used today for the current residents. The shelly black midden, and refuse piles along the bay of Ancon denote the archaeological site there, the necropolis however, is in a different location.

The necropolis was located inland of the bay towards the southern portions (Menzel 1977). The necropolis was not only the burial ground, but a settlement site for the inhabitants during the Huari empire, or the Middle Horizon Epoch B. The midden that makes up the deposit at the necropolis differs significantly from the deposit closest to the bay. The shell remains are scarce and the dirt is much browner and more compact ranging in side from 1.2 to 2 meters above the desert floor. Uhle encountered numerous house posts, stone foundations, and mud walls still intact at the necropolis. Llama dung was found within the walls of a courtyard indicating possible trade with highland individuals. Llamas were considered to be a highland dwelling animal but could be kept on the lowland coastal areas if provided with proper resources and temperatures.

Ancon is thought to have prospered from the Middle Horizon 1 B to the Inca empire, and then declined in the Late Intermediate Period Epoch 4 (Menzel 1977). During this time, the cultures in the north coast declined and became more segregated from surrounding areas. Before the decline, a large wall was built around Ancon during the Middle Horizon Epoch 4, this was found out due to the Middle Horizon graves being few under and outside of the wall, and the later graves appearing at a higher elevation and inside of the walls. Three concentric circular walls surrounded the ancient town, during

the Huari Empire, it was likely that the city of Ancon became a middle man for the distribution of resources especially shellfish and marine foods.

Within the settlement, burial internments from the Middle Horizon Epochs 1B, 2 and 3 were in the sterile soil beneath the midden (Menzel 1977). The graves from the Middle Horizon 2B and 3 were found in the first few layers of midden deposit. Other areas of the site including the areas closer to the sea were found to have graves from the Middle Horizon Epoch 4 and Later Intermediate Period Epoch 3. These graves were found more centrally located within the midden layers. During the times of regional isolation, the people of Ancon relied more on shellfish and marine resources creating such an overburden of shell midden compared to the surrounding less shelly deposits (Late Intermediate Period). Ancon itself, has a very poor clean water supply for irrigation and agriculture. This indicates a high probability of trade between neighboring regions for access to resources and food not found within the walls of Ancon.

Inside of section P, Uhle uncovered six burials dating from the Middle Horizon 2B where adult remains were found with gold and silver indicating not status but distinction (Menzel 1977). During the 3rd and 4th Epochs of the Middle Horizon, gold and silver use in burials receded and the headdresses became plainer and textile based. At least one of the mummy bales in Ancon had silver frontlets (headdress) inside the bale, and dated to the Late Intermediate Period Epoch 3. These mummy bales with false heads were very common burial practices during the Epoch 2 to the Middle Horizon. By the Middle Horizon Epoch 1B, the use of mummy bales had receded.

One of the most interesting parts of the bioarchaeological history at Ancon is the change in the burial practices (Menzel 1977). The burial practices changed during almost every phase, including the burial internment vessel, and the style of internment. In Epoch 1B the burials were placed in long shallow graves with the individuals extended, indicative of the individuals living in the area before the Huari influence. When the Huari empire took over the area of Ancon (Epoch 2) burial posturing became much different with the individuals buried in seated position inside of a mummy bale with the knees by the face and a false head adorning the top of the bale. During Epoch 1B, the transition into Huari ruling, the burial practices reflect older traditions and some newer traditions indicating that the Huari influence did not dominate local culture until a later period. During Epoch 2A, the depth of the burial chambers changed allowing up to three mummy bales.

The Epoch 2B and 3 held some of the most interesting burial practices at Ancon (Menzel 1977). The deep, 2-3 meter tombs, with circular narrow shafts, was the final resting place of one individual inside of the mummy bale. Within Epoch 2B, special extra shafts at the top of the columns were built into to accommodate other individuals and their artifacts. The late Middle Horizon Epoch 2B and Epoch 3 had fewer extra chambers, and were sometimes only accompanied by artifacts at the top and not the other deceased individuals.

Middle Horizon 4 had shallower graves that were rectangular in shape (Menzel 1977). Some burial artifacts were recessed into the lower walls and occasionally

secondary burials were placed in the upper reaches of the shaft. Similar to the earlier tomb style, the chambers could hold anywhere from one to three adult individuals inside of their mummy bales. Throughout time, the burial practices became much simpler and less time consuming.

Ancon is desert like but it sits very close to the Bay of Ancon, which contributed to the variety of marine foods at the site, evident by the fishing hooks and nets that were found in the archaeological assemblages (Slovak and Paytan 2009). The close proximity to water gave them access to a varied diet that included other food such as lucuma, quinoa, mani, avocado, and beans (Menzel 1977). Other food such as corn (C4 plants), lama, and guinea pigs have been found in the archaeological record. Corn was probably brought in from the Chancay and Chillon Valleys because Ancon could not support the water needed for intensive corn cultivation (Menzel 1977).

During the Middle Horizon in Ancon, life changed drastically. Marine resources were once staples of daily lives and during the Middle Horizon, they became scarce in the archaeological record (Menzel 1977). The bulk of the population reorganized and moved into the Necropolis of Ancon which was a flat plain north of the village. Burial practices around this time also changed in that the single and simple burials were replaced with chambered tomb burials that held grave goods and were markers of the wealthy. During this time the burials of the wealthy also held items that were diagnostic for the Wari empire, a highland group (Menzel 1977). This could indicate increased trade between the two groups and a possible reason for dietary and cultural change. Other postulations for

the decrease in marine resources involve El Nino and climactic changes that damaged the marine resources.

For one study, Slovak and Paytan (2009a) looked at nitrogen, oxygen, and carbon stable isotopes. Bone records these signatures for the last 10-15 years of life, while teeth show the signatures from the first few years of life (tooth development). Carbon isotopes look at the bulk protein intake of an individual (ie where they are getting it from), nitrogen isotopes look at marine versus terrestrial sources of food, and oxygen isotopes are best used to look at weaning practices since it involves water (Slovak and Paytan 2009a).

What was found during this study was that there was greater dietary diversity in the Early Middle Horizon than in the Late Middle Horizon (Slovak and Paytan 2009a). The carbon 13 ratios fell very close to the marine protein lines, which was to be expected given the close proximity to the ocean. Some indications of people having more C4 protein were seen in the stable isotope results. Most of the dietary energy came from C4 plants (maize), which is interesting given that C3 plants are found at the site.

The nitrogen isotope results showed that marine resources were being consumed (Slovak and Paytan 2009a). There was also greater diversity in the diet during the Late Middle Horizon. There was a greater decline in N15 over time and the variability of these values could have indicated that the people at Ancon were relying less on marine resources and more on C4 plants and protein from terrestrial animals.

Overall, the diet at Ancon was mixed with marine foods, C4 foods, and some C3 foods (Slovak and Paytan 2009a). There was an abundance of seafood eaten at the site, and the C4 foods could have been corn and/or corn fed animals that were being raised. Most of the plants found were C3, which gives an interesting view of the trade and communication between the Anconero's and other people residing in Peru. Caries, or abscesses in populations have been correlated with an increasing dependency on C4 or corn. Many of the individuals looked at for the stable isotope studies (27/32) were seen to have at least one carious lesion or more. This supports the isotope research that during the Middle Horizon, corn was consumed at a higher rate.

Other indicators of health and trade at the site of Ancon have to do with resource distribution during the EMH (Slovak and Paytan 2009a). Slovak and Paytan (2009a) noted that in the Early Middle Horizon there was variation in the calcium carbonate and collagen values indicating that some people had easier access to maize while others did not. This could be because of trade, status, or other reasons. They noted that the one individual with the highest carbon collagen value in the whole sample was buried in the only "high status" tomb during the EMH. The stability of the nitrogen 15 values indicate that marine resources still played an important role throughout the occupation of Ancon, it was noted that the EMH did have a population that received a bulk of its dietary protein from marine resources.

In the LMH, the carbon and nitrogen isotopic signatures were switched (Slovak and Paytan 2009a). During this time, the carbon 13 values increased, meaning that there

was an increase in C4 plants consumption, such as corn. The nitrogen levels still showed that marine resources were important to the diet, but that they were significantly decreased from the signatures in the EMH. This indicates that the bulk of the dietary protein was from a mix of marine resources and C4 plants. Notable to the research was that corn, being so prevalent during the LMH, was not able to be grown at the site because of a lack of fresh water and usable land (Slovak and Paytan 2009a). This could indicate the people at Ancon had access to other areas of land, or that trade was increased between the coast and valley. Slovak, Paytan and Wiegand (2009b) examined strontium isotopes in another paper and noted that there was at least one individual who migrated from the Wari/Ayacucho areas and that could help to explain the interrelated trade network, modification of burials, and Wari artifacts (Slovak et al. 2009b).

Tate Chulpaca

Unfortunately, due to common practice during the early 1900's there is a paucity of information on this site, and the archaeology.

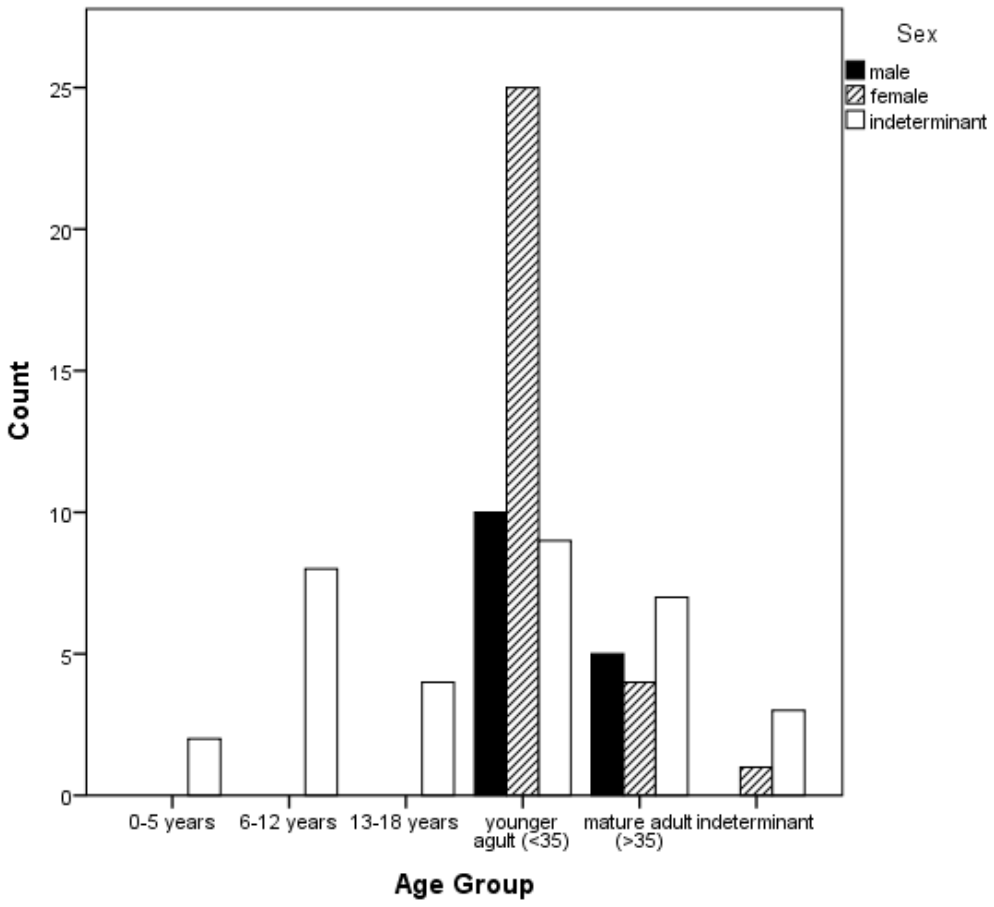

Figure 4: Distribution of individuals at Tate-Chulpaca

Tate Chulpaca is a coastal lowland site. From this site, there was a total of 91 skeletons, of which 79 could be scored for orbital lesions. Figure 3 shows that the composition of the site consists of 15 males and 30 females, and 34 indeterminate sex individuals (14 under the age of 18, and 20 adults). There are two children between the ages of 0 and 5, eight children between the ages of six and 12, and four children between the ages of 13 and 18. For the adults, there are 44 under age 35, and 16 over the age of

35. There are also an extra five individuals that could not be aged at all, because they were represented skeletally by elements that had no diagnostic criteria for age.

Cuzco

Uhle and Hrdlicka both had very difficult times during their explorations because of the climate and atmospere in the highland of Peru. Due to the hardships that Uhle faced in the highlands, the sample that he collected was much smaller than the coastal and desert sites he visited (Rowe,). The collection from Cuzco, while small and lacking in strict provenience and associations are important to the understanding of the imperial class and the inner workings at Cuzco.

Built by the first Inca emperor, Pachakuti, the town was built in the shape of a Puma with the tail being the downward side of the valley (Menzel 1977). The river, Vilcanota-Urubamba River flowed east of Cuzco. The head of the puma was made up of the walls of the fortress Sascahuaman. The higher class nobility were the individuals who lived within the walls in Cuzco. A ceremonial plaza was tucked into the lower limbs of the puma. Cuzco was characterized by numerous large storage places that were a very important part of Inca government. The government collected raw materials to distribute to local people to turn into finished products such as textiles, and precious metals.

Inca style artifacts were pottery made with certain types of clay and pigment, wooden cups, and tunics. Rowe has suggested that these wooden cups and the tunics may have been gifts from the government to the middle class citizens of Cuzco.

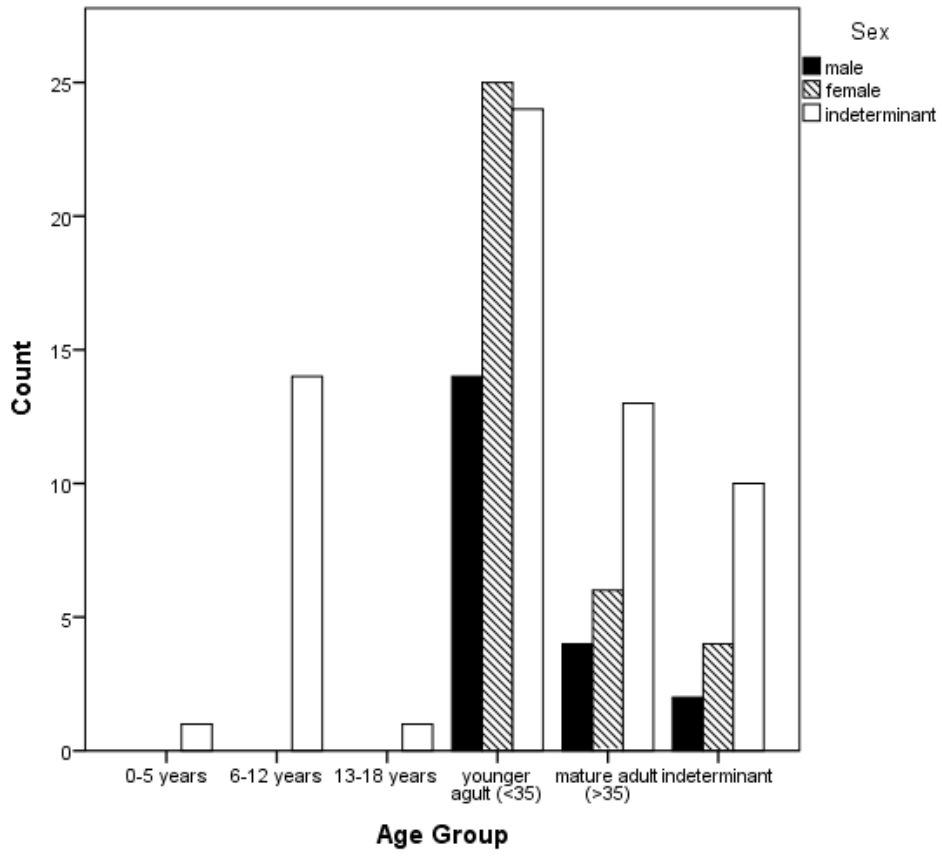

Figure 5: Distribution of individuals at Cuzco

Cuzco is a highland site in Southern Peru. From this site, there was a total of 131 skeletons, of which 118 could be scored for orbital lesions. The composition, shown in Figure 5, of the site consists of 20 males and 35 females, and 63 indeterminate sex individuals (16 under the age of 18, and 34 adults). There was one child between the ages of 0 and 5, 14 children between the ages of six and 12, and one child between the ages of 13 and 18. For the adults, there are 63 under age 35, and 23 over the age of 35.

There are also an extra 16 individuals that could not be aged at all, because they were represented skeletally by elements that had no diagnostic criteria for age.

Marca Huamachuco

Once again, as in Tate-Chulpaca, there is a paucity of information regarding the archaeology that Uhle completed while in the area. It was noted by Menzel (1977) that Uhle had a very difficult time in the highland regions, which is why some of the samples from these sites are so small.

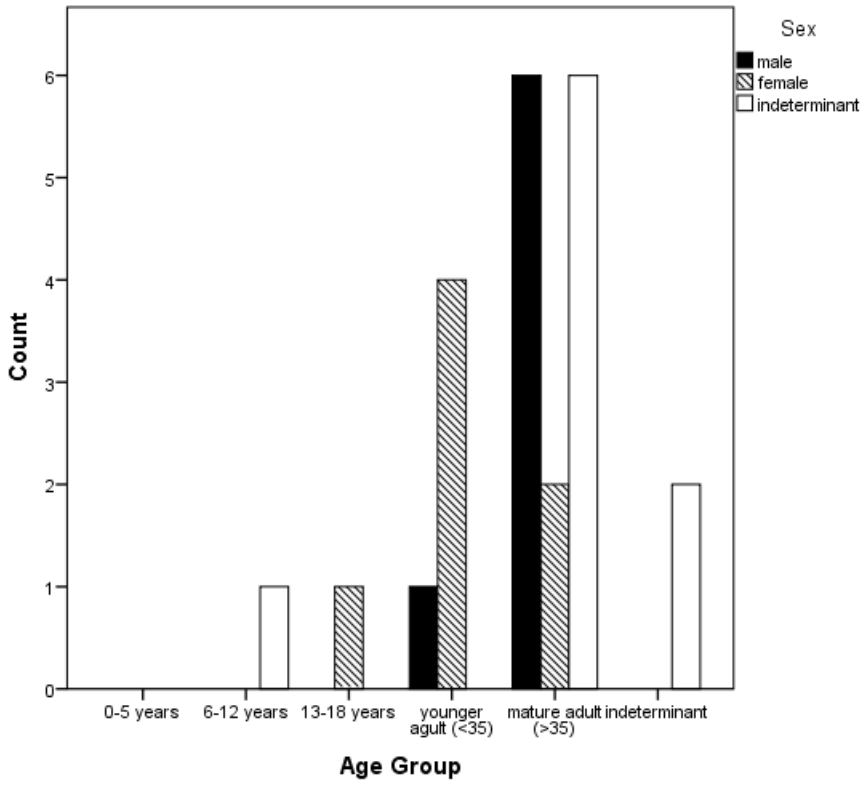

Figure 6: Distribution of individuals at Marca Huamachuco

Marca Huamachuco is a highland site in Northern Peru, Figure 6 above shows the distribution of individuals from the site. From this site, there was a total of 25 skeletons, of which 23 could be scored for orbital lesions. The composition of the site consists of seven males and seven females, and nine indeterminate sex individuals (six under the age of 18, and three adults). There is one child between the ages of 0 and 5, one child between the ages of six and 12, and five children between the ages of 13 and 18. For the adults, there are five under age 35, and 14 over the age of 35. There are also an extra two individuals that could not be aged at all, due to the fact that they were represented skeletally by elements that had no diagnostic criteria for age.

A total of approximately 302 usable skeletal samples will be viewed and the previous pathological designations will be compared to the new ones that are being assessed. In the past few years Wilczak (2008) has created new designations for diagnosing orbital lesions based on the macroscopic examination of specific bone changes, and their etiology. Each of the 302 crania will be viewed, non-invasively, for the lesions and scores will be assessed for each specimen by the system created by Wilczak (2008).

CHAPTER 4 - METHODS

Paleopathology has changed drastically since its inception in the mid19th century. Over the years, the advancements in the understanding of the progression of diseases and their etiology have led to the reassessment of previous methods for diagnosing pathological conditions. Since the original idea that cribra orbitalia was a manifestation of toxins in the early 1900's, to the iron deficiency anemia hypothesis in the 1960's it is obvious that the etiology has been misunderstood. Now it is imperative that new methods be tested and the previous research be reassessed using current methods. Current ideas on etiology involve megaloblastic anemia, and B vitamin deficiencies, which challenges previous research. The previous assumption that anemia causes all or even most of the orbital lesions seen in skeletal remains is inaccurate; it is necessary now to reclassify lesions in terms of their individual etiologies in the interest of more accurate differential diagnoses. A new method by Wilczak and Jeney (2008) will be employed to macroscopically examine the lesions and categorize them according to major categories such as porosity, cribrotic lesions, and inflammatory lesions.

4.1 Age Estimation

The human body follows a sequence of bone development, or ontogeny, that occurs in a predictable pattern for most individuals. This pattern is the basis for methods used to estimate age at death. Under ideal conditions, age estimations would occur using

both cranial markers, and postcranial markers for age. This is because cranial age estimation is not nearly as accurate as postcranial age estimation. The age ranges can be quite large when only cranial aging methods are used, and due to both early suture closure and cranial deformation, the results can be skewed.

In the subadult phase, the skeleton is dynamic and responds to many different internal and external stimuli. Adult cranial age estimation is much different than that of juveniles. Suture closure is one of the defining characteristics of an adult cranium. For instance, the basilar suture is one of the sutures that has the narrowest range of closure, and can be used in distinguishing an adult from a child. The closure of the basilar suture has a narrow range of 17-19 years and can be a preliminary estimate of adult age (McKern and Stewart 1957).

As an adult ages, the cranial bones fuse together in a distinct pattern that can help estimate age at death. Another method for estimating age at death for adults was created by Perizonius (1984). This method involves suture closure, and is accurate in that most scientists can agree on the importance of suture closure, but the range of variation is so wide it can be unreliable when compared to the postcranial age at death methods. Perizonius used 5 stages of closure to estimate age. There was much overlap in the original 5 stages and Buikstra and Ubelaker (1994) modified the original method to include only four stages: (0) open, (1) partially closed and continuous, (2) thinner lines, less zigzags, moderate closure, and (3) complete closure. These four stages are used to score 10 ectocranial sites. A one centimeter focal point at every site is used and usually

the left side is scored for standardization purposes. If one side of the skull has been affected by trauma, taphonomic processes or anything else, it is acceptable to use the right side.

Unfortunately, for the samples in this thesis, only crania were available to be examined as Uhle did not bring back complete skeletons. When aging the cranium, there are two major periods of time to consider, childhood where the skull is developing, and adulthood where the skull begins to fuse together. The first stage is characterized by dynamic and active growth and the latter stage is characterized by wear and rugosity. The first major step that needs to be taken in age at death estimation is to figure out which type of growth stage one is looking at (active or non active) in order to determine applicable methods.

This composite method of scoring certain cranial sites was a modification of several researchers' work (Perzonius , Buikstra and Ubelaker . A caveat associated with this method is that if you are missing a scoring area, then a composite score is impossible to achieve. Another problem is that if all sutures are open, there is no accurate age range, and adults whose sutures are completely closed immediately fall into the older adult group which may not be true (craniosynostosis). Lovejoy and Meindl (1985) did note that the lateral anterior scores perform as a stronger predictor of age at death then the vault sites did.

Adult Aging

Many of the individuals from all of the Peruvian sites examine in this thesis did not have associated mandibles, had non-articulating mandibles, or were missing a significant number of teeth. In cases where teeth were present, the wear patterns were very extreme even for younger individuals. Unfortunately due to the lack of teeth, and the extreme wear on the occlusal surface in the Peruvian sample, it was determined that tooth wear would be unreliable. There was also no consistency between the occlusal wear. In some cases, mature adults (>35 years) based on suture closure had very strong and intact teeth with no dentin exposure. Other times, young adults (<35 years) would have such extreme dental wear that would normally accompany older individuals. Figure ---- below shows the conditions of some of the Peruvian remains.

Figure 7: Lack of teeth and condition of remains (Cat # 2920)

Due to the fact that the skeletal remains from Peru were mostly crania, cranial aging methods were used (Figure 7). For adults, the Buikstra and Ubelaker (1994) revised suture closure method was used. McKern and Stewart (1957) who noticed that the closure of the basilar between 17-19 years, Schuer and Black (2004) narrowed the range of closure for the basilar suture to approximately 16 years of females and 18 for males. This means that some individuals with a closed basilar suture can be younger than 18. Figure 7 below demonstrates an open basilar suture.

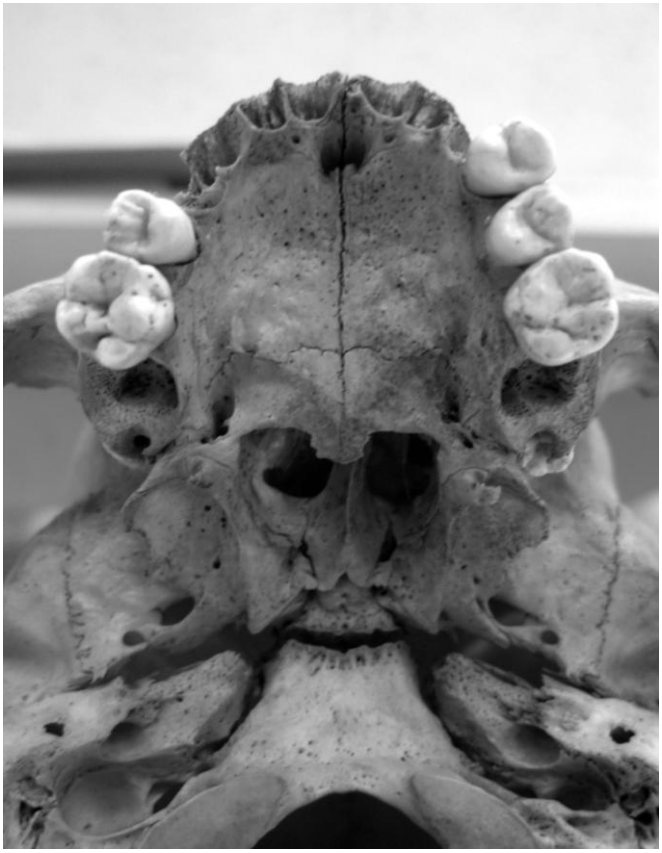

Figure 8: Catalog number 3287 Cuzco, open basilar suture

In cases where fusion of the basilar had already occurred completely, adult age estimation was done using suture closure. This composite method of scoring certain cranial sites was a modification of several researchers' work (Perzonius 1984, Buikstra and Ubelaker 1994). A caveat associated with this method is that if you are missing a scoring area, then a composite score is impossible to achieve. Another problem is that if all sutures are open, there is no accurate age range, and adults whose sutures are completely closed immediately fall into the older adult group, which may not be true (craniosynostosis). Lovejoy and Meindl (1985) did note that the lateral anterior scores perform as a stronger predictor of age at death then the vault sites did. Figures 9 and 10 below demonstrate open and partially closed cranial sutures.

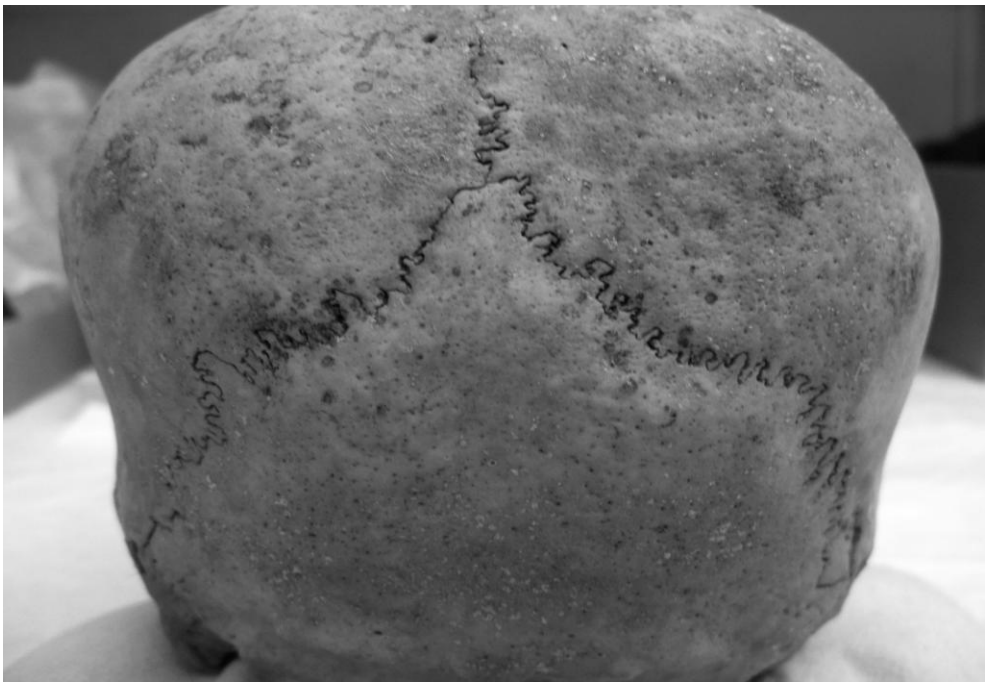

Figure 9: Showing open cranial sutures (Cat # 3047)

Figure 10: Demonstrating partially closed cranial sutures (Cat # 3030)

A one centimeter locus was used on each cranium by placing the two thumbs parallel and then spreading them to approximately a one centimeter area. In cases where the vault scores and the lateral anterior scores differed, the lateral anterior scores were used as they have a higher accuracy rate (Meindl and Lovejoy 1985).

Age Groups

When creating age groups for the individuals, the adult samples were classified by the suture scores into 2 categories; younger adult (18-35 years of age), and mature adult (35+ years of age). If an individual was given an age of 35, they were assumed to be at the younger end of that range and placed within the 18-35 category.

Notable to the research within this thesis is the fact that cranial suture closure is not as accurate as postcranial methods. It should be noted that in any case where an estimate of age at death is needed, that one method is usually insufficient. The cranium, pelvis, and dental wear are just some of the areas that are affected by aging. In cases where the age is ambiguous, most of the focus should be put on the more accurate postcranial methods rather than suture closure methods alone, which was unable to be completed for this thesis due to a lack of post cranial remains. It is because of this difficulty that the adult age categories used were broad.

Juvenile Age Estimation

Juvenile age estimation was completed using the methods of Ubelaker (1989a) for dental eruption. For juveniles, dental aging is the most accurate estimation, figure ---- shows dental aging from the Peruvian collection. This method considers the emergence of teeth from both the mandible and the maxilla from 5 months in utero until 35 years of age. Children in late adolescence, under the age of 18 and demonstrating an unfused basilar suture, were scored against the chart to find the estimated age range. Similarly, just as adults were placed into categories, if a child was given an age of 9 +/- 24 months, they were considered to be 9, and placed within the 6 to 12 year old group. For juveniles,

there are three age ranges, 0-5 years, 6-12 years, and 13-18 years. The mean age was the deciding factor of placing an individual into an age category. Figure 11 shows a typical juvenile mandible used for age estimation.

Figure 10: Aging Using Dental Eruption (cat # 1846)

Age Groups

Due to the wide age range of the individuals in the sample, it was necessary to bin the ages into groups for statistical purposes. General age categories were constructed based on the singular age estimation methods. There were two categories for adults: younger adult (<35), and mature adult (>35). Juveniles were placed into three categories: 0-5 years, 6-12 years, and 13-18 years. These categories correspond with infant, juvenile, and adolescent respectively. When the skeletal specimen was unable to be given an age assessment, they were classified as indeterminate. This could be due to a specimen having the whole vault mummified, or if the sample was only a portion of an orbit with no diagnostic criteria associated with it.

4.2 Sex estimation

Sexual dimorphism is a marked difference between the physical characteristics between males and females (White 2000). Testosterone and estrogen create different features in the human body, most notably in the skull and pelvis. Males will have more testosterone than females, and females will have more estrogen than males. These hormones create marked differences in the skeleton especially during puberty. During normal growth and the onset of adulthood, the skeleton responds to the increase of hormones and the amount of physical activity the person endures. These qualities help to create the dimorphism we see between the sexes in the skeleton, but it should be noted that there is no one defining characteristic that makes an individual male or female.

These differences can be quantified and observed to estimate sex in skeletal remains. For instance, males will tend to have larger muscle mass and therefore will have larger muscle markings and attachment sites (Skelton 1996, Acsadi and Nemeskeri 1990, Krogman and Iscan 1986, and Wolfe et al. 1994). The increased testosterone and heightened muscle activity presents itself in the skull as a large glabella, a pronounced mental eminence, large robust mastoids, and a pronounced nuchal crest. Other traits that are characteristic of males are shallow and broad palates, blunt and rounded supraorbital ridges, square eye orbits, a vertical forehead, and small frontal and parietal eminences. Male skulls tend to be thicker as well because of the increase in testosterone.

Females, because of the increased estrogen and lower levels of testosterone, will show more pedamorphic features and have more gracile muscle markings and attachment sites (Skelton 1996, Acsadi and Nemeskeri 1990, Krogman and Iscan 1986, and Wolfe et al. 1994). They show smaller mastoids and supercilliary arches. The orbits are sharp, and rounded with higher arches than in the males. Their foreheads are also rounded and smooth with larger frontal and parietal eminences, and the skull remains thinner. The methods used to estimate the sex of an individual uses these sexually dimorphic characteristics to classify crania.

There are some caveats associated with estimating sex on a skull. There is a vast amount of variation in all populations around the world. Some men tend to look more gracile skeletally, and some women can look more robust. There are certain populations

that are known to have very gracile males, and very robust females. For the Peruvian samples examined in this thesis, all of the individuals were fairly robust, which can skew the effectiveness of the methods if it is not taken into consideration.

The Acsadi and Nemeskeri (1970) method, one of the standards in Buikstra and Ubelaker (1994), uses a scale of 1 to 5, with 1 being extremely feminine and 5 being extremely masculine. The scale is based on a continuum of traits that are expressed differently in males and females. Scores are based on overall robusticity of cranial features. They looked at specific areas including the eye orbits, occipital area, mandible and mastoid processes.

For adult sex estimation, the Acsadi and Nemeskeri (1970) method was used. All five traits were scored: nuchal crest, supraorbital ridge, supraorbital margin, mastoid process, and mental emminence. If some traits were missing, the score was not taken. For many cases, the mandibles did not articulate properly with the skulls, so the mental emminence scores were not used. Sex estimates of male and female were only given when there were three or more characteristically male or female traits. For cases where the specimens scored threes for all five traits, they were classified as indeterminate. Also, if many scores were on both the male and female sides (1,3,3,4,3), the individual was places into the indeterminate category. For this thesis, it was important to make sure the sex estimations were as accurate as possible, because many authors have said that iron deficiency and certain lesions are more common in one sex than in the other. The

Peruvian skeletons are very robust in general for males and for females, making the Acsadi and Nemeskeri method more challenging, especially with the complete lack of postcranial remains from these sites.

Since sex estimation for juveniles is unreliable, all individuals under the age of 18 were scored as indeterminate for sex. The skeleton is not fully developed and estimating sex could skew the data. Being that there is no accurate way to estimate sex on juvenile or child remains, it was not completed for this thesis.

4.3 Scoring System

Orbital lesions manifest differently depending on the pathology causing them and normal biological variation. For instance, in cases of hyperostotic cribrotic lesions, the diploe begins to push through the outer table of the skull, whereas in other orbital lesions the only indications may be porosity or increased vascularity. Each orbit was viewed and scored using the Wilczak and Jeney (2008) method, and the more severe orbit was the orbit used for scoring and classification. Lesions of the orbit were diagnosed in eight classification categories based on the location and type of change: hyperostotic cribra orbitalia (CO), possibly healing cribra orbitalia (CO?), anterior porosity (AP), posterior porosity (PP), inflammation (INF), anterior inflammation with porosity (APIF), and

inflammation that extended from the posterior orbit to the anterior orbit (IFANT). In cases where the orbits were damaged, the remaining orbit was scored.

Porotic Hyperostosis/Vault Porosity

Most individuals at the sites viewed for this research exhibited some vault porosity, and some exhibitied signs of marrow hypertrophy on the vault. Two difference categories were used for these manifestations, one for porotic hyperostosis and one for simple vault porosity. In cases where there was clear marrow hypertrophy or signs of healing hypertrophic lesions, a presence score for porotic hyperostosis was given. For instances where there was simple vault porosity, vault porosity was coded as present or absent, and the notes indicate the locations of the porous lesions.

Lesion Types

There are eight major types of lesions most commonly seen that have been described by Wilczak and Jeney (2008), Wliczak (2009), and Wilczak and Hopkins (2010). These separate lesions types were all lumped together in previous works as. cribra orbitalia without differentiating between different bone changes forming the lesions. The Wilczak and Jeney (2008) method classifies only lesions with clear expansion of the diploe through the outer table as "true" or hyperostotic cribra orbitalia. Due to the previous associations that these lesions had with anemias, the authors saw fit to restrict the lesions classification to those that were most likely anemic in origin. Those cases where there was not diploic expansion are referred to as another lesions type based on the manifestation of the lesion.

CO or active cribra orbitalia is a manifestation where there were coalesced pores, and diploic expansion of the anterior orbit, including the anterior lateral corner. CO? indicated coalesced pores on the anterior orbit that can include the anteriolateral corner without the signs of diploic expansion. Based on the morphology of the lesions, CO? is believed to be a healing phase of hyperostotic cribra orbitalia (Wilczak and Hopkins, 2010). Figure 12 below shows typical hyperostotic lesions associated with cribra orbitalia, figure 13 shows the healing form of cribra orbitalia.

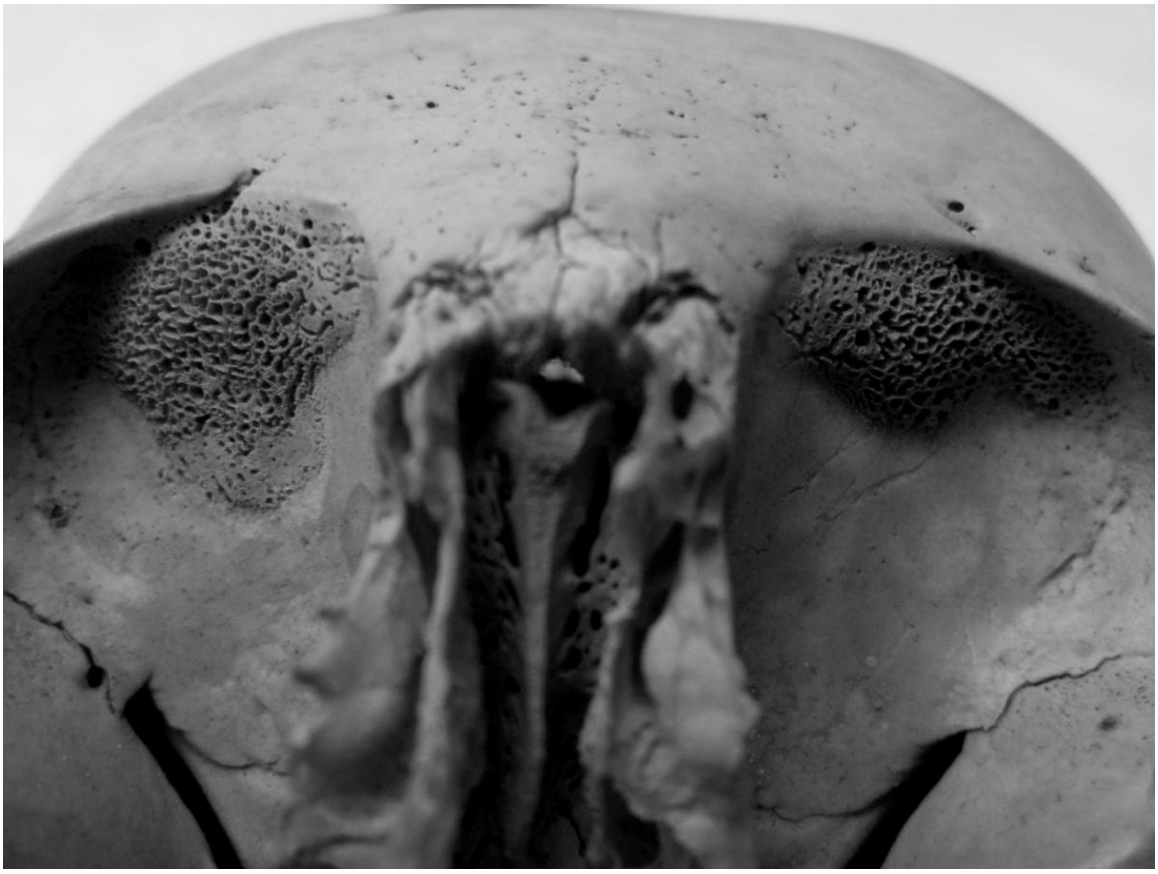

Figure 12: Hyperostotic cribra orbitalia (CO) (Cat # 2032)

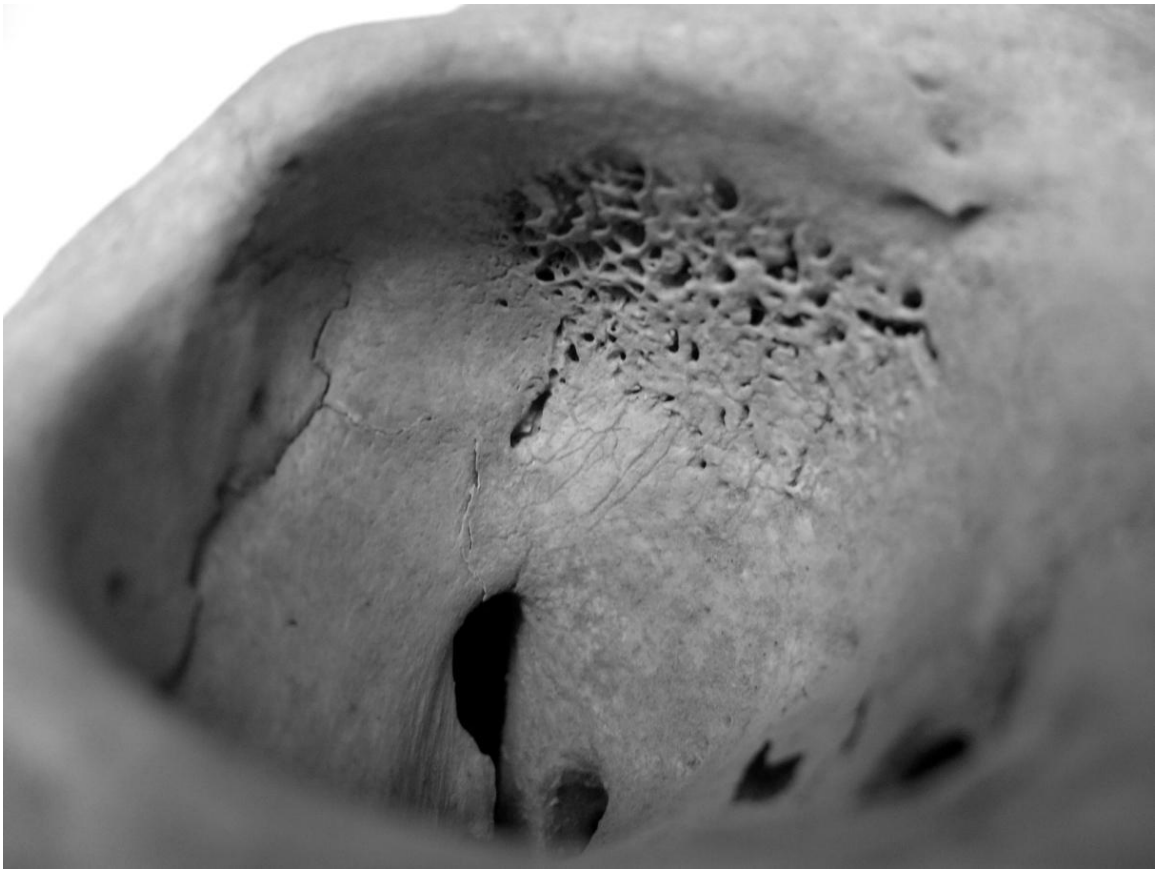

Figure 13:Possibly healing cribra orbitalia (CO?) (Cat# 2044)

In addition to the two lesions types that are considered to be active and possibly healing cribrotic lesions, there are two other general categories of lesion types: porous and inflammatory. These lesions present within the bone can present much differently than the "classic" cribrotic lesions. While numerous lesions types can occur singly or at the same time mimicking the look of a cribrotic lesion, there are numerous causes to explain their manifestations. The possibility of considering multiple causes for orbital lesions in a population is only possible when true cribrotic lesions are more narrowly defined as those with diploic expansion or coalesced pores without hypertrophic marrow.

There are four main categories that deal with porosity in the anterior and posterior orbit. AP is anterior porosity that is not coalesced. It may have some slight vascular impressions, but clear increases in vascular channel formation are scored separately as inflammatory lesions. PP is posterior porosity that does not manifest with vascular impressions. Figure 14 below shows anterior and posterior porosity.

Figure 14: Demonstrating anterior and posterior porosity (Cat# 3174)

CV is a type of porosity that expresses in a curvilinear pattern that can affect a portion of the anterior but mostly the posterior, and may display vascular channels. APIF, the coded name for anterior porosity with inflammation, is a term that indicates porosity and vascular channels with no coalesced pores that mainly affects the anterior orbit. APIF is also used when the curvilinear porosity (CV) extends significantly onto the anterior orbit beginning from the anteriolateral corner. Figure -- shows both curvinilear porosity and APIF.

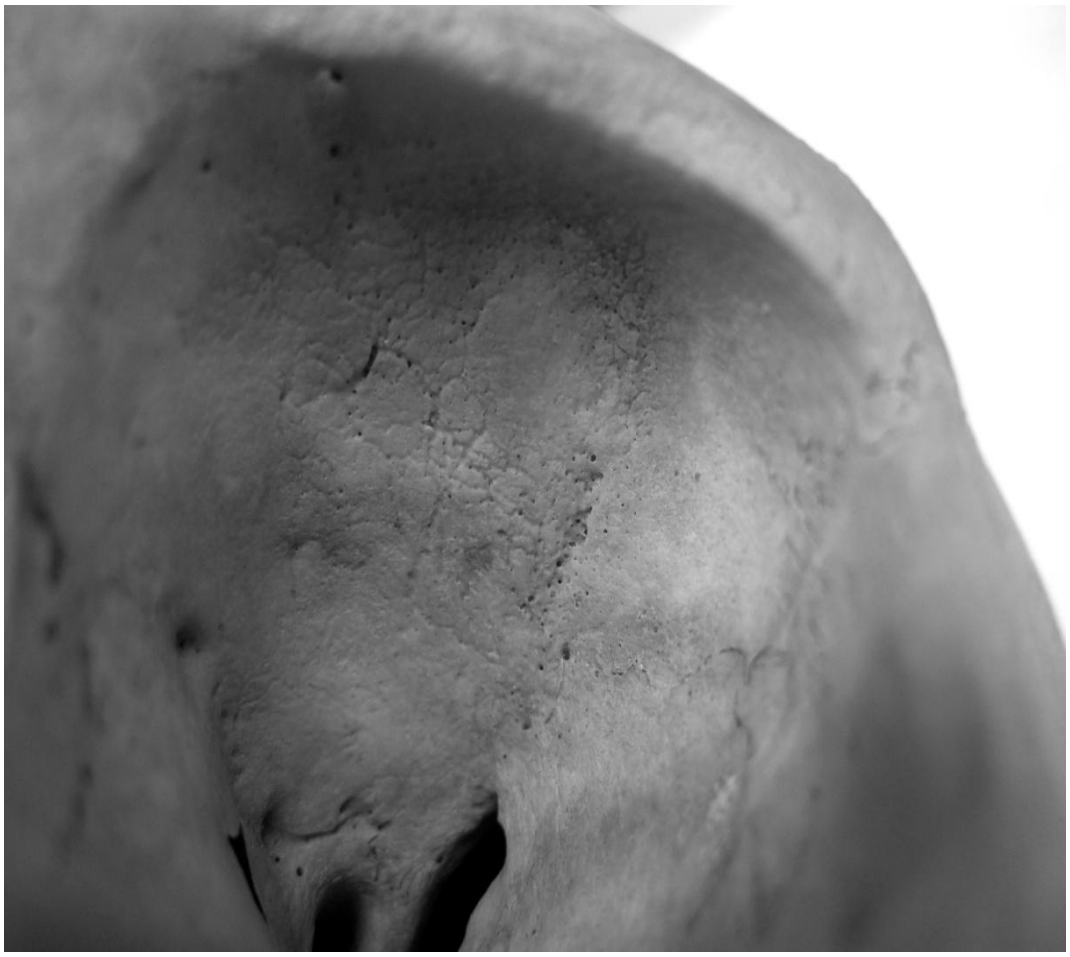

Figure 15: Curvilinear porosity and APIF (Cat# 3080)

Figure 16: Inflammatory lesion (Cat#3180)

Two categories of diagnosis involve vascular lesions with or without porosity. Figure16- above shows a typical inflammatory lesion from Peru. IF is an inflammatory lesions that has diffuse porosity and increased vascularity along the posterior orbit, and can slightly affect the anterior. IFANT is used when inflammatory lesions (IF) include extensive anterior involvement. The primary difference between IFANT and APIF is that the former is an extension of posterior inflammation that does not show the concentration along the anterior margin while the latter is most severe in the anteriolateral corner and may extend across the anterior orbital margin.

Scoring System for The Primary, Secondary and Tertiary Classification

Each orbit was scored and given a classification or classifications in cases where lesions co-occurred. Each orbit can have a primary, secondary, and tertiary classification based on the severity of the lesion. A primary lesion will be the most severe, a secondary lesion will be slightly less severe, and a tertiary lesion may not be extreme at all. In some of the analyses, the primary lesions were collapsed into five major lesion types . . . .[and indicate which of the eight went into each type]

4.4 Hypotheses

The hypotheses that I will be testing are: (1) adults and juveniles/adolescents will have significantly different prevalence of cribrotic lesions, (2) coastal populations will have a higher frequency of orbital lesions than highland groups, (3) orbital lesions will include forms other then hyperostotic and healing cribra orbitalia , and (4) that there will be a correlation between individuals with hyperostotic orbital and vault lesions. In addition to the four major hypotheses that I have, I am also going to look at geographical patterning of the lesions, and to determine which regions have the highest frequencies of orbital lesions.

4.5 Statistical Methods

Due to the nominal nature of the data, Chi Square tests and Fisher's Exact tests will be used to compare sex/age/site location to the types of lesions found in the individuals. Statistical tests were run for this thesis using SPSS™ grad pack version 20. Due to the violation of the chi square assumption, a Fisher's test was needed and the add-

on pack for exact tests was used. Basic calculations of effect size were used based on the calculated statistical values.

CHAPTER 5 – RESULTS

5.1 Marca Huamachuco

Marca Huamachuco is a highland Peruvian site located in the northern portion of Peru. Due to common practice in archaeology from the early 1900‘s, not a lot of information was published about the site or the archaeology within it. Because this site is located within the highlands, previous research predicts that there will be few if any cases of hyperostotic or healing cribra orbitalia. Viewing this site in terms of the hypotheses for this thesis, (1) true cribrotic lesions will be almost nonexistent, (2) the prevalence of all types of orbital lesions will be lower compared to coastal populations, (3) children/juveniles will have a higher prevalence and more severe manifestation of orbital lesions.

Distributions by Sex

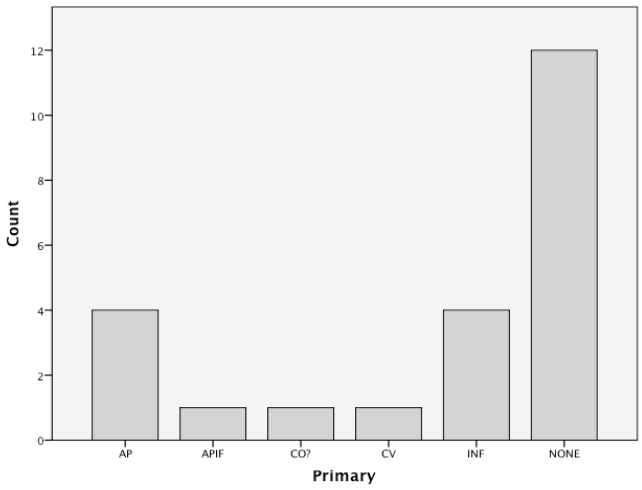

Figure 17: Primary Lesion Type at Marca Huamachuco

Figure 17 shows the primary lesion distribution for Marca Huamachuco. While populations were scored for all eight lesion categories, some lesions did not occur and those categories do not appear in the graph. Four individuals displayed anterior porosity, one showed anterior porosity with inflammation, and four showed lesions consistant with inflammation. One individual had a curvilinear lesion, and one had a case of probable healing cribra orbitalia. Twelve individuals within the site had no lesions.

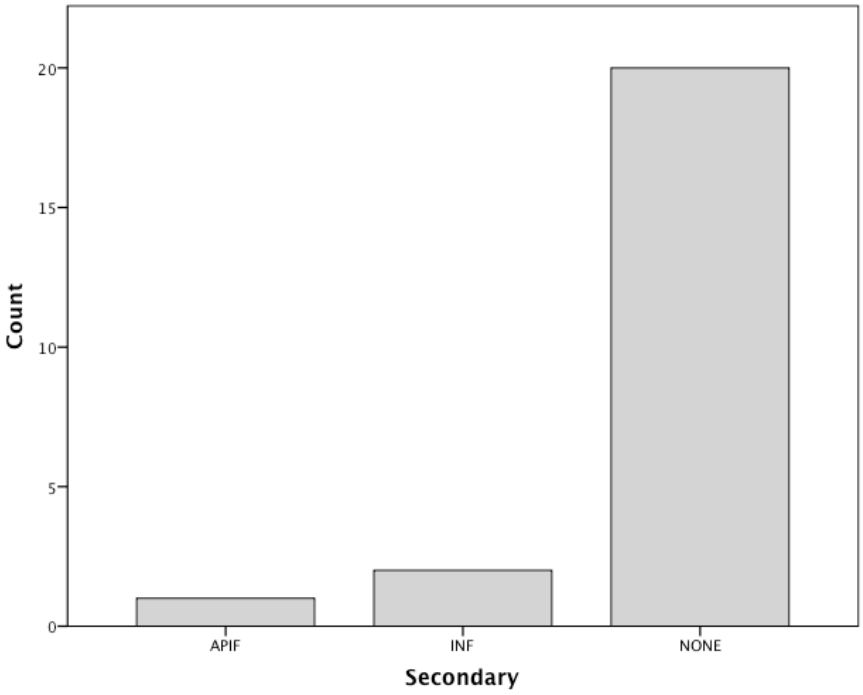

Figure 18: Secondary Lesions at Marca Huamachuco

Only three individuals shows secondary lesions in this highland site as shown in figure 18, they were consistent with inflammatory lesions (both anterior porosity with

inflammation and inflammation). There were no tertiary lesions associated with the population for Marca Huamachuco.

Analysis by Sex

Table 2: Primary lesion type by sex

Primary Lesion Type By Sex

Sex		Frequency	Percent
male	Porosity	2	28.6
	Inflammation	2	28.6
	None	3	42.9
female	CO?	1	14.3
	Porosity	1	14.3
	Inflammation	2	28.6
	None	3	42.9
indeterminant	Porosity	2	33.3
	Inflammation	1	16.7
	None	3	50.0
Indet	None	3	100.0

The numerous categories of lesion type, were condensed into five categories for this analysis: cribra, possible healing cribra, inflammatory, porous, and no lesions (Table 2). Of the females in the site, one had a possibly cribrotic lesion, one had a porous lesion, two had an inflammatory lesion, and three had no lesions. Male individuals had two porous primary lesions, two inflammatory, and three individuals with no lesions. The individuals that were indeterminate for sex, both ambiguous adults and individuals under 18, showed two individuals with porous lesions, one with an inflammatory lesion, and six with no orbital lesions.

There is no statistical significance when looking at the differences in lesion types by sex using a Fisher's exact, $\chi 2$ (9, n=23) = 6.159, p = .891.

Analysis By Age

Table 3: Primary lesion type by age groups

Primary Lesion Type By Age Groups

Age Group		Frequency	Percent
6-12 years	None	1	100.0
13-18 years	None	1	100.0
younger agult (<35)	CO?	1	20.0
	Porosity	1	20.0
	Inflammation	2	40.0
	None	1	20.0
	Total	5	100.0
mature adult (>35)	Porosity	4	28.6
	Inflammation	3	21.4
	None	7	50.0
	Total	14	100.0
indeterminant	None	2	100.0

The above table shows that the individuals under the age of 18 showed no signs of orbital lesions (Table 3). Mature adults (>35) had the highest prevalence of lesions, followed by young adults (<35). There was one individual under 12 that had no lesion, and one individual between 13 and 18 that had no lesion. The young adults had one individual displaying a possible healing cribrotic lesion, one individual with a porous lesion, two with inflammatory lesions, and one without lesions. The mature adults had four individuals with porous lesions, three with inflammatory lesions and seven with no lesions. The two indeterminate aged individuals both had no evidence of orbital lesions.

A Fisher's exact test for independence indicated no significant association between age group and primary lesion type, $\chi 2$ (3, n=21) = 2.445, p = .362.

5.2 Ancon

Ancon is a coastal lowland site located on the bay of Ancon. The hypotheses associated with this site are (1) true cribrotic lesions will be almost nonexistent, (2) the prevalence of all types of orbital lesions will be lower compared to coastal populations, (3) children/juveniles will have a higher prevalence and more severe manifestation of orbital lesions.

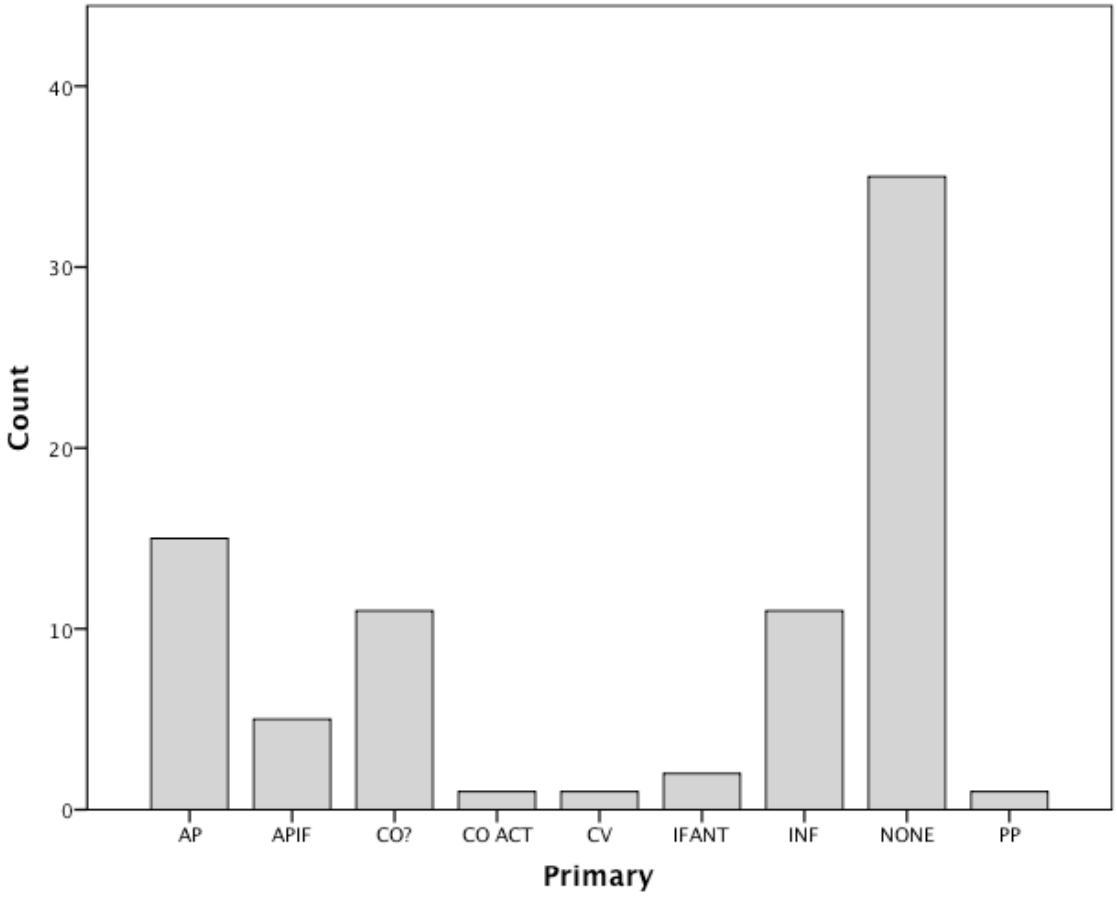

Figure 19: Primary lesions at Ancon

This chart shows the distribution of primary lesion types at Ancon (Figure 19). Out of a total of 100 individuals within the collection at the Phoebe Hurst Museum, 82 of them were able to be scored using the Wilczak and Jeney (2008) method. There were 15 individuals with anterior porosity, five with anterior porosity and inflammation. One individual had an active cribrotic lesions, and one had a curvilinear lesion, 11 had possible healing cribrotic lesions. Two had inflammatory lesions that extended onto a majority of the anterior orbit, 11 had inflammatory lesions, one had posterior porosity and 35 had no lesions.

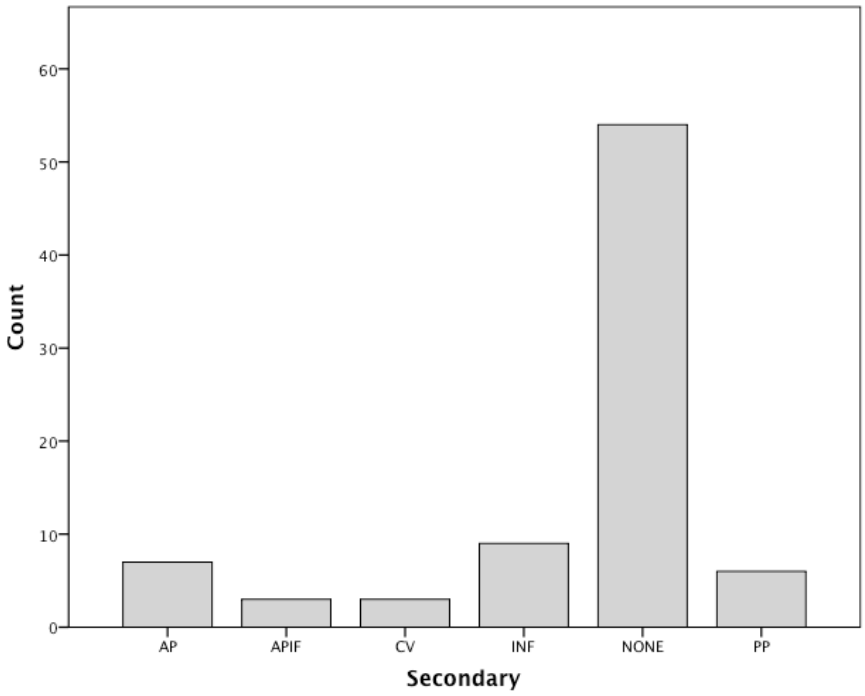

Figure 20: Secondary lesions at Ancon

Secondary lesions at this site were also common (Figure 20). Seven individuals had anterior porosity, three had anterior porosity with inflammation, and three had curvilinear lesions. Nine individuals had inflammatory lesions, six had posterior porosity, and 54 individuals had no lesions.

Not many individuals showed signs of tertiary lesions. Within the males, there was one individual with anterior porosity, and 21 with no tertiary lesion. The females had no tertiary lesions. The indeterminate individuals showed one person with an inflammatory tertiary lesion, three with posterior porosity, and 36 with no lesions.

Analysis By Sex

Table 4: Primary lesions by sex

Primary Lesion Type By Sex

Sex		Frequency	Percent
male	CO?	1	4.5
	Porous	4	18.2
	Inflammation	9	40.9
	None	8	36.4
female	CO?	3	15.0
	Porous	2	10.0
	Inflammation	2	10.0
	None	13	65.0
indeterminant	CO	1	2.5
	CO?	7	17.5
	Porous	10	25.0
	Inflammation	8	20.0
	None	14	35.0

When using the condensed categories, the males had one individual with a healing cribrotic lesion, four with porous lesions, nine with inflammatory lesions and eight individuals with no lesions (Table 4). Females had three healing cribrotic lesions, two

porous lesions, two inflammatory lesions, and 13 individuals with no lesions. Indeterminate sexed individuals had one sign of active cribra, seven individuals with healing cribra, ten with porous orbital lesions, eight with inflammatory lesions, and 14 individuals with no lesions.

Looking at the differences in lesion types by sex in Ancon, there was no statistical significance when using a Fisher's exact, $\chi 2$ (6, n=82) = 10.668, p = .090.

Analysis by Age

Table 5: Primary lesion type by age

Primary Lesion Type

Age Group		Frequency	Percent
0-5 years	CO	1	14.3
	CO?	1	14.3
	Porous	4	57.1
	None	1	14.3
6-12 years	CO?	3	27.3
	Porous	3	27.3
	Inflammation	1	9.1
	None	4	36.4
13-18 years	CO?	1	25.0
	Porous	1	25.0
	Inflammation	1	25.0
	None	1	25.0
Young Adult (<35)	CO?	3	6.5
	Porous	6	13.0
	Inflammation	16	34.8
	None	21	45.7
Mature Adult (>35)	CO?	2	20.0
	Porous	2	20.0
	Inflammation	1	10.0
	None	5	50.0
indeterminant	CO?	1	25.0
	None	3	75.0

The above table shows that the only individual displaying a hyperostotic cribrotic lesion is in the 0-5 category (Table 5). Possibly healing cribrotic lesions were seen in one individual between 0-5 years, three individuals between 6-12 years, one individual 13-18 years, three young adults (<35), and two individuals considered to be mature adults (>35), and one indeterminate aged individual (adult). Porous lesions were seen in five individuals 0-5 years, two individual between 6-12 years, one 13-18 year old, six young adults, and two mature adults. Inflammatory lesions were seen in one 6-12 year old individual, one 13-18 year old, 16 young adults, and one mature adult. One individual aged 0-5, four individual between 6-12, one between the ages of 13 and 18, 21 young adults, five mature adults, and three individuals of indeterminate age had no orbital lesions.

A Fisher's exact test indicated significant association between age group and lesions, $\chi 2$ (15, n=82) = 22.641, p = .024.

Tate-Chulpaca

Tate Chulpaca is a site located inland off the coast of Peru. Due to the common practice in archaeology from the early 1900's, very little information has been published about this site or the archaeology that Uhle completed while there.

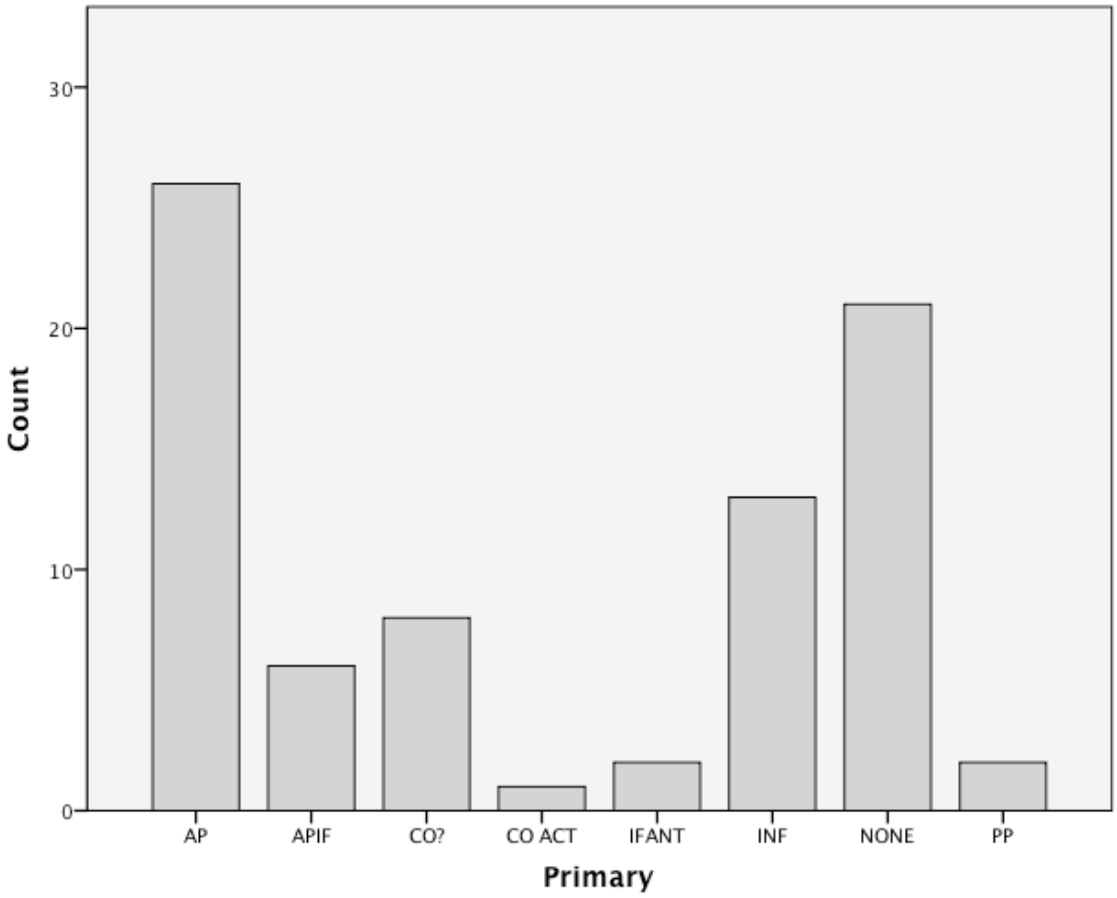

Figure 21: Primary lesion types at Tate Chulpaca

Individuals from Tate-Chulpaca demonstrated numerous porous and inflammatory lesions (Figure 21). There were 26 individuals with anterior porosity, six with anterior porosity and inflammation. One individual had hyperostotic cribrotic lesions and eight had possible healing cribrotic lesions. Two had inflammatory lesions that severely affected the anterior orbit, and 13 had inflammatory lesions. Two had posterior porosity, and 21 individuals had no lesions.

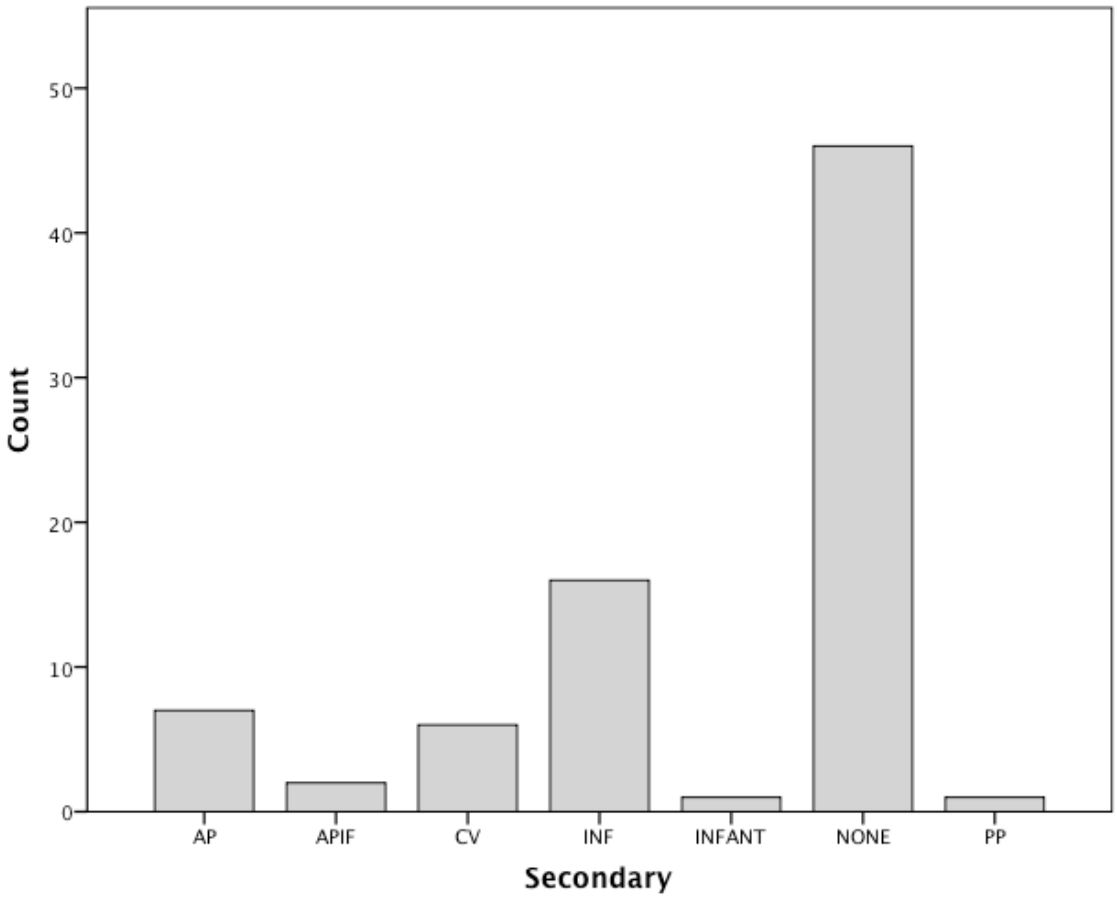

Figure 22: Secondary Lesions at Tate-Chulpaca

Secondary lesions were seen in numerous individuals from this site (Figure 22). There were seven individuals with anterior porosity, and two with anterior porosity with inflammation. Six individuals had curvilinear lesions, 16 had inflammatory lesions, and one had an inflammatory lesions that affected a majority of the anterior orbit. One individual had posterior porosity, and 46 individuals had no secondary lesions.

Tertiary lesions were seen in only three individuals. One had anterior porosity, one had a curvilinear lesion and one had an inflammatory lesion.

Analysis By Sex

Table 6: Primary lesion type by sex at Tate-Chulpaca

Primary Lesion Type By Sex

Sex		Frequency	Percent
indeterminant	CO	1	3.0
	CO?	2	6.1
	Porosity	12	36.4
	Inflammation	10	30.3
	None	8	24.2
male	CO?	3	20.0
	Porosity	6	40.0
	Inflammation	4	26.7
	None	2	13.3
female	CO?	3	10.0
	Porosity	9	30.0
	Inflammation	7	23.3
	None	11	36.7

Sex distribution of orbital lesions in Ancon is as follows (Table 6). There were three females with possibly healing cribrotic lesions, nine with porous lesions, seven with inflammatory lesions and 11 individuals with no lesions. Males had three possibly healing cribrotic lesions, six with porous lesions, four with inflammatory lesions and two individuals with no lesions. Indeterminate sexed individuals had one case of hyperostotic cribra, two healing cases of cribra, 12 porous lesions, ten inflammatory lesions and eight individuals with no lesions.

A Fisher's exact test indicated no significant association between sex and lesion type, $\chi 2$ (6, n=82) = 10.668, p = .065.

Analysis By Age

Table 7: Primary lesion type by age at Tate-Chulpaca

Primary Lesion Type By Age

Age Group		Frequency	Percent
0-5 years	Inflammation	2	100.0
6-12 years	CO?	1	12.5
	Porosity	5	62.5
	Inflammation	2	25.0
13-18 years	CO	1	25.0
	Porosity	2	50.0
	None	1	25.0
younger agult (<35)	CO?	6	13.6
	Porosity	15	34.1
	Inflammation	11	25.0
	None	12	27.3
mature adult (>35)	CO?	1	6.3
	Porosity	3	18.8
	Inflammation	6	37.5
	None	6	37.5
indeterminant	Porosity	3	60.0
	None	2	40.0

The primary lesions types were distributed as follows (Table 7). The 0-5 year olds had two individuals with inflammatory lesions. The 6-12 year olds had one with possible healing cribra, five with porous lesions, and two with inflammatory lesions. 13-18 year olds had one with active cribra, two with porous lesions, and one with no lesion. Young adults had six with healing cribrotic lesions, fifteen with porous lesions, eleven with inflammatory lesions and 12 with no lesions. Mature adults had one with a healing cribrotic lesion, three with porous lesions, six with inflammatory lesions and six with no

lesions. Adults of indeterminate age were seen to have one individual with an inflammatory lesion and three with no lesions.

Looking at the differences between adults/children and lesion types, there is no statistical significance using a Fisher's exact test, $\chi 2$ (3, n=74) = 3.901, p = .0254

5.4 Cuzco

Cuzco is a southern highland site in the inland of Peru. The hypotheses associated with this site are (1) hyperostotic cribra orbitalia will be almost nonexistent, (2) the prevalence of all types of orbital lesions will be lower compared to coastal populations, (3) children/juveniles will have a higher prevalence and more severe manifestation of orbital lesions.

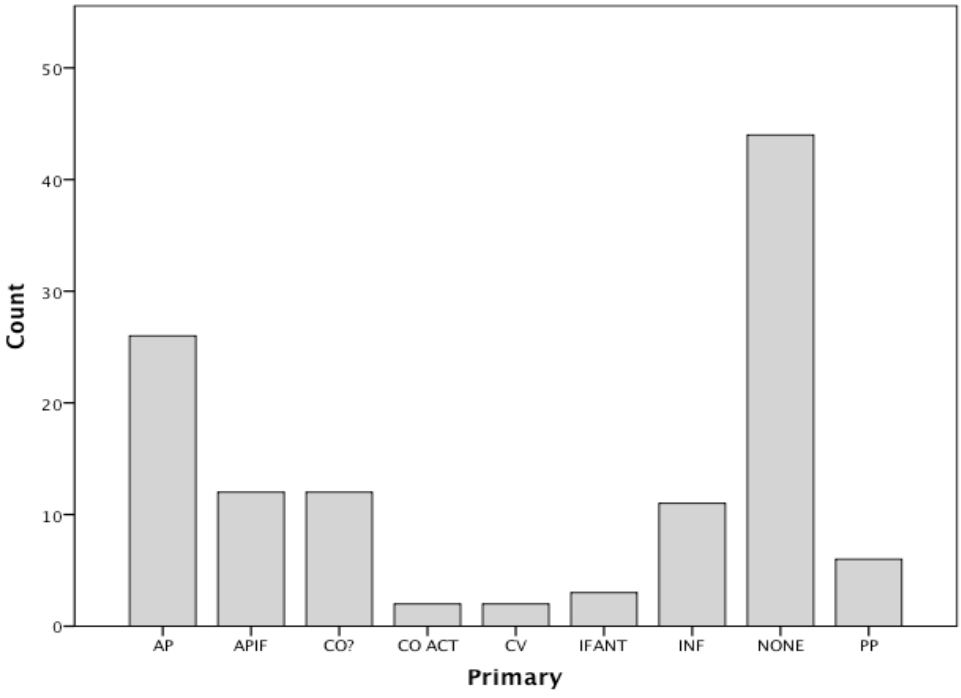

Figure 23: Primary lesion types at Cuzco

This chart shows the distribution of primary lesion types in Cuzco (Figure 23). Out of a total of 131 individuals within the collection at the Phoebe Hurst Museum, 118 of them were able to be scored using the Wilczak and Jeney (2008) method. There were 26 individuals with anterior porosity, 12 with anterior porosity and inflammation. Two individuals had hyperostotic cribrotic lesions and 12 had healing cribrotic lesions. Three individuals had inflammatory lesions that severely affected the anterior orbit, two had curvilinear lesions, and 11 had inflammatory lesions. Six individuals showed lesions consistent with posterior porosity and 44 had no lesions at all.

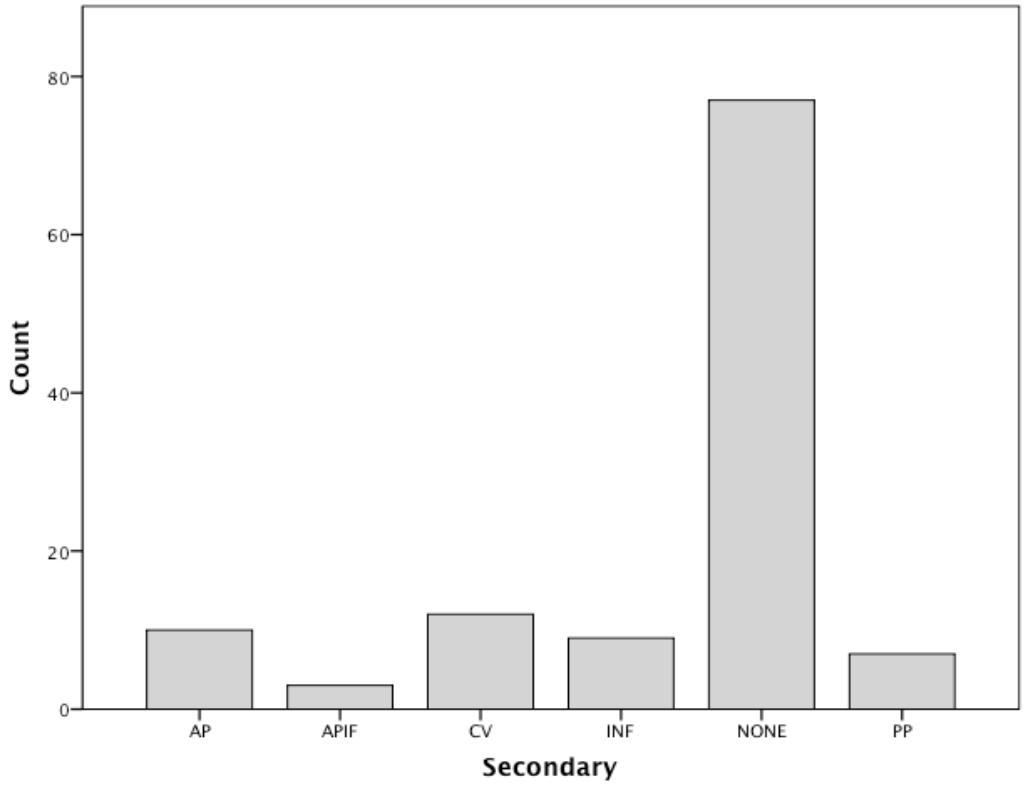

Figure 24: Secondary Lesions at Cuzco

Secondary lesions were seen in numerous individuals at Cuzco (Figure 24). There were ten with anterior porosity, three with anterior porosity and inflammation. Twelve individuals had curvilinear leisons, and nine had inflammatory lesions. Seven individuals showed signs of posterior porosity and 77 had no secondary lesions.

Tertiary lesions were seen in four individuals. Three had curvilinear lesions and one had and inflammatory lesion.

Analysis by Sex

Table 8: Sex by primary lesion type at Cuzco

Sex by Lesion Types

			Lesions				
			CO	Porous	Inflammation	None	Total
Sex	male	Count	2	7	5	6	20
		% within Sex	10.0%	35.0%	25.0%	30.0%	100.0%
	female	Count	3	13	8	11	35
		% within Sex	8.6%	37.1%	22.9%	31.4%	100.0%
	indeterminant	Count	9	14	13	27	63
		% within Sex	14.3%	22.2%	20.6%	42.9%	100.0%
Total		Count	14	34	26	44	118
		% within Sex	11.9%	28.8%	22.0%	37.3%	100.0%

When using the condensed categories, males had two individuals with possible healing cribra, seven with porous lesions, five with inflammatory lesions, and six individuals with no lesions (Table 8). Females had three individuals with healing cribra, 13 with porous lesions, eight with inflammatory lesions, and 11 with no lesions. The indeterminate individuals had two with a hyperostotic cribrotic lesions, seven with

healing cribra, 14 with porous orbital lesions, 13 with inflammatory lesions, and 27 individuals with no lesions.

Looking at the differences between sex and lesion types, there is statistical significance using a Fisher's exact test, $\chi 2$ (10, n=118) = 20.592, p = .006.

Analysis by Age

Table 9: Age Groups By Primary Lesion Types at Cuzco

Age Group and Primary Lesions

			Lesions			
			CO	Porous	Inflammation	None
Age Group	0-5 years	Count	0	0	1	0
		% within Age Group	0.0%	0.0%	100.0%	0.0%
	6-12 years	Count	2	5	2	5
		% within Age Group	14.3%	35.7%	14.3%	35.7%
	13-18 years	Count	1	0	0	0
		% within Age Group	100.0%	0.0%	0.0%	0.0%
	younger agult (<35)	Count	9	17	14	23
		% within Age Group	14.3%	27.0%	22.2%	36.5%
	mature adult (>35)	Count	0	8	7	8
		% within Age Group	0.0%	34.8%	30.4%	34.8%
	indeterminant	Count	2	4	2	8
		% within Age Group	12.5%	25.0%	12.5%	50.0%
Total		Count	14	34	26	44
		% within Age Group	11.9%	28.8%	22.0%	37.3%

Table 9 shows that in the 0-5 year category, there is one individual with an inflammatory lesion. For those 6-12 years, there are two individuals with active cribra, five with porous orbital lesions, two with inflammatory lesions, and five with no lesion. For the 13-18 year olds, one had healing cribra orbitalia. Young adults (<35 years)

shows nine individuals with healing cribra, 17 with porous lesions, 14 with inflammatory lesions, and 23 with no lesion. Mature adults have 8 individuals with porosity, seven with inflammation, and eight with no lesions. Indeterminate individuals show two healing cribrotic lesions, four porous lesions, two inflammatory lesions, and 8 individuals with no lesions.

Looking at the differences between age groups and lesion types, there is no statistical significance using a Fisher's exact test, $\chi 2$ (15, n=118) = 14.818, p = .448.

Results by Geographic Location

Hrdlicka (1916), and many other authors including have stated that highland populations do not have the high number of orbital lesions that are seen in coastal and lowland populations (Table 10).

Table 10: Site Location and the Presence of Lesions

Site Location and the Presence of Lesions

			Lesion or No Lesion		
			Yes	No	Total
Site Location	Highland	Count	85	56	141
		% within Site Location	60.3%	39.7%	100.0%
	Lowland	Count	104	56	160
		% within Site Location	65.0%	35.0%	100.0%
Total		Count	189	112	301
		% within Site Location	62.8%	37.2%	100.0%

There is no statistical significance between the number of highland or lowland Peruvians who present or do not present orbital lesions $\chi 2$ (1, n=301) = .714, p = .406.

Table 11: Primary Lesion Types by Geographic Location

Lesions

Site Location			Frequency	Percent
Highland	Valid	CO	15	10.6
		Porous	39	27.7
		Inflammation	31	22.0
		None	56	39.7
Lowland	Valid	CO	21	13.0
		Porous	43	26.7
		Inflammation	40	24.8
		None	56	34.8

When considering the difference between the highland and lowland locations, as seen in Table 11, in terms of general lesion types, there was no statistical significance $\chi 2$ (3, n=301) = 1.141, p = .767.

Results by Age

Table 12: Adults/Children with or without lesions

			Lesions	
			None	Total
AgeNew	Child to Adolescent (<18)	Count	14	54
		% within AgeNew	25.9%	100.0%
	Adult (>18)	Count	83	221
		% within AgeNew	37.6%	100.0%
Total		Count	97	275
		% within AgeNew	35.3%	100.0%

Looking at the differences between adults and children who manifest and do not manifest lesions (Table 12), there is statistical significance at the .05 level. $\chi^2(3,N=275)=9.333,p=.023$.

Table 13: Adults/Children and Primary Lesion Type

		Lesions			
		CO	Porous	Inflammation	None
AgeNew	Child to Adolescent (<18)	11	20	9	14
		20.4%	37.0%	16.7%	25.9%
	Adult (>18)	22	56	60	83
		10.0%	25.3%	27.1%	37.6%

The difference is lesion types by adults and children, regardless of geographic location, shows statistical significance $\chi 2$ (3, n=275) = 9.637, p = .021 using a Chi Square test (Table 13). While the sample size is fairly small for children (N=54), there is still a difference.

Table 14: Adults/Children and primary lesions

Adults/children and Primary Lesion Types

		Primary Lesion Type				
		CO	CO?	Porosity	Inflammation	None
AgeNew	Child to Adolescent (<18)	4	7	20	9	14
		7.4%	13.0%	37.0%	16.7%	25.9%
	Adult (>18)	0	22	56	60	83
		0.0%	10.0%	25.3%	27.1%	37.6%

The difference is lesion types by adults and children when taking into account the differences between hyperostotic and healing cribra (Table 14), shows statistical significance $\chi 2$ (4, n=275) = 22.456, p = .001 using a Chi Square test.

5.8 Specific Orbital Lesions in Highland and Lowland Peru

It has been stated in many other articles that the prevalence of cribra orbitalia is higher in lowland areas than in highland areas possibly due to the increased access to marine parasites and poor sanitation. When this was tested, it was not shown to be true for populations in this study.

Table 15: Cribra orbitalia highland Vs. lowland Peru

Site Location and Presence of Cribra

		Cribra or Not	
		Yes	No
Site Location	Highland	15	126
		10.6%	89.4%
	Lowland	21	139
		13.1%	86.9%

Looking at the prevalence of cribra orbitalia in the highlands and lowlands between all the individuals (Table 15), with or without lesions, shows that there is no statistical significance $\chi 2$ (1, n=301) = .440, p = .594 using a Chi Square test.

Inflammatory Lesions in Highland and Lowland Peru

Similar to cribrotic lesions, it has been postulated that inflammatory lesions will be more prevalent in lowland coastal populations than in highland populations due to the same stressors that cause cribrotic lesions, parasites and chronic inflection causing depletion of nutrients. This thesis shows that this is not the case for the individuals in these Peruvian cities.

Table 16: Inflammatory Lesions Highland Vs. Lowland

Site Location and Inflammatory Lesions

		Inflammatory Lesions	
		Yes	No
Site Location	Highland	31	110
		22.0%	78.0%
	Lowland	40	120
		25.0%	75.0%

Looking at the prevalence of inflammatory lesions in the highlands and lowlands using all of the individuals with or without lesions (Table 16), shows that there is no statistical significance $\chi 2$ (1, n=301) = .378, p = .539 using a Chi Square test.

Correlations Between Cribrotic Orbital Lesions and Vault Lesions

Table 17: Correlations between cribra orbitalia and vault porosity

Cribra and Vault Porosity

		Vault Porosity		
		yes	no	unknown
Cribra or Not	Yes	59	16	7
		72.0%	19.5%	8.5%
	No	150	54	14
		68.8%	24.8%	6.4%

Blom and coauthors (2004) also noticed a correlation between hyperostotic orbital and vault lesions. Other researchers have noticed similar trends as well. For this project, using a Chi Square test (Figure 17), there was not a statistically significant result

correlation cribra with porotic hyperostosis $\chi 2$ (2, n=300), =1.171, p = .579.

CHAPTER 6 – DISCUSSION

Cribra orbitalia is a pathological condition often seen and associated with archaeological sites (Walker et al. 2009). This type of orbital lesion was associated with anemia since the 1960's, and since then, other research has changed the etiological understanding of cribrotic lesions in the past half of a century (Angel 1966, Walker et al. 2009, Walper 2004). Cribra orbitalia has been diagnosed as any porosity within the orbit, and was once described as "sieve-like" by Schultz (2001:133) and like coral growth by Hrdlicka (1914), but not all porous lesions are necessarily caused by anemia. The fact that it has become synonymous with anemia, when some these lesions are in fact not related to most forms of anemia is why this new classification system has been created. The ability to differentially classify orbital lesions can lead to new understandings of health in the past. The major problems associated with these types of hyperostotic lesions is that they have numerous etiologies and can be tricky to differentially diagnose. However, the hyperostotic lesions are the ones that are most likely to be related to certain types of anemia like megaloblastic anemia. Orbital lesions can manifest in the bone as porosity, hyperostotic lesions, and increased vascularization. However, these impressions left in the bone can be caused by normal variation as well, the severity of the lesions can help researchers to determine if the lesions is pathological or within the realm of normal variation.

Differential diagnosis has been a major focal point in paleopathology within the current literature for good reason. Many diseases can create similar manifestations on bone, such as the orbital lesions associated with both cribra orbitalia and scurvy (Marks and Hamilton 2007:217). These similar lesions necessitate a differential diagnosis in order to properly understand the nature of disease itself and proper diagnosis which is why the Wilczak and Jeney (2008) method lends itself to this research. Differential diagnosis views all possible causes for the lesions, pathognomic (diagnostic) features, compiles a detailed summary of the lesion and its physical form, and then chooses a best fit diagnosis.

The history of cribrotic lesions and other types of orbital lesions has driven many researchers to reevaluate methodology. This thesis follows in the same direction. Due to the changing nature of the etiology ideas and the numerous manifestations of orbital lesions, it is very important to continue to reevaluate and challenge previous methodology that may be incorrect in light of new research. Cribra orbitalia has been the focus of many famous scientists and each one has contributed to the overall understanding of a complex disease. Hrdlicka described this type of lesion as a condition that manifests during infancy and affects the cranium (1914). Even in 1913, he thought it was a reaction to a toxin and not a nutritional or degenerative pathology. In 1966, J. Lawrence Angel began to associate porotic hyperostosis and cribra orbitalia with inherited forms of anemia, especially thalassemia and sicklemia (Angel 1966). New World lesions were most likely caused by iron deficiency anemia due to a regimented

diet lacking in nutritional value, and/or nutritional depletion due to lactation. In the 1980's-1990's Patty Stuart-Macadam published a series of papers delving into both the etiology and manifestations of both cribra orbitalia and porotic hyperostosis (1985, 1987a, 1987b, 1991, 1992). By 1992, it was seen that bioavailability of dietary iron was only one small component to the overall health and the manifestation of these lesions. If it is possible to have cribrotic orbital lesions without iron deficiency anemia, other causes must be examined and tested.

Throughout all of the research that has been compiled during the writing of this thesis, numerous challenges to the research and the data analysis were encountered. One of the major limitations of this study is that during the early 1900's archaeological standards were not as rigorous as they are today. Most anthropologists traveling to other countries were looking just for the best specimens they could find. Uhle was a product of his time and like similar scholars of that age, the focus was on the most impressive skeletal remains. Most of the post cranial bones were left behind due to the desire from the states to have the skulls, and the thoughts that the skulls were the most important portion of the body. Children and pathological remains from adults were some of the most highly sought after remains to bring back home to study.

Both Hrdlicka and Uhle, during their time in Peru, collected mostly skulls. The postcranial remains were not as highly sought after, and in the case of Hrdlicka when he was traveling through Peru, he was brought to homes where there were nothing but skulls littering the floors. Part of this was due to the high rates of looting in Peru during the

Spanish occupation, and part due to the nature of the secondary burials in which the skulls were removed from the original tombs and placed in other tombs.

Through this research, the expected findings was that children and adults will have significantly different prevalence rates of cribra orbitalia, and that coastal populations will have higher prevalence of cribra orbitalia as compared to the highland populations. Walker and coauthors (2009) supported that childhood anemia would most likely be the cause for severe lesions because most active porotic lesions with no remodeling were found in juvenile and adolescent remains. Adults were found to have signs of remodeled lesions without the severity of the childhood lesions (Stuart-Macadam 1985; Walker 1985, 1986).

In terms of geographical patterning associated with cribra orbitalia, it has been shown that coastal populations are more likely to manifest orbital lesions than the highland groups. Walker et al. (2009) noted that parasites and chronic sickness can also cause anemia by depleting B12. It has been shown that pre-Columbian mountain populations in Peru showed no orbital lesions when they were examined in the early 1900's (Hrdlicka 1914, Blom et al 2005). Blom and coauthors found in the 2005 study that individuals were more likely to have childhood anemia in arid environments. They also were able to show that children buried in the lower altitudes, possible raised in lowland areas, who were closer to the coast, and had a diet heavy in marine resources were less likely to die.

Each of the sites tested was analyzed individually first to look at the inter site differences between people. For Marca Huamachuco, Ancon, Cuzco, and Tate-Chulpaca there were no significant differences at the .05 alpha level between the sexes and the types of orbital lesions present. When analyzing by age, Marca Huamachuco, and Cuzco, were not significant at an alpha level of .05, but both Ancon and Tate-Chulpaca showed statistical significance indicating that there was a difference in the ages of individuals and the orbital lesions that they manifested.

In terms of geographical patterning of orbital lesions between the highlands and the lowlands in Peru, it was seen that there was not statistical significance . Many other scholars, including Hrdlicka, thought that the geography would influence the types of lesions seen. For the individuals sampled in this thesis, that was not seen to be the case when viewing all of the presence/absence of lesions in the different areas. When all of the lesions were viewed in terms of their diagnosis, the same result occurred, no statistical significance between highland and lowland individuals .

In terms of sex, a statistically significant result of p=.022 (Fisher) occurred when testing for the differences between adults/children and the presence or absence of lesions. This coincides with other research stating that orbital lesions, especially true cribrotic lesions, are seen most often in children rather than adults. The difference is lesion types by adults and children when taking into account the differences between active and healing cribra, shows statistical significance, p = .021 using a Chi Square test.

The prevalence of numerous lesions types is said to vary between highland and lowland Peru. Two of the most important diagnoses that have been seen to differ significantly from highland to lowland regions are: cribra orbitalia and inflammatory lesions. It was originally postulated that the high altitude hypobaric environment can cause and aggravate anemia. This thesis showed that the current sample from the PAHMA showed no statistical differences between highland and lowland Peruvians in terms of cribrotic lesions. This indicates that even though individuals in highland Peru may have been acclimatized to the low oxygen environment, cribrotic lesions still occur, leading to the agreement with other researchers that an iron based anemia etiology is not an appropriate diagnosis for cribra orbitalia.

Inflammatory lesions were said to be most common in lowland coastal populations due to parasite load and chronic infections from these parasites. Looking at the prevalence of inflammatory lesions in the highlands and lowlands within the sample for this thesis, there was no significance at the .05 level.

The differing prevalence rates from inflammatory lesions and cribrotic lesions in highland Peru indicate that there is more occurring than originally thought. While the diets of ancient Peruvians in each of these locations is vastly different, the lesion types are not. Blom and coauthors (2005) postulated that a more maize-based diet could lead to anemia. Ancon, while unsuitable for growing corn had very similar numbers of cribrotic lesions as highland Peru where corn is a staple crop. Originally, highland populations were seen to have no orbital lesions at all (Hrdlicka 1916), but this new method shows

that there are clearly orbital lesions present in highland Peru. It could be that if Hrdlcika was looking for the coral growth appearance, that he may not have scored these types of lesions. The vastly different methods between Hrdlicka and myself can also account for the variability in prevalence of lesions between highland and lowland Peru.

Blom and coauthors (2005) found a high number of individuals with the pathology along the coastal regions of Peru. Specifically that 23.1% (n=402) of adults in the sample exhibited signs of cribra orbitalia (Blom 2005). In children, the prevalence was 81.8% (n=66). In this study, the prevalence of cribrotic lesions in lowland coastal Peru was significantly lower (22.2% for children and 10.3% for adults). Blom et al. did find an association between cribra orbitalia and porotic hyperostosis which was not found to be statistically significant in this thesis (p=.301).

Throughout this thesis and data analysis, it was seen that this research differed significantly from previous research. Since the method used in this thesis was not used on the other sites studied, it is understandable that the result vary. It is important to be consistent when using a method so that the data is replicable. Since orbital lesions are so often classified in archaeological sites, standardized methodology, in particular in terms of seperating out "true cribrotic" lesions from other types of lesions, would assist in creating consistency within the field of paleopathology. Over the last 100 years, the understanding of these lesions has changed drastically. As the understanding of the lesions evolves, so must the methods. That is what makes this thesis so integral to the current research in the field. Now that multiple classes of lesions have been seen to be caused by a number of condition, some just by normal variation, it is integral to take the step to classify them separately. While this is still just an early attempt to separate these lesions and see if they can tell scholars anything about health and wellness, further research and study on these different classifications of lesions through time and locations could add to the current understanding of orbital lesions. Future research in Peruvian collections would benefit from being analyzed using this macroscopic and non destructive method as there are so many sets of skeletal remains spanning numerous time periods all over North and South America.

The research completed in this thesis shows that orbital lesions and their manifestations can vary between the sexes, age groups, and the geographical location of the individuals. Even though numerous tests were not statistically significant, that does

not indicate that these results are less important or incorrect. These results are just a stepping-stone for further research to be completed on more areas in Peru, and for other museums housing Peruvian skeletal materials to allow researchers to adapt this method to view orbital lesions. More research on collections from around the world can give a more accurate and reliable view of skeletal health.

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APPENDIX 1 - Orbital Lesions Method Complete

Phoebe Hearst Museum Scoring System (Wilczak and Jeney 2008)

Anterior/Corner and Posterior Porosity: Both are scored on Pore size, Density and Percent affected

Pore size: RAPS (right anterior pore size), LAPS (left anterior pore size), RPPS (right posterior pore size), LPPS (left posterior pore size)

0 = no porosity
1 = pinpoint
2 = pinpoint to 0.5 mm
3 = > 0.5 mm
4 = coalesced (also include if pore density low but at least 2 pores overlap)

Pore Density: RAD (right anterior density), LAD (left anterior density), RPD (right posterior density), LPD (left posterior density)

0 = no porosity
1 = low < 15 / cm sq.
2 = moderate 15-25
3 = high 25-50 cm sq.
4 = coalesced, generally high density with at least ¼ of area showing coalescence to give cribriform appearance.

Porosity % affected: RAP (right anterior percent), LAP (left anterior percent), RPP (right posterior percent), LPP (left posterior percent)
0 = none
1 = ¼
2 = ¼-1/2
3 = > ½

Diploic expansion: RADip (right anterior diploe), LADip (left anterior diploe), RPDip (right posterior diploe), LPDip (left posterior diploe)

0 = none
1 = slight, barely discernible
2 = clearly present
3 = severe exceeds 1 mm in height

Vascular Channels: RAV (right anterior vascularity), LAV (left anterior vascularity), RPV (right posterior vascularity), LPV (left posterior vascularity)

0 = none
1 = barely discernible to slight
2 = mild, clear channels but on less than 1/3
3 = moderate, clear channels on 1/3 -1/2
4 = severe, clear channels on greater than ½

Plaque(Plaq) = well-defined surface deposit of smooth bone
0 or blank = no plaques present
Record # of discrete plaques if present

Plaque size = maximum diameter of bone plaques
Plaque Location (PlaqL): anterior or posterior orbit
Blank = no plaques present

Classifications

CO active = Coalesced, plus Diploic expansion on anterior including ant/lat corner
CO? = Coalesced on anterior orbit including anteriolateral corner
AP = Anterior affected by porosity (not coalesced) can have slight vascular impressions
PP = Porosity on posterior, no vascular channels.
IF = inflammatory lesion with diffuse porosity and vascular channels across posterior, may also partially affect anterior but if extensive use **IFANT**
APIF = porosity and vascular channels with no coalesced pores in ant lateral corner
CV= curvilinear posterior with possible anterior affected with porosity and/or vascular channels. Mostly limited to posterior orbit, if significant extension onto anterior surface, code with **APIF**.

APPENDIX 2 - Coding for Data Sheet

Usable
1. Yes
2. No

Sex
1. Male
2. Female

Age
1. 0-5 years
2. 6-12 years
3. 13-18 years
4. Younger adult (<35)
5. Mature adult (>35)

Les (Lesions)
1. CO
2. Porous
3. Inflammation
4. None

Primary Lesion Types
1. CO
2. CO?
3. Porous
4. Inflammatory
5. None

Vault Porosity
1. Yes
2. No

Porotic Hyperostosis
1. Yes
2. No

APPENDIX 3 - Raw Data

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
1753	Ancon	1	41.1+/-10 years	5	1	2	3	AP	INF	NONE	2	2	1	1	2	2	0
3029	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	<1	<2	<1	<1	<1	<1	0
3030	Ancon	1	<35	4	1	3	4	INF	NONE	NONE	2	2	<1	<1	<1	<1	0
3031	Ancon	3	<35	4	1	1	2	CO?	CV	PP	4	4	4	4	3	4	0
3032	Ancon	3	10+/-30 months	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3033	Ancon	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3034	Ancon	3	4+/-12 months	1	1	2	3	AP	PP	NONE	2	2	1	1	1	1	0
3035	Ancon	1	32+/- 8.3 years	4	1	1	2	CO?	INF	NONE	4	4	4	4	1	3	0
3035A	Ancon				2												
3036	Ancon	3	32+/- 8.3 years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3037	Ancon	3	indeterminate	6	1	1	2	CO?	INF	NONE	4	4	4	3	3	0	
3038	Ancon	1	<35	4	1	3	4	INF	APIF	NONE	2	2	1	1	1	0	
3039	Ancon				2												
3040	Ancon																
3041	Ancon	3	indeterminate	6	1	4	5	NONE	NONE	NONE	2						
3042	Ancon	3	15+/-36 months	3	1	4	5	NONE	NONE	NONE	0	2	0	<1	0	<1	0
3043	Ancon	1	32+/-8.3 years	4	1	4	5	NONE	NONE	NONE	3	2	1	1	1	1	0
3044	Ancon	3	<35	4	1	4	5	NONE	NONE	NONE		2		<1		<1	
3045	Ancon	3	32+/-8.3 years	4	1	4	5	NONE	NONE	NONE		0		0		0	
3046	Ancon	1	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3047	Ancon	1	<35	4	1	3	4	INF	CV	AP	<1	2	<1	1	<1	2	0
3048	Ancon	1	<35	4	1	2	3	AP	NONE	NONE	1	2	1	1	1	1	0
3051	Ancon	3	3+/-12 months	1	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3052	Ancon				2												
3053	Ancon	3	5+/-16 months	1	1	1	2	CO?	PP	NONE	3	3	2	2	3	2	0
3054	Ancon	3	6+/-24 months	2	1	4	5	NONE	NONE	NONE	2	2	<1	<1	<1	<1	0
3055	Ancon	3	15+/-36 months	3	1	3	4	IFANT	AP	NONE	2	2	1	1	2	2	0
3056	Ancon	3	7+/-24 months	2	1	3	4	INF	AP	NONE	2	2	1	1	1	1	0
3056A	Ancon				2												
3057	Ancon	2	<35	4	1	2	3	AP	NONE	NONE	3	3	1	1	1	1	0
3058	Ancon	3	4+/-12 months	1	1	2	3	AP	NONE	NONE	2	2	1	1	2	3	0
3058A	Ancon				2												
3058B	Ancon				2												
3059	Ancon	3	5+/-16 months	1	1	1	1	CO ACT	INF	PP	4	4	4	4	3	3	3
3059A	Ancon				2												
3060	Ancon	3	10+/-30months	2	1	1	2	CO?	APIF	INF	4	4	4	1	3	2	0

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0	1	2	1	2	1	1	1	0	0	2	2	1	Pos			.25x.50crt	2	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	2	2	0	-	-	-	-	2	1	2	1
0	1	1	1	1	1	1	1	0	0	2	2	0	-	-	-	-	2	1	2	1
0	1	1	2	2/3	1	2/2	1	2/2	0	2	1	0	-	-	-	-	2	1	2	1
0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	1
0	1	1	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	1
0	1	1	2	0	1	0	1	0	0	1	1	0	-	-	-	-	2	1	2	1
0	2	2	1	1/2	1	1	1	0	0	2	2	0	-	-	-	-	2	1	2	1
																				1
0	1	1	<2	<2	<1	<1	<1	0	0	2	2	0	-	-	-	-	2	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	-	2	2	2	1
0	1	2	2	2	1	1	1	0	0	2	2	0	-	-	-	-	2	1	2	1
																				1
																				1
1												0	-	-	-		3	3	2	1
0	1	1	0	0	0	0	0	0	0	1	1	0	-	-	-		2	2	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-		2	1	2	1
0	1	1		<1		<1	<1	<1	0	0	1	0	-	-	-		2	2	2	1
0			0		0		0		0	1	0	0	-	-	-		2	2	2	1
0	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-		2	1	2	1
0	1	1	2	2	1	1	2	0	0	2	2	0	-	-	-		2	1	2	1
0	0	1	0	0	0	0	0	0	0	2	2	0	-	-	-		2	1	2	1
0	0	0	1	1	1	0	1	0	0	2	2	0	-	-	-		2	1	2	1
																				1
0	1	0	4	4/1	1	1/1	1	0	0	1	2	0	-	-	-		2	1	2	1
0	1	1	0	0	0	0	0	0	0	1	1	0	-	-	-		2	1	2	1
0	2	1	1	1	1	1	1	0	0	2	2	0	-	-	-		2	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	2	2	0	-	-	-		2	1	2	1
																				1
0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-		2	1	2	1
0	1	1								0	0	0	-	-	-		2	2	2	1
																				1
1	1	1	2	2	1	1	1	0	0	2	1	0	-	-	-		2	1	2	1
1	1	1	2	2	1	1	1	0	0	2	1	0	-	-	-		2	1	2	1
0	2	1	1	1	1	1	1	0	0	2	2	0	-	-	-		2	1	2	1

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
3061	Ancon				2												
3062	Ancon	3	5+/-16 months	1	1	2	3	AP	PP	NONE	2	1	1	3	3	0	
3063	Ancon	1	32 +/-8.3 years	4	1	3	4	APIF	INF	NONE	1	2	1	1	1	0	
3064	Ancon	1	34.7+/-7.8 years	4	1	3	4	IFANT	NONE	NONE	<1	<1	<1	<1	<1	0	
3065	Ancon	1	32+/-8.3 years	4	1	3	4	INF	APIF	NONE	<1	<1	<1	<1	<1	0	
3066	Ancon	3	39.4+/-9.1years	5	1	3	4	APIF	NONE	NONE		0		0		0	
3067	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	0	<1	0	<1	0	<1	0
3068	Ancon	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3069	Ancon				2												
3070	Ancon	3	7+/-24 months	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3071	Ancon	1	36.2+/-6.2 years	5	1	4	5	NONE	NONE	NONE		0		0		0	
3072	Ancon	3	indeterminant	6	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3074	Ancon	3	32+/-8.3 years	4	1	3	4	INF	AP	NONE	0	2	0	1	0	1	0
3075	Ancon	2	32+/-8.3 years	4	1	3	4	APIF	PP	NONE	1	1	1	1	1	0	
3076	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3077	Ancon	3	15+/-36 months	3	1	2	3	AP	NONE	NONE		2		1		1	
3078	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	2	2	<1	<1	<1	<1	0
3079	Ancon	3	11+/-30 months	2	1	1	2	CO?	NONE	NONE	4	4	4	4	3	2	0
3080	Ancon	1	<35	4	1	3	4	CV	AP	NONE	1	1	<1	1	<1	2	0
3081	Ancon	3	30.5+/-9.6 years	4	1	3	4	INF	NONE	NONE	0	0	0	0	0	0	0
3081A	Ancon				2												
3082	Ancon	1	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3083	Ancon	2	32+/-8.3 years	4	1	4	5	NONE	NONE	NONE	0	<1	0	<1	0	<1	0
3083A	Ancon				2												
3084	Ancon	1	32.0+/-8.3 years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3085	Ancon	3	3+/-12 months	1	1	2	3	AP	PP	NONE	2	3	1	1	1	0	
3088	Ancon	3	34.7+/-7.8years	4	1	3	4	APIF	NONE	NONE	2	<1	<1	<1	<1	0	
3088A	Ancon				2												
3090	Ancon	1	34.7+/-7.8 years	4	1	3	4	INF	AP	NONE	2	2	1	1	2	2	0
3090A	Ancon				2												
3091	Ancon	3	6+/-24 months	2	1	1	2	CO?	AP	PP	3	3	1	1	3	3	0
3093	Ancon	3	30.5+/-9.6 years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3094	Ancon	1	41.1+/-10 years	5	1	2	3	AP	PP	NONE	2	0	1	0	1	0	
3095	Ancon	1	34.7+/-7.8years	4	1	3	4	INF	NONE	NONE	0	<1	0	<1	0	<1	0
3096	Ancon	2	41.1+/-10	5	1	1	2	CO?	INF	NONE	4	3	4	1	3	2	0
3096A	Ancon				2												

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
																				1
0	1	1	2	2	2	1	1	0	0	0	0	0	-	-	-	1	1	1		1
0	1	1	2	2/3	1	1	1	0	0	2	2	0	-	-	-	2	2	2		1
0	2	2	1	2	1	1	1	0	0	3	3	0	-	-	-	2	2	1		1
0	1	1	<1	1	<1	<1	1	0	0	1	2	0	-	-	-	2	2	1		1
0		2		0		0		0		0	2	0	-	-	-	2	2	1		1
0	0	1	0	0	0	0	0	0	0	2	1	0	-	-	-	3	3	3		1
0	1	1	0	0	0	0	0	0	0	0	0	0	-	-	-	3	3	3		1
																				1
0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	2	1		1
0		0		0		0		0			0	0	-	-	-	2	2	1		1
0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	3	3	3	2	1
0	0	1	2	2	1	1	1	0	0	1	2	0	-	-	-	2	2	1	2	1
0	0	1	2	2	1	1	1	0	0	2	2	0	-	-	-	2	2	1		1
0	1	1	0	0	0	0	0	0	0	1	1	0	-	-	-	2	2	1		1
												-	-	-	-	2	1	1		1
0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-					1
0	1	1	4/<1	<1	1/<1	<1	1/<1	<1	0	1	1	0	-	-	-	2	2	1		1
0	1	1	2	2	<1	1	<1	1	0	1	2	0	-	-	-	2	2	1		1
0	2	2	0	<1	0	<1	0	<1	0	2	2	0	-	-	-	2	2	1		1
																				1
0	0	0										0	-	-	-	2	2	1		1
0	1	1	2	2	<1	<1	<1	<1	0	2	2	0	-	-	-	2	2	2		1
																				1
0	1	1	<2	<1	<1	<1	<1	<1	0	2	1	0	-	-	-	2	2	1		1
0	1	1	2	2	2	1	1	0	0	0	0	0	-	-	-	2	2	1		1
0	1	1	2	2	<1	<1	<1	<1	0	1	1	0	-	-	-	2	2	1		1
																				1
0	1	1	2	2	1	1	1	0	0	2	2	0	-	-	-	2	2	1		1
																				1
0	0	0	2/1	2/1	1/3	1/3	1/1	1/1	0	1	1	0	-	-	-	2	2	1		1
0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	2	1		1
0	1	1	<1	2	<1	1	<1	1	0	2	2	0	-	-	-	2	2	1	1	1
0	1	1	2	1	1	1	1	0	0	2	2	0	-	-	-	2	2	1	2	1
0	1	1	1	1	1	1	1	0	0	2	2	0	-	-	-	2	2	1	1	1
																				1

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
3099	Ancon	2	30.5+/-9.6 years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3100	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3102	Ancon	2	41.1+/- 10 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3103	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3107	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE		0			0		
3105	Ancon	2	32.0+/-8.3years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3109	Ancon				2												
3110	Ancon	1	36.2+/-6.2years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3111	Ancon	3	8+/-24 months	2	1	2	3	AP	NONE	NONE	2	3/4	1	1	1	3	0
3112	Ancon	3	<35	4	1	3	4	INF	NONE	NONE							
3113	Ancon	1	<35	4	1	4	5	NONE	NONE	NONE	2		<1		<1		0
3114	Ancon	3	<35	4	1	2	3	AP	NONE	NONE	0	3	1	1	0	0	1
3116	Ancon	3	15+/-36 months	3	1	1	2	CO?	INF	NONE	4	4	4	2	3	4	0
3117	Ancon	2	<35	4	1	1	2	CO?	NONE	NONE	<2	4	<1	4	<1	1	0
3118	Ancon	1	<35	4	1	2	3	AP	INF	NONE	2	2/1	1	1/1	1	1/2	0
3119	Ancon	3	6+/-24 months	2	1	2	3	AP	NONE	NONE	2	2	1	1	1	1	0
3120	Ancon	3	11+/-30 months	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3121	Ancon	2	36.2+/-6.2 years	5	1	1	2	CO?	INF	NONE	4	4	4	3	2	0	0
3122	Ancon	3	<35	4	1	3	4	INF	CV	NONE	0	2	0	1	0	1	0
3123	Ancon	2	<35	4	1	3	4	APIF	NONE	NONE	1	1	1	1	2	2	0
3125	Ancon	2	unknown	6	1	4	5	NONE	NONE	NONE		0		0		0	
3126	Ancon				2												
3127	Ancon				2												
3128	Ancon	3	<35	4	1	2	3	AP	NONE	NONE	0	2	0	1	0	1	0
3129	Ancon	2	30.5+/-9.6 years	4	1	2	3	AP	NONE	NONE	2	0	1	0	1	0	0
3132	Ancon	1	36.2+/-6.2 years	5	1	4	5	NONE	NONE	NONE	0	<1	0	<1	0	<1	0
3145	Ancon	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3146	Ancon	3	3+/-12 months	1	1	2	3	PP	AP	NONE	2	2	1	1	1	1	0
3226	Cuzco	2	<35	4	1	3	4	INF	NONE	NONE	0	0	0	0	0	0	0
3227	Cuzco	2	<35	4	1	1	2	CO?	CV	NONE	4	3	4	1	1	1	0
3228	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3229	Cuzco	3	36.2+/-6.2 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3230	Cuzco	2	32+/-8.3 years	4	1	3	4	INF	AP	NONE	0	1	0	1	0	1	0
3232	Cuzco				2												
3233	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3234	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0		0	0		0	0

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	1
0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	-	2	1	2	1
0	1	<1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	-	2	2	2	1
0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	1
0	1		<1		<1		<1	0	0	0	1	0	-	-	-	-	2	1	2	1
0	0	2	<1	<1	<1	<1	<1	0	0	2	2	0	-	-	-	-	2	1	2	1
0	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	1
0	1		0	0	<1	0	0	0	0	0	1	0	-	-	-	-	2	1	2	1
		0	0	0	0	0	0	0	0	2	2	0	-	-	-	-	2	1	2	1
1	0	0	0	0	0	0	0	0	0	1	2	0	-	-	-	-	2	1	2	1
0	1	0	0	<1	<1	<1	<1	0	0	1	1	0	-	-	-	-	2	1	2	1
0	1	1	<2		<1		<1	0	0		2	0	-	-	-	-	2	2	2	1
0	1	2	<1	<1	<1	<1	<1	0	0	1	2	0	-	-	-	-	2	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	-	2	1	2	1
0	0	0										0	-	-	-	-	1	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	2	2	0	-	-	-	-	1	1	2	1
0	1	1	2	<1	1	<1	<1	0	0	1	2	0	-	-	-	-	2	1	2	1
0	1	0	0	<1	0	0	0	0	0	1	0	0	-	-	-	-	1	1	2	1
0		0	0		0	0	0	0	0	0	0	0	-	-	-	-	2	2	2	1
																				1
0	1	0	<1	0	<1	0	<1	0	0	1	2	0	-	-	-	-	3	3	2	1
0	1	<1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	-	1	1	2	1
0	1	<1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	-	2	1	2	1
0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	1
0	1	1/1	1/1	3/1	3/1	1/1	1/1	0	0	1	1	0	-	-	-	-	2	2	2	1
0	1	<1	<1	<1	<1	<1	<1	0	0	2	2	0	-	-	-	-	2	2	2	2
0	0	2	2	1	1	1	1	0	0	1	2	0	-	-	-	-	2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	2
0	1	<1	<1	<1	<1	<1	<1	0	0	1	2	0	-	-	-	-	2	1	2	2
																				2
0	0	1	0	0	0	0	0	0	0	1	1	0	-	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	1	0	0	-	-	-	-	2	1	2	2

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADjp
3235	Cuzco	2	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	1	1	1	1	1	0	0
3236	Cuzco	1	32+/-8.3 years	4	1	3	4	APIF	CV	NONE	1	1	1	1	1	0	0
3238	Cuzco	2	<35	4	1	2	3	AP	NONE	NONE	2	1	1	1	1	0	0
3239	Cuzco	3	7+/-24 monts	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3240	Cuzco	1	41.1+/-10 years	5	1	3	4	INF	PP	NONE	1	1	1	1	2	2	0
3241	Cuzco	3	10+/-30 months	2	1	2	3	AP	NONE	NONE	1	2	1	1	1	3	0
3242	Cuzco	2	41.1+/-10 years	4	1	4	5	NONE	NONE	NONE	0	1	0	1	0	1	0
3243	Cuzco	3	9+/-24 months	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3244	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3245	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3247	Cuzco	3	10+/-30 months	2	1	3	4	INF	AP	NONE	<1	1	1	<1	1	0	
3248	Cuzco	1	indeterminant	6	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3249	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3250	Cuzco	3	41.1+/-10 years	5	1	3	4	APIF	PP	NONE	1	0	1	0	2	0	
3251	Cuzco	3	41.1+/-10 years	5	1	3	4	APIF	CV	NONE	0	1	0	1	0	1	
3252	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3253	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3255	Cuzco	2	32+/-8.3 years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3256	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3258	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3259	Cuzco	3	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3260	Cuzco	2	41.1+/-10 years	5	1	2	3	PP	NONE	NONE	0	0	0	0	0	0	
3261	Cuzco	2	36.2+/-6.2 years	4	1	2	3	AP	CV	NONE	2	1	1	1	1	0	
3262	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3263	Cuzco	3	43.4+/-10.7 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3264	Cuzco	3	32+/-8.3 years	4	1	2	3	AP	INF	NONE	2		1	2		0	
3266	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3267	Cuzco	1	32+/-8.3 years	4	1	3	4	INF	APIF	NONE	0	1	0	1	0	0	
3268	Cuzco	3	41.1+/-10 years	5	1	3	4	INF	AP	NONE	1	0	1	0	1	0	
3269	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3270	Cuzco	3	36.2+/-6.2 years	5	1	4	5	NONE	NONE	NONE	<1	0	<1	0	<1	0	
3271	Cuzco	3	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
3272	Cuzco	3	<35	4	1	3	4	APIF	NONE	NONE	2	2	1	1	1	0	
3273	Cuzco	3	41.1+/-10 years	5	1	2	3	PP	AP	NONE	2	1	1	1	1	0	
3274	Cuzco	2	<35	4	1	3	4	APIF	CV	NONE	4	2	1	1	1	0	
3275	Cuzco	1	43.4+/-10.7 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
	0		<1		<1		<1		0		0		0	-	-	-	2	1	2	2
0	0	0	1	1	1	1	1	2	0	0	1	1	0	-	-	-	2	1	2	2
0		0		0		0		0		0		0	0	-	-	-	2	2	2	2
0	1	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	2	2	2
0	0	0	2		1		2		0		2	1	0	-	-	-	2	1	2	2
0	0	0	0	<1	0	<1	0	<1	0	0	0	2	0	-	-	-	2	2	2	2
0	0	0	0	1	0	1	0	1	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	1	0	-	-	-	2	1	2	2
0	0	1	0	0	0	0	0	0	0	0	0	1	0	-	-	-	2	2	2	2
0	1	1	1	1	1	1	1	1	0	0	2	2	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	1	0	-	-	-	3	3	2	2
0	0	0	0	0	0	0	0	0	0	0	0	1	0	-	-	-	2	1	2	2
0	1	1	1	0	1	0	1	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	0	1	0	1	0	1	0	0	0	1	0	-	-	-	2	1	2	2
0	1	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-	3	3	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	1	2	2
0	1	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	2	2	2
0	0	1	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	1	1	1	1	1	1	0	0	1	1	0	-	-	-	2	1	2	2
0	0	0	0	<1	<1	<1	<1	<1	0	0	2	2	0	-	-	-	2	1	2	2
0	0	0	0	1	0	1	0	1	0	0	2	1	0	-	-	-	2	2	2	2
0	0	2	2	0	1	0	1	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	2	2	2
0	1	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	2	2	2
	1		1	1	1	1	1	1	0	0	2	2	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	2	2	2
0	1	1	0	<1	0	<1	0	<1	0	0	2	2	0	-	-	-	2	2	2	2
0	1	0	2	1	1	1	1	1	0	0	2	2	0	-	-	-	2	1	2	2
0	1	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	2	2	2
0	0	1	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	0	<1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	2	1	2	2
0	0	0	2/3	2	1	1	1	1	0	0	1	1	0	-	-	-	2	1	2	2
0	0	0	2	2	1	1	1	1	0	0	1	1	0	-	-	-	2	1	2	2
0	0	0	0	<1	0	<1	0	<1	0	0	0	0	0	-	-	-	2	1	2	2

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
3276	Cuzco	3	6+/-24 months	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3277	Cuzco	2	<35	4	1	3	4	INF	AP	NONE	1	1	1	1	1	1	0
3278	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3279	Cuzco	3	41.1+/-10 years	5	1	3	4	APIF	NONE	NONE	0	2	0	1	0	1	0
3280	Cuzco	2	41.1+/-10 year	5	1	2	3	CV	APIF	NONE	1	1	1	1	1	1	0
3281	Cuzco	3	41.1+/-10years	4	1	3	4	IFANT	NONE	NONE	1	2/3	1	1	1	1	0
3282	Cuzco	3	9+/-24 months	2	1	3	4	INF	AP	NONE	1	1	1	1	1	1	0
3283	Cuzco	2	<35	4	1	2	3	CV	AP	NONE	0	1	0	1	0	1	0
3284	Cuzco	2	45.5+/-8.9 years	5	1	2	3	AP	PP	NONE	2	2	1	1	1	1	0
3285	Cuzco	2	<35	4	1	2	3	AP	INF	NONE	1	1	1	1	1	1	0
3286	Cuzco	3	41.1+/-10 years	5	1	3	4	INF	NONE	NONE	0	0	0	0	0	0	0
3287	Cuzco	3	12+/-36 months	2	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3288	Cuzco	2	41.1+/-10 years	5	1	2	3	AP	CV	NONE	2	2	1	1	1	1	0
3289	Cuzco	2	<35	4	1	1	2	CO?	CV	NONE	4	4	4	4	3	3	0
3290	Cuzco	3	32.0+/-8	4	1	2	3	AP	NONE	NONE	1	1	1	1	1	1	0
3291	Cuzco	1	<35	4	1	1	2	CO?	INF	NONE	4	4	4	4	3	3	0
3292	Cuzco	1	<35	4	1	2	3	AP	NONE	NONE	0	2	0	1	0	3	0
3293	Cuzco	2	indetermin ate	6	1	4	5	NONE	NONE	NONE	0	<1	0	<1	0	<1	0
3294	Cuzco	3	indeterminate	6	1	1	2	CO?	NONE	NONE	4		4		3		0
3295	Cuzco	3	indeterminate	6	1	4	5	NONE	NONE	NONE	0		0		0		0
3296	Cuzco	3	10+/-30months	2	1	1	1	CO ACT	NONE	NONE	3	4	2	4	3	3	1
3297	Cuzco	1	indeterminate	6	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3298	Cuzco				2												
3299	Cuzco	3	32+/-8.3years	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3300	Cuzco	3	11+/-30months	2	1	4	5	NONE	NONE	NONE	<1	<1	<1	<1	<1	<1	0
3301	Cuzco	1	32+/-8.3 years	4	1	3	4	APIF	NONE	NONE	2	1	1	1	1	1	0
3302	Cuzco				2												
3303	Cuzco	2	indeterminate	6	1	3	4	APIF	NONE	NONE	0	1	0	1	0	1	0
3304	Cuzco	3	<35	4	1	1	2	CO?	INF	NONE	4	4	4	4	3	3	0
3305	Cuzco	2	<35	4	1	4	5	NONE	NONE	NONE	2	<1	<1	<1	<1	<1	0
3306	Cuzco	3	indeterminate	6	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
3307	Cuzco	3	<35	4	1	3	4	INF	AP	NONE	1	1	1	1	2	2	0
3309	Cuzco	1	32+/-8.3years	4	1	2	3	AP	NONE	NONE	2	2	1	1	1	1	0
3310	Cuzco				2												
3311	Cuzco	3	32+/-8.3 years	4	1	2	3	AP	NONE	NONE	0	2	0	1	0	1	0
3312	Cuzco	1	45.5+/-8	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0	1	1	0	1	0	1	0	1	0	0	1	1	0	-	-	-	2	2	2	2
0	1	0	<1	1	<1	1	<1	1	0	0	1	1	0	-	-	-	2	1	2	2
0	0	0		0		0		0		0		1	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	1	0	-	-	-	2	1	2	2
0	0	0	0	2	0	1	0	1	0	0	1	1	0	-	-	-	2	1	2	2
0	1	2	0	<1	0	<1	0	<1	0	0	0	1	0	-	-	-	2	2	2	2
0	0	1	0	1	0	1	0	1	0	0	1	2	0	-	-	-	2	1	2	2
0	0	0	2	2	1	1	1	1	0	0	0	0	0	-	-	-	2	1	2	2
0	0	1	2	2	<1	<1	<1	<1	0	0	1	0	0	-	-	-	2	1	2	2
0	1	1	1	1	1	1	1	1	0	0	1	1	0	-	-	-	2	1	2	2
0	0	1	0	2	0	1	0	1	0	0	1	2	0	-	-	-	2	1	2	2
0	1	1	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	1	2	2
0	1	1	1	0	1	0	1	0	0	0	0	0	0	-	-	-	2	1	2	2
0	0	1	2	2	1	1	1	1	0	0	0	1	0	-	-	-	2	1	2	2
0	0	0	0		0		0		0		0		0	-	-	-	2	1	2	2
0	1	1	1	2	1	1	1	1	0	0	1	2	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	1	0	-	-	-	2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	3	3	2	2
	0		0		0		0		0		0		0	-	-	-	3	3	2	2
	0												0	-	-	-	3	3	2	2
2	1	1	4	4	4	4	3	3	2	2	1	1	0	-	-	-	2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0	0	-	-	-	3	3	2	2
																				2
0	0	0	0	0	0	0	0	0	0	0	1	1	0	-	-	-	2	1	2	2
0	1	1	<1	<1	<1	<1	<1	<1	0	0	1	2	0	-	-	-	2	1	2	2
0	0	1	0	2	0	1	0	1	0	0	1	1	0	-	-	-	2	1	2	2
																				2
0	1	2		0		0		0		0		1	0	-	-	-	2	2	2	2
0	1	1	1	2	1	1	1	1	0	0	2	2	0	-	-	-	2	1	2	2
0	1	0	0	0	0	0	0	0	0	0	1	0	0	-	-	-	2	1	2	2
0	1	1											0	-	-	-	2	1	2	2
0	1	1	2	2	1	1	1	1	0	0	2	2	0	-	-	-	2	1	2	2
0	1	1	<1	<1	<1	<1	<1	<1	0	0	1	1	0	-	-	-	2	1	2	2
																				2
0	0	1	0	0	0	0	0	0	0	0	0	1	0	-	-	-	2	1	2	2
0	0	1	0	3	0	<1	0	<1	0	0	1	1	0	-	-	-	2	1	2	2

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
3313	Cuzco	2	<35	4	1	1	2	CO?	INF	CV	4	4	4	3	3	0	
3314	Cuzco	3	<35	4	1	1	2	CO?	INF	CV	4	4	4	3	3	0	
3315	Cuzco	3	10+/-30 months	2	1	1	1	CO ACT	INF	CV	4	4	4	3	3	1	
3316	Cuzco	1	32+/-8.3 years	4	1	1	2	CO?	PP	NONE	4	4	4	3	2	0	
3317	Cuzco	2	<35	4	1	2	3	AP	CV	NONE	0	2|0	1	0	1	0	
3318	Cuzco	1	<35	4	1	2	3	AP	CV	NONE	3	2/3	1	0	2	0	
3319	Cuzco	2	<35	4	1	3	4	IFANT	NONE	NONE	1	1|1	1	1	1	0	
3320	Cuzco	2	32+/-8.3 years	4	1	3	4	APIF	NONE	NONE	0	0|0	0	0	0	0	
3321	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0|0	0	0	0	0	
3322	Cuzco	1	<35	4	1	2	3	AP	PP	NONE	0	2|0	1	0	2	0	
3323	Cuzco	1	32+/-8.3 years	4	1	2	3	PP	NONE	NONE	0	0|0	0	0	0	0	
3324	Cuzco	1	32+/-8.3 years	4	1	4	5	NONE	NONE	NONE	0	0|0	0	0	0	0	
3325	Cuzco	3	5+/-16 months	1	1	3	4	IFANT	AP	NONE	1	1|1	1	1	1	0	
3328	Cuzco	3	10+/-30 months	2	1	2	3	AP	NONE	NONE	2	2|1	<1	1	<1	0	
3329	Cuzco	2	<35	4	1	3	4	INF	NONE	NONE	0	0|0	0	0	0	0	
3330	Cuzco	3	15+/-36 months	3	1	1	2	CO?	NONE	NONE	4	4|4	4	3	3	0	
3331	Cuzco	1	32+/-8.3 years	4	1	2	3	AP	NONE	NONE	3	3|1	1	1	1	0	
3332	Cuzco	3	indeterminate	6	1	3	4	APIF	NONE	NONE	1	1|1	1	2	2	0	
3333	Cuzco	3	indeterminate	6	1	2	3	AP	NONE	NONE	1	1|1	1	1	1	0	
3334	Cuzco				2												
3335	Cuzco	3	indeterminate	6	1	4	5	NONE	NONE	NONE	0	0|0	0	0	0	0	
3336	Cuzco				2												
3337	Cuzco	3	indeterminate	6	1	2	3	AP	NONE	NONE	3	3|1	1	1	1	0	
3338	Cuzco	2	indeterminate	6	1	2	3	AP	NONE	NONE	N/A	0|N/A	0	N/A	0	N/A	
3339	Cuzco	3	indeterminate	6	1	1	2	CO?	APIF	NONE	3	1|1	1	2	1	0	
3350	Cuzco	3	10+/-30 months	2	1	2	3	AP	INF	NONE	0	1/3	0	1	0	2/1	0
3351	Cuzco	3	6+/-24 months	2	1	2	3	AP	NONE	NONE	2	2	1	1	1	1	0
3352	Cuzco	2	indeterminate	6	1	2	3	PP	AP	NONE	1	1	1	1	1	1	0
3353	Cuzco	2	36.2+/-6.2 years	5	1	2	3	AP	NONE	NONE	1	1|1	1	1	1	1	0
3354	Cuzco	3	<35	4	1	4	5	NONE	NONE	NONE	0	0|0	0	0	0	0	0
3355	Cuzco				2												
3356	Cuzco	3	indeterminate	6	1	4	5	NONE	NONE	NONE	0	0|0	0	0	0	0	0
3357	Cuzco				2												
3358	Cuzco				2												
3359	Cuzco	1	<35	4	1	4	5	NONE	NONE	NONE	0	0|0	0	0	0	0	0
3360	Cuzco	3	32+/-8.3 years	4	1	1	2	CO?	NONE	NONE	4	4|4	4	3	3	0	

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0	0	0	2	0	1	0	1	0	0	1	2	0	-	-	-		2	1	2	2
0	1	0	1	1	1	1	1	0	0	2	2	0	-	-	-		2	1	2	2
0	0	0	2	1	1	1	1	0	0	1	1	0	-	-	-		1	2	2	2
0	1	0	2	1	1	1	1	0	0	2	1	0	-	-	-		2	1	2	2
0	0	0	2	1	1	1	1	0	0	1	1	0					2	1	2	2
0	0	0	1	1	1	1	1	0	0	1	1	0					2	1	2	2
0	1	2	2	1	1	1	1	0	0	2	1	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	1	0	0					2	1	2	2
0	0	1	0	0	<1	0	<1	0	0	1	0	0					2	1	2	2
0	0	2	2/3	1	1	1	1	0	0	1	1	0					2	1	2	2
0	1	1	1	1	1	1	1	0	0	2	1	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	1	1	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	1	1	0					3	1	2	2
0	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				2	1	2	2
																				2
0	1	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				2	1	2	2
0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				2	1	2	2
0	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				3	3	3	2
0	1	1	<1	<1	<1	<1	<1	0	0	1	2	0					2	2	2	2
0	2	2	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A					2	2	2	2
0	0	0	2/3	1	1	1	1	0	0	1	0	0					3	3	2	2
0	0	0	<1	<1	<1	<1	<1	0	0	0	1	0					2	2	2	2
0	0	1	0	0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	1	0	0 0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	1	1	0					3	3	3	2
0	0	0	0	0	0	0	0	0	0	1	1	0					3	3	3	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	2	2	2

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
3361	Cuzco	3	36.2+/-6.2 years	5	1	2	3 AP	CV	INF	0	2	3	1	1	3	3	0
3362	Cuzco	3	indeterminate	6	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	0
3363	Cuzco	3	6+/-24 months	2	1	2	3 AP	PP	NONE	2	2	1	1	2	2	0	
3364	Cuzco	3	32+/-8.3 years	4	1	2	3 PP	INF	NONE	0	<1	0	<1	0	<1	0	
3365	Cuzco	1	<35	4	1	3	4 APIF	PP	NONE	2	0	1	0	1	0	0	
3366	Cuzco				2												
3368	Cuzco	3	<35	4	1	2	3 AP	NONE	NONE	N/A	1	N/A	1	N/A	1	N/A	
3369	Cuzco				2												
3370	Cuzco				2												
3371	Cuzco	1	36.2+/-6.2 years	5	1	2	3 PP	NONE	NONE		0		0		0		
3372	Cuzco				2												
3373	Cuzco	3	<35	4	1	1	2 CO?	CV	NONE	4	4	4	4	3	3	0	
3374	Cuzco	2	<35	4	1	2	3 AP	NONE	NONE	0	3/1	0	1	0	1	0	
3375	Cuzco	3	<35	4	1	4	5 NONE	NONE	NONE		0		0		0		
3378/337	Cuzco	3	36.2+/-6.2 years	5	1	3	4 APIF	CV	NONE		2	1	1	1	1	0	
1842	Marca Huamchuco	1	41.1+/-10 years	5	1	2	3 AP	NONE	NONE	1	2	1	0	<1	<1	0	
1843	Marca Huamchuco				2					-	-	-	-	-	-	-	
1844	Marca Huamchuco	1	32.0+/-8.3 years	4	1	3	4 INF	NONE	NONE	0	3	0	1	0	<1	0	
1845	Marca Huamchuco				2					-	-	-	-	-	-	-	
1846	Marca Huamchuco	Indet	6+/-24 months	2	1	4	5 NONE	NONE	NONE	1	1	<1	<1	1	1	0	
1847	Marca Huamchuco	Indet	Indet	6	1	4	5 NONE	NONE	NONE	1	1	<1	<1	<1	<1	0	
1848	Marca Huamchuco	Indet	check	6	1	4	5 NONE	NONE	NONE	<1	<1	<1	<1	<1	<1	0	
1852	Marca Huamchuco	2	<35	4	1	2	3 AP	INF	NONE	1	1	2	2	3	3	0	
1858	Marca Huamchuco	3	43.4+/-10.7 years	5	1	2	3 AP	NONE	NONE	2	0	1	0	<1	0	0	
1859	Marca Huamchuco	2	41.1+/-10 years	5	1	3	4 INF	NONE	NONE	0	0	0	0	0	0	0	
1860	Marca Huamchuco	3	36.2+/-6.2 years	5	1	2	3 AP	NONE	NONE	1	0	<1	0	2	0	0	
1861	Marca Huamchuco	2	43.4+/-10.7 years	5	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	
1862	Marca Huamchuco	1	43.4+/-10.7 years	5	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	
1863	Marca Huamchuco	3	36.2+/-6.2 years	5	1	4	5 NONE	NONE	NONE	0	1	0	1	0	<1	1	
1864	Marca Huamchuco	3	36.2+/-6.2 years	5	1	3	4 INF	NONE	NONE	0	0	0	0	0	0	0	
1865	Marca Huamchuco	1	36.2+/-6.2 years	5	1	2	3 CV	APIF	NONE	0	0	0	0	0	0	0	
1881	Marca Huamchuco	1	41.1+/-10 years	5	1	3	4 INF	NONE	NONE	1	1	1	1	<1	<1	0	
1866	Marca Huamchuco	1	41.1+/-10 years	5	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	
1867	Marca Huamchuco	2	<35	4	1	1	2 CO?	INF	NONE	4	4	3	2	3	2	0	
1868	Marca Huamchuco	2	32+/-8.3 years	4	1	4	5 NONE	NONE	NONE	<1	<1	<1	<1	<1	<1	0	
1869	Marca Huamchuco	3	41.1+/-10 years	5	1	4	5 NONE	NONE	NONE	<1	0	<1	0	<1	0	0	

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0	1	0	2/1	1	1 1	1 1	1 1	0	0	2	2	0					2	1	2	2
0	0	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A					2	2	2	2
0	0	0	1 1	1 1	1 1	1 1	1 1	0	0	0	0	0					2	2	2	2
0	1	1	2	1	1	1	1	0	0	1	2	0					2	1	2	2
0	1	2	2	1	1	1	1	0	0	1	1	0					2	1	2	2
																				2
0	N/A	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A					3	3	3	2
																				2
0		0	2			1		1	0		0		2				2	1	2	2
																				2
0	1	2	1/4	1	1	1	1	0	0	1	1	0					1	1	2	2
0	0	0	1	0	1	0	1	0	0	1	2	0					1	1	2	2
0		0	0		0		0		0	0	0	0					2	1	2	2
0	0	0	2	1	1	1	1	0	0	0	0	0					2	1	2	2
0	0	0	1	0	1	<1	<1	0	0	1*	1*	0	0				2	2	2	2
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-					2
0	2	2	0	0	0	0	0	0	0	2	2	3	ant	<1mm			2	2	2	2
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-					2
0	0	2	0	0	0	0	0	0	0	0	2	0					2	2	2	2
0	0	0	-	-	-	-	-	-	-	-	-	0					2	2	2	2
0	1	1	0	-	0	-	-	0	-	0	-	0					2	2	2	2
0	0	0	1	1	<1	<1	<1	0	0	1	1	0					2	2	2	2
0	?	?	0	0	0	0	0	0	0	0	0	1		2-3	2		2	1	2	2
0	0	0	0	0	0	0	0	0	0	2	2	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	0	1	1	1	1	0	0	0	1	0	0					2	2	2	2
1	0	0	0	1	0	1	1	0	0	0	0	0					2	1	2	2
0	0	0	2	0	1	0	0	0	0	0	0	0					2	2	2	2
?	0	0	2	2	1	1	1	0	0	0	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	1	1	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	1	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	1	0	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	2	0					2	2	2	2
0	0	0	0	0	0	0	0	0	0	0	0	0					2	1	2	2

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
1870	Marca Huamchuco	1	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	<1		<1		1		
1871	Marca Huamchuco	2	32+/-8.3 years	4	1	3	4	APIF	NONE	NONE	0	0	0	0	0	?	
1872	Marca Huamchuco	2	15+/-36 months	3	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
1873	Marca Huamchuco	3	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
2887	Tate-Chulpaca	2	<35	4	1	2	3	AP	INF	NONE	2	2	2	3	3	0	
12-2888	Tate-Chulpaca				2												
2890	Tate-Chulpaca	2	>35	4	1	2	3	AP	CV	NONE	1	1 1	1	1	1	0	
2891	Tate-Chulpaca	1	>35	4	1	4	5	NONE	NONE	NONE	0 0	0	0	0	0	0	
2892	Tate-Chulpaca	2	>35	4	1	2	3	AP	INF	CV	1	3	1	1	1	0	
2893	Tate-Chulpaca	2	<35	4	1	4	5	None	NONE	NONE	0	0 0	0	0	0	0	
2894	Tate-Chulpaca	1	<35	4	1	3	4	INF	NONE	NONE	0	0 0	0	0	0	0	
2895	Tate-Chulpaca	1	<35	4	1	1	2	CO?	INF	NONE	0	4	0	0	3	0	
2897	Tate-Chulpaca	2	<35	4	1	3	4	INF	CV	AP	2	1 1	1	1	1	0	
2898	Tate-Chulpaca	2	41.1+/-10 years	5	1	2	3	AP	CV	NONE	2	2/3	1	1	1	0	
2899	Tate-Chulpaca	1	32.0+/-8.3 years	4	1	2	3	AP	INF	NONE	1	3/1	1	1	1	0	
2900	Tate-Chulpaca	2	<35	4	1	2	3	AP	NONE	NONE	1	1 1	1	1	1	0	
2901	Tate-Chulpaca	3	10+/-30 months	2	1	3	4	IFANT	NONE	NONE	1	2	1	1	1	0	
2902	Tate-Chulpaca		indeterminate	6	1	2	3	AP	NONE	NONE	0	0	0	0	0	0	
2902 A	Tate-Chulpaca	3	indeterminate	6	1	2	3	AP	NONE	NONE	3/4	N/A	1	N/A	1	N/A	0
2902 B	Tate-Chulpaca	3	indeterminate	6	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	
2902C	Tate-Chulpaca	3	indeterminate	6	2												
2902D	Tate-Chulpaca	3	indeterminate	6	2												
2902E	Tate-Chulpaca	3	indeterminate	6	2												
2903	Tate-Chulpaca	2	indeterminate	6	1	2	AP		NONE	NONE	<1	2	<1	2	<1	3	0
2904	Tate-Chulpaca	2	<35	4	1	4	5	NONE	NONE	NONE	<1	2	<1	<1	<1	<1	0
2905	Tate-Chulpaca	2	32.0+/-8.3years	4	1	4	5	NONE	NONE	NONE	0		0		0		0
2906	Tate-Chulpaca	3	36.2+/-6.2 years	5	1	3	4	INF	AP	NONE		2	2 1	1	1	1	0
2907	Tate-Chulpaca	2	41.1+/-10 years	4	1	3	4	APIF	NONE	NONE		2 0	<1	0	<1	0	0
2908	Tate-Chulpaca	3	15+/-36 months	3	1	1	1	CO ACT	INF	NONE	4	4	4	4	3	3	0
2909	Tate-Chulpaca	3	indeterminate	6	1	4	5	NONE	NONE	NONE	0		0		0		0
2910	Tate-Chulpaca	2	45.5+/-8.9 years	5	1	3	4	APIF	NONE	NONE	2	3	1	1	1	1	0
2911	Tate-Chulpaca	3	<35	4	1	1	2	CO?	INF	NONE	4	3	4	1	1	1	0
2912	Tate-Chulpaca	2	<35	4	1	2	3	AP	INF	NONE	1	2/3	1	1	1	1	0
2913	Tate-Chulpaca	2	<35	4	1	1	2	CO?	NONE	NONE		2	4 1	4	1	3	0
2913A	Tate-Chulpaca	3			2												
2915	Tate-Chulpaca	1	32+/-8.3 years	4	1	2	3	AP	NONE	NONE	3	2 1	1	1	1	0	

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0		0	0		0	0		0	0		0	0					2	2	2	2
?	0	0	<1	1	<1	1	<1	1	0	0	0	0					2	1	2	2
0	0	0	0	0	0	0	0	0	0		2	2					2	2	2	2
0	2	0	0	0	0	0	0	0	0	0	0	0	1	<1mm			2	2	2	2
2	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				2	1	2	1
																				1
0	0	1	1	1	1	1	1	0	0	1	1	0					2	2	2	1
0	1	0	0	0	0	0	0	0	0	1	1	0					2	2	2	1
0	1	1	2	1	1	1	1	0	0	1	2	0					2	1	2	1
0	0	0	0	0	0	0	0	0	0	1	1	0					2	2	2	1
0	0	1	1	1	1	1	1	0	0	1	2	0					2	1	2	1
0	1	2	1	0	1	0	1	0	0	0	2	0					2	1	2	1
0	1	1	1	1	1	1	2	0	0	1	2	0					1	1	1	1
0	1	1	2	2	1	1	1	0	0	2	2	0					2	1	2	1
0	1	2	1	1	1	1	1	0	0	2	2	0					2	2	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	1	1	0					2	1	2	1
0	2	2	1	1	1	1	1	0	0	2	2	0					2	1	2	1
0	0	0	N/A	N/A	N/A	N/A	N/A	0	N/A	N/A	N/A	N/A	N/A				3	2	2	1
N/A	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				3	2	2	1
0	0	0	0	0	0	0	0	0	0	0	0	0	0				3	2	2	1
																				1
																				1
0	0	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				3	3	2	1
0	0	0	0	0	0	0	0	0	0	0	0	0	0				2	1	2	1
0	0	2	2	<1	1	1	2	0	0	2	2	0	0				3	3	2	1
0	0	0	2	0	0	0	0	0	0	0	2	0	0				2	1	2	1
0	0	0							0		0	0	0				2	2	2	1
1	2	2	2	1	1	1	1	0	0	2	2	0	0				2	1	2	1
	0	0	0	0	0	0	0	0	0	0	0	0	0				3	3	2	1
0	3	2	1	1	1	1	1	0	0	2	2	0	0				2	1	2	1
0	1	1	1	1	1	1	1	0	0	2	2	0	0				2	1	2	1
0	1	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				2	1	2	1
0	1	0	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A				2	1	2	1
																				1
0	0	1	2	1	1	1	1	0	0	2	2	0	0				2	1	2	1

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
2916	Tate-Chulpaca	3	10+/-30 months	2	1	1	2 CO?	NONE	NONE	2/3	2/3	1	1	1	1	1	0
2917	Tate-Chulpaca	3	7+/-24 months	2	1	2	3 AP	INF	NONE	2	1	1	1	1	1	1	0
2918	Tate-Chulpaca	3	<35	4	1	3	4 APIF	CV	NONE	2	2 2	1	1	1	1	1	0
2919	Tate-Chulpaca	2	41.1+/-10 years	5	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	0
2920	Tate-Chulpaca	1	<35	4	1	2	3 AP	INF	NONE	1	1/3	2	1/1	1	1/1	0	
2921	Tate-Chulpaca	1	41.1+/-10 years	5	1	3	4 INF	APIF	NONE	1	1	1	1	1	1	0	
2922	Tate-Chulpaca	1	41.1+/-10 years	5	1	3	4 APIF	INF	NONE	1	2/3	1	1	1	1	0	
2923	Tate-Chulpaca	3	36.2+/-6.2 years	5	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	
2924	Tate-Chulpaca	2	<35	4	1	3	4 INF	AP	NONE	0	1	0	1	0	1	0	
2925	Tate-Chulpaca	3	15+/-36 months	3	1	4	5 NONE	NONE	NONE	0	0	0	0	0	0	0	
2925A	Tate-Chulpaca				2												
2926	Tate-Chulpaca	3	32.0+/-8.3 years	4	1	2	3 AP	NONE	NONE		2		1		1		
2927	Tate-Chulpaca	2	<35	4	1	1	2 CO?	INF	NONE	4	4	4	4	2	3	0	
2929	Tate-Chulpaca	3	32+/-8.3 years	4	1	4	5 NONE	NONE	NONE	<1	<1	<1	<1	<1	<1	0	
2930	Tate-Chulpaca	3	<35	4	1	4	5 NONE	NONE	NONE	1	1	1	1	1	1	0	
2931	Tate-Chulpaca	2	<35	4	1	1	2 CO?	NONE	NONE	4	4	4	4	3	3	0	
2932	Tate-Chulpaca	3	32+/-8.3years	4	1	3	4 IFANT	CV	NONE	0	2 0	1	0	1	0		
2936	Tate-Chulpaca	3	7+/-24 months	2	1	2	3 AP	INF	NONE	2	3	1	1	3	0		
2937	Tate-Chulpaca	2	<35	4	1	2	3 AP	CV	INF	4	4	1	1	1	2	0	
2938	Tate-Chulpaca	3			2												
2939	Tate-Chulpaca	3	<35	4	1	2	3 AP	NONE	NONE	4	4	1	1	2	1	0	
2941	Tate-Chulpaca	3	15+/-36 months	3	1	2	3 AP	INF	NONE	0	1 0	1	1	0	1	0	
2942	Tate-Chulpaca	3	4+/-12 months	1	1	3	4 APIF	NONE	NONE	2	1	2	1	3	1	0	
2943	Tate-Chulpaca	2	<35	4	1	4	5 NONE	NONE	NONE	0	<1	0	<1 co	0	<1 co	0	
2944	Tate-Chulpaca	1	<35	4	1	2	3 AP	INF	NONE	2	4	1	1	1	3	0	
2947	Tate-Chulpaca	3	32+/-8.3years	4	1	3	4 INF	AP	NONE	0	2 0	1	1	0	1	0	
2948	Tate-Chulpaca	3	8+/-24 months	2	1	2	3 PP	INFANT	NONE	2	2 1	1	1	3	3	0	
2948A	Tate-Chulpaca				2												
2950	Tate-Chulpaca	3	5+/-16months	1	1	3	4 INF	NONE	NONE	0	0	0	0	0	0	0	
2952	Tate-Chulpaca	3	36.2+/-6.2 years	5	1	4	5 NONE	NONE	NONE	0	0 0	0	0	0	0	0	
2954	Tate-Chulpaca	1	<35	4	1	2	3 PP	AP	NONE	2	2	1	1	1	1	0	
2955	Tate-Chulpaca	3	10+/-30months	2	1	2	3 AP	PP	NONE	<1	2	<1	1	<1	1	0	
2956	Tate-Chulpaca	3	10+/-30months	2	1	3	4 APIF	NONE	NONE	1/2	2/3	1	1	1	1	0	
2957	Tate-Chulpaca	3	32+/-8.3 years	4	1	3	4 INF	NONE	NONE	1	1	1	1	1	1	0	
2958	Tate-Chulpaca	1	<35	4	1	1	2 CO?	INF	NONE	0	4	0	4	0	2	0	
2959	Tate-Chulpaca	3	41.1+/-10 years	5	1	4	5 NONE	NONE	NONE	1	<1	<1	<1	<1	<1	0	

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RFV	LPV	Plaq	PlaqL	PlSizeL	PlSizeR	PHYP	VaultPor	Trac	LOC
0 (start	1	0	0	0	0	0	0	0	0	2	2	0					2	1	2	1
0	2	1	0	1	0	1	0	0	0	2	1	0					2	1	2	1
0	2	1	1	1	2	1	1	0	0	2	2	0					2	1	2	1
0	0	0	0	0	0	0	0	0	0	0	1	0					2	1	2	1
1	0	2	2	1	1	1	1	0	0	2	2	0					2	1	2	1
1	1	N/A	2	N/A	1	N/A	1	N/A	0	N/A	2	0					2	1	2	1
0	1	N/A	1	N/A	1	N/A	1	N/A	0	N/A	2	0					2	1	2	1
0	0	0	2	0	1	0	1	0	0	0	0	2 0					2	2	2	1
0	1	1	0	10	1	0	1	0	0	2	2	0					1	1	2	1
0	1	1	0	<1	0	<1	0	<1	0	1	1	0					2	1	2	1
																				1
0		0		0		0		0			0	0					2	1	2	1
0	1	1	1	2	1	1	1	0	0	2	2	0					2	1	2	1
0	0	0	0	0	0	0	0	0	0	0	2	2 0					2	1	2	1
0	2	2	2	1	1	1	1	0	0	1	1	0					2	1	2	1
0	0	0	0	0	0	0	0	0	0	1	1	0					2	1	2	1
0	1	2	2	1	1	1	1	0	0	2	2	0					2	2	2	1
	2		2		1		1	10		2		0					2	2	2	1
	2	2	0	2	0	1	0	1	0	2	2	0					2	1	2	1
																				1
0	0	0	<1	<1	<1	<1	<1	<1	0	0	0	0					2	1	2	1
0	1	0	1	0	1	0	1	0	0	1	2	0					2	1	2	1
0	2	1	1	0	1	0	1	0	0	2	2	0					2	1	2	1
0	1	1	0	0	0	0	0	0	0	0	0	0					2	1	2	1
0	1	1	1	1	1	1	1	0	0	1	2	0					2	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	2	2	0					2	2	2	1
0	3	3	1	1	3	2	2	0	0	2	2	0					2	1	2	1
																				1
0	1	1	<2	0	<1	0	0	0	0	2	2	0					2	1	2	1
0	1	1	<2	<2	<1	<1	<1	0	0	2	2 0						2	1	2	1
0	0	0	2	3	1	1	1	0	0	0	0	0					2	1	2	1
0	1	1	1	1<1	1	1<1	1	0	0	1	1	0					2	1	2	1
0	2	1	0	0	0	0	0	0	0	1	1	0					2	1	2	1
0	2	1	1	1	1	1	1	0	0	2	2	0					2	1	2	1
0	0	0	1	0	1	0	1	0	0	0	2	0					2	1	2	1
0	0	0	1	0	1	0	1	0	0	0	0	0					2	1	2	1
0	1	0	0	0	0	0	0	0	0	0	0	0					2	1	2	1

CatNum	Site	Sex	Age	AgeGr	USE	Les	Ples	Primary	Second	Tertiary	RAPS	LAPS	RAD	LAD	RAP	LAP	RADip
2960	Tate-Chulpaca	1	41.1+/-10 years	5	1	1	2	CO?	APIF	NONE	4	4	4	3	3	0	
2961	Tate-Chulpaca				2												
2962	Tate-Chulpaca				2												
2963	Tate-Chulpaca				2						0		0		0		
2964	Tate-Chulpaca	2	<35	4	1	4	5	NONE	NONE	NONE	<1	0	<1	0	<1	0	0
2965	Tate-Chulpaca	2	<35	4	1	3	4	INF	NONE	NONE	0	0	0	0	0	0	0
2966	Tate-Chulpaca	3	36.2+/-6.2 years	5	1	2	3	AP	NONE	NONE	<1	2	<1	1	<1	1	0
2967	Tate-Chulpaca	2	<35	4	1	2	3	AP	NONE	NONE	2	2	1	1	1	1	0
2968	Tate-Chulpaca	2	<35	4	1	3	4	INF	AP	NONE	1	1	1	1	1	1	0
2969	Tate-Chulpaca	3	9+/-24 months	2	1	2	3	AP	INF	NONE	2	2/4	1	1/1	1	3/1	0
2970	Tate-Chulpaca	3	41.1+/-10 years	5	1	3	4	INF	AP	NONE	2	2	0	0	0	0	0
2971	Tate-Chulpaca	2	<35	4	1	4	5	NONE	NONE	NONE	0	0	0	0	0	0	0
2972	Tate-Chulpaca	3	41.1+/-10 years	5	1	2	3	AP	NONE	NONE	2	2	1	1	1	1	0
2973	Tate-Chulpaca	2	41.1+/-10 years	5	1	4	5	NONE	NONE	NONE	1	2	1	1	1	1	0
2974	Tate-Chulpaca	2	<35	4	1	4	5	NONE	NONE	NONE	0		0			0	0
2975	Tate-Chulpaca	2	<35	4	1	3	4	INF	AP	NONE	1	2	1	1	1	1	0
2976	Tate-Chulpaca	1	<35	4	1	2	3	AP	NONE	NONE		2		1		1	
2977	Tate-Chulpaca				2												
2978	Tate-Chulpaca	2	32+/-8.3 years	4	1	4	5	NONE	NONE	NONE	<1	2	<1	<1	<1	<1	0
2979	Tate-Chulpaca	2	<35	4	1	4	5	NONE	NONE	NONE	0		0		0		0
2980	Tate-Chulpaca	1	36.2+/-6.2	5	1	4	5	NONE	NONE	NONE		3		1		1	
2981	Tate-Chulpaca	3	15+/-36 months	3	1	2	3	AP	NONE	NONE	1	1	1	1	1	1	0
2982	Tate-Chulpaca	1	36.2+/-6.2 years	5	1	3	4	INF	NONE	NONE	1	1	1	1	1	1	0

LADip	RAV	LAV	RPPS	LPPS	RPD	LPD	RPP	LPP	RPDip	LPDip	RPV	LPV	Plaq	PlaqL	PISizeL	PISizeR	PHYP	VaultPor	Trac	LOC
0		0	0	0	0	0	0	0	0	0	1	1	0				1	1	2	1
																			1	1
																			1	1
0		0																	1	1
0	1	1	0	0	0	0	0	0	0	1	1	0					2	1	2	1
0	1	2	<1	<1	<1	<1	<1	0	0	2	2	0					1	1	2	1
0	0	1	1	2 1	<1 or 1		<1	0	0	1	2	0					2	1	2	1
0	1	0	0	0	0	0	0	0	0	1	1	0					2	1	2	1
0	1	1	<1	<1	<1	<1	<1	0	0	2	2						2	1	2	1
0	1	2	<1	<1	<1	<1	<1	0	0	2	2	0					2	1	2	1
0	1	1	2	3	1	<1	<1	0	0	2	1	0					2	1	2	1
0	1	1	0	0	0	0	0	0	0	2 1	2	0					1	1	2	1
0	0	0	<1	<1	<1	<1	<1	0	0	2	2	0					2	1	2	1
0	1	1	0	0	0	0	0	0	0	1	2	0					2	1	2	1
	0		<1			<1		0		0		0							1	1
0	1	1	1	1	1	1	1	0	0	2	2	0					2	1	2	1
0	1	1	0	0	0	0	0	0	0	2	2	0					2	1	2	1
																			1	1
0	1	1	<1	<1	<1	<1	<1	0	0	1	1	0					2	1	2	1
0		2		<1		<1		0		1		0					2	1	2	1
		2	2	1	1	1	1	0	0	2	1	0					2	1	2	1
0	1	<1	N/A	N/A	N/A	N/A	N/A	0	N/A	1	N/A	0					2	1	2	1
0	1	1	1	<1	1	<1	1	0	0	2	1	0					2	1	2	1
0	0	0	1	1	1	1	1	0	0	2	2	0					2	1	2	1