CRIBRA ORBITALIA, NUTRITION AND PATHOGENIC STRESS IN PREHISTORIC SKELETAL REMAINS FROM THE PENDER ISLAND CANAL SITES (DeRt 1, DeRt 2), BRITISH COLUMBIA, CANADA

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Abstract

The presence of cribra orbitalia in prehistoric skeletal material is usually cited as an indicator of dietary stress. It is identified by porous openings of various size and frequency in the orbital roof of an affected skull. There is strong evidence to suggest that the orbital lesions are formed during a childhood episode of anemia. The dietary explanation, however, has never been completely accepted for the occurrence of cribra orbitalia on the Northwest Coast where the diet appears to have been iron-rich. Recent research suggests that iron deficiency may be more closely related to infection and pathogenic stress than to diet. Lowered iron levels are a natural defense the body employs against infection. If the body fails to maintain a balance between lowered iron levels as a defense and increased physiological needs, iron deficency anemia will ensue .

This research provides an analysis of cribra orbitalia in a sample of 83 prehistoric crania from the Pender Island Canal Sites (DeRt 1, DeRt 2) on North Pender Island, in the Gulf Islands of British Columbia. The sample is examined for the presence and severity of cribra orbitalia. Statistical analysis is used to identify patterns of occurrence by sex, age, and time period. These data provide a basis for examining both the traditional dietary model, and the alternative pathogenic stress model.

Prehistoric diet is estimated through archaeological, ethnographic, stable carbon isotopic, and botanical evidence. Nutritional adequacy is estimated through a literature-based nutritional analysis of traditional foods. Potential environmental pathogens are identified by using archaeological, ethnographic, and modern clinical evidence.

The results of this study indicate a high prevalence of cribra orbitalia in the Pender Canal skeletal sample. Dietary and pathogenic data suggest that

pathogenic stress best explains the high occurrence of cribra orbitalia at Pender Canal. It is proposed that conditions associated with aggregation and sedentism at the site are responsible for the high prevalence. Inadequate sanitation, crowded living conditions, and contaminated water sources probably provided ideal conditions for pathogen growth and transmission. Children would be particularly susceptible to iron deficiency anemia at weaning because of increased pathogen exposure, low iron stores, and high growth demands.

Dedication

For my father, who has always encouraged me to find my own path in life.

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Chapter I
Introduction

Introduction

Evidence of anemia in past human populations can be identified by the presence of a skeletal paleopathological entity known as cribra orbitalia. Cribra orbitalia is identified by porous openings of various size and frequency in the orbital roof of an affected skull. The cribrotic lesions are also frequently associated with increased thickness of the diploic, or middle, layer of bone. The lesions are believed to be the result of increased red blood cell production in the red marrow which is associated with anemia (Caffey 1937). Since the presence of cribra orbitalia provides evidence that an anemia occurred during the affected individual's lifetime, it is considered to be a good nutritional stress marker for assessing the health of past populations (for example, see Goodman *et al.* 1984a). Consequently, high frequencies of cribra orbitalia in North and South American archaeological skeletal populations have commonly been identified as evidence for a dietary deficiency of iron (for example, see El Najjar *et al.* 1976; Norr 1984; Palkovich 1984, 1987).

The nutritional deficiency explanation has not been completely accepted for all areas, however. For example, the occurrence of cribra orbitalia on the Northwest Coast where the diet appears to have been iron-rich has never been adequately explained (Cybulski 1977, 1990, 1994). Recent research suggests that iron deficiency may, in fact, be more closely related to infection and high pathogen exposure than to diet (Kent 1986; Kent and Dunn 1993; Kent and Lee 1993; Stuart-Macadam 1982, 1991, 1992a, 1992b; Walker 1986; WHO 1972).

Aim and Scope of the Thesis

Cribra orbitalia has been observed in the skeletal collection from the Pender Island Canal Sites (DeRt 1, DeRt 2), at a fairly high frequency. The aim of this research is to determine the likelihood that the anemia which caused cribra orbitalia in the Pender skeletal population was the result of a nutritional deficiency or environmental pathogens. The assumption that cribra orbitalia is the result of an anemia is not under investigation in this thesis, as it is well established and generally accepted amongst researchers (for a review see Stuart-Macadam 1991, 1992a).

Two models can be derived to explain the presence of cribra orbitalia in the Pender skeletal population: iron sequestering as a defence mechanism against a high pathogen load; or, inadequate dietary iron due to malabsorption or insufficient intake. The first model emphasizes environmental influences and will hereafter be referred to as the "environmental anemia" explanation, while the second focuses on diet and will be referred to as the "nutritional anemia" explanation. In order to test these two models, a multidimensional approach will be undertaken. All individuals in the Pender Island skeletal sample (hereafter, referred to as the Pender sample) will be examined for the presence of diagnostic lesions in order to determine the frequency and severity of cribra orbitalia. The Pender sample will be characterized by determining the distribution of cribra orbitalia by sex, age at death, and chronological time period. Any patterns or significant differences between these categories will be evaluated statistically. The sufficiency of the prehistoric diet in terms of dietary availability of iron will be examined through a literature-based nutritional analysis of traditional foods. Potential environmental pathogens to which this population may have been subjected will be discussed. Other possible factors

that may have affected maintenance of adequate iron levels will be identified and assessed.

This research contributes to an understanding of the etiology of cribra orbitalia in the Gulf of Georgia region of British Columbia through an assessment of native diet. Very little is known about the "condition" of life and the environmental and dietary stresses of prehistoric Northwest Coast peoples. The central focus of this thesis is to examine the frequency of cribra orbitalia in the Pender population and to determine if the diet was nutritionally adequate. There has been a general lack of information on the subject of nutritional adequacy in the literature for the Northwest Coast. Research in this area has involved reconstructing native diet (Chisholm 1986; Suttles 1968, 1974; Turner 1975, 1976), as well as providing a nutritional analysis of native foods (Kuhnlein 1982; Kuhnlein and Turner 1986, 1991; Lazenby and McCormack 1985; Norton *et al.* 1984; Nuxalk Food and Nutrition Program 1984; Rivera 1969). There have also been several unpublished theses on faunal remains from the Pender Canal Sites (for example, Hanson 1991; Maxwell 1989). There has been no comprehensive assessment of the nutritional adequacy of native diet based on Northwest Coast archaeological evidence. This research provides a better understanding of the state of health and nutrition of the prehistoric inhabitants of Pender Island specifically, but it is hoped that this study will be applicable to other coastal groups leading similar lifestyles.

Organization of Chapters

Chapter 2 provides background for an analysis of the bioavailability of iron in the Pender diet by examining factors that are known to inhibit or enhance iron absorption. Included in this chapter is a comprehensive review of the literature on iron metabolism, dietary requirements of iron, and anemias. A

description of cribrotic lesions, as well as the etiology of cribra orbitalia is discussed. Chapter 3 provides background information for the Pender Canal sites, including archaeological as well as ethnohistoric information. Chapter 4 provides an outline of the methods used to collect and analyse the skeletal and dietary data. The methods used in determining sex and estimating age of the skeletal sample are explained, as well as the criteria for scoring the severity of the orbital lesions. The compilation of archaeological and ethnohistoric evidence for native diet reconstruction is discussed. In Chapter 5, the results of the skeletal analysis are summarized; as well there is discussion of the applicability of the "nutritional" and "environmental" models to this population. Distribution of cribra orbitalia by sex, age, and time period is given. An analysis of the adequacy of the prehistoric Pender diet in terms of iron availability is made by reviewing the archaeological and ethnohistoric evidence. Potential pathogenic influences from the environment are identified. Chapter 6 summarizes and concludes the findings of the thesis research and offers suggestions for further research.

Chapter II

Literature Survey: Iron Deficiency, Diet, and the Skeleton

Background

Terminology and Description of Orbital Lesions

The term "cribra orbitalia" was first introduced by Welcker (1888 cited in Stuart-Macadam 1992b:152) to describe lesions on the superior surface of the eye orbits. Cribra orbitalia is characterized by an expansion of the spongy diploic bone and a corresponding thinning of the outer table that manifests as porous lesions on the surface of the bone (Figure 1). Lesions can vary from minor porous openings to a severe honey-combed appearance to the bone (Nathan and Haas 1966a, 1966b). Tissue hypoxia caused by anemia stimulates the bone marrow to produce more red blood cells to compensate for reduced circulating iron. Bony changes are thought to occur from the compensatory expansion of the red bone marrow exerting pressure on surrounding bone (Caffey 1937). Eventually, marrow hyperplasia increases the marrow space and erodes the compact bone leaving cribrotic lesions.

Cribra orbitalia almost always occurs bilaterally and occasionally co-occurs with similar lesions on the frontal, parietal or occipital bones, often referred to as porotic hyperostosis. When vault lesions occur, cribrotic lesions are almost always present (Stuart-Macadam 1982, 1987a, 1989a, 1989b). The term cribra orbitalia refers specifically to the orbital lesions, while porotic hyperostosis usually includes both the vault lesions and the orbital lesions. Cribra orbitalia is believed to be the initial osseous expression of porotic

Figure 1. Illustration of Cribra Orbitalia
from Pender Canal (Identification No. 168).

hyperostosis (Hengen 1971). Post-cranial manifestions of iron deficiency anemia may occur, although these changes are generally believed to be minimal (Caffey 1967; Moseley 1971; Shahidi and Diamond 1960). Post-cranial evidence for iron deficiency anemia has been reported to occur in long bones, ribs, phalanges, and metacarpals (Aksoy *et al.* 1966; Cybulski 1977; Lanzkowsky 1968). Red blood cell production in long bones and ribs occurs only in infants and young children, while the thin marrow spaces between the tables of the skull remain an important site of red blood cell production throughout life (Moseley 1971). The initial and primary involvement of the skull is probably due to the thinness of the compact bone surrounding the marrow spaces.

Etiology

Many explanations have been proposed for the cause of the vault and orbital lesions, but it is now widely accepted that porotic hyperostosis is the result of either an acquired or genetic anemia (Angel 1964, 1966, 1967; El Najjar *et al.* 1976; El Najjar and Robertson 1976; Hengen 1971; Lallo *et al.* 1977; Mensforth *et al.* 1978; Moore 1929; Stuart-Macadam 1982, 1985, 1987a, 1987b; Zaino 1964, 1967). The presence of the lesions with genetic anemias such as thalassemia, and sickle cell anemia has been known for some time, however, its occurrence in pre-Columbian America was unexplained because none of the genetic anemias are known to have occurred there prior to European contact. Malaria can cause anemia but is not believed to have occurred on the Northwest coast prior to contact. According to Boyd (1990), malaria was probably introduced by recently infected traders or trappers from other endemic areas of North America.

The idea that an acquired iron deficiency could be responsible for the lesions on the skeleton was suggested by Girdany and Gaffney (1952 cited in Stuart-Macadam 1982) and Shahidi and Diamond (1960). They found that radiographs of skull changes in infants with acquired iron deficiency were identical to those with genetic anemias. These clinical findings led to changes in the interpretation of lesions in skeletal remains. It was suggested that the cranial lesions found in archaeological samples could be the result of an acquired anemia, rather than a genetic one (Moseley 1961). Henschen (1961) suggested that more than one factor may be responsible: nutritional conditions as well as chronic infection might produce the orbital and vault lesions. The research of Nathan and Haas (1966b) indicated that nutritional factors best explained the wide temporal and spatial occurrence of cribra orbitalia in both human and nonhuman primates. Later researchers, including Hengen (1971), Carlson *et al.* (1974), El-Najjar *et al.* (1976), Lallo *et al.* (1977), Mensforth *et al.* (1978), Stuart-Macadam (1982, 1985, 1987a, 1987b) emphasized the idea that an acquired iron deficiency was responsible for the lesions in the majority of cases and this hypothesis has now gained wide popularity.

Physical anthropologists have speculated that a combination of nutritional deficiencies, inadequate absorption of iron, and infection contributed to anemic responses in past populations. All of these interpretations have generally included diet as a major factor in the development of the lesions (for example Henschen 1961; Nathan and Haas 1966b; El-Najjar *et al.* 1976). El-Najjar *et al.* (1976) found a differential prevalence of porotic hyperostosis among different ecological zones, and argued that it occurred more frequently in groups with a predominantly maize diet because phytates in the diet inhibited iron absorption. Hengen (1971) pioneered the idea that an interaction between diet, parasitic

infection, and infectious disease was responsible in the development of porotic hyperostosis. His approach was followed by Carlson *et al.* (1974) who speculated that weanling diarrhea, poor diet, and parasitic infection combined in the development of iron-deficiency anemia in Nubian populations. Lallo *et al.* (1977) hypothesized that microbial infection, and malabsorption due to weanling diarrhea acted synergistically in the production of iron deficiency anemia in prehistoric Ohio skeletons. Walker (1986) also suggested that the high occurrence of porotic hyperostosis among prehistoric California Channel Islanders was a result of the interaction between diet and diarrheal infections. Other researchers including Palkovich (1986), Mensforth *et al.* (1978), Perzigian *et al.* (1984), Norr (1984), and White (1986) also cite combined causes for the prevalence of cribra orbitalia. Despite the presence of disease, all of these researchers suggest that diet is significant in the etiology of iron-deficiency anemia. Porotic hyperostosis and cribra orbitalia, therefore, is usually cited as a good indicator of nutritional stress in past populations (for example, El Najjar *et al.* 1976; Goodman *et al.* 1984a, 1984b; Lallo *et al.* 1977; Mittler and Van Gerven 1994; Shipman *et al.* 1985).

Temporal and Spatial Distribution of the Porotic Lesions

Stuart-Macadam (1992b) has identified a trend in the spatial and temporal distribution of the porotic lesions. The frequency of the porotic lesions increases after the appearance of agriculture in many areas of the world, and decreases toward the 20th Century (Stuart-Macadam 1992b). Kennedy (1984) examined preagricultural remains from India and Sri Lanka and found only one occurrence of porotic hyperostosis, however there was an increase in frequency in agricultural remains. Lallo *et al.* (1977) found porotic hyperostosis to be

more prevalent in Illinois after the appearance of agriculture. Angel (1971) examined skeletons from Catal Huyuk and found a high frequency among adults in the Early Neolithic trading town. Hengen (1971) noted a significant decrease in prevalence of cribra orbitalia in Wurtemberg, Germany from the twelfth to the early twentieth century.

Porotic lesions occur throughout the world, however, they seem to be more prevalent in specific temporal and geographic locations. Hengen's (1971) analysis of 5,698 skulls found that cribra orbitalia more commonly occurred in skeletons that originated in countries closer to the equator. The lesions are also more likely to occur in coastal and lowland areas. Cribra orbitalia was more common in canyon bottom sites than higher altitude sites in the American Southwest (El-Najjar *et al.* 1976). They attribute this difference to inhibited iron absorption from the high maize diet in canyon bottoms. The porotic lesions were also found to be more common in lowland sites than highland sites in Ecuador (Ubelaker 1984b). Cribra orbitalia has been found to be more prevalent in coastal areas rather than highland areas of Peru (Hrdlicka 1914). Angel's (1972) examination of adult and juvenile skeletons from Greece and Turkey, found a higher prevalence in farming populations located in marshy areas than in farming populations living in dry areas. He also found a similar pattern in the occurrence between highland and lowland sites in the Neolithic and Bronze Age populations of Greece and Turkey (Angel 1972). A higher prevalence of cribra orbitalia was found among the Santa Barbara Channel Islanders than among mainland groups of the same time period (Walker 1986). Appendix A is a compilation of the frequency and distribution of porotic hyperostosis and cribra orbitalia worldwide by the above mentioned researchers.

Age and Sex Distribution

Stuart-Macadam (1985) suggests that porotic hyperostosis represents a childhood episode of anemia in which the lesions have not undergone complete remodelling. In young children, all of the available marrow space is occupied with red marrow. With anemia, an increase in the production of red blood cells can cause an expansion of the marrow space outwards. Childrens' bones are more plastic than adults and are more readily altered through marrow hyperplasia (Lanzkowsky 1968). In adults, increased production of red blood cells occurs through expansion of red marrow into fatty yellow marrow cavities, and shortening cellular maturation time, so marrow expansion can occur without exerting this kind of pressure on the bone (Stuart-Macadam 1985). Lesions formed in childhood are believed to undergo remodelling over time so a decreasing prevalence with age is expected (Lanzkowsky 1968; Stuart-Macadam 1985).

Modern studies indicate that iron deficiency anemia is most likely to occur in young children and premenopausal women (Scrimshaw 1991). Osteological studies of cribra orbitalia correspond well with the modern age distribution of iron deficiency anemia. The lesions occur more frequently in children than in adults (Carlson et al. 1974; Cybulski 1977; El-Najjar 1976; Hengen 1971; Nathan and Haas 1966b; Stuart-Macadam 1982). The sexual distribution, however does not fit the pattern. With a higher prevalence of iron deficiency anemia among females worldwide, one would expect to see sexual disparity in the occurrence of cribra orbitalia in skeletal samples (Stuart-Macadam 1985). In the majority of cases, however, there are no statistically significant differences in occurrence between males and females (Stuart-Macadam 1985). According to Stuart-Macadam (1985), the relatively undifferentiated sexual occurrence of the lesions

in most studies, combined with the age distribution, further supports the idea that the lesions reflect a growth period anemia.

Modern clinical evidence suggests that osseous changes do not always occur with an episode of anemia. Influential factors in the development and extent of skeletal changes in anemic individuals include: individual variation, age, distribution of red marrow, the nature of bone, and the severity and duration of the anemia itself (Stuart-Macadam 1982). It is estimated that 50-75% of patients with anemia actually exhibit bony changes radiographically (Stuart-Macadam 1982:235), therefore, what appears archaeologically actually under-represents the amount of anemia in the population (Stuart-Macadam 1991, 1992b).

Development of Iron Deficiency Anemia

Iron and Iron Metabolism

Iron is an essential mineral required in the manufacture of new red blood cells. A major component of red blood cells, iron transports oxygen from the lungs to active respiring tissues. The human body has adopted an efficient strategy to maintain optimal levels of iron. Losses of iron are minimal and maintenance is achieved primarily through dietary absorption.

The majority of iron in blood is associated with hemoglobin which is a conjugated protein consisting of globin (a protein with four polypeptide chains), and heme (four non-protein pigments, each containing ferrous iron). Iron in hemoglobin remains within the circulating red blood cell for approximately 120 days until the plasma membrane becomes too fragile for the cells to function. The worn out cell is removed from circulation by macrophages in the liver, spleen, and bone marrow (Lewis 1981; Wintrobe 1974, 1980). The hemoglobin

molecule is split and the majority of iron is either reused in the manufacture of new red blood cells, or stored as hemosiderin or ferritin in the liver, spleen, bone marrow, heart and kidney (Crichton 1971, 1975). The rest of the molecule is converted into other substances and either reused (globin), or eliminated through sweat, urine, feces, or blood (Fairbanks and Beutler 1988). Loss amounts to about 1 mg a day in a normal adult male (Cook 1990:303). This efficient recycling process ensures that little additional iron needs to be obtained through diet.

The amount of body iron varies along the lines of sex, age, iron status, season, and time of day (Wadsworth 1992). Generally a 70 kg adult male will have a total of about 3-5 grams of iron in his body (Thompson 1988:158). Body iron can be categorized as either functional or storage. Functional iron is generally identified as circulating iron while storage iron acts as a reserve from which losses of functional iron can be replaced. Functional iron accounts for about two thirds of body iron, mainly in the form of hemoglobin in red blood cells, myoglobin in tissue, intracellular heme enzymes in cells, and transport iron bound to transferrin in plasma (Fairbanks and Beutler 1988). Storage iron exists as ferritin and hemosiderin in the liver, spleen and bone marrow (London 1980:176). There is also a very small percentage of iron stored as lactoferrin, a compound found in breast milk, mucosal tissues, and leukocytes (Scrimshaw 1991).

Tissue Hypoxia

When dietary iron becomes insufficient to meet an individual's iron needs, or when iron needs are elevated due to blood loss, the body responds by first drawing upon iron stores and increasing dietary absorption. When this

adjustment is insufficient, hemoglobin synthesis is impaired and tissue hypoxia results. Tissue hypoxia triggers an increased production of erythropoetin (a hormone produced in various body tissues) which stimulates increased red blood cell production in the bone marrow. The result can be an expansion in cellular marrow, which may invoke a bony response as evident in porotic hyperostosis.

Iron Absorption

Balance is crucial in iron metabolism. Too much iron can be toxic and too little can lead to anemia (Arthur and Isbister 1987; Kent and Stuart-Macadam 1992). Iron is a "free radical", making it unstable, readily donating or accepting electrons. In humans, iron is almost always bound to proteins thus making it stable (Griffiths 1987a). In hyperferremic individuals, however, as much as 35% of iron may be free, accumulating in the liver, spleen and heart (Weinberg 1989:10) which can lead to heart disease (Sullivan 1989).

Loss of iron must be balanced through absorption. The average loss of iron for normal adults through the skin, urinary, and gastrointestinal tracts is estimated to be in the range of 0.6 to 1.6 mg per day (Green *et al.* 1968:336). When stored iron is diminished, iron absorption increases and when storage iron is excessive, iron absorption decreases. The average American diet contains about 6 mg of iron per 1000 calories, or between 10 and 30 mg of iron per day (Wintrobe 1974:155). Assuming 1 mg of iron is required to replace the amount lost per day, then 5 to 10% of available dietary iron actually needs to be absorbed. Healthy individuals absorb about 5 to 10% of dietary iron while those who are iron deficient will absorb 10 to 20% (Wintrobe 1974:155).

Little is known about the actual mechanisms that determine how and what percentage of iron is absorbed, however it is known that iron is absorbed in the

small intestine with the most efficient absorption occurring in the upper portion. Iron absorption occurs in two distinct steps: mucosal uptake at the brush border of the mucosal cell; and, transfer of iron from the mucosal cell to the lamina propria where it can enter plasma. Within plasma, iron is bound with the transport protein transferrin and is passed into the storage or marrow sites. The remainder of the iron combines with apoferritin (a protein) to form mucosal ferritin. Some of the ferritin will be taken into the blood, but most will remain in the mucosal cells for a few days before being destroyed (Figure 2). Detailed descriptions of iron absorption and systemic iron loss are provided by Fairbanks and Beutler (1988); Layrisse (1975); Layrisse *et al.* (1974); Lewis (1981); London (1980); and Wintrobe (1974).

Dietary iron is primarily divided into two types: heme and non-heme. The behaviour of iron during digestion and absorption is largely dependent on the type of iron. Most dietary iron is in the form of non-heme iron. Legumes, nuts, whole grain cereals, breads, fruits and vegetables which constitute the principal component of most diets, contain non-heme iron. Heme iron comes from hemoglobin, myoglobin and other heme proteins found in meat. Approximately 40% of the iron in meat, oysters, clams, and poultry is in the form of heme with the remaining 60% as non-heme (Monsen 1981:320).

Heme iron is highly bioavailable because of the way it is absorbed. It is unaffected by the presence of other dietary constituents, unlike non-heme iron. Consequently, the percentage absorption of heme iron can be 5 to 10 times higher than that from non-heme iron (Cook 1990:303). Because absorption of heme iron is constant and independent of meal composition, the contribution to the diet can be readily calculated (Monsen and Balintfy 1982).

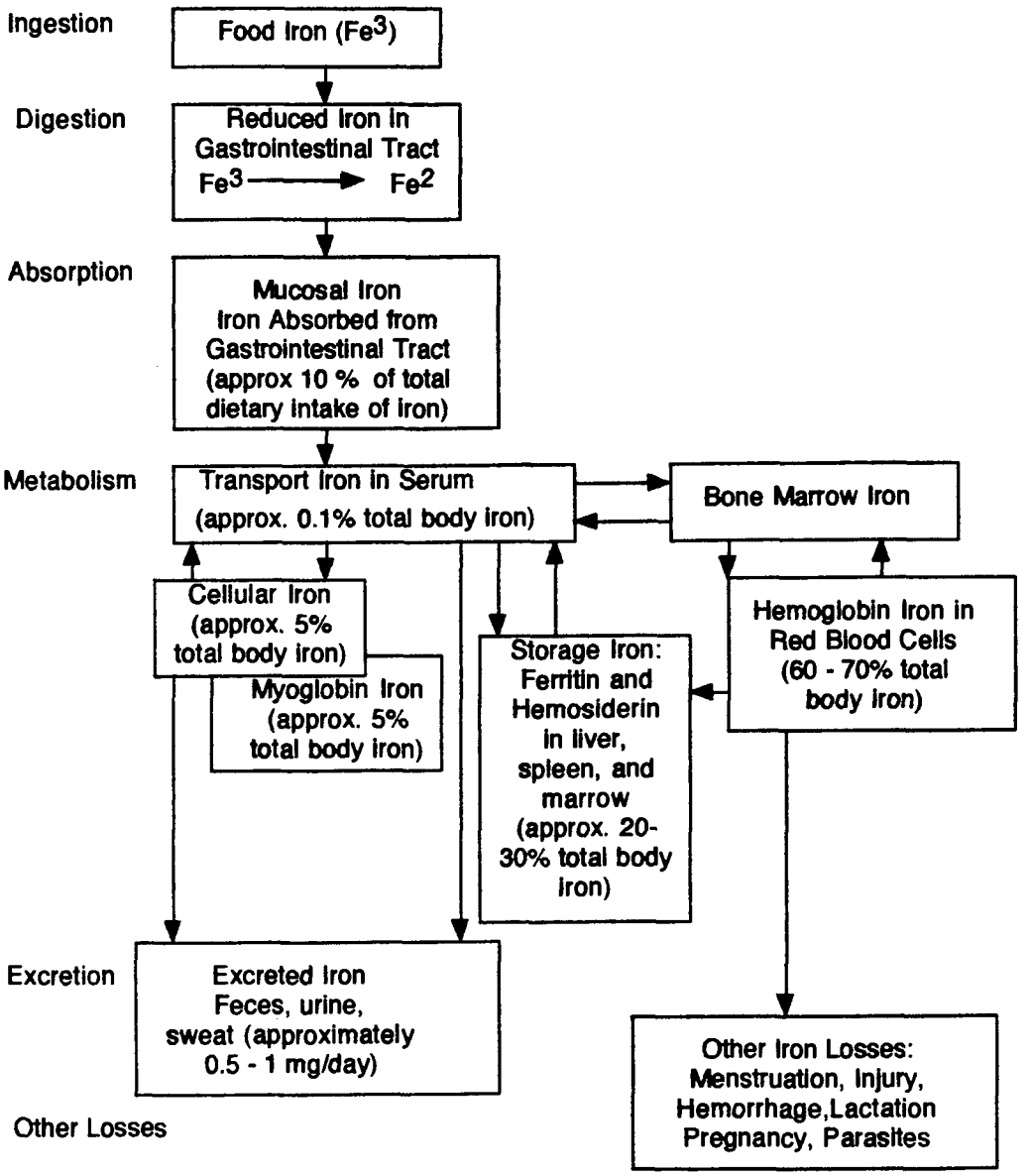


Figure 2. Iron Pathways. Adapted from Mitchell *et al.* (1968:65).

The bioavailability of non-heme iron is largely determined by the presence of enhancers or inhibitors of absorption in the diet. The most important inhibitors reducing bioavailability of non-heme iron include phytates and fiber found in grains and corn; phosphates or phosphoproteins found in egg yolks; oxalates found in some leafy green vegetables, and tannins found in tea (Callender 1975; Cook 1990; Fairbanks and Beutler 1988; Fernandez and Phillips 1982a, 1982b; Frolich and Lyso 1983; Layrisse 1975; Monsen 1981). These substances either attach large molecular compounds to the iron, or combine to form insoluble complexes reducing absorption of non-heme iron (Monsen 1981).

Some foods may facilitate absorption of non-heme iron while having no effect on heme iron (Wintrobe 1974). The major enhancers of non-heme iron absorption are meat and organic acids such as citric, lactic, and especially ascorbic acid (Vitamin C) which is the strongest facilitator of non-heme iron absorption (Bothwell *et al.* 1989; Cook 1990; Fairbanks and Beutler 1988). Foods such as meat, fish and poultry enhance the absorption of iron from non-heme sources. Animal muscle breaks down to yield amino acids, of which cysteine, lysine, and histidine enhance the absorption of iron (Hazell 1985). Cysteine in fish muscle, for example, reduces iron from a ferric to a ferrous state and improves its solubility (Layrisse *et al.* 1974; WHO 1972). The amino acids in muscle form unstable bonds with iron, allowing the iron to be readily taken up by the absorptive cells of the mucosal epithelium (Hazell 1985). Ascorbic acid converts ferric iron to a ferrous state and maintains its solubility in the alkaline environment of the gut. It also forms a bond with ferric chloride keeping it stable at an alkaline pH in the acid environment of the stomach (Bothwell *et al.* 1989). Non-heme iron absorption can increase threefold with

the addition of ascorbic acid (Bothwell *et al.* 1989). Although the addition of ascorbic acid can increase non-heme iron absorption from a single meal, there is some suggestion that continued ascorbic acid supplementation has no effect on total body iron stores (Hunt *et al.* 1994).

Extraneous Iron

Extraneous iron from water can contribute to daily minimum requirements but the dietary effect of this type of iron is not well understood. Extraneous irons in soil and water are usually in an ionized state and can only be absorbed if they are bound with organic compounds (Safe Drinking Water Committee 1980). Nevertheless, groundwater may contain large amounts of ferrous iron which has better bioavailability than ferric iron found in surface waters (Safe Drinking Water Committee 1980). Zoeteman and Brinkmann (1975) found that, in extreme cases, more than 10% of total dietary iron can be obtained from drinking water.

Anemias

Anemia is not actually a disease, but rather, an indicator of a disease. Anemia can be either hereditary, or acquired. An acquired anemia is the most common type and is usually due to one or more of the following: blood loss, ineffective absorption of dietary nutrients, chronic disease or chronic inflammation (Kent and Stuart-Macadam 1992).

Clinical manifestations of anemia are primarily due to the body's response to hypoxia. The intensity of the manifestations vary depending on the severity of the anemia and the presence of co-existing diseases. Individuals may be asymptomatic in the early stages of iron deficiency, but as the disease becomes

chronic, several manifestations may develop, including fatigue, depression, irritability, headache, pallor, inflammation of the tongue, and marrow hyperplasia, among others (Fielding 1975; Callender 1981; Mowinski Jennings 1992, Wintrobe 1974). The skeletal changes that may occur with anemia are due to marrow expansion in response to an anemic stimulus (Mensforth *et al.* 1978).

Hereditary anemias can cause skeletal changes, however, their occurrence has been geographically restricted in the past. Hereditary anemias are not known to have been present on the Northwest Coast prior to contact, and are not considered a cause of cribra orbitalia in the Pender skeletal sample (see Cybulski 1977 for a possible exception regarding the Haida).

Erythroblastosis Foetalis

Erythroblastosis foetalis is an acquired hematological disorder which is sometimes referred to as Hemolytic Disease of the Newborn (HDN). It results when there is fetal-maternal ABO, or Rh incompatibility (Wintrobe 1974). Bone changes primarily occur in the post-cranium as growth arrest lines at the ends of long bones (Brenner and Allen 1963; Caffey 1973; Moseley 1971). A thickening of the frontal and parietal bones has been found to occur as a result of a widening of the diploë (Caffey 1973).

This disorder cannot be considered a factor in the Pender Canal population, since there is only a very small proportion of the Rh negative alleles in the Northwest Coast populations (Hulse 1955). The presence of Rh negative alleles on the Northwest Coast is believed to reflect the time depth of European admixture (Hulse 1955).

Nutritional Anemias

Iron Deficiency

Anemia from a dietary iron deficiency is viewed as the most widely distributed type (Scrimshaw 1991; Wintrobe 1974). Iron deficiency anemias are present worldwide, and common even in countries with relatively good nutritional standards (Wadsworth 1975). Women of reproductive age, infants, and children are the most vulnerable because of increased iron losses or heavy demands for growth (Davidson *et al.* 1975). The recommended daily intake of iron is provided in Table 1. Even with abundant dietary iron, iron deficiency anemia can occur from inadequate absorption of iron, nutritional deficiencies, and blood loss (Robinson 1972).

Absorption is affected by the nature of the diet. A joint study by a group of experts from the Food and Agricultural Organization of the United Nations and the World Health Organization (FAO/WHO 1988) has identified three categories of diet based on iron bioavailability. A low bioavailability diet consists largely of cereals, roots, tubers, and iron inhibiting foods such as corn or beans. Meat, fish or ascorbic acid rich foods are negligible in this type of diet. Total absorption of iron from a low bioavailability diet is around 5 percent. An intermediate bioavailability diet is similar to a low bioavailability one, but with enhanced absorption from the addition of ascorbic acid rich foods and meat and fish. Total iron absorption from this type of diet is approximately 10 percent. A high bioavailability diet is diversified and contains generous amounts of meat, fish, poultry and/or ascorbic acid rich foods. As much as 15 percent of the total dietary intake of iron can be absorbed from this type of diet.

A diet rich in animal foods and low in iron inibitors has the best iron bioavailability.

Diet is not the only factor in determining iron absorption. Absorption of iron varies according to the need of the individual. Iron deficient males have been found to absorb 21% more heme iron and 20% more non-heme iron than iron replete males consuming the same diet (Cook 1990:304). Alternatively, hyperferremia results in decreased iron absorption (Cook 1990). With a total elimination of iron from the diet, which is highly unlikely, a dietary deficiency without concomitant blood loss would take a considerable time to develop (Arthur and Isbister 1987). It is estimated that it would take approximately 6 to 8 years for an adult male to deplete his iron stores and develop iron deficiency solely from a lack of dietary iron, or from malabsorption (Hoffbrand 1981). Even in infants, anemia due to gastrointestinal blood loss is commonly misclassified as a dietary iron deficiency anemia (Fairbanks and Beutler 1988). The efficiency of iron recycling and iron absorption suggests that iron deficiency anemia very rarely results from an inadequate dietary intake of iron (Arthur and Isbister 1987; Fairbanks and Beutler 1988; Hoffbrand 1981).

The onset of iron-deficiency anemia can be insidious and the symptoms gradual. Mild and moderate forms of iron deficiency anemia may be asymptomatic. Some individuals may accommodate well to the anemia and recognition may take several years (Wintrobe 1974:649). As the body attempts to compensate for the anemia, there is often a concomitant increase in cellular marrow that can lead to bony changes like those seen in cribra orbitalia. The effects are most notable in the skull, although they have been noted in the post-cranium as well (Aksoy *et al.* 1966; Cybulski 1977).

Folate Deficiency

Folate is required for red blood cell formation and maturation and a deficiency can cause anemia (Wintrobe 1974). Normal body stores of folate vary and studies indicate an individual's stores can suffice for anywhere from three weeks to three months (FAO/WHO 1988:55; Wintrobe 1974:142). The recommended daily intake (RDI) of folate is provided in Table 2. The most common causes of folic acid deficiency are poor nutrition, or malabsorption from drugs or alcohol (Wintrobe 1974). Green leafy vegetables, liver, meat, fish, and legumes are all good sources of folic acid.

Vitamin B_{12} Deficiency

Vitamin B_{12} aids in the production of red blood cells in the red bone marrow. The recommended daily intake of Vitamin B_{12} is provided in Table 3. A dietary deficiency of Vitamin B_{12} is unusual because the body's stores can last for several years (Wintrobe 1974). Only strict vegans are at risk of developing a deficiency of this vitamin. B_{12} deficiency is nearly always a result of defective absorption but may also be due to lack of intrinsic factor, or other various small intestinal disorders (Wintrobe 1974)). Vitamin B_{12} deficiency can also result from fish tapeworm (*Diphyllobothrium latum*) infestation in humans (Nyberg *et al.* 1958).

Vitamin A Deficiency

Experiments suggest that there is a causal relationship between vitamin A, iron metabolism, and anemia (Bloem *et al.* 1990; Hodges *et al.* 1978, 1980). It is still inconclusive, however, how vitamin A deficiency interferes with normal iron metabolism. There is some suggestion that a deficiency in Vitamin A will

affect the ability of the iron to bind with carrier or intracellular proteins (Hodges *et al.* 1980) . Vitamin A is in fruits and vegetables in a carotenoid form, or in foods of animal origin as retinol (FAO/WHO 1988). The recommended daily intake (RDI) of Vitamin A is provided in Table 4.

Other Related Factors:

Dietary Deficiencies During Accelerated Demand

Neonatal stores, in combination with a high bioavailability of iron in human breast milk, provides sufficient quantities for an infant during the first six months of life (FAO/WHO 1988:43). By around six months, infants' stores of neonatal iron start to deplete and they begin to require iron from external sources. Between four months and one year, an infant's body iron stores increase from about 290 mg, to about 400 mg (WHO 1970:51-52). This amounts to an increase of about 0.5 mg per day during these 8 months. This demand, when combined with daily losses averaging around 0.5 mg per day makes the total iron required for absorption to be the same as an adult male (WHO 1970:52). This increased need for iron continues into adolescence (Dallman *et al.* 1980; Herbert 1987).

During pregnancy, iron requirements amount to an average of 2.5 mg per day over the term of the pregnancy (Wintrobe 1974:648). During this period, iron reserves of women are drained, and without a sufficient absorptive adjustment an anemia may result. Clinical research suggests that as much as a five-fold increase in absorption can occur to compensate for the high demands during pregnancy (King *et al.* 1987). Requirements are less in the early stage of pregnancy, and rise during the last half of pregancy due to expansion of red blood cell mass (Fairbanks and Beutler 1988; Herbert 1987). There are no

additional demands for iron during lactation as daily loss of iron as lactoferrin is about 0.5 to 1 mg, making the total daily requirement roughly equal to that of an adult woman of childbearing age (Wintrobe 1974:649).

Blood Loss

Anemia can be caused by acute or chronic blood loss. Acute blood loss can be caused by sudden hemorrhage due to trauma or surgical complications and disease. Chronic blood loss is associated with extremely heavy menstrual bleeding, hemorrhoids, diarrhea, ulcers, and parasites. Every 2 ml of blood contains 1 mg of iron (Fairbanks and Beutler 1988:206). Iron loss through bleeding is usually compensated by an increase in dietary absorption.

The amount of blood lost through menstruation varies among women, with a small proportion of women having high losses (Milne *et al.* 1990; Fairbanks and Beutler 1988). Basically women have an additional average daily loss of 0.5 mg of iron over the normal daily loss (Cook 1990:303; FAO/WHO 1988:37) which is usually offset through increased absorption. In fact women are believed to have a natural safeguard against anemia by having a great ability to absorb up to twice as much iron as men from the same diet (Wadsworth 1992). Additionally, losses of body iron may diminish through a reduction in menstrual blood loss and cells lost from the surface of the skin in individuals with iron deficiency anemia (Jacobs and Butler 1965; Milne *et al.* 1990).

Moderate to heavy consumption of fish can increase normal bleeding times in both males and females (Herold and Kinsella 1986; Howelingen *et al.* 1987; Sullivan 1989). A two minute increase in normal bleeding time has been observed in at least one study (Houwelingen *et al.* 1987). The exact reason is unknown, however it is believed that the increased bleeding time results from

reduced platelet aggregation (Herold and Kinsella 1986). Despite increased bleeding time, hemoglobin values are still reported to be within the normal range (Houwelingen *et al.* 1987). The development of anemia from increased bleeding time in various studies of fish eating groups is rare and has been reported in only one case (Houwelingen *et al.* 1987:434). This condition would be most critical to women of menstrual age and those individuals suffering from injury or parasitic infection.

Parasites can be a major source of blood loss, often affecting an entire population. Parasitic and bacterial infections that cause internal bleeding have been demonstrated to cause iron deficiency anemia among contemporary groups (Cohen 1989; Haddock *et al.* 1991; Scrimshaw 1991; Scrimshaw and Tejada 1970). Parasitic infections are most common in areas where inadequate sanitation, overcrowding, and various other cultural and ecological health hazards provide an environment for bacterial proliferation (Dunn 1968, 1972; Kent 1986; Scrimshaw 1991). In many rural Guatemalan villages, for example, the modern parasitic infection rate is 100% of the population (Scrimshaw and Tejada 1970:211). Evidence suggests similar parasitic conditions were also present in past populations (Reinhard 1992). Hookworm (*Anclystoma*) has been recovered from coprolites and mummified remains in Peru, Brazil, and Tennessee (Allison *et al.* 1974; Faulkner *et al.* 1989; Ferreira *et al.* 1980 cited in Reinhard 1992:242). Hookworm and schistosomiasis have been implicated as possible factors of iron deficiency anemia in modern and prehistoric Nubia (Carlson *et al.* 1974). Tapeworm (*Diphyllobothrium pacificum*) has been recovered from coprolites on the coast of prehistoric Chile (Ferreira *et al.* 1984 cited in Reinhard 1992:236; Patrucco *et al.* 1983). Hydatid cyst disease and trichinosis, also caused by helminth parasites have been reported from

prehistoric skeletons (Ortner and Putschar 1981; Williams 1985; Zimmerman and Aufderheide 1984). Thorny-headed worm (*Acanthocephala)* has been found in human coprolites from Utah (Moore *et al.* 1969).

Anemia of Chronic Disorders or Infection

Anemia from chronic disorders is very common and is identified as being second only to iron-deficiency anemia in prevalence (Wintrobe 1974). Mild anemias can be caused by chronic infection, rheumatoid arthritis, fractures, severe tissue injury, and neoplastic diseases such as cancer (Cartwright 1966; Lee 1983; Weinberg 1977, 1984; Wintrobe 1974). The anemia is a physiological compensatory response initiated by the body's defense to an infection (Cartwright 1966). The anemia is usually mild in degree and not progressive in severity (Cartwright 1966).

Nutritional Immunity

There is increasing evidence to suggest that lowered iron levels may be an adaptation to protect the body against infection (see for example Dallman 1987; Griffiths 1987a, 1987b; Griffiths and Bullen 1987; Kent and Weinberg 1989; Lauffer 1992; Lee 1983; Payne 1988; Strauss 1978; Weinberg 1974, 1977, 1984, 1992a, 1992b). Iron is required by bacterial and viral pathogens to survive and reproduce in the host's tissues (Weinberg 1992a, 1992b). The body responds to pathogens, neoplasia and inflammation by decreasing absorption of iron and preventing the release of iron from storage into circulating blood (Weinberg 1992a 1992b). Studies have shown a consistent immediate decrease in iron absorption in children with infections or who have been injected with vaccines (Weinberg 1974). A reduction in plasma iron, therefore, strengthens

the host's defense against microbial invasion. Studies have also shown the opposite to be true: when iron is added, microbial growth is enhanced (Kluger and Rothenburg 1979; Payne 1988; Weinberg 1974, 1992a, 1992b). Anemia, despite the presence of iron stores is a paradoxical situation that Cartwright (1966:371) refers to as "iron starvation in the face of plenty". This attempt by the host to withhold iron from invaders has been termed a "nutritional immunity" (Weinberg 1974:952) although it is more commonly referred to as an "anemia of chronic disorders" (Cartwright 1966:371) or an anemia of chronic infection.

Clinical research indicates that a large number of cases of anemia of chronic infection are commonly misdiagnosed as iron deficiency anemia and incorrectly treated (Arthur and Isbister 1987). Iron therapy may prove to be disadvantageous and sometimes fatal to a patient with an infection (Becroft *et al.* 1977; Kent 1992; Kent *et al.* 1990; Weinberg 1992a). This lowered iron level can be an adaptive strategy and probably does not have any deleterious effects unless extra demands from growth spurts, pregnancy, or blood loss upset the precarious iron balance resulting in iron-deficiency anemia (Stuart-Macadam 1991, *in press*). There is some suggestion that the interaction of fever and reduced iron are part of a coordinated defense response by the host to combat bacterial and viral infections (Kluger and Rothenburg 1979). An elevated body temperature has been shown to interfere with bacteria's ability to produce iron-transport compounds (siderophores) thus decreasing the ability of the pathogenic bacteria to sequester adequate iron for growth from the host (Kluger and Rothenburg 1979; Weinberg 1984). The same leukocyte-derived protein is believed to be responsible for the development of fever and reduction of serum iron (Kluger and Rothenburg 1979).

Summary

This chapter has attempted to demonstrate two points: that cribra orbitalia is indicative of an acquired iron deficiency in the majority of skeletal remains; and, that anemia as a result of a dietary deficiency of iron is highly unlikely. Cribrotic lesions result from hyperplasia of the red bone marrow which expands the diploic space and thins the outer table through pressure atrophy. The lesions are believed to form in childhood when there is little room for marrow expansion and the bones are more plastic. Most researchers include diet as a major factor in the development of cribra orbitalia, however there is some question whether diet is always responsible. Absorption of iron is determined primarily by the iron status of the individual (Herbert 1987; Wintrobe 1974) but is also influenced by total iron intake, bioavailability of non-heme iron, gastrointestinal state, and the presence of infection or disease. The body has evolved an efficient system of iron acquisition and even an individual with a diet very low in iron can acquire what is needed through increased absorption.

Iron deficiency anemia is prevalent even in areas with good nutrition and high dietary iron (Wadsworth 1975). Lowered iron levels do not necessarily lead to an anemia and can protect the body against infection. Iron deficiency anemia is almost always due to malabsorption, blood loss, or infection. A review of the literature on iron metabolism (Fairbanks and Beutler 1988; Hoffbrand 1981; Layrisse 1975; Wintrobe 1974, 1980) medical and clinical studies, (Arthur and Isbister 1987; FAO/WHO 1988; Griffiths 1987b; Payne 1988; Sullivan 1989; Weinberg 1974, 1984, 1992a, 1992b; WHO 1972, 1975) and the archaeological distribution of cribra orbitalia (Angel 1971; Cybulski 1977, 1990, 1994; El Najjar *et al.* 1976; Goodman *et al.* 1984; Hrdlicka 1914; Kent 1986; Nathan and Haas 1966a, 1966b; Stuart-Macadam 1982; Walker 1986)

indicates that factors other than diet play a role in the development of iron deficiency anemia in past and present human populations.

Table 1. Recommended Daily Dietary Intake of Iron (mg/day)

Group	External Losses	Menses	Pregnancy Cost	Growth	Fe Required	Daily Iron Intake Required[a]
Adult males [b] (50-100 kg)	0.65-1.3				0.65-1.3	6.5-13
Non-menstruating women [b] (45-70 kg)	0.6-0.9				0.6-0.9	6-9
Menstruating women [b] (45-70 kg)	0.6-0.9	0.1-1.4			0.7-2.3	7-23
Lactating women [c]	1.1				1.1	11
1st Trimester Pregnancy [d] (55 kg prior to pregnancy)	0.8				0.8	8
2nd Trimester Pregnancy [d]	0.8		3.6		4.4	44
3rd Trimester Pregnancy [d]	0.8		5.5		6.3	63
Adolescent boys [b] (50-100 kg)	0.65-1.3			0.35-0.7	1-2	10-20
Adolescent girls [b] (45-70 kg)	0.6-0.9	0.1-1.4		0.3-0.45	1-2.7	10-27
Children [e]					0.4-1.0	4-10
Infants [e]					0.5-1.5	5-15

a Estimate based on 10% iron absorption from diet.
b Estimates from Fairbanks and Beutler (1988: 207).
c Estimates by a Joint FAO/WHO Expert Consultation, Food and Agriculture Organization of the United Nations (FAO/WHO 1988:37-38).
d Estimates by a Joint FAO/WHO Expert Group (WHO 1970: 53).
e Estimates by the Committee on Iron Deficiency (1968: 407-412).

Table 2. Recommended Daily Dietary Intake of Total Folate

Group	Age	Recommended Level of Intake (μg/kg body weight/day)
Infants	0-3 months	3.6
	3-6 months	3.6
	6-12 months	3.6
Children	1-6 years	3.3
	6-12 years	3.3
	12-16 years	3.3
Males	16+ years	3.1
Females	16+ years	3.1
Pregnant women		7.0
Lactating women		5.0

Adapted from a Joint FAO/WHO Group of Experts, Food and Agriculture Organization of the United Nations (FAO/WHO 1988:58).

Table 3. Recommended Daily Dietary Intake of Vitamin B_{12}

Group	Age (years)	Recommended Level of Intake (μg/day)
Infants	0-1	0.1
Children	1-10	0.1 -1.0 [a]
Pregnant Women		1.4
Lactating Women		1.3
Others	10+	1.0

[a] Safe level of intake estimated at 0.04 ug/kg/day.

Adapted from a Joint FAO/WHO Group of Experts, Food and Agriculture Organization of the United Nations (FAO/WHO 1988:71).

Table 4. Recommended Daily Dietary Intake of Vitamin A

Group	Age (years)	Level of Intake (μg retinol equivalents/day)
Both Sexes	0-1	350
	1-6	400
	6-10	400
	10-12	500
	12-15	600
Boys	15-18	600
Girls	15-18	500
Men	18 +	600
Women	18 +	500
Pregnant Women		600
Lactating Women		850

Adapted from a Joint FAO/WHO Group of Experts, Food and Agriculture Organization of the United Nations (FAO/WHO 1988:25).

Chapter III
Materials

Introduction

The Pender Canal sites are located on Pender Island which is situated in the southern Gulf Islands of British Columbia between Vancouver Island and the mainland (Figure 3). The skeletal sample comes from the Pender Canal sites, DeRt 1 and DeRt 2, located on the the North side of an artificial canal which runs between Port Browning and Bedwell Harbour. The construction of the canal in 1902-03 split the island into North and South halves and allowed small marine craft access between Shark Cove and Bedwell Harbour. Prior to the construction of the canal, a narrow, swampy isthmus, or landbridge, connected the North and South Islands. The construction of the canal and subsequent building of a road and bridge to rejoin the two islands created four remnants of what had probably once been a single site (Hanson 1991). The four midden deposits that remain of this site are known as DeRt 1, DeRt 2, DeRt 18, and DeRt 19 (Figure 4). There is also a midden site on Mortimer Spit (DeRt 3), and just south of DeRt 2 there is a fortification site on Ainslie Point.

Archaeological Investigations

Although the sites were known as early as the construction of the canal, the first excavations were not undertaken until some years later. In 1957 and 1958 the Provincial Museum excavated two test pits at DeRt 2 (Kew 1959). Excavations were again undertaken in 1971 at DeRt 1 in the form of several test pits as part of Simon Fraser University's "Salvage '71" Project (McMurdo 1971). Subsequent erosion from the canal necessitated more thorough

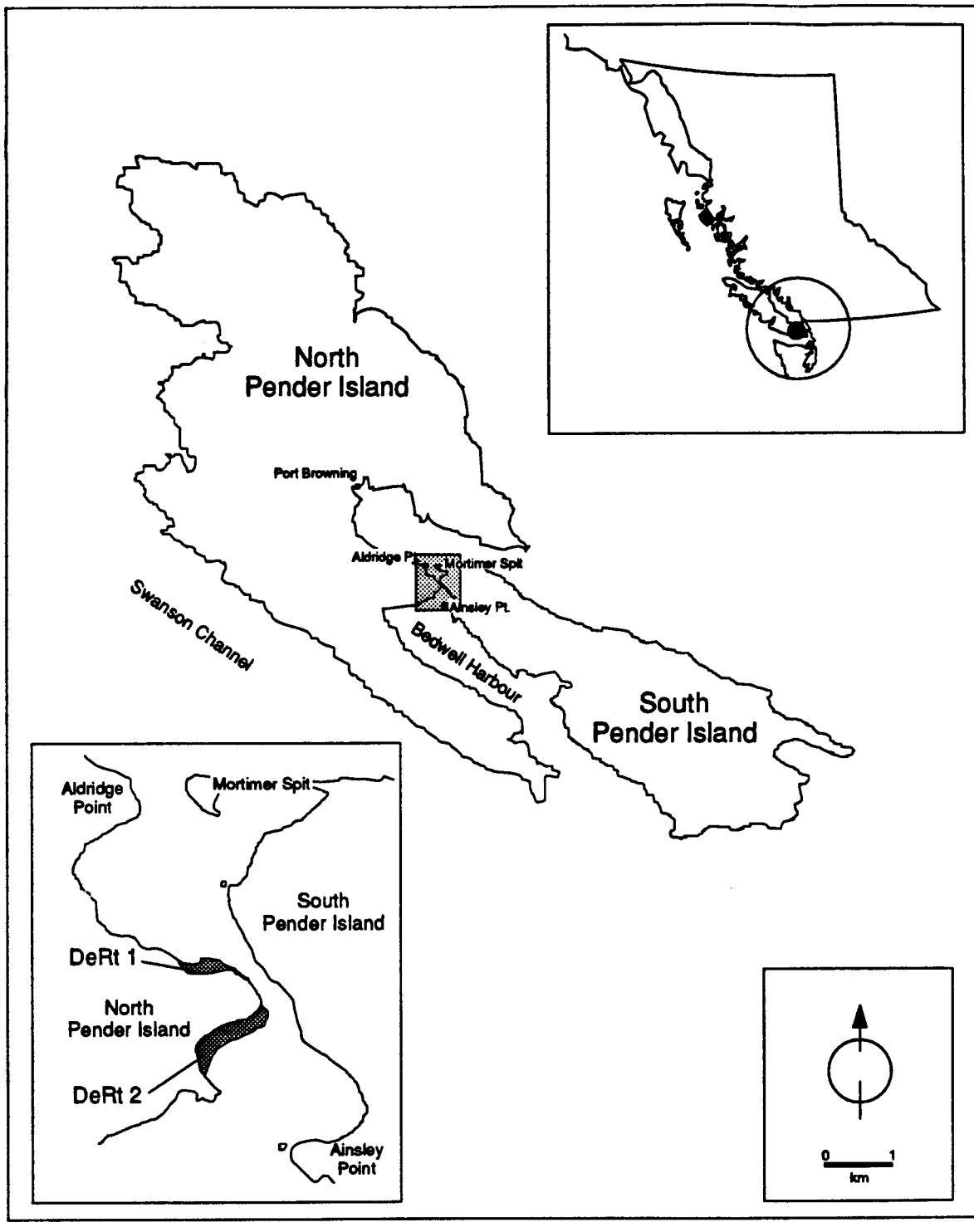


Figure 3. Map showing the location of North and South Pender Islands. DeRt 1 and DeRt 2 are indicated by arrows.

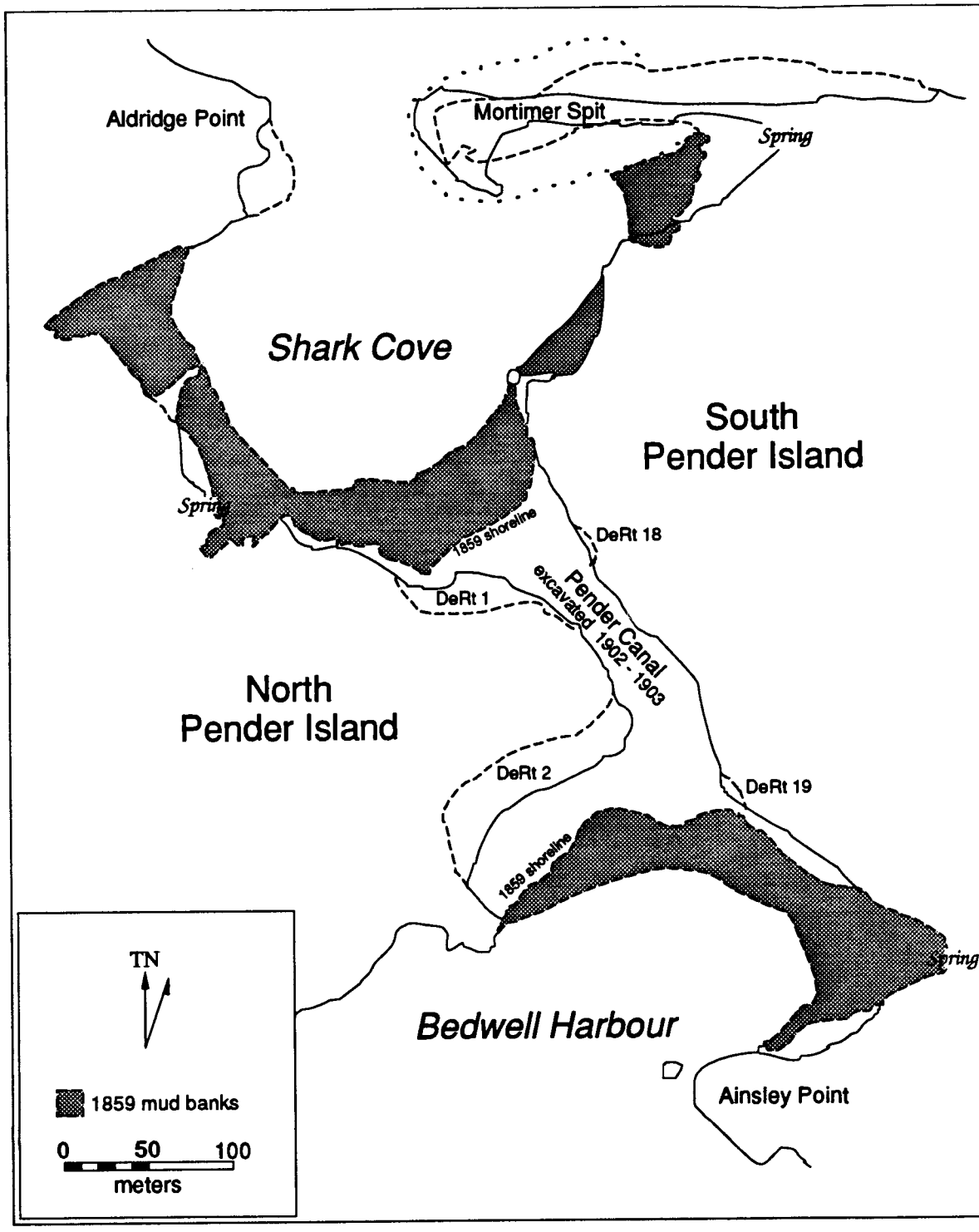


Figure 4. Map showing the locations of Pender Canal sites. Both the present and pre-canal shorelines are indicated, as mapped by Captain G.H. Richards and the Officers of the H.M.S. Plumper in 1858 - 1860 (adapted from Carlson 1986: 4).

excavation of the sites and excavations continued at DeRt 1 and DeRt 2 over three field seasons from 1984 to 1986 as a joint project between the Heritage Conservation Branch and Simon Fraser University under the direction of Dr. R. L. Carlson. The skeletal collection used in this analysis was recovered from 1984 to 1986, with the exception of one skeleton salvaged in 1987.

Evidence from burial areas provide the majority of information for the site. Houses were probably built close to high tide levels; rises in sea level, as well as construction of the canal have obscured almost all traces of these living areas (Carlson 1993). Preservation of the burials at DeRt 2 is largely a result of the slightly higher elevation (Carlson 1993).

The date of occupation of the sites is estimated to be between 5200 and 400 B.P. with a winter village type of settlement indicated during the Mayne and Locarno Beach phases (Carlson and Hobler 1993). Faunal analysis by D. Hanson (1991) and $\partial\ ^{13}C$ studies by B. Chisholm (1986) indicate a full maritime adaptation throughout the occupation periods.

Contemporary Environmental Profile

Pender Island is an area of abundant and diverse resources. Rocky shores, secluded coves, and mud, sand and gravel bayshores provide a habitat for shellfish and many other types of fish. Many of the resources are available year-round. The fauna and flora on the islands have changed since the time of site occupation, largely due to the influence of settlers (Hanson 1991). A description of changes in fauna can be found in Hanson (1991).

North Pender Island currently has several sources of freshwater. Three freshwater lakes which supply drinking water include: Majic Lake, Buck Lake, and Roe Lake. Majic Lake and Buck Lake, however, were artificially created in the 1960's by damming the outlet of swamps (Mourdant 1981). Several

freshwater springs exist on the island today with three in close proximity to the Pender Canal Sites (Carlson 1986:4, Figure 4). It is not known if these springs existed at the time of occupation, however they did exist as early as 1858 when Captain G.H. Richards mapped the pre-canal shoreline at Pender. A number of short creeks which flow during the rainy season (Mourdant 1981) may have provided freshwater for the earlier inhabitants as well. Pender Island water quality is currently problematic due to high levels of naturally occurring minerals. High levels of iron pose a particular problem at Pender, occurring both in groundwater and freshwater lakes (Mourdant 1981:18). Ground water samples from a well location presently near the Pender Canal sites has iron levels exceeding the recommended standard (Ministry of Environment n.d.). It is reasonable to assume that these naturally occurring minerals also existed in the groundwater-fed springs around the site in the past, and may have contributed to the iron content of the diet.

Ethnographic References

The Pender Island sites are located in Straits (Saanich) territory. There is no specific ethnographic data concerning use of the Pender Canal sites. The first description of Pender Island was by the Spanish explorer Juan Pantoja y Arriaga in 1791 when he piloted the ship Santa Saturna into Haro Strait passing between Saturna and Pender Islands, into the Gulf of Georgia and then into Rosario Strait (Pethick 1980:54). Moon (1985) gives an interesting ethnohistoric account of Pender Island through local informants memory and stories passed down from early settlers.

The site is believed to have been occupied by the ancestors of the East Saanich Band of the Coast Salish (Carlson 1993). Barnett (1955) stated that the Saanich went to South Pender Island to fish for salmon and the Cowichan went to

North Pender Island to fish for herring, halibut, porpoise and seal. Jenness (n.d.) noted that people from Duncan would fish for sockeye salmon just off Pender Island. There are no salmon reef-netting stations within the immediate vicinity of the Pender Canal Sites. The recovered faunal elements suggest that salmon were caught and processed at another location and brought to the Pender Canal sites (Carlson 1985; Hanson 1991)

The location of the Pender Canal sites are consistent with ethnographic village site descriptions. Jenness (n.d.: 3-4) describes Saanich village sites as being adjacent to bays with sand or gravel beaches and forested lands behind. Suttles (1974:46) states that, in addition to being adjacent to sand or gravel beaches, village sites were situated so as to provide protection from northeast and southwest winds. The sites of DeRt 1 and DeRt 2 at Pender Canal meet both of the above criteria. In addition, Mortimer Spit and Ainslie Point allowed lookout and defense locations on Bedwell Harbour and Port Browning. The landbridge between North and South Pender Islands allowed quick escape to the opposite bay. The above combined features, together with the presence of freshwater streams near the site and the diversity of species at the site all served to make this an ideal area for a semi-permanent or permanent village (Hanson 1991:82).

Skeletal Collection

Over 100 individuals were recovered from the Pender Canal sites. An accurate estimation is impossible at this time due to the fragmentation caused by continuous interments. Burials consisted of single inhumations and multiple interments. Some burials were placed under cairns. In addition, a number of miscellaneous, single element bones were recovered and recorded. The majority of the burials recovered are from DeRt 2, however, a few (6) come from DeRt

1. Almost all burials came from the shell midden and are from layers which have been radiocarbon dated to 5200-1100 BP . The majority of the burials are from levels which contain deposits of the Mayne and Locarno Beach Phases of local culture history.

Summary

Archaeological investigations suggest a winter or possible year-round village occupation of the Pender Canal sites. Radiocarbon dates indicate great time depth in site use extending over a period of approximately 4500 years. The archaeological evidence from the site largely consists of burials and accompanying artifacts, although there is some evidence of houses from the later period of occupation (Carlson 1993).

The Pender skeletal collection is ideal for analysis for several reasons, the foremost being its large size. The collection is also well dated, having forty-five radiocarbon dates, most of these directly from the skeletal material. The site has had a fairly comprehensive faunal analysis done, indicating the animal protein component of the diet (Hanson 1991), which, when combined with the stable carbon isotope analysis (Chisholm 1986) gives some indication of what animal foods were being eaten and the relative percentage of them. There is a large body of data, both historical, ethnographic, and archaeological, providing a picture of life on the Northwest Coast at the time of European contact.

Skeletal research on cribra orbitalia has been done on many collections from the Northwest Coast, however, no research has been done on a prehistoric sample size this large from the Gulf of Georgia region. Cybulski (1990; 1994) has noted a higher incidence of cribra orbitalia in the Gulf of Georgia region and the Queen Charlotte Islands as compared to other regions on the coast, and

has suggested that this may be a reflection of different population densities. The analysis of the Pender Island collection will add to this body of data.

The primary drawback to the Pender Canal skeletal collection is the high degree of fragmentation. Multiple interments at the site and fragmentation have resulted in mixing of individuals. This limits the amount of sex and age information that can be derived. The nature of this research, however, draws primarily on the frequency of skeletal lesions and in this regard the sample is well suited to the analysis.

Chapter IV
Methods

Introduction

The nature of this research necessitated data collection from two areas: skeletal and dietary. The skeletal data allows a determination of cribra orbitalia in the Pender sample. This can be used to infer the prevalence of iron deficiency anemia in the population. Dietary data indicates the types of species that were available and possibly eaten by the individuals from the site. Through this data, a fairly reliable assessment of nutritional adequacy of diet can be obtained.

Skeletal Sample Collection

Individual burials and single elements in the Pender skeletal collection were examined, assigned identification numbers and entered into an Excel 4.0 spreadsheet . The sample in this analysis was selected from the Pender collection based on the preservation of at least one eye orbit. Partial orbits were only included if enough of the orbit was preserved for a reliable assessment. In order to eliminate any possibility of duplication of individuals, only those specimens with the presence of a glabella point (mid-frontal) were included. The resulting sample of 12 Right, 17 Left, and 54 paired orbits represents 83 individuals (Appendix B).

Data Collection

Although many of the individuals in the sample had previously been aged and sexed by students or other physical anthropologists, these data were

recollected by the analyst. It was hoped that this would provide a more consistent categorical classification of sex and age.

It would have been ideal to study the prevalence of porotic hyperostosis as well as cribra orbitalia, but this was not possible due to time constraints and fragmentation. Many of the skull fragments exhibited porotic hyperostosis with severe vault thickening but the prevalence could not be quantified because this would have required major cranial reconstruction. Severe vault thickening and porosity were noted when obvious (see Appendix B).

Aging and Sexing of Skeletal Material

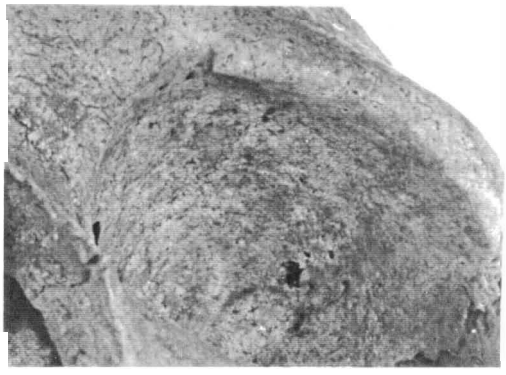
Determining the age and sex of individuals in the sample was hindered by the exclusion of post-cranial bones. Sex was estimated by a visual assessment of the skull using standard morphological criteria (Bass 1987; Brothwell 1972). No metrical data was collected. Sex estimation was aided by studying the post-cranial skeletons of four individuals in the sample (ID Numbers 59, 99, 113, 147). A set of six primary and six secondary sexual characteristics was determined for the four adult skulls in light of diagnostic post-cranial indicators of sex (in Bass 1987; Brothwell 1972). These twelve sex distinctions were in turn applied to the remaining crania in the sample. Each of the skeletal characteristics was visually assessed and rated as either Male, Female, Indeterminate, or Not Present. Sex was conservatively assigned to all adult individuals based on a table worked out by the analyst (Table 5). Generally, an individual with four primary characteristics of a sex, was assigned to that sex. The conservativism of this method yielded a high number of individuals of indeterminate sex.

Age at death was estimated by use of dental eruption sequence (Brothwell 1972). Age estimates were classified into 10 categories: Fetus to 6 months; 6

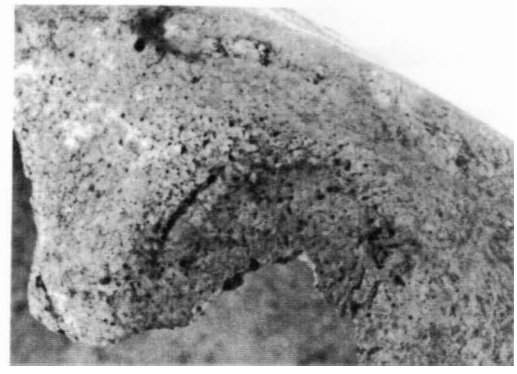
months to 2 years; 2-4 years; 4-6 years; 6-8 years; 8-12 years; Adult (18+); Old Adult; and Indeterminate. All individuals with a third molar were considered to be young to mid-adult unless they had definitive characteristics of old age. Old adults were differentiated from young to mid-adults by the presence of characteristic degenerative changes such as severe tooth loss, severe tooth wear, endo-cranial suture closure, and alveolar resorption. For analytical purposes, the ages were lumped into three general categories: Children (0 to 6 years); Juvenile (6 to 18 years); and Adult (18+). The general categorization aided in comparing results with those of other researchers (for example, Cybulski 1977). The more specific age categories collected in this analysis were done primarily so that other researchers might use them for comparative purposes in the future.

Scoring of Orbital Lesions

Crania were examined macroscopically for the presence of cribra orbitalia. Scoring of lesion severity was based on a revised version of Nathan and Haas (1966a) and Stuart-Macadam's (1982) criteria (Table 6). Each of the categories of severity is clearly defined with little potential for overlap. Cribra orbitalia is readily distinguished from post-mortem damage. Capillary-like impressions on the bone, believed to be indicative of healed lesions (Stuart Macadam 1982; Webb 1989) were noted in the analysis, however they were not included in the scoring. Figure 5 illustrates the identified orbital lesion types. The lesions were scored according to the most advanced type in each case. Observations on cribra orbitalia severity were repeated and the level of concordance for scoring was found to be 89.7%.



NORMAL



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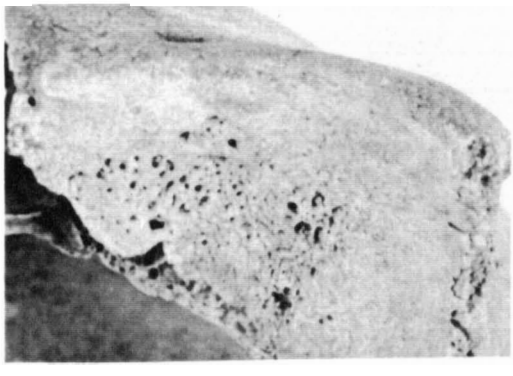

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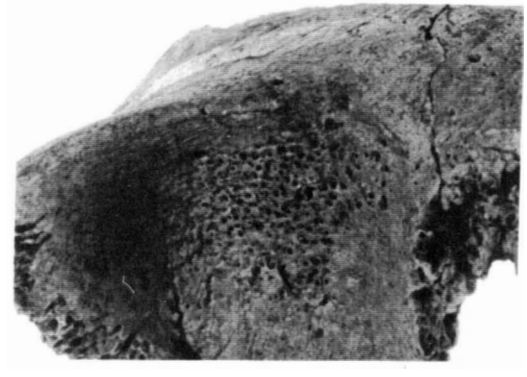

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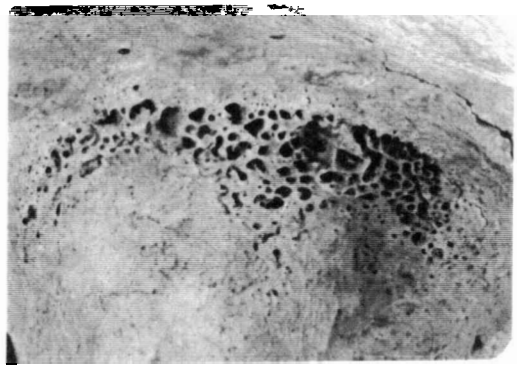

SEVERITY TYPE 4

Figure 5. Orbital Lesion Severity Types Identified in the Pender Canal Skeletal Sample.

Analysis

Chronological time period for the burials that did not have radiocarbon dates was estimated by R. Carlson based on stratigraphic position, depth, and proximity to C-14 dated material. Nineteen of the individuals in the selected sample have radiocarbon dates. Where possible, each burial was placed into one of six one-thousand-year time periods. Table 7 provides a list of the burials in the sample with actual radiocarbon dates.

Statistical analysis of the sample and the production of figures was aided by using Statview 4.0 and Systat 5.2.1. Comparison of the proportions of two different groups was tested by Chi-square analysis, and Fisher's Exact test where possible. The results of the statistical analysis can be found in Chapter 5.

Dietary Data Collection

Archaeological, ethnographic, and historical sources have been used to compile information on food resources for the Pender Canal Sites and the Straits Salish. The use of a number of sources of data provides a more realistic picture of prehistoric Salish diet. Archaeological data which pertains to food resources is drawn from human bones, faunal remains, artifacts of subsistence procurement and preparation, and features such as hearths. Ethnographic description provides details on species eaten, procurement strategies, cooking and preservation techniques, and food taboos (Barnett 1955; Boas 1909; Drucker 1951; Eells 1976; Jenness n.d.; McIlwraith 1948; Suttles 1974; and others). It is assumed that once techniques of food procurement develop, use would persist over time (Hobler 1990). The use of ethnographic analogy for food processing can be considered sound in this case, since many of the patterns or techniques noted ethnographically can be identified archaeologically, for example, stone

boiling or steaming of food in pits. Faunal data provides corroborative evidence for ethnographic information.

Although the presence of available foodstuffs can be identified, it is not possible to determine which foods were actually being eaten or the quantities consumed by the Pender Island inhabitants. It has to be assumed that individuals were eating available foods in sufficient quantities to maintain health. A stable carbon isotope analysis of fifty individuals from the Pender Canal Sites indicates that the majority of dietary protein was derived from marine resources (Chisholm 1986:123). Although providing information on the protein component of the diet, the stable isotope analysis does not indicate the relative percentage of plant or animal foods in the Pender diet. This must be estimated from ethnographic and historical sources.

It has been fairly well established that foods of animal origin were the principal means of subsistence prehistorically on the Northwest Coast (Folan 1984; Jenness n.d.; Murdock 1967; Suttles 1974), however, plants must have been important as well (Norton 1981). Some vitamin and minerals cannot be derived from animal foods, for example Vitamin C. Animal foods have been emphasized almost to the exclusion of plants in ethnographic and historical accounts and current paleo-diet reconstructions (for example, see Chisholm 1986; Chisholm *et al.* 1983; Folan 1984; Hanson 1991). This exclusion is partly due to the fact that animal remains are more visible archaeologically, while plant foods rarely preserve. Some researchers have addressed the importance of plants in native diet (for example, Kuhnlein and Turner 1991; Norton 1981; Turner 1975, 1981; Turner and Kuhnlein 1975, 1982). The retention of the memory of their use and preparation up to the present day indicates that a number of plant foods may have always been important on the coast. There are a number of ethnohistoric accounts for the traditional use of plants (see for

example, Gunther 1945, 1974; Suttles 1974). The importance of plants for essential carbohydrates, as well as a source of vitamins in the winter months cannot be disregarded.

Further support for the importance of plants on the Northwest Coast comes from the site of Ozette, located on the north coast of Washington state. Ozette has yielded approximately 30,000 seeds of salmonberry and red elderberry (cited in Norton 1981:435). An analysis of coprolites from the site indicates that approximately 10% of the diet was plant material (cited in Norton 1981:435).

It has been argued that, although animal protein may provide a reliable and important source of energy, it cannot be the major source of kilojoules (Noli and Avery 1988). According to Noli and Avery (1988:399) no more than 20 - 50% of the diet can be derived from animal protein, without the addition of carbohydrates and fats. Over-consumption of protein may have been occasional and short-term, but extended reliance on a protein diet would have caused death from protein poisoning (Noli and Avery 1988). Carbohydrates and fats, as well as being good sources of energy, are essential in preventing protein poisoning. Carbohydrates or fat-rich foods, therefore, probably provided the basic source of energy in prehistoric coastal dwelling peoples (Noli and Avery 1988). This may account for the extreme cultural importance of marine fats such as eulachen grease, as well as carbohydrate-rich camas in the largely marine-protein diet on the Northwest Coast.

Dietary plant resources from Pender Island have been determined through the range of species available to the inhabitants, and ethnohistoric information on Coast Salish plant utilization. A botanical analysis of traditional flora in the vicinity of the Pender Canal sites has been done by Brooke (in Carlson 1985:Appendix II). Some information is also derived from historical or

ethnographic sources suggesting the importance of various plants (Gunther 1945, 1974; Rivera 1949). The majority of the floral information was derived from studies of traditional plant foods used by Northwest Coast peoples (Keeley *et al.* 1982; Kuhnlein and Turner 1991; Norton 1981; Norton *et al.* 1984; Turner 1975). Nutritional information on plants was obtained from a compendium of sources found in Turner and Kuhnlein (1991) and analysis done by other researchers (Holland *et al.* 1991; Keeley *et al.* 1982; Kuhnlein and Turner 1986). Table 8 provides a partial list of native plant food resources that are currently available in the vicinity of the Pender Canal sites. Appendix C provides the nutrient composition of selected plants that would have been available to the Pender inhabitants.

The meat component of the diet at the Pender Canal sites is compiled from the archaeological evidence, in particular, the faunal analysis lists. Faunal lists from DeRt 1 and DeRt 2 indicate that a wide range of both terrestrial and marine animal foods was available (see Duff n.d.; Galdikas-Brindamour 1972, Hanson 1985,1986, 1991; for a list of fauna recovered). Recent studies assert that, like plants, shellfish resources have been underemphasized on the Northwest Coast (Belcher 1985; Moss 1993). According to Moss (1993), the general impression that shellfish played a minor role in Northwest Coast food economies is probably due to ethnographic bias. Many food sources are gender-specific and male informants and ethnographers may have unknowingly produced biased reports as a result (Moss 1993). Archaeological data, however confirms the importance of shellfish at Pender. Faunal analysis indicates that fish and shellfish were the main meat constituents in the diet (Hanson, 1986, 1991). Tools indicative of sea mammal hunting and fishing have been recovered archaeologically (Carlson 1986) supporting the ethnohistorical data on the importance of marine foods in the diet. Table 9 is a partial list of animal foods

utilized by the inhabitants based on the faunal lists from the Pender Canal sites (Hanson 1986, 1991). The nutrient values of a select number of animal foods can be found in Appendix D.

In many cases it was not possible to obtain nutritional information on specific native fauna or flora, so generalized nutrient values have been substituted. Although there can be some differences in actual nutrient composition between native and modern species, it is felt that this information is quite sufficient for determining the nutritional content of native foods.

Table 5. Criteria used for Assigning Sex to Adult Cranial Elements

Number of Primary Criteria [a]	Number of Secondary Criteria [b]	Sex Determination
1	0-6	Indeterminate
2	0-6	Indeterminate
3	< 3	Indeterminate
3	3 or more	Male or Female
4	2 or more	Male or Female
5	0-6	Male or Female
6	0-6	Male or Female

a Primary Criteria include: General size and architecture; size of mastoid process; frontal angle; size of supraorbital ridge; musculature of occipital area; gonial angle.

b Secondary Criteria include: shape and height of orbits; shape and height of nasal aperture; musculature of malars; size of occipital condyles; shape of chin.

Table 6. Criteria for Scoring Severity of Cribra Orbitalia Lesions

Score	Description
0	No evidence of cribra orbitalia
1	Porotic Type: A small number of scattered, fine foramina forming a round or elongated cluster.
2	Cribrotic Type: Small as well as large foramina (isolated).
3	Trabecular Type: Linkage of foramina into a trabecular structure.
4	Advanced Trabecular Type: Outgrowth in trabecular form from the outer table surface with involvement of a substantial area ($> 1cm^2$).

Adapted from Nathan and Haas (1966a) and Stuart-Macadam (1982).

Table 7. Radiocarbon Dates for Sampled Skeletal Remains from the Pender Canal Sites

Identification Number	Field Burial Number	Years BP
5	84-5a	3780 ± 180
19	84-12	5170 ± 220
34	84-27	3260 ± 200
39	84-31	4320 ± 220
41	84-33	4430 ± 170
46	84-36 (Orig. No.84-34c)	3370 ± 280
47	84-37	3140 ± 200
51	84-41	3940 ± 140
57	84-44	1420 ± 90
64	85-1	1710 ± 190
69	85-1a	4070 ± 150
73	85-4	1340 ± 150
85	85-12	1460 ± 130
90	85-17	3520 ± 170
111	85-36	3600 ± 160
112	85-37	3380 ± 150
113	85-38	3630 ± 140
126	86-10	3040 ± 60[a]
139	86-24	2620 ± 50[a]

[a] Conventional C-14 dates. All others are AMS dates. All dates are uncalibrated (from Carlson 1993; Carlson and Hobler 1993).

Table 8. Native Plant Food Resources Presently in the Vicinity of Pender Canal Sites

Common Name [a]	Taxonomic Name
Broadleaf Maple	*Acer macrophyllum*
Red Alder	*Alnus rubra*
Flowering Currant	*Ribes sanguineum*
Salal	*Gaultheria shallon*
Trailing Blackberry	*Rubus ursinus*
Red Huckleberry	*Vaccinium parvifolium*
Sweet Cicely	*Osmorhiza brevitas*
Wild Strawberry	*Fragaria bracteata*
Licorice Fern	*Polypodium glycyrrhiza*
Sword Fern	*Polystichum munitum*
Bracken Fern	*Pteridium aquilinum*
Clover	*Trifolium wormskjoldii*
Stinging Nettle	*Urtica dioica*

[a] Plants cited from Brooke (in Carlson 1985: Appendix II)

Table 9. Native Animal Food Resources from Pender Canal Sites

Common Name [a]	Taxonomic Name
Duck	Cygninae sp.
Herring	Clupeidae sp.
Sea Perch	Embiotocidae sp.
Rockfishes	*Sebastes* sp.
Salmonids	*Oncorhyncus* sp.
Sardine	Clupeidae sp.
Sharks and Rays	*Squalus* sp.
True Cods	Gadidae sp.
Deer/Wapiti	Cervidae sp.
Dog	*Canis* sp.
Seal	*Pinnipedia* sp.
Barnacle	*Balanus* sp.
Chiton	*Mopalia* sp.
Red Rock Crab	*Cancer productus*
Sea Urchin	*Strongylocentrotus* sp.
Blue Mussel	*Mytilus edulis*
Butter Clam	*Saxidomus giganteus*
Littleneck Clam	*Protothaca staminea*
Cockle	*Clinocardium nuttali*
Dogwinkles	*Nucella* sp.

[a] Faunal list taken from Hanson (1985, 1986, 1991).

Chapter V

Results and Discussion

Introduction

The results from both the skeletal and dietary analysis are provided and discussed in this chapter. The probable etiology of the anemia which caused the lesions is discussed by examining dietary evidence for the "nutritional" model and pathogenic information for the "environmental" model.

Skeletal Results

Of the 83 individuals in the sample, 60 (72.3%) were adult, 4 (4.8%) were 12-18, 5 (6%) were 0-6, and 14 (16.9%) were not aged because of poor preservation. Of the 60 adult crania, 17 appeared adult female, 24 adult male, and 19 were of indeterminate sex. The bias towards adult individuals in this sample can be explained by differences in preservation of orbits, that is, adults were more frequently extracted from the Pender Collection for this analysis because they had preserved orbits. It is not known whether this age bias exists in the Pender collection as a whole. A breakdown of the age and sex categories of the sample is presented in Table 10 and Figures 6 and 7. Appendix E provides a more detailed breakdown by specific age group.

The sample of 83 crania was examined for both the presence and severity of cribra orbitalia. Statistical analysis of the data is conditional because the indeterminate age and sex categories were excluded from the tests. A total of 31 individual crania (37.3%) was found to exhibit lesions with varying degrees of severity. In agreement with studies by other researchers, cribra orbitalia at Pender is usually symmetrical and bilateral in expression; three of the cases

(9.6%) had unilateral expression in comparison with 10% of cases in a study by Stuart-Macadam (1982), and 9.5% by Nathan and Haas (1966a). Of the three cases of unilateral expression, the left orbit was affected in two, and the right orbit was affected in one. The lesions are usually localized to the anterior area of the orbit. Cribra orbitalia occurred in 6 (7.2%) females, 5 (6%) males, 7 (8.4%) unsexed adults, 3 (3.6%) unsexed 6-18 year olds, 2 (2.4%) children, and 8 (9.6%) indeterminate sex and age groups. About 35% of the adult female group was affected, while only 21% of the adult male group was affected but this difference is not statistically significant (x^2=1.060; DF=1; P=.3032; Fisher's Exact P-Value =.4757). Within the 0-6 year old group 40% were affected and within the 6-18 year old group 75% were affected. The variability of occurrence among the 0-6 year olds, 6-18 year olds, and adults is not statistically significant (x^2=3.525; DF=2; P=.1716). The occurrence of cribra orbitalia in the total sample is reported by age group, sex and percentage of each group affected in Table 11 and Figures 6 and 7.

Within the four categories of severity, the porotic type (severity type 1) is by far the most frequent and the advanced trabecular type (severity type 4) the least frequent. The distribution by sex and age for each score of severity is given in Table 12. In the three cases of unilateral expression of cribra orbitalia, scores of 2 and 1 occurred in the left orbits of two adult males and a score of 3 occurred in the right orbit of one adult male. The small sample size for each score of severity precluded any tests for statistical significance by sex or age. Approximately 6 adult individuals exhibited what have been considered "healed" lesions by other researchers (Stuart-Macadam 1982; Webb 1989). Although these have not been included in the scoring, Appendix B indicates which individuals exhibit this type of lesion.

Table 10. Combined Age and Sex Categories of the Pender Canal Sample

Sex\Age	0-6 years	6-18 years	Adult	Indeterm.	Total
Male			24 (28.9%)		24 (28.9%)
Female			17 (20.5%)		17 (20.5%)
Indeterm.	5 (6%)	4 (4.8%)	19 (22.9%)	14 (16.9%)	42 (50.6%)
Total	5 (6%)	4 (4.8%)	60 (72.3%)	14 (16.9%)	83 (100%)

Table 11. Prevalence of Cribra Orbitalia in the Pender Canal Sample by Age Group and Sex

Age Group	Number of Affected Skulls	% of Group Affected
0-6 years	2/5	40
6-18 years	3/4	75
Adult females	6/17	35.3
Adult males	5/24	20.8
Adults of Indeterminate Sex	7/19	36.8
Indeterminate Age/ Indeterminate Sex	8/14	57.1
Total Sample	31/83	37.3

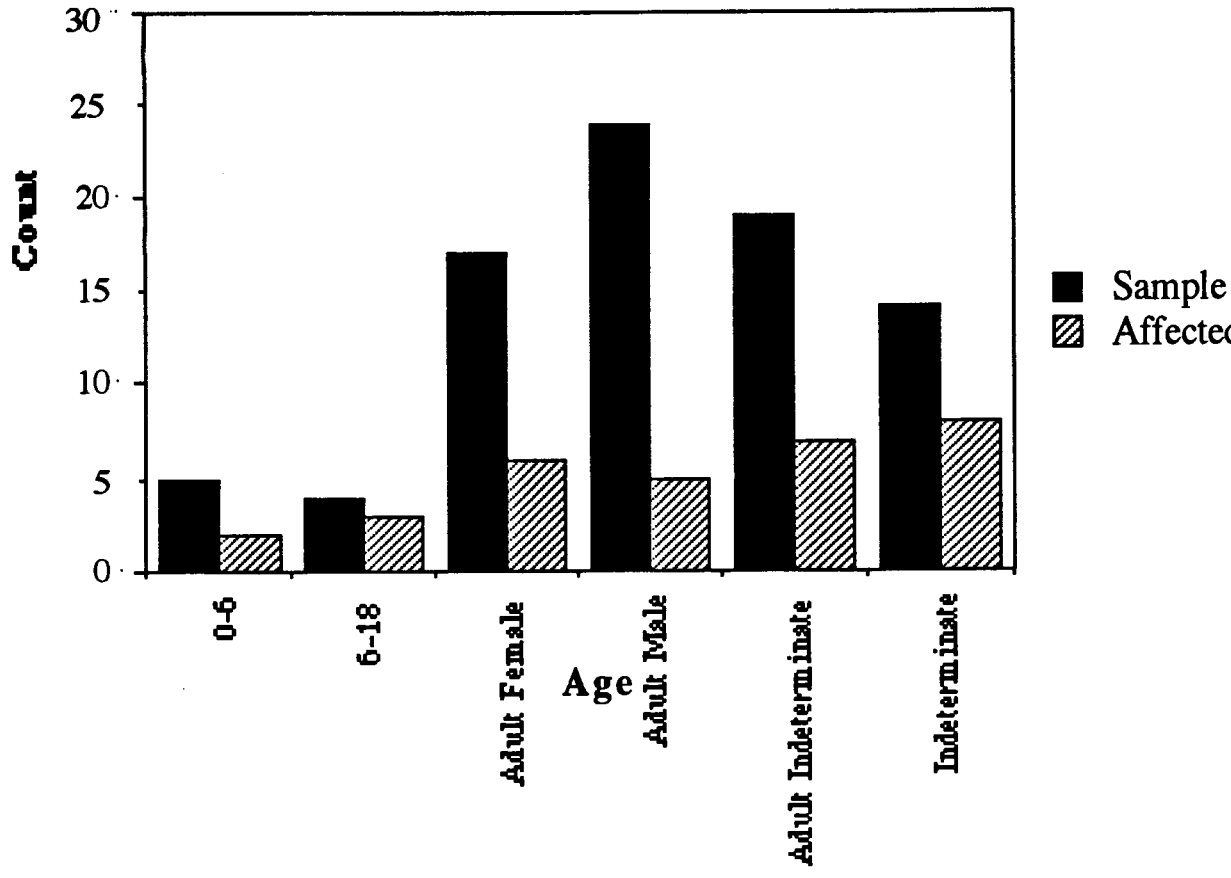

Figure 6. Distribution of Sample by Age Group.

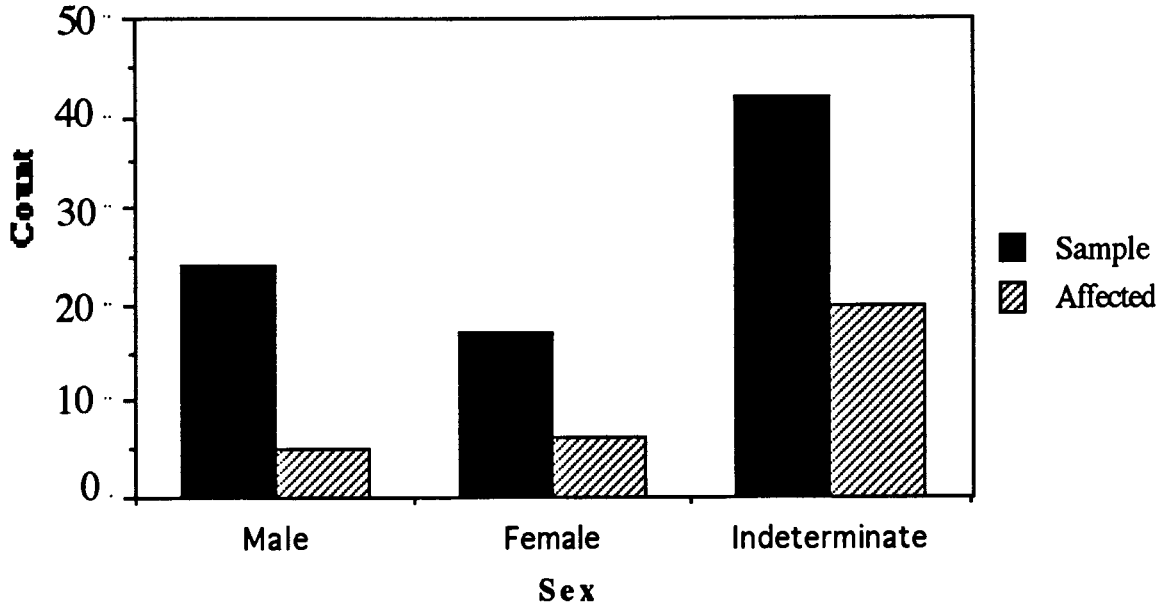

Figure 7. Distribution of Sample by Sex.

Table 12. Cribra Orbitalia Severity by Sex and Age

Severity Score	Adult Females	Adult Males	Indet. Adults	0-6 Years	6 - 18 Years	Indet.	Total for Each Type
1	5	2	7	1	0	5	20
2	1	1	0	1	1	3	7
3	0	2	0	0	1	0	3
4	0	0	0	0	1	0	1
Total	6	5	7	2	3	8	31

Spatial and Temporal Distribution

The temporal distribution of the sample ranges from about 5,000 to 400 years BP, with the largest groups dating between 3,000 and 4,000 years BP and 1,000 and 2,000 years BP. Table 13 indicates the temporal distribution of the sample, as well as the relative frequencies for the occurrence of cribra orbitalia within and between each time period. The frequency of affected individuals is fairly high in almost all the time periods, with the most frequently affected group in the sample dating between 3,000 and 4,000 years BP. From 2,000 to 3,000 years BP there appears to be a marked decrease in occurrence of cribra orbitalia, but this difference is not statistically significant (x^2=8.918; DF=5; P=.1124). The small number of affected individuals may be related to the smaller sample size within this time period, or it could be due to sampling error. There may not be a random distribution of representative individuals within each of the thousand year time periods in the sample. The large number of individuals not assigned to a time period, as well as the small sample size within each time period makes any inferences about frequency changes through time extremely difficult. The temporal distribution of cribra orbitalia does indicate, however,

that a high frequency of cribra orbitalia goes back quite far in time. A high prevalence can be found on the coast for the last 4,000 years and perhaps further back as well. With only a small number of individuals dated earlier than 4,000 BP, any inferences regarding a greater time depth for the high occurrence of cribra orbitalia would be speculative.

Table 13. Temporal Distribution of Individuals at Pender Canal

Time Period	Number of Crania Affected	% Affected in Time Period	% of Affected Sample
0-1000 BP	0/1	0	0
1000-2000 BP	11/21	52	35
2000-3000 BP	1/12	8	3
3000-4000 BP	13/36	36	42
4000-5000 BP	2/4	50	6
5000-6000 BP	1/1	100	3
Unknown	3/8	38	10

The spatial distribution of individuals with cribra orbitalia is extremely varied. Figure 8 indicates the approximate location of individuals with cribra orbitalia. There is no indication of a particular pattern of burying affected individuals.

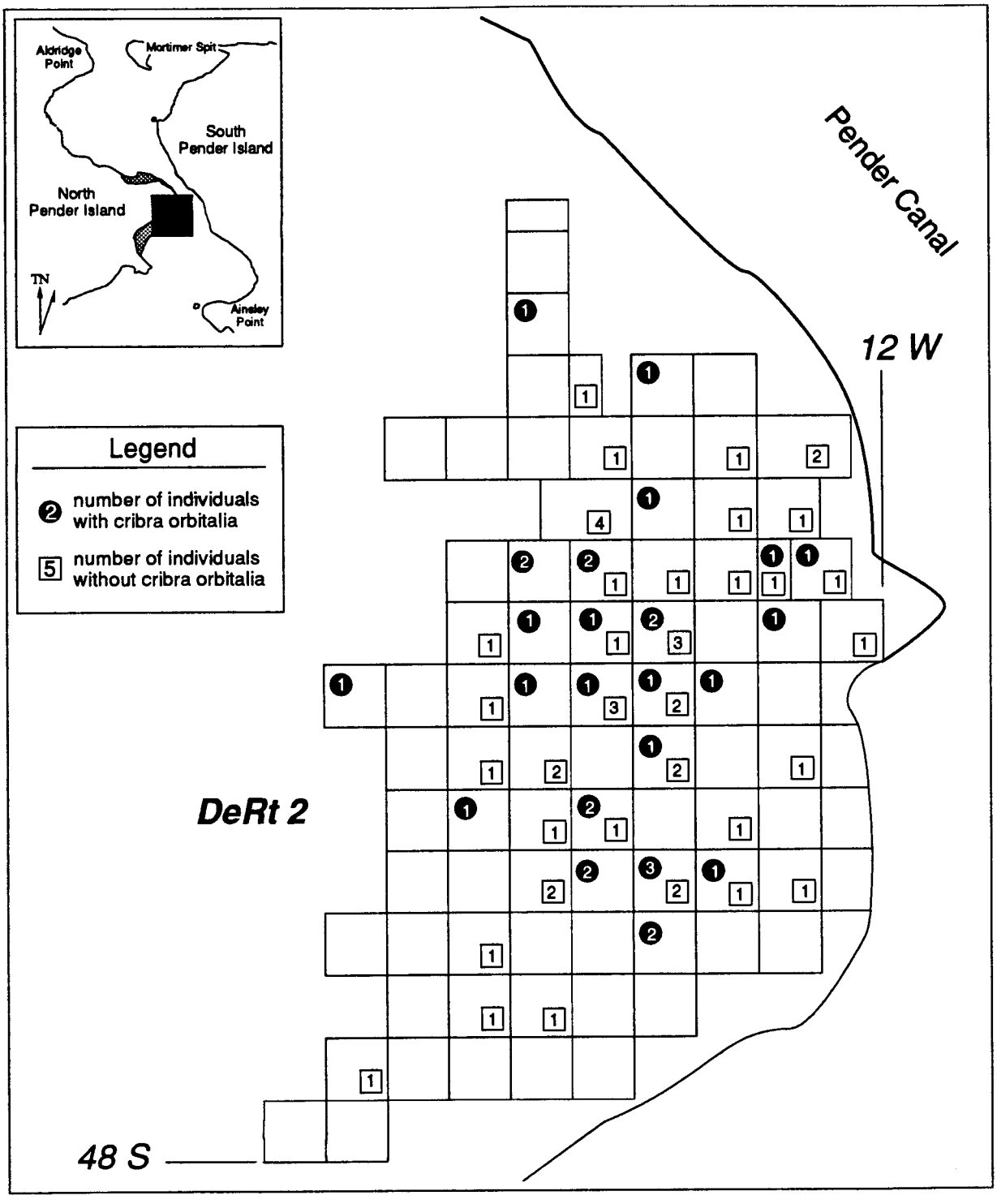

Figure 8. Spatial Distribution of Individuals.

Evaluation of Potential Causes of Cribra Orbitalia in the Pender Population

Dietary Iron

As outlined in Chapter 2, the critical issue relating to dietary iron status is not the amount of iron ingested, but rather, the "bioavailability" of iron. "Bioavailability" refers to the proportion of the nutrient that can be absorbed and used or stored (Holland *et al.* 1991:15). The bioavailability of iron is determined by the type of iron, the presence of inhibiting or enhancing agents in the meal, and the iron status of the individual. Heme iron has the highest bioavailability since it is relatively unaffected by other components of the diet and is absorbed at a higher rate. The absorption of heme iron is somewhat dependent on the iron status of the individual; an individual with no iron stores can absorb approximately 20% more dietary heme iron than an individual with sufficient stores (Monsen 1981:321; Monsen and Balintfy 1982:307).

Sources of heme iron in the Pender Canal diet would have been abundant. Shellfish, fish, and animal tissues and organs all contain heme iron. In addition to being a good source of dietary iron, these foods would also have increased dietary absorption of iron from non-heme sources.

A stable carbon isotopic analysis of individuals from the Gulf Islands, including 32 individuals from the Pender sample, indicates that approximately 93% of the protein in the diet was obtained from marine resources (Chisholm 1986: 123). An earlier analysis using stable carbon isotopes found this same dietary pattern for marine protein intake to be in place on the coast for the last 5,000 years (Chisholm 1986; Chisholm *et al.* 1983). Ethnographic descriptions of subsistence along the coast at the time of European contact also provide detail

on the types of foods and the relative proportion of marine foods in the diet. Folan (1984) examined the frequency of food items mentioned in the journals of John Jewitt during his captivity by the Nootka and estimated a marine protein proportion in line with that of Chisholm *et al.* (1983). Since archaeological and stable isotopic analysis indicates that the Pender inhabitants were fully maritime adapted with the majority of their protein obtained from marine resources, it seems reasonable to identify heme iron as a significant contributor of total dietary iron.

Rich sources of non-heme iron available to the inhabitants would likely have included berries, nettles, dried fruit, inner tree bark, clover roots, silverweed roots, fern roots, camas, and seaweed. The enhancing effects of ascorbic acid in berries would have increased the absorption of iron from non-heme dietary sources. There appears to be few inhibitors of non-heme iron absorption, such as the tannins or phytates which adversely affect the agriculturalist's diet.

In terms of seasonal availability, fish and shellfish would have been available throughout the year. The mild climate in the Pacific Northwest meant that fresh plant foods could also be obtained almost year-round (Rivera 1949). Plant utilization probably varied seasonally, being higher in the summer and spring when fresh fruits and greens were available. Much of the diet could be stored for year-round use, with the exception of stalks or shoots which were probably eaten fresh when available in the spring. Roots and rhizomes were probably cleaned, cooked, and dried for the winter (Turner 1975). The prehistoric technique of preserving plants has been shown as an efficient method of preserving essential nutrients, principally iron and Vitamin C (Norton 1981; Norton *et al.* 1984). Dried berries are a particularly high in ascorbic acid (Norton *et al.* 1984).

Through an analysis of the types of foods available to the Pender inhabitants, the iron content, and the dietary bioavailability of iron, it is more than probable that the recommended daily requirements for iron could be met through diet. Approximately 5-10 clams per day would have provided an adequate supply of dietary iron (from Monsen 1981). The large amount of heme-rich foods in the diet provided not only a good dietary supply with a much higher absorption rate, but also served to enhance the absorption of dietary non-heme iron. According to FAO/WHO (1988) criteria, the Pender diet would be classified as a "high bioavailability" diet with an absorption rate of approximately 15%. An added dietary source of iron at Pender could have come from the highly mineralized groundwater.

Other Dietary Nutrients Required for Prevention of Anemia

Other necessary nutrients essential to prevent anemia include Vitamin A, Vitamin B_{12}, and Folic Acid. As mentioned in Chapter 2, a deficiency of any one of these nutrients may indirectly cause anemia through impaired red blood cell formation, or iron absorption. In order to determine if inadequate nutrition was a potential cause of anemia at Pender it is necessary to examine the possibility that any of these nutrients could have been absent, or deficient in the diet.

Vitamin A is found in high amounts in traditional Salish foods, and so it is reasonable to assume that it was also high in the prehistoric Pender diet. Green vegetables can be expected to be high in carotene (Kunhlein and Turner 1986; 1991). Bracken fern leaves, wild rosehips, fireweed, and stinging nettle are all very high in Vitamin A. Eulachen grease, an important dietary item to historic Northwest Coast groups, is an extremely good source of Vitamin A. A study done by Kuhnlein *et al.* (1982:158), indicates it would take less than 50 g (2.5-

3.5 tablespoons) to meet the adult daily needs for Vitamin A. According to ethnohistoric descriptions, the Salish and other Northwest Coast groups ate eulachen grease or sea mammal oil with most things in their diet, including berries, fish, and greens (Barnett 1955; MacIlwraith 1948; Suttles 1968).

An analysis of the Vitamin B_{12} content in the foods utilized by the Pender inhabitants suggests that this vitamin was plentiful. Meat, fish and shellfish are rich sources of Vitamin B_{12}. With a diet consisting largely of marine foods, it seems highly unlikely that there would have been a dietary deficiency of Vitamin B_{12}.

Dietary sources of folic acid appear to have been widely available to the prehistoric inhabitants at Pender. Data on the occurrence of folate in foods is severely lacking in the literature, however, folate is generally abundant in many of the same foods which contain ascorbic acid (Clark *et al.* 1992). Green leafy vegetables, fruits, root vegetables, salmon, and organ meats are good sources for this nutrient (Douglas 1993; Kuhnlein and Turner 1987; Tsui and Nordstrom 1990). Folate may be destroyed by cooking, especially boiling, so the amount that is absorbed is difficult to quantify (Wintrobe 1974). Clinical research suggests that persons can subsist on diets with suboptimal levels of folate without ill effects (Wintrobe 1974). A deficiency of folic acid in pregnancy has been linked to stillbirth, miscarriage, as well as fetal malformation resulting in *spina bifida* and *anencephaly* in newborns (Douglas 1993; WHO 1972). One would expect to find a high prevalence of these birth defects in the sample if such a deficiency was endemic, however, this does not appear to be the case. Dietary sources of folic acid appear to have been highly available in the food supply of the inhabitants so it appears unlikely that there was a dietary deficiency of this nutrient.

The Pender diet probably provided sufficient nutrients for growth and development. The Pender natives, like the historic Coast Salish, probably used multiple resources to provide themselves with a varied and ample diet throughout the year. The mainstays of traditional Coast Salish diet were fish, shellfish, meat, root crops, tubers, and berries (Rivera 1949). All of these foods would have been prepared for storage. Traditional methods of processing and storage have been shown to provide highly nutritious foods (Keely *et al.* 1982; Kuhnlein *et al.* 1982; Norton *et al.* 1984). From all indications, Northwest Coast traditional diet was healthy (Kennelly 1986, Kuhnlein *et al.* 1982; Kuhnlein and Moody 1989; Norton *et al.* 1984; Nuxalk Food and Nutrition Program 1984; Rivera 1949; Turner 1981). In fact, many researchers have advocated a return to traditional native foods for their superior nutritional value (Kennelly 1986; Kuhnlein and Turner 1991; Kuhnlein *et al.* 1982; Nuxalk Food and Nutrition Program 1984; Turner 1981; Turner and Kuhnlein 1982).

Dietary Factors Affecting Children

An important aspect of nutrition that needs to be addressed is the diet of children. Clinical evidence suggests that cribra orbitalia is formed during a childhood episode of anemia. The nutritional status of children may be affected by prematurity, prolonged exclusive breastfeeding, multiparity, food taboos, and food shortages.

In premature infants there is often insufficient fetal stores of iron. It is possible that this may have been a factor in the occurrence of cribra orbitalia in the Pender skeletal sample. However, the large number of affected individuals would suggest that premature births would have had to have been in the order of 37% which seems unlikely.

Prolonged exclusive breast feeding can lead to an iron deficiency anemia. After about 6 months of age, an infant requires dietary supplementation because breast milk alone no longer supplies sufficient iron for growth (Pochedly and May 1987; Wadsworth 1992; Zitelli 1987). It is unlikely that anemia would develop until after about 6 months of age because full term infants generally have sufficient iron stores from birth (Dallman 1987; Wadsworth 1992). Up until this time, breast milk supplies sufficient amounts of iron (Pochedly and May 1987; Saarinen *et al.* 1977; Schneider and Worthington-Roberts 1981). Breast milk, although low in iron, has an extremely high absorption rate with an average of approximately 49% (Committee on Nutrition 1976:765). The period of increased need after 6 months of age usually coincides with dietary supplementation of mashed or watery solid foods, although breast feeding may continue for some time. The high bioavailability of iron from breast milk drops after the introduction of solid foods (Brock 1980; Committee on Nutrition 1979)

There is no archaeological evidence for the length of breast feeding, nor the types of weaning foods introduced. Ethnohistoric evidence suggests that supplementation of the diet occurred quite early although breast feeding of children may have continued for some time after. McIlwraith (1948) says the Bella Coola added soft pounded salmon and water to an infant's diet as soon as possible although breast feeding may have continued until a child was two or three years of age. Barnett (1955:130) states that Coast Salish newborns were given a clam to suck on shortly after birth, which suggests that infants were introduced to soft adult foods very early in life. A child was given soft solid foods such as duck when it began to sit up although nursing continued until the baby walked or later (Suttles 1974:497). Eells (1985:296) says infants from Puget Sound were nursed until they were three years or older. The types of foods a child was weaned on are unknown, although they probably were not

severely different from adult foods. They would have been chewed up or watery.

Successive pregnancies may drain a woman's iron, leading to anemia. The influence of the maternal iron status on the iron status of the fetus is controversial (Wadsworth 1992). Low maternal iron stores can influence the iron stores of the newborn (King *et al.* 1987), however, adequate iron levels have been found in infants born to anemic mothers (Krawinkel *et al.* 1990). Multiparity may have been a factor in the iron deficiency of infants at Pender, however it seems unlikely because of the high bioavailability of iron in the diet. The increased iron demands of pregnancy, and blood loss at childbirth would have been compensated through an increase in dietary absorption from the diet. The relationship between maternal iron stores and iron content of breast milk is not clear either (Schneider and Worthington-Roberts 1981). The general consensus appears to be that maternal nutritional state is independent of the nutritional quality of the milk, except in cases of severe maternal malnourishment (Jelliffe and Jelliffe 1978; Schneider and Worthington-Roberts 1981; Worthington-Roberts *et al.* 1985).

Occasional shortages of food, or episodes of food avoidance probably would not have had a significant impact on the iron status of the Pender Island individuals. Suttles (1968) suggested that episodes of food scarcity may have existed along the coast prehistorically, but this is controversial (Boyd 1990). The efficiency of iron metabolism indicates that such an episode would have to border on starvation for a prolonged period of time in order to cause an iron deficiency in adults. It is probable, however, that food shortages would have had a significant impact on the health of children. If food fluctuations did lead to iron deficiency anemia in the population we would expect to find temporal changes in the prevalence of cribra orbitalia. However, the occurrence is fairly

constant, with no statistically significant differences between time periods. The wide diversity of foods available throughout the year make it unlikely that starvation would have been a major cause of iron deficiency anemia. The so-called "starvation" periods referred to historically may actually refer to shortage of preferred resources (Boyd 1990; Rivera 1949). Rivera (1949:20) says the historic Salish of Puget Sound said they were "starving" when forced to subsist solely on stored foods in winter during episodes of poor weather.

Cultural factors, such as food taboos or limited access to foods by certain individuals would have influenced the iron status of an individual. Although ethnographic sources indicate cultural food taboos existed in most Northwest Coast groups during certain ritual periods of an individual's life, there is no mention that they adversely affected the health of a child or adult (Boas 1966; MacIlwraith 1948; Suttles 1974). In addition, similar stable isotope results between individuals indicates that restricted social access to foods, if it did exist, did not seem to have had any long-term effect on the individual. All individuals show a diet high in marine foods suggesting that there was equal access to iron-rich sources. Lazenby and McCormack (1985) have suggested that differences in $\partial\ ^{13}C$ values between adults and children on the Northwest Coast may be due to a recognition of the toxic effects of hypervitaminosis D associated with high salmon consumption. However, even with reduced salmon intake, the other components of the diet should still have provided sufficient vitamins and minerals to prevent iron deficiency anemia in children and pregnant women. Of the three infants analysed from the Pender Canal skeletal collection, one 2-4 year old was found to be 1% heavier isotopically than the female average (Chisholm 1986:107). This actually indicates a heavier reliance on marine foods. Chisholm suggests the value may indicate that the child has not yet been

weaned, since breast milk may have a heavier isotopic value in relation to the mother's isotopic value.

Although blood loss can be a major source of iron loss and was a potential factor in Northwest Coast populations, it cannot be considered a major factor in the children or adults at Pender. Severe acute blood loss would cause death, and chronic blood loss (with the exception of severe parasitic infections) should be compensated for by increased dietary absorption of iron.

Environmental Anemia

According to Stuart-Macadam (1992b) and Kent (1992) the flexibility of iron metabolism, clinical studies on iron deficiency anemia, the hypoferremic response to pathogens, and the archaeological distribution of cribra orbitalia worldwide suggests that the past prevalence of porotic hyperostosis is directly related to the total pathogen load of a population. The incidence of parasitic, infectious, viral and fungal diseases increases with a heavy pathogen load. Parasitic infestations, bacterial infections, and chronic diseases such as tuberculosis and osteomyelitis occurred in the past, and are present day problems in many areas today. Stuart-Macadam (1988, 1991, 1992a,1992b) postulates that an "anemia of chronic disease", rather than an "iron deficiency anemia" was common in the past contributing to a lowered iron status among populations exposed to high pathogen loads. With continual and repeated exposure to pathogens there is an increased risk that the lowered iron status could lead ultimately to an iron deficiency anemia. Children and women are particularly vulnerable because of increased physiological requirements.

The total pathogen load can be seen as a function of several environmental and cultural factors including: climate, topography, demography, hygiene, food, seasonality, customs, and economy (Stuart-Macadam 1992b). An examination

of environmental and social factors that are indicated at Pender archaeologically suggests that it is highly probable that the prehistoric inhabitants of Pender were exposed to a high number of pathogens, including viral, protozoal and helminthic bacteria.

Although the severe impact of Old World crowd diseases on native peoples on the Northwest Coast is well known (Boyd 1990), very little is known about indigenous New World diseases. Newman (1976) suggests that the native peoples had their own share of infectious communicable diseases prior to contact. It is likely that native immunity against such infective agents was very effective as well (Newman 1976). According to Newman (1976:669) probable diseases of the New World include: bacillary and amoebic dysentery; viral influenza and pneumonia; various arthritides; viral fevers; roundworms; non-venereal syphilis; bacterial pathogens such as streptococcus and staphylococcus; salmonella and other food poisoning agents.

Winter and possibly year-round occupation at Pender would have provided ideal conditions for pathogenic growth. Sedentism and aggregation promote the maintenance and transmission of pathogenic organisms through person-to-person contact and inadequate sanitation (Kent 1986, 1992; Kent and Dunn 1993; Schmidt and Roberts 1981; Stuart-Macadam 1992b). Sedentism with inadequate sanitation results in fecal mound buildup, which not only promotes pathogenic survival, but further maintains human populations in chronic contact with pathogens (Schmidt and Roberts 1981). Moist soils, such as those in the Pacific Northwest would be particularly conducive to pathogenic growth by sustaining the pathogen while between hosts (Schmidt and Roberts 1981).

Many cultural practices of the prehistoric inhabitants probably aided in the maintenance of environmental pathogens. Storage of foods has a tendency to attract arthropods and rodents which promote the life cycle of pathogens

(Schmidt and Roberts 1981; WHO 1985). Stored fish, grease, and berries, and other foods would, no doubt, have attracted pathogens as well as carriers of pathogens.

Cultural food preferences can also be a cause of pathogenic infection. Jewitt (1976:118) expressed extreme distate at the Nootka preference for putrid, rotting fish and whale meat. Such dietary foods would increase the risk of bacterial infection. Raw or inadequate cooking of infected seafood is a common cause of parasitic and pathogenic infection in adults (Ensminger *et al.* 1994). Inadequately cooked or raw fish may contain parasites such as tapeworm which can infect humans. Unhygienic conditions in food preparation and storage can be a factor in the transmission of such organisms as *Escherichia coli, Staphylococcus aureus, and Salmonella typhimurium* (Ensminger *et al.* 1994). Ethnographic sources indicate that the Nootka usually ate their food with fingers from communal trays or troughs (Eells 1985; Jewitt 1976; King in Cook 1967: Appendix III). This method of food consumption certainly would have increased the risk of spreading infection within a household.

Certain environmental hazards also pose pathogenic risks to human health. Paralytic shellfish poisoning, for example, occurs in mussels and clams on the Northwest Coast regularly as a result of red tide. The toxins in shellfish can cause reactions ranging from minor illness to respiratory failure (Dale and Yentsch 1978). There is some evidence to suggest that individuals who habitually eat shellfish with low levels of toxin can build up an immunity (Dale and Yentsch 1978). The extent to which paralytic shellfish poisoning affected the health of Northwest Coast natives is unknown, however, it is more than probable that they were aware of its effects and avoided shellfish during those times.

Drinking water may be a source of chronic infection. The danger of contaminating fresh water sources increases with sedentism or aggregation.

Water may be contaminated with giardia by animal or human fecal material. Other free living organisms that may occur in water supplies include fungi, algae, protozoa, and nematodes (WHO 1984).

Human social and behavioural patterns play an important role in the epidemiology of infection. Historically, the most common Coastal Salish house was a large shed-roof type which was occupied by several related families (Jenness n.d.; Suttles 1990). The close contact between co-habitating individuals would have aided in the transmission of pathogens. The Coast Salish practice of going barefoot (Drucker 1965; Eells 1985; Suttles 1990), probably predisposed those individuals to infection through cuts and abrasions. Domestic animals such as dogs also act as vectors for disease. Dogs were present at Pender and probably contributed to the transmission of pathogens. Some parasites that are maintained by invertebrate or vertebrate hosts other than humans can be transmitted to humans. The resultant diseases are called zoonoses (Schmidt and Roberts 1985). Dogs are a good example of carriers of such organisms; the zoonosis hydatid disease occurs in humans when they become accidentally infected with larval tapeworms through ingestion of eggs in dog feces (Schmidt and Roberts 1985). Dogs are also carriers of fleas, lice, and mites, which readily become companions of humans as well. Aside from being important pests, fleas and lice have been vectors for some of the worst diseases in history, including the plague and typhus (Schmidt and Roberts 1985). It seems probable that fleas, lice, and flies were vectors for at least minor infections in the New World as well.

Infants are particularly susceptible to infection at the time of weaning because of new exposure to pathogens (Stuart-Macadam 1992). Supplementary feeding interferes with the beneficial effects of exclusive breastfeeding. Newborns are immunologically immature and less resistant to infection than

adults (Marks 1985). While nursing, human milk confers a protective effect against invasive organisms (Griffiths and Bullen 1987; Worthington-Roberts *et al.* 1985). Anti-infectious agents in human milk and colostrum include, lactoferrin, bifidus factor, lactoperoxidase, and various immunoglobins (Worthington-Roberts *et al.* 1985). Breast milk contains large amounts of unsaturated binders for iron, folic acid, and vitamin B_{12} (Schneider and Worthington-Roberts 1981). Lactoferrin, an iron binding protein, similar to transferrin, inhibits growth of several pathogens, including *Escherichia coli* and staphylococcus by depriving them of iron (Brock 1980; Bullen 1981a, 1981b, Bullen *et al.* 1972; Oram and Reiter 1968; Schneider and Worthington-Roberts 1981; Worthington-Roberts *et al.* 1985). The result is increased immunity. *Staphylococcus aureus* colonizes many babies in the first two weeks of life without ill effects. With increasing exposure to pathogens, and loss of immunity from mother's milk, children become immunologically vulnerable to infection. Bacterial infections are common in many children today (Friedman 1987; Marks 1985), and probably were in the past, as well. Streptococcus and staphylococcus infection can be introduced through contaminated food or water droplets (Zitelli *et al.* 1987). Superficial skin infections occur with relatively high frequency in childhood when organisms enter through a wound, cut, abrasion, or insect bite (Zitelli *et al.* 1987). Close contact between mother and infant can favour the transmission of pathogens.

Introduction of supplementary foods can be a source of infection which may affect the utilization of iron (Wadsworth 1992). Both weaning foods and containers can be contaminated (Raphael 1984). Weaning foods often contain high amounts of water which may not only be a good source of iron, but also a good source of infection. A high level of iron in water serves as a nutrient for some organisms, enhancing their virulence (Weinberg 1989; 1992a). When

infants are weaned from sterile milk to food and water contaminated with microorganisms they can contract weanling diarrhea which can lead to anemia (Gordon *et al.* 1963).

With the introduction of supplementary foods and concomitant pathogens, it would be advantageous for an infant to have lowered iron levels (Stuart-Macadam 1992b). In fact, despite a diet adequate in iron, there is some evidence to suggest that many, if not all, infants experience a temporary "physiological iron lack" in the first two years of life (Beal *et al.* 1962; Burman 1972; Stuart-Macadam 1992b; Wadsworth 1992; WHO 1972). In a World Health Organization study (WHO 1972), a group of infants and young children treated for anemia through dietary fortification had attained normal hemoglobin levels by the age of 2 to 3 years. However, anemia also disappeared by the same age in a group of children whose diet was not fortified (WHO 1972). This suggests that the low hemoglobin levels in early childhood are only temporary and physiological and have little to do with a dietary deficiency of iron (Wadsworth 1992). Stuart-Macadam (1992b), suggests that the "physiological anemia" that occurs in infants may actually be an evolutionary advantage. If this hypothesis is accepted, then lowered iron levels in infants is not a pathological disease but a physiological defense.

The large number of adults with cribra orbitalia suggests a childhood episode of anemia did not necessarily lead to death. Whether there was a high mortality rate in Pender infants from anemia is unknown, since the small number in the sample, as well as conditions of preservation, preclude such an observation. The infants and children who exhibit cribra orbitalia skeletally may have just as likely succumbed to the pathogenic infection as the anemic condition.

There is evidence of increased prevalence of infection in breast-fed infants with the onset of weaning (Bullen 1981b). Exclusive breastfeeding of newborns

at Pender would have conferred immunity against pathogens. However, it is very likely that as the introduction of solid foods increased there was a loss of immunity against these pathogens. As infants increase in age, they begin to explore the world around them, thus exposing themselves to new pathogens. Infants and young children have low iron stores to begin with, and the non-specific immune response of decreasing intestinal absorption, and/or associated weanling diarrhea would have put them at risk for anemia. The sample from Pender Island suggests a high prevalence of infection within the 0-6 and 6-18 years of age categories, although this is not statistically significant. The youngest age of occurrence in the sample is approximately 4 years old which may correspond with the age at which the child was most susceptible to anemia due to a combination of increased pathogenic invasion, and high iron demands for growth.

A cultural group successfully maintaining itself in an environment through many generations can be assumed to have had access to adequate food and nutrition (Kuhnlein 1984). The Pender diet appears to have been nutritionally adequate, yet anemia still appears to have been fairly high. Since only one half to two-thirds of clinical patients with anemia have actually been found to exhibit bony changes radiographically (Stuart-Macadam 1982:235) the number of individuals with anemia in the Pender population is probably under-represented by the skeletal lesions. Taking this into consideration, it is probably safe to assume that at least one half of the population at the Pender Island sites would have been anemic at some point in their lives. This suggests that lowered iron levels may have been beneficial to the inhabitants. Since the environmental conditions would have been more or less constant, it is probable that lowered circulating iron levels persisted throughout the life of the individual with iron

deficiency anemia developing only in individuals with precariously low iron stores and high physiological demands.

Chapter VI

Summary and Conclusions

The Pender Canal skeletal sample was studied to investigate the relationship between cribra orbitalia and nutritional deficiencies, and cribra orbitalia and environmental pathogens. The objective of this study was to determine the nutritional adequacy of the Pender Canal diet in terms of iron bioavailability, and to assess the pathogen load of the population. This was achieved by integrating dietary, ethnographic, archaeological, and skeletal data.

This research has shown: 1) that cribra orbitalia occurs at a relatively high frequency (37%) and appears to occur independent of age, sex, and time period; 2) that prehistoric Pender diet was diverse and abundant with a particularly heavy reliance on iron-rich marine foods; 3) that the Pender diet was probably nutritionally adequate and contained sufficient vitamins and minerals to prevent anemia; and, 4) that many of these individuals lived through an episode of childhood anemia, as evident in the large number of adults with the skeletal pathology.

Iron withholding as a defense against infection is a more probable cause of anemia at Pender Canal. Children in the first few years of life are particularly prone to an "anemia of infection" because of new exposure to pathogens at time of weaning. Children have low iron stores and high physiological demands after the age of 6 months which puts them at a high risk for anemia if subjected to a high number of pathogens. A combined hypoferremic (iron deficient) response and/or weanling diarrhea probably resulted in reducing dietary iron absorption. An individual who is hypoferremic in an area where there is a heavy or chronic pathogen load will have an advantage over an iron replete individual (Kent and Weinberg 1989; Stuart-Macadam 1988, 1991, 1992b, n.d.). Because pathogens

are dependent upon iron, lowered iron levels would be advantageous to the host and disadvantageous to the invader. This is not to suggest that anemia, *per se* is advantageous, for in many cases a chronic or severe anemia can be fatal. However, in areas of heavy pathogen loads, the benefits of lowered iron levels may outweigh the possible risks of anemia (Stuart-Macadam n.d.). The large number of adults in the Pender sample with cribra orbitalia suggests that many children were living through this initial episode of anemia. On the other hand, the small number of infants and children in the sample is not necessarily indicative of low infant mortality. Conditions of preservation or cultural burial customs may be responsible for the small number of infants and young children in the sample.

Research by Cybulski (1977, 1990) indicates that there was a higher frequency of cribra orbitalia in skeletal remains from the Gulf of Georgia region and the Queen Charlotte Islands than in other areas of the coast in both the historic and prehistoric periods. Approximately 31% of the skulls in the Strait of Georgia affected by cribra orbitalia in the prehistoric period (Cybulski 1990:58). The results of this analysis supports Cybulski's findings in terms of high regional frequency of cribra orbitalia. Age related occurrence was found to be similar to Cybulski's (1977) findings in the historic period as well, with the highest occurrence within the 6-18 year age category. This thesis research also indicates that a high prevalence of cribra orbitalia has considerable time depth in the Gulf of Georgia region. Approximately 37% of the 40 skulls dating between 5000 and 3000 years ago from the Pender Canal sample are affected by cribra orbitalia. Cybulski (1990, 1994) has suggested that since diet, culture and environment were very similar throughout the Northwest Coast, population size, density and sanitation may account for the regional differences in occurrence of cribra orbitalia.

The differences in regional frequency could be a reflection of differences in pathogen load as a result of high population levels, or different environmental conditions. Alternatively, the differences could reflect inadequate sampling of Northwest Coast skeletal populations. An analysis of diet, environment, and cultural items at Pender suggests a higher pathogen load in the Gulf of Georgia region may be due to increasing population density, and aggregation or sedentism with inadequate sanitation. Boyd (1990:146) estimates that the pre-contact population of the Central and South Coast Salish was approximately 30,000. A more reliable resource base may have allowed a higher population in these areas than in others on the coast (Boyd 1990). Archaeological evidence from Pender indicates that there was an increase in the frequency of luxury goods, for example, labrets, beginning about 5,000 years ago (Carlson and Hobler 1993). This may suggest that the Gulf Islands were a center for the early development of the Northwest Coast cultural complexes sometime between 4,000 and 2,500 years ago (Carlson 1993). The period between 3,000 and 4,000 years ago initiates this trend with the highest overall frequency of cribra orbitalia. This period coincides with the presence of complex art which may reflect increasingly complex social hierarchies (Carlson 1991). These cultural changes may have contributed to population growth and greater aggregations. This would have had a positive effect on growth and transmission of pathogens. A change in archaeological items at Pender after 2200 BP suggests that the site may have been used as a seasonal campsite, rather than a village (Carlson 1993b). Although not statistically significant, a lower prevalence of cribra orbitalia in the Pender Canal sample between 3,000 and 2,000 years ago may indicate a decreased pathogen load due to decreasing sedentism and aggregation at the site. This explanation, however, does not adequately explain the increased prevalence which once again occurs between 2000 and 1000 years ago. There is no

archaeological evidence for an increase in site usage after 2200 BP. Alternatively, as mentioned in Chapter 5, the low occurrence in the period between 3,000 and 2,000 years BP could be the result of a non-random distribution of individuals within this time period.

Regional differences in the frequency of cribra orbitalia could be due to different levels of environmental pathogens in the water. High levels of iron in drinking water can have two different effects: it can be a source of dietary iron; and, it can provide a natural breeding ground for pathogenic organisms (Weinberg 1989, 1992a). It is possible that a higher pathogen load at Pender Canal and Blue Jackets Creek is a direct result of high iron levels in drinking water. In order to test this hypothesis it would be necessary to identify the source of drinking water and the iron content from various archaeological sites on the coast to see how this relates to the prevalence of cribra orbitalia within skeletal populations.

Differences in frequency by region may also be caused by inadequate sampling. Cribra orbitalia may appear to be high in the Gulf of Georgia region, however this may only be a reflection of the sample size. Further studies of skeletal remains from other regions on the coast may find that high frequencies of cribra orbitalia are not specific to the Queen Charlotte Islands, or the Gulf of Georgia region.

This study illustrates the necessity of considering numerous causes of anemia as well as the importance of understanding that diet may not be the cause of cribra orbitalia in a skeletal population. Recent medical and anthropological studies suggest that diet is probably not a major factor in the development of iron deficiency anemia (Cook 1990; Fairbanks and Beutler 1988; Weinberg 1974, 1992a). Research suggests that even groups consuming iron-rich diets suffer from iron deficiency anemia (Wadsworth 1975). It has been suggested

that we must include infectious disease as a possible factor when interpreting cribra orbitalia (Cybulski 1994; Hengen 1971; Kent 1986, 1992; Stuart-Macadam 1992a, 1992b). Dietary, hygienic, cultural and environmental factors need to be investigated prior to determining the cause of cribra orbitalia in skeletal material. It is possible that individuals with the pathology may have acquired an iron deficiency as part of their adjustment to the chronic pathogen load in their environment (Hengen 1971; Stuart-Macadam 1992a, 1992b; Kent 1986, 1992). Iron deficiency anemia may ensue as the body tries to maintain a balance between lowered iron absorption and greater physiological demands for iron. Individuals with low or absent iron stores are at high risk of developing anemia under a high pathogen load.

This thesis research supports the "environmental" anemia model for the occurrence of cribra orbitalia in the Pender population. Cribra orbitalia at Pender may be more closely related to factors associated with increased aggregation of people and sedentism, rather than dietary subsistence. If lowered iron levels confer immunity against infection, then their occurrence in certain environmental situations could be seen as adaptive rather than maladaptive (Stuart-Macadam 1991, 1992a). If this non-traditional perspective is accepted, then the high occurrence of cribra orbitalia at Pender Island should be interpreted as a healthy, adaptive response to environmental stress, rather than as an indicator of nutritional stress.

Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Angel (1971)	Catal Huyuk - Konya Plain Early Neolithic			-	41			143	41*	malaria at Catal Huyuk (pop. 5000) may have been responsible for high occurrence
Angel (1971)	Nea Nikomedeia, Macedonia Early Neolithic			-	60			20	60*	
Carlson *et al.* (1974)	Nubia 350 BC-AD 1400	24/75	32	37/210	18			285	21.4*	parasites, weanling diarrhea and poor nutrition all factors
Cule and Evans (1968)	Wales Bronze Age	1/1	100					1	100**	inadequate dietary intake of iron by mother or infant
Cybulski (1977)	British Columbia Coast Post-contact	25/94	27	32/360	9			454	13**	chronic disease/blood loss may have been responsible, although dietary deficiency cannot be discounted due to disruption of Aboriginal eating habits by Europeans

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Cybulski (1990)	British Columbia Coast					36/261	14	261	14**	chronic disease/blood loss, intestinal parasites, multiple childbirths, crowded living conditions may have been responsible
	Prehistoric									
El-Najjar *et al.* (1976)	Arizona, New Mexico							539	34*	population in canyon bottom depended heavily on maize which interfered with absorption
	Anasazi									
El-Najjar *et al.* (1976)	Canyon	28/36	78	24/31	77				78**	
	Basketmakers									
El-Najjar *et al.* (1976)	Canyon	8/15	53	18/28	64				84**	emphasis on maize inhibited absorption of iron
	Puebloans									
El-Najjar *et al.* (1976)	Canyon	6/10	60	7/13	54				57**	emphasis on maize inhibited absorption of iron
	Chaco Canyon (900-1156 A.D.)									

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** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

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Author/date	Location/ Time Period	Frequency Children n	Children %	Adults n	Adults %	Age not stated n	Age not stated %	Sample Size (N)	Total % Affected	Comments
El-Najjar *et al.* (1976)	Canyon	3/7	43	0/6	0				23**	diet of squash, and gourds low in dietary iron, maize inhibited iron absorption
	Inscription House (1250-1300 A.D.)									
El-Najjar *et al.* (1976)	Sage Plain	6/7	85	3/5	60				75**	wild and domesticated foods- mazie inhibited iron absorption
	Navajo Reservoir (700-1100 A.D.)									
El-Najjar *et al.* (1976)	Sage Plain	5/12	42	4/15	27				33**	wild and domesticated foods- maze inhibited iron absorption
	Gran Quivira									
El-Najjar & Robertson (1976)	Arizona	1	100					1	100*	severe iron deficiency as a result of maize dependent diet - inhibited absorption
	1200 A.D.									
Goodman *et al.* (1984b)	Dickson Mounds, Illinois							238	37**	increase of cribra orbitalia through time associated with increased reliance on maize
	Late Woodland-Middle Mississippian									

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Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Goodman *et al.* (1984b)		6/44	14						14**	
	Late Woodland (non-agricultural)									
Goodman *et al.* (1984b)		29/93	31						31**	
	Mississippian Acculturated Late Woodland									
Goodman *et al.* (1984b)		52/101	51						51**	
	Middle Mississippian (agric.)									
Hengen (1971)	Wurttemberg, Germany	11/16	69	140/373	38			389	39**	iron deficiency anemia due to poor hygienic conditions
	5th Century-1922									
Hirata (1990)	Edo, Japan (Tokyo)					81	-		*81	incidence and severity of cribra orbitalia a reflection of hygienic, social and environmental circumstances in 17th Century
	Medieval									

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
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Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Jerszynska (1991)	Cedynia, Poland Medieval	57/90	63						63**	
Mensforth *et al.* (1978)	Libben Site, Ohio Late Woodland	107/241	44					241	44*	constitutional factors, diet, and infectious disease all operative in the pathogenesis of porotic hyperostosis
Nathan & Haas (1966a)	America, Europe various dates	67/104	64	155/614	19			718	31**	
Norr (1984)	Isthmus of Panama (8000 B.C.-1550 A.D.)							153	14*	increase in frequency of porotic hyperostosis over time coincides with increasing dependence on maize
Norr (1984)	Pre-Ceramic-Archaic (8000-2500 B.C.)	8/44	24	0/50	0				12*	

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
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Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Norr (1984)		3/5	60	2/23	9				18*	
	Agricultural Villages (300 B.C. - A.D. 500)									
Norr (1984)		2/10	20	6/22	27				33*	
	Agricultural Chiefdoms (A.D. 500-1550)									
Norr (1984)		0/3	0	1/3	33				33*	
	Caribbean Coast (A.D.600-900)									
Palkovich (1987)	Arroyo Hondo, New Mexico Coalition Period Pueblo	14/44	31					44	31*	endemically inadequate diets affecting pregnant females and their fetuses the underlying cause of porotic hyperostosis in infants and children
Rathbun (1984)	Iran/Iraq							535	12**	higher level of cribra orbitalia in metal ages may be due to higher pathogen exposure or periods of dietary stress in urban areas
	Palaeolithic to Metal Ages									

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Rathbun (1984)	Iran/Iraq Neolithic					12/56	21		21**	
Rathbun (1984)	Iran/Iraq Chalcolithic					5/18	28		28**	
Rathbun (1984)	Iran/Iraq Bronze/Iron Age					106/46	23		23**	
Smith *et al.* (1984)	Levant Epipaleolithic and Neolithic							191	64**	chronic disease associated with increased population density and permanent settlements by late Neolithic are responsible for lesions
Smith *et al.* (1984)	Natufian	1/2	50	4/11	40				38**	

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Smith *et al.* (1984)	Chalcolithic	4/4	100	2/2	100				100**	
Smith *et al.* (1984)	Middle Bronze	5/5	100	22/35	63				68**	
Smith *et al.* (1984)	Roman-Byzantine	13/21	62	23/36	65				63**	
Smith *et al.* (1984)	Early Arab	28/33	84	20/42	47				64**	
Stuart-Macadam (1982)	Poundbury Camp, England Romano-British	75/206	36	143/546	26			752	31**	possible parasitism, infectious disease, and lead poisoning

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
Walker (1986)	Channel Islands, Southern California Prehistoric to Historic							432	35**	high nutrient losses associated with diarrheal diseases from exposure to fish-borne parasites and contaminated water rather than nutritional inadequacy
Walker (1986)	Mainland Coast Prehistoric to Historic					18/79	23		23**	
Walker (1986)	Santa Cruz Island Prehistoric to Historic					40/172	23		23**	
Walker (1986)	Santa Rosa Island Prehistoric					60/154	43		43**	
Walker (1986)	San Miguel Island Prehistoric to Historic					26/37	70		70**	

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

Appendix A. Worldwide Distribution of Porotic Hyperostosis

Author/date	Location/ Time Period	Frequency Children n	%	Adults n	%	Age not stated n	%	Sample Size (N)	Total % Affected	Comments
White (1988)	Belize, Central America Postclassic to Historic							-	32-43	ecological factors & maize processing technique combine to increase survival rate of parasites in population resulting in blood loss and chronic disease

N = sample size
n= number of individuals with porotic hyperostosis/cribra orbitalia
- = no information given
* porotic hyperostosis cited
** cribra orbitalia cited
*** combined cribra orbitalia and/or porotic hyperostosis - author did not cite nature of pathology

Appendix B. Pender Skeletal Sample

Key to Appendix B:

Orbits:

1 Both Orbits present
2 Right Orbit only present
3 Left Orbit only present

Time Period:

0 Unknown
1 0 - 1000 BP
2 1000 - 2000 BP
3 2000 - 3000 BP
4 3000 - 4000 BP
5 4000 - 5000 BP
6 5000 - 6000 BP

Age Estimate:

0 Fetus to 6 months
1 6 months to 2 years
2 2 years to 4 years
3 4 years to 6 years
4 6 years to 8 years
5 8 years to 12 years
6 12 years to 18 years
7 Adult (18+)
8 Old Adult
9 Indeterminate

DeRt 2 Units Equivalents:

Unit	Provenience	
0	Unknown	
3	30-28W	44-46S
9	26-24W	42-44S
10	24-22W	42-44S
15	26-24W	40-42S
18	20-18W	40-42S

23	24-22W	38-40S
24	22-20W	38-40S
25	20-18W	38-40S
26	18-16W	38-40S
27	16-14W	38-40S
30	26-24W	36-38S
31	24-22W	36-38S
32	22-20W	36-38S
38	26-24W	34-36S
39	24-22W	34-36S
41	20-18W	34-36S
43	16-14W	34-36S
45	30-28W	32-34S
47	26-24W	32-34S
48	24-22W	32-34S
49	22-20W	32-34S
50	20-18W	32-34S
51	18-16W	32-34S
54	26-24W	30-32S
55	24-22W	30-32S
56	22-20W	30-32S
57	20-18W	30-32S
59	16-14W	30-32S
60	14-12W	30-32S
62	24-22W	28-30S
63	22-20W	28-30S
64	20-18W	28-30S
65	18-16W	28-30S
66	16-15W	28-30S
67	15-13W	28-30S
68	23-20W	26-28S
69	20-18W	26-28S
70	18-16W	26-28S
71	16-14W	26-28S
75	22-20W	24-26S
77	18-16W	24-26S
78	16-13W	24-26S
80	22-20W	22-24S
81	20-18W	22-24S
83	24-22W	20-22S
86	80W	60S

Appendix B. Pender Skeletal Sample

ID No.	Year	Burial No	Orbits	Location	Level	Time Period	Unit	Age at Death	Age Estimate	Sex	CO Score R	CO Score L	Comments
2	1984	2	1	2	1	0	10	7	Adult	M	0	0	
5	1984	5a	1	2	5	4	78	8	Adult	F	0	0	capillary like impress. in both R & L
16	1984	M9	3	2	5	2	45	9	Indeterminate	I		2	
17	1984	10	1	2	6	2	78	7	Adult	I	0	0	
19	1984	12	1	2	5	6	81	7	Adult	M	0	2	
25	1984	M17b	3	2	3	2	50	9	Indeterminate	I		1	
34	1984	27	1	2	12	4	54	8	Adult	I	0	0	
37	1984	30a	1	2	3	2	50	7	Adult	M	0	0	
39	1984	31	1	2	17	5	77	7	Adult	F	0	0	
41	1984	33	1	2	7	5	83	7	Adult	F	2	2	
44	1984	34c	1	2	4	0	80	7	Adult	I	0	0	
46	1984	36	1	2	7	4	75	7	Adult	M	0	0	
47	1984	37	1	2	10	4	49	8	Adult	F	0	0	
49	1984	39	1	2	13	4	49	7	Adult	M	0	0	
51	1984	M41	1	2	18	4	49	7	Adult	I	0	0	
52	1984	42	1	2	5	2	65	7	Adult	I	0	0	deformed
57	1984	44	1	2	4	2	66	7	Adult	I	0	0	Dwarf
59	1984	46	1	2	11	4	48	7	Adult	M	0	1	Sex type specimen
64	1985	1	3	1	20	2	10	7	Adult	I		1	
69	1985	1a	1	2	17	5	64	2	0-6 years	I	0	0	
73	1985	4	1	2	7	2	0	7	Adult	M	3	0	deformed
74	1985	5	3	2	13	4	15	7	Adult	F	0	0	
76	1985	7	1	2	5	3	39	8	Adult	F	0	0	
79	1985	9a	1	2	10	3	63	8	Adult	M	0	0	
81	1985	10a	3	2	17	4	63	7	Adult	I		0	
83	1985	10b	1	2	11	4	63	7	Adult	I	1	1	
85	1985	12	1	2	7	2	86	3	0-6 years	I	0	0	
86	1985	13	2	2	11	3	23	7	Adult	M	0		
90	1985	17	2	2	10	4	41	7	Adult	M	3		
91	1985	18	1	2	8	3	41	7	Adult	F	0	0	

Appendix B. Pender Skeletal Sample

ID No.	Year	Burial No	Orbits	Location	Level	Time Period	Unit	Age at Death	Age Estimate	Sex	CO Score R	CO Score L	Comments
92	1985	19	1	2	8	2	62	8	Adult	I	1	1	
94	1985	21	1	2	11	4	23	7	Adult	M	0	0	capillary like impress. in both R & L
96	1985	23	3	2	8	3	51	9	Indeterminate	I	1	1	
97	1985	24	1	2	9	4	70	7	Adult	I	0	0	pathological
98	1985	25	1	2	9	4	39	7	Adult	I	0	0	capillary like impress. in R. orbit
99	1985	26	2	2	15	4	69	7	Adult	F	1		Sex type specimen
100	1985	27	1	2	12	4	30	3	0-6 years	I	1	1	
103	1985	29b	2	2	10	3	56	7	Adult	M	0		
109	1985	34	3	2	9	3	68	7	Adult	F		0	
110	1985	35	1	2	11	4	68	7	Adult	M	0	0	
111	1985	36	1	2	16	4	68	7	Adult	F	0	0	
112	1985	37	1	2	16	4	56	8	Adult	F	1	1	
113	1985	38	1	2	12	4	55	7	Adult	F	1	1	Sex type specimen
116	1986	2	1	1	3	1	34	8	Adult	F	0	0	
123	1986	8	2	2	15	4	31	7	Adult	M	0		
126	1986	10	1	2	7	4	34	7	Adult	M	0	0	
127	1986	11	1	2	13	4	41	O	0-6 years	I	0	0	
129	1986	13	1	2	11	4	57	7	Adult	F	1	1	
131	1986	15	2	2	9	4	57	7	Adult	M	0		capillary like impress. in both R & L
134	1986	19	1	2	17	4	57	7	Adult	F	0	0	capillary like impress. in both R & L
135	1986	20	3	2	6	2	27	7	Adult	F		0	
137	1986	M22	1	2	5	3	57	7	Adult	M	0	0	capillary like impress in both R & L
138	1986	23	1	2	5	2	57	4	6-18 years	I	3	3	
139	1986	24	1	2	19	3	3	6	6-18 years	I	0	0	
140	1986	26a	3	2	18	4	24	9	Indeterminate	I		1	
142	1986	28	1	2	4	2	59	7	Adult	M	1	1	
146	1986	32a	2	2	9	4	32	7	Adult	I	1		
147	1986	32b	1	2	9	4	32	7	Adult	M	0	0	Sex type specimen
150	1986	35	3	2	8	4	71	7	Adult	M	I	0	
152	1986	37	1	2	13	4	25	7	Adult	I	1	1	

Appendix B. Pender Skeletal Sample

ID No.	Year	Burial No	Orbits	Location	Level	Time Period	Unit	Age at Death	Age Estimate	Sex	CO Score R	CO Score L	Comments
154	1986	39	2	2	9	2	18	3	0-6 years	I	2		
155	1986	40	1	2	10	4	26	7	Adult	M	0	0	
158	1986	45a	1	2	9	4	60	7	Adult	I	0	0	
161	1986	46	3	2	15	4	18	5	6-18 years	I		2	
162	1986	47	1	2	2	2	43	7	Adult	M	0	0	
168	1986	52a	1	2	23	5	32	6	6-18 years	I	4	4	
170	1986	53a	2	2	1	0	25	9	Indeterminate	I	1		
172	1986	53c	3	2	1	0	25	9	Indeterminate	I		0	p.h. thickened vault
174	1986	55	1	2	8	4	25	8	Adult	M	0	0	
175	1986	56	2	2	1	2	67	7	Adult	I	0		p.h. thickened vault
176	1986	57	3	2	6	2	67	8	Adult	F		1	
179	1986	60	1	2	4	2	26	7	Adult	I	1	1	p.h. thickened vault
180	1986	61	3	2	7	2	25	7	Adult	M		0	p.h. thickened vault
181	1985	M114	1	2	6	3	38	9	Indeterminate	I	0	0	
182	1984	M89	3	2	ns	0	49	9	Indeterminate	I		2	
183	1984	M00	3	2	3	2	66	9	Indeterminate	I		1	
336	1986	M287	1	2	13	3	9	9	Indeterminate	I	0	0	
378	0	M00	1	2	n.s.	0	0	7	Adult	I	1	1	has capillary like impress. with co
412	1987	1	1	2	n.s.	0	0	8	Adult	I	0	0	
430	1985	M130	2	2	3	2	68	9	Indeterminate	I	0		
457	1984	M42	3	2	9	0	47	9	Indeterminate	I		0	
664	1984	M102	3	2	7	3	50	9	Indeterminate	I		0	
693	1986	26b	2	2	17	4	24	9	Indeterminate	I	2		

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Black Raspberry *Rubus leucodermis*	fresh	0.7	18.0	-	4	5.0
Blue Elderberry *Sambucus cerulea*	fresh	1.0	33.3	-	-	-
Bog Blueberry Vaccinium sp.	fresh	0.2	-	-	-	6.40
Common Saskatoon *Amelanchier alnifolia*	fresh	0.5	15.7	-	86	-
Common Saskatoon *Amelanchier alnifolia*	dried	6.06	-	-	-	-

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Common Wild Gooseberry *Ribes divaricatum*	fresh	0.7	40.2	-	29	-
Evergreen Huckleberry /Winter Huckleberry *Vaccinium ovatum*	fresh	0.4	66.6	-	-	-
Evergreen Huckleberry /Winter Huckleberry *Vaccinium ovatum*	dried	1.38	289.7	-	-	-
High Bush Cranberry *Viburnum edule*	fresh	0.3	13.4	-	6	-
Kinnikinnick *Arctostaphylos uva-ursi*	fresh berries	0.7				

- = not stated, or unknown

Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Mountain Bilberry *Vaccinium membranaceum*	fresh	0.2	6.6	-	1	-
Red Elderberry *Sambucus racemosa*	fresh	1.1	81.0	-	30	-
Red Huckleberry *Vaccinium parvifolium*	fresh	0.4	37.0	-	8	-
Salal *Gaultheria shallon*	fresh	0.7	68.5	-	-	-
Salal *Gaultheria shallon*	dried	3.6	570	-	-	-

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Salmonberry *Rubus spectabilis*	fresh berries	0.6	30.4	-	79	-
Salmonberry *Rubus spectabilis*	dried berries	5.0	371	-	-	-
Salmonberry *Rubus spectabilis*	shoots peeled	0.3	7.5	-	-	-
Soapberry *Shepherdia canadensis*	fresh	0.5	-	-	-	-
Stink Currant *Ribes bracteosum*	fresh	0.8	27.5	-	4	-

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Strawberry *Fragaria* sp.	fresh	1.0	59	-	8	20.0
Swamp Gooseberry *Ribes lacustre*	fresh	0.4	58.2	-	3	-
Thimbleberry *Rubus parviflorus*	fresh	0.9	78	-	-	-
Trailing Wild Blackberry *Rubus ursinus*	fresh	0.6	28.3	-	4	
Bracken Fern *Pteridium aquilinum*	shoots	0.8	15.0	-	-	-

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Bracken Fern *Pteridium aquilinum*	greens	-	-	-	1,250	-
Common Horsetail *Equisetum arvense*	shoots	2.9	50	-	18	-
Lady Fern *Athyrium filix-femina*	greens	0.8	8.9	-	134	-
Licorice Fern *Polypodiaceae glycyrrhiza*	roots	4.4	-	-	-	-
Hazelnut *Corylus* sp.	fresh	3.4	1.0	-	7	-

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Wild Crabapple *Pyrus fusca*	fresh	0.6	8.0	-	4	-
Wild Rose Hips Rosa sp.	fresh	-	-	-	358	-
Arrowhead/Wapato *Sagittaria latifolia*	fresh	6.6	5.0	-	0	-
Blue Camas/Common Camas *Camassia quamash*	fresh	1.6	4.0	-	-	-
Perennial Clover *Trifolium wormskioldii*	steamed roots	4.2	-	-	0.3	-

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Silverweed *Potentilla pacifica*	steamed	3.5	-	-	0.2	-
Wild Onion *Allium nuttalli*	fresh bulbs	1.5	15.0	-	-	-
Wild Onion *Allium* sp.	fresh tops	6.0	27	-	-	-
Yampah/ Carrot *Perideridia gardneri/ Lomatium utriculatum*	dried	7.5	3.0	-	-	-
Cow Parsnip/Indian Celery *Heracleum lanatum*	fresh peeled stalks	0.3	3.5	-	7.5	16.1

- = not stated, or unknown
Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Fireweed *Epilobium angustifolium*	fresh leaves	2.1	128	-	1,700	-
Fireweed *Epilobium angustifolium*	fresh shoots	0.5	-	-	4	-
Stinging Nettle *Urtica dioica*	greens	3.0	89.8	-	-	-
Stinging Nettle *Urtica dioica*	fresh leaves	1.0	75.0	-	2,248	-
Giant Kelp *Macrocystis integrifolia*	fresh fronds	1.2	-	-	-	-

- = not stated, or unknown

Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix C. Nutritional Content of Select Native Plant Foods (per 100 g fresh/dry weight)

Common Name/ Taxon cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Giant Kelp *Macrocystis integrifolia*	dried fronds	9.3	-	-	-	-
Laver Porphyra sp.	dried fronds	10.5	11.6	-	263	-

- = not stated, or unknown

Data compiled from Ensminger *et al.* 1994; Holland *et al.* 1991; Keely *et al.* 1982; Kuhnlein and Turner 1986, 1991; Rivera 1949; USDA Agric. Handbook 8-11 1976.

Appendix D. Nutritional Content of Select Native Animal Foods (per 100 g fresh weight)

Common Name/ Species cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Duck *Anas platryhynchos*	roasted meat, fat & skin	2.7	0	2.0	-	-
Herring *Clupea harengus*	grilled	1.0	Tr.	11	34	10
Sockeye Salmon *Oncorhynchus nerka*	steamed	0.8	Tr.	6.0	20-150	29
Sockeye Salmon *Oncorhynchus nerka*	baked	1.20	Tr.	-	20-150	-
Sockeye Salmon *Oncorhynchus nerka*	BBQ	0.7-1.9	-	-	-	17-27
Coho Salmon *Oncorhynchus kiutsch*	half-smoked	0.6-0.9	-	-	-	7-24
Coho Salmon *Oncorhynchus kiutsch*	smoked & dried	0.7-1.0	-	-	-	4-21

Tr = Trace
- = data not given
Nutritional sources include: Ensminger *et al.* 1994; Kennelly 1986; Kuhnlein *et al.* 1982; Monsen 1981.

Appendix D. Nutritional Content of Select Native Animal Foods (per 100 g fresh weight)

Common Name/ Species cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Cod *Gadus morhua*	baked	0.4 - 1.0	Tr.	2.0	2	12
Cod *Gadus morhua.*	boiled	1.8	Tr.	Tr.	2	Tr.
Eulachen Grease *Thaleichthys pacificus*	oil	Tr.	0	-	1,985	-
Deer/Wapiti (Venison) *Cervidae* sp.	roasted meat	7.8	0	0.8	-	6
Crab *Cancer pagarus*	boiled	1.3	Tr.	Tr.	Tr.	20
Blue Mussel *Mytilus edulis*	boiled	7.7	Tr.	22.1	-	-
Hardshell Clam -	raw meat & liquid	9.4	10.0	19.10	-	15

Tr = Trace
- = data not given
Nutritional sources include: Ensminger *et al.* 1994; Kennelly 1986; Kuhnlein *et al.* 1982; Monsen 1981.

Appendix D. Nutritional Content of Select Native Animal Foods (per 100 g fresh weight)

Common Name/ Species cited in nutritional study	Preparation	Iron mg	Vitamin C mg	Vitamin B12 ug	Vitamin A RE	Folate ug
Cockle *Cardium edule*	boiled	26-40	Tr.	Tr.	-	-
Dogwinkles *Littorina littorea*	boiled	2.9	Tr.	6.9	-	-

Tr = Trace
- = data not given
Nutritional sources include: Ensminger *et al.* 1994; Kennelly 1986; Kuhnlein *et al.* 1982; Monsen 1981.

Appendix E. Specific Age Distribution of Pender Canal Sample

Age Group	Number Affected	% of Group Affected
0-6 months	0/1	0
2-4 years	0/1	0
4-6 years	2/3	66
6-8 years	1/1	100
8-12 years	1/1	100
12-18 years	1/2	50
Adult	15/49	31
Old Adult	3/11	27
Unknown	8/14	57

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