TUBERCULOSIS IN POLYNESIA:
A discussion of its occurrence before initial European contact

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By
Katherine K. Suzuki

Thesis Committee:
C. Fred Blake, Chairperson
Nina L. Etkin
Michael Pietrusewsky
We certify that we have read this thesis and that, in our opinion, it is satisfactory in scope and quality as a thesis for the degree of Master of Arts in Anthropology.

THESIS COMMITTEE

C. Fred Blake  
Chairperson

M. Peterson
Abstract

Infectious diseases continue to be of great importance to human societies due to the social, economic, political, and psychological disruptions they cause. In the question of infectious disease in prehistoric populations, a bioarchaeological approach situated within a political-economic context provides a comprehensive and effective framework to assess globally significant diseases, such as tuberculosis. Important aspects of tuberculosis epidemiology can be learned from the reconstruction of past events, especially in areas where tuberculosis has been assumed to be a European introduction. The \textit{M. tuberculosis} complex is capable of causing skeletal changes which are therefore indicators of this disease in archaeological human remains. In the Pacific region, there have been notably few opportunities to assess pre-European skeletal remains for tuberculosis due to the lack of archaeological remains available for study. The osteological evidence that does exist is ambiguous but suggestive of pre-European presence of tuberculosis. Archaeological data support population densities that may have been sufficient to support an infectious pathogen such as \textit{M. tuberculosis}. The field of molecular genetics has been successfully applied in the recovery of tuberculosis aDNA and offers methods that can significantly reduce the margin of uncertainty about the presence of tuberculosis in the Pacific. After reviewing the available data, it is determined that the lack of conclusive evidence should not preclude the possibility of pre-European tuberculosis in this region.
TABLE OF CONTENTS

Abstract................................................................................................................................iii
List of Tables ..................................................................................................................................v
List of Figures .................................................................................................................................vi
Introduction ........................................................................................................................................1
Chapter 1: Tuberculosis....................................................................................................................9
    General description of *M. tuberculosis* ............................................................................... 10
    Current epidemiological trends of tuberculosis................................................................. 15
    Factors influencing susceptibility ......................................................................................... 19
Chapter 2: Introduction of disease ..................................................................................................21
    The Americas ......................................................................................................................... 21
    Hawai‘i ................................................................................................................................. 29
    Tuberculosis during European contact .............................................................................. 33
    The problematic assumptions of “virgin soil” ..................................................................... 36
Chapter 3: The physical anthropology of tuberculosis ..................................................................39
    Osteological consequences of tuberculosis ........................................................................ 39
    Case studies .......................................................................................................................... 41
Chapter 4: Archaeology and demographics of pre-European Polynesia .....................................44
    Analyses of Polynesian sites ................................................................................................. 44
    Origins of TB in the Americas .............................................................................................. 48
Chapter 5: Molecular biology and tuberculosis .......................................................................51
    Tuberculosis genome ........................................................................................................... 51
    Polymerase chain reaction .................................................................................................... 53
    PCR and ancient DNA .......................................................................................................... 55
    Tuberculosis aDNA ................................................................................................................ 58
Chapter 6: Discussion of the evidence .........................................................................................61
    Demography .......................................................................................................................... 61
    Skeletal lesions ...................................................................................................................... 63
    Diet of Polynesians ............................................................................................................... 64
    Summary ................................................................................................................................. 65
    Alternative origins for TB in Polynesia .............................................................................. 68
Conclusion ........................................................................................................................................71
References .........................................................................................................................................76
<table>
<thead>
<tr>
<th>Table</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Tuberculosis data in regions designated by the World Health Organization (2004)</td>
<td>15</td>
</tr>
<tr>
<td>1.2</td>
<td>Tuberculosis data for Oceania by country and conventional cultural region (World Health Organization 2004)</td>
<td>16</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Model for interpreting stress in archaeological populations</td>
<td>5</td>
</tr>
<tr>
<td>1.2</td>
<td>Conventional regions of the Pacific.</td>
<td>17</td>
</tr>
<tr>
<td>3.1</td>
<td>Polynesian island groups with skeletal samples of possible tuberculosis</td>
<td>42</td>
</tr>
</tbody>
</table>
Introduction

Infectious diseases have been, and continue to be, of intrinsic importance to human societies. Infectious disease pathogens have obliged human populations to adapt both genetically and culturally to the social, economic, political, and psychological disruptions they cause. The 1970s saw the development of a newly emerging sub-discipline in anthropology that addresses the multi-faceted interaction between sociocultural, biological, and ecological factors in the etiology and prevalence of infectious disease (Inhorn and Brown 1997). This sub-discipline of medical anthropology continues to address these issues through different theoretical perspectives and methodologies that work to holistically illuminate the complex interactions between humans and the pathogens that affect them.

As noted by Inhorn and Brown (1997), there is no single theoretical paradigm that dominates the field of medical anthropology. Most anthropological studies on infectious disease involve an ecological perspective that focuses on the interaction between pathogenic agent and host within a given ecosystem in which the environmental factors include both physical and sociocultural aspects. For example, physical environmental factors, such as altitude, temperature, soil type, and presence of certain animals, can intersect with human behavioral factors, such as subsistence strategies, housing types, and community mobility, to affect the rates of certain parasitic infections (Inhorn and Brown 1997). Nutrition, energy, i.e. protein and calories, flows, and migrations are also research foci within this paradigm (Goodman and Leatherman 1998).

Sociocultural perspectives focus on how human behavior affects infectious disease transmission. This approach recognizes that not all human behavior is adaptive in the evolutionary sense as many behaviors can actually promote the spread of disease. One such
model proposes the human behavior can affect disease transmission by creating exposure to the disease agent, shedding the pathogen from an infected human host, creation of habitats that promote or complete the transmission cycle, and through the spatial diffusion of these transmission systems (Inhorn and Brown 1997). One of the best-known examples of this type of anthropological application in an infectious disease problem is the case of kuru in the eastern highlands of New Guinea, in which studies linked the mortuary practices of the Fore to the prevalence of a particular neurological degenerative condition caused by prion proteins.

Medical anthropologists also employ an ethnomedical perspective that addresses the motivations for certain health-related human behaviors. Culture provides a theoretical system for understanding and manipulating the diseases the cause human illness and death. Ethnomedical approaches seek to identify the social, cultural, and psychological correlates of human behavior that relate to and affect infectious disease outcomes, such as indigenous beliefs about etiology, diagnosis, and cure.

A growing and extremely important current rippling through medical anthropological studies is the application of a political economy perspective. Political economy addresses how global systems and history intersect with local systems and histories in order to establish a context within which to understand certain human behaviors. Social relations of power and institutional control of fundamental resources, access to these resources, and differential distribution of resources provides a critical and perhaps corrective (Goodman and Leatherman 1998) groundwork for the interpretation of health outcomes. Human biologies are not outside the scope of political economy. This theoretical perspective can used to examine biological variation in terms of social relations, access to resources, production, and distribution in regard to health stressors that are a result of these social processes. The connection between regional
and global processes also reveals social forces that influence inequality, and these inequalities affect the disease process (Armelagos et al. 2005; Farmer 1999). Global economic processes have created significant disparities in wealth that are at the core of differential health outcomes. Human biologies, then, can be framed in the understanding of how separate communities are connected through larger historical and political-economic processes.

Biological perspectives can also be applied through both micro- and macro-evolutionary studies. Microevolutionary biological approaches focus on how humans adapt to infectious disease pathogens at the level of the gene. The classic example of genotypes that are evolutionarily selected for is the heterozygous condition for sickle cell anemia in areas where the malaria-causing *Plasmodium* is present (Inhorn and Brown 1997). Due to the pathogenesis and life cycle of this parasite, this particular genotype confers significant resistance to death from this disease, and there is a statistical correlation between malaria prevalence and sickle-cell gene frequencies in these areas (Inhorn and Brown 1997).

Similarly, a theoretical perspective of co-evolution attends to human biological adaptations to pathogens, but also includes the cultural adaptations that likewise influence the adaptations and evolutionary processes of the pathogens themselves, as well as any involved peripheral species. Etkin (2003) provides an illustrative example of this application through extensive studies on malaria among the Hausa in Nigeria, which include patterned use of certain plant species as both food and medicine, genetic erythrocyte abnormalities that confer malarial resistance, and addressing the complex biocultural interaction of humans, plants, and pathogens.

On a macro-scale, biological approaches can address epidemiological patterns throughout human evolution through the use of the epidemiological transition model. This is a broad-stroke perspective that traces the ecological and social relationships between humans and their
pathogens beginning with the Paleolithic (Armelagos et al. 2005). Notably, Paleolithic populations would have shared common pathogens with their primate ancestors, as well as would have been exposed to zoonotic pathogens due to their foraging subsistence strategies. Sparse and mobile populations would have limited any endemic infectious diseases. The shift to agricultural subsistence economy is associated with the advent of infectious disease. Armelagos et al. (2005) note that public health measures, improved nutrition, and medicine have brought a second epidemiological transition marked by the decline of infectious disease and a rise in chronic and degenerative diseases. Furthermore, these authors claim that human populations are embarking on a third epidemiological transition characterized by the reemergence of infectious diseases previously thought to be under control as well as the emergence of novel diseases.

Different biological approaches such as paleopathology and paleoepidemiology attempt to reconstruct epidemiological patterns of disease transmission among prehistoric and historic human populations. The goal of these reconstructions is largely to establish the antiquity of various infectious pathogens. Of particular importance to this work is the framework of bioarchaeology. Bioarchaeology represents the interface between archaeology and skeletal biology, and encompasses paleopathology. Skeletal material can be used to independently measure outcomes in order to test hypotheses and reflects a commitment to the study of process. Bioarchaeology can also incorporate framing hypotheses in a political-economic context, using the analysis of skeletons to measure the effects of social, political, and economic transformations on health and illness. In addition, more recent advances in skeletal biology have resulted from being able to examine the skeleton at the organ, tissue, cellular, and molecular levels. Analyses of bone histology and chemistry to study various bone pathologies, the use of stable isotopes to
reconstruct diet and determine age of weaning, and the DNA identification of pathogens provide new dimensions in which to apply bioarchaeology (Armelagos 2003).

Bioarchaeology allows for a multi-faceted approach to health and disease questions in the archaeological record. As discussed previously, culture as a component of environment influences the disease process in many ways. By using bioarchaeological techniques and methods to reconstruct cultural behavior, the efficacy of these behaviors can be assessed vis-à-vis the evidence available. In addition, examining stress indicators can be used to evaluate the ability of a cultural system to respond to stressors. Importantly, multiple indicators of stress, i.e. congenital defects, growth disruptions, nutritional deficiencies, infections, degenerative conditions, trauma, and patterns of cranial and postcranial skeletal morphology are essential factors in reconstructing patterns of adaptation. Bioarchaeology can be the key element in archaeological investigation due to the ability to determine such issues as the impact of cultural practices on human adaptation that it allows (Armelagos 2003).

![Diagram](image)

**Figure 1.1** Model for interpreting stress in archaeological populations (adapted from Larsen 1997)
In considering the tremendous importance of infectious disease in human societies, there are a number of approaches and theoretical perspectives available within which to frame an inquiry. In the question of infectious disease in prehistoric populations, a bioarchaeological approach provides a comprehensive and effective framework which will be used to present an argument for the pre-European occurrence of tuberculosis in certain regions of the Pacific.

Tuberculosis is currently recognized as one of the most significant "re-emerging" infectious diseases across the globe (Roberts and Buikstra 2003; Balamurugan et al. 2004; Dutt 2006). An important aspect of understanding the current epidemiology of infectious disease, such as tuberculosis, is the reconstruction of disease occurrence in both historic and prehistoric populations (Inhorn and Brown 1997). In this bioarchaeological endeavor, the fields of physical anthropology, archaeology, and molecular genetics offer important data, techniques, and methods.

The region of the Pacific, designated as Oceania, has presented little and ambiguous skeletal data regarding the presence of tuberculosis before European contact. There are a number of difficulties associated with this lack of published research which include poor preservation of skeletal remains, as well as repatriation considerations. However, the established osteological presentation of tuberculosis, i.e., lesions, especially on the ribs, vertebrae, ossa coxae, femora, and joints, indicates there are features that can provide differential diagnoses of tuberculosis in Pacific skeletal remains. In addition, there are physical anthropological data that suggest tuberculosis infection in skeletal remains dated to the pre-contact period (Pietrusewsky et al. 1991). Therefore, the possibility of tuberculosis in the Pacific before European contact is a topic that requires further inquiry.
The field of molecular genetics has also been applied in the paleopathological assessment of tuberculosis in various regions of the world, including North and South America, Egypt, and Europe (Clark et al. 1987; Zink et al. 2004; Spigelman et al. 2002). For example, *M. tuberculosis* ancient DNA (aDNA) has been successfully extracted and identified in both Andean (before 1220 A.D.) and Egyptian (3500-2800 B.C.) mummified tissue (Konomi et al. 2002; Zink et al. 2005). These studies attest to the antiquity of this particular pathogen complex.

Techniques and knowledge from the molecular genetic studies of tuberculosis could be applied to certain Pacific skeletal samples, as important questions have been raised as to the endemicity of tuberculosis in areas where it has been assumed to be a European introduction.

The geographic region of the Pacific is discussed using two simultaneous and general distinctions. The first distinction is ultimately derived from Europeans as a consequence of the great voyages of Pacific exploration, and divides the Pacific into the conventional regions of Melanesia, Micronesia, and Polynesia. The modern definition of Polynesia, as the islands found within the vast triangle subtended by Hawai‘i in the north Pacific, New Zealand in the southwest, and Easter Island (Rapa Nui) in the far southeast, dates to the early 1800s. Polynesia is set apart from Melanesia, the islands of the southwestern Pacific from New Guinea to Fiji, and from Micronesia, islands north of the equator ranging from the Marianas and Palau in the west to the Marshall Islands in the east (see Figure 1.1 and Table 1.2 for modern polities that lie within these conventional boundaries).

Roger Green (1991) has proposed that a more meaningful way to partition Oceania is between Near Oceania- comprised of New Guinea, the Bismarck Archipelago, and the Solomon Islands- and Remote Oceania, which is comprised of all of Micronesia, the Melanesian archipelagoes of Vanuatu, Loyalty Islands, New Caledonia, Fiji, as well as all of Polynesia. This
distinction recognizes the long history of Pleistocene human occupation of Near Oceania beginning at least 40,000 years ago, and the relatively late expansion of Austronesian-speaking peoples into Remote Oceania after approximately 2000 B.C. Nevertheless, the island cultures found within the vast triangle of Polynesia display a certain cultural cohesiveness that allows the term “Polynesia” to retain salience.

The purpose of this paper is to present data from physical anthropology, archaeology, and molecular genetics supporting the hypothesis that tuberculosis was, in fact, present in areas of Polynesia before European contact. The epidemiology of tuberculosis, osteological presentation of tuberculosis in archaeological remains, archaeological data that describe important social, demographic, and nutritional aspects of pre-contact Polynesians, and aDNA studies involving the *M. tuberculosis* complex will be articulated in order to present a case for the presence of tuberculosis. Due to the sampling bias of the data available, Hawai‘i presents the most compelling case. The presence of tuberculosis in pre-contact Polynesian populations, if established, may have significant implications for understanding population movement, social structure and environment, and population interaction, as well as the co-evolution of the *M. tuberculosis* pathogen with human hosts in the Pacific region. This information can be further extrapolated to aid in the understanding of current tuberculosis epidemiology.
CHAPTER 1
Tuberculosis

This mycobacterial disease is identified as one of the most important infectious diseases in world today. With approximately one-third of the world’s population infected with bacteria of the *Mycobacterium tuberculosis* complex, 8-9 million new cases annually, and 2-3 million deaths each year, tuberculosis (TB) is a major cause of death worldwide (Watts and Lifeso 1996; Chan and Iseman 2002; Zink et al 2004). The factors attributable to the epidemiology of TB are numerous and complex. Sociological, ecological, geographic, as well as molecular and microbiological changes and aspects contribute to a population’s susceptibility to TB. While commonly classified as a “reemerging” infectious disease, it has been noted this label can be attributed to the lack of interest in a disease that appeared to only be affecting the developing world. Therefore, while TB is “reemerging” in industrialized countries, it has continued to infect and take the lives of the poor and marginalized, despite the numerous medical advances of the 20th century (Roberts and Buikstra 2003; Farmer 1999).

Kochi (2001) notes that there is striking difference in patient profiles when comparing developing and industrialized nations. In industrialized countries, tuberculosis patients tend to be elderly and illness is usually an endogenous reactivation of past infection. The small percentage of cases that result from recent infection occur mainly in ethnic minorities and migrants (Kochi 2001; Raviglione and O’Brien 2005). In contrast, patients with tuberculosis in developing nations span nearly all age groups, and the risk of infection remains high. The greatest incidence and mortality, more than 80% of tuberculosis in the developing world, occurs in the age group that is also the most economically productive (15-59 years). In addition, it is estimated that less than half of these are covered by treatment services.
General description of *M. tuberculosis*

TB is an infectious disease caused by rod-shaped bacteria (bacilli) in the genus *Mycobacterium*, family Mycobacteriaceae, and the order Actinomycetales. Mycobacteria, including *M. tuberculosis*, are often neutral with Gram's staining procedures. However, once stained, the bacilli cannot be decolorized by acid alcohol, a characteristic justifying their classification as acid-fast bacilli. Acid fastness is due mainly to the organisms' high content of mycolic acids, long-chain cross-linked fatty acids, and other cell-wall lipids. In the mycobacterial cell wall, these mycolic acids (lipids) are linked to underlying arabinogalactan and peptidoglycan, both sugar-protein polymer molecules. This structure confers very low permeability of the cell wall, thus reducing effectiveness of most antibiotics. Another glycoprotein molecule in the mycobacterial cell wall, lipoarabinomannan, is involved in the pathogen-host interaction as a virulence factor, and facilitates the survival of *M. tuberculosis* within macrophages (Raviglione and O'Brien 2005).

There are a number of mycobacteria capable of infecting humans, but there is a distinct group which is considered regularly pathogenic to humans. The sequencing of the entire *Mycobacterium tuberculosis* genome (Cole et al. 1998) has allowed the deciphering of the "*M. tuberculosis* complex," which consists of the closely related organisms *M. tuberculosis*, *M. bovis*, *M. africanum*, *M. canettii*, and *M. microti*, with the first three being most important for human infection (Roberts and Buikstra 2003; Zink et al., 2004).

Tuberculosis is a biphasic disease. Infection is transmitted via respiratory route through aerosolized droplets and can occur through coughing, sneezing, and speaking. There can be up to 3000 infectious nuclei per cough, and the droplets can remain suspended in the air for several hours. In addition, the intimacy and duration of contact with an individual with a case of
tuberculosis, the degree of infectiousness of the case, and the shared environment of the contact are all important determinants of transmission (Raviglione and O'Brien 2005). While the majority of inhaled bacilli are trapped in the upper airways and expelled by ciliated mucosal cells, a fraction (usually <10%) reach the alveoli. Once the pathogenic bacteria have access to terminal air passages, nonspecifically activated alveolar macrophages ingest the bacilli. The bactericidal activity of the macrophage and the number and virulence of the bacilli (with virulence partially linked to the bacterium's lipid-rich cell wall and to its glycolipid capsule) determines the events following phagocytosis. The development of pulmonary tuberculosis in the first five years following primary infection is termed primary tuberculosis; the development of disease after five years is diagnosed as post-primary tuberculosis (Fanning 1999; Roberts and Buikstra 2003).

In the initial stage of host-bacterium interaction, the host's macrophages contain bacterial multiplication by producing proteolytic enzymes and cytokines that break down cell walls causing lysis of the bacterial cell. If this response is inadequate, the bacilli begin to multiply. When this occurs, their growth quickly kills the macrophages, which lyse and release their pathogenic contents. Non-activated monocytes are then drawn from the bloodstream to the site and ingest the bacilli released from the lysed macrophages. These initial stages of infection are usually asymptomatic.

About two to four weeks after infection (also the time required to produce visible colonies in lab culture, see Dubos 1952 and Watts and Lifeso 1996), two additional host responses to \textit{M. tuberculosis} develop: a tissue-damaging response and a macrophage-activating response. The tissue-damaging response destroys non-activated macrophages that contain multiplying bacilli. The macrophage-activating response is a cell-mediated phenomenon
resulting in the activation of macrophages that are capable of killing and digesting tubercle bacilli. Although both of these responses can inhibit mycobacterial growth, it is the balance between the two that determines the form of tuberculosis that will develop.

Expression of disease can happen decades after initial infection. In laboratory cultures, tubercle bacilli grow much slower than typical microbial species, which can produce visible colonies in as little as one day. Tubercle bacilli, however, can take over two to four weeks (Dubos 1952; Watts and Lifeso 1996). This relatively slower rate of growth may contribute to the elongated temporal windows of disease expression. Little is actually known about the molecular basis for the bacteria’s dormancy and/or reactivation; however it is often correlated with advanced age, compromised immune function, and/or prolonged and consistent exposure (Cole et al. 1998). Infection results from immune response to the toxic constituents and products of the bacteria so that inoculations of dead bacilli can still elicit the formation of characteristic tubercles (Dubos and Dubos 1952). Symptoms include coughing (often with blood), weakness and lethargy, difficulty breathing, chills/night sweats, weight loss, fever, pallor, and chest pains. It is important to note the latency of symptom development, as infected individuals may not reveal signs or symptoms of a pathogen that is nonetheless present.

Following infection, the primary site of which is the lungs, the cell-mediated response is evoked and the accumulation of large numbers of activated macrophages at the site of the primary lesion cause granulomatous lesions (tubercles) to be formed. These granulomas inhibit replication and spread of the bacteria. Depending on the adequacy of this immune response, 90% of infected persons do not develop clinical disease (Dutt 2006). Generally, there is intense initial inflammation surrounding the infected areas coordinated with the development of a new kind of tissue surrounding the bacilli in an effort to isolate them. This cluster of tissue cells
constitutes the tubercle which expands and/or coalesces with adjacent tubercles. If the bacilli reach excessive concentrations, the immunologic reaction can cause extensive tissue damage and/or necrosis which results in solid caseous lesions (Manabe and Dannenberg, Jr. 2006). A network of fibrous tissue may form around the tubercle completely isolating it from the rest of the body. Although *M. tuberculosis* can survive, its growth is inhibited within this necrotic environment by low oxygen tension and low pH. If the bacilli survive and continue to kill immune cells within the tubercle, it becomes caseous. It is possible that the tubercle becomes impregnated with fibrous tissue and later calcifies which produces a hard nodule, renders the bacilli inactive (Dubos and Dubos 1952), and produces scar tissue in the lungs. However, some of this region may persist as a necrotic locus in which tubercle bacteria can survive.

Physiological and environmental stressors as well as re-introduction of the bacteria can trigger the development of open clinical disease generated from these areas and/or the development of new foci (Dubos and Dubos 1952), a process known as reactivation (Roberts and Buikstra 2003).

If the lesion remains caseous and active, it can eventually be penetrated by the blood. The lesion can then burst open into a bronchus discharging its contents, and this leaves a cavity in the lung tissue. The discharged contents contain viable bacilli and toxic materials which then become disseminated into the lung bronchus and/or the blood. The introduction into the blood can then result in the bacilli being transported to other parts of the body to establish new foci (Dubos and Dubos 1952). Hematogenous infection can spread to virtually any other tissue and/or organ of the body (Baron et al., 1996; Fanning 1999). Non-respiratory presentation is most common in the lymph nodes, genitourinary, skeletal, abdominal, and central nervous systems. In TB involving bone tissue, the spine is the most common site (about 50%) of infection. Large joints, such as the hip, knee, shoulder, elbow, and wrist are less common.
Musculoskeletal presentation of extrapulmonary tuberculous bacilli can be detected through lesions on the bone tissue, large areas of bone destruction, and paravertebral abscess formation and deformity. Lesions indicative of skeletal TB consist of osteomyelitis and arthritis, and tend to be the most common at metaphyses. Abscesses surround the infected site (known as “cold” abscesses) and may rupture forming sinus tracts. Healing of skeletal TB is often indicated by the formation of fibrous scar tissue and calcifications. Tuberculous osteomyelitis is further characterized by the lack of bone regeneration (sclerosis) or periosteal reaction. Features in the spine include rarefaction of vertebral endplates, loss of disc height, osseous destruction, and new bone formation. Narrowing of disc space occurs as a late phenomenon and sharp kyphosis can occur (Watts and Lifeso 1996). Biopsies of bone samples from those with skeletal TB show fewer organisms than those with pulmonary TB (Ganguli 1963; Fanning 1999; Leonard, Jr. and Blumberg 2006).

Little is known about how tuberculosis triggers an immune response. There may be genetic factors in the *M. tuberculosis* genome that influence virulence (Donoghue et al., 2004). According to Raviglione and O’Brien (2005), a number of genes have been identified in the *M. tuberculosis* genome that are believed to confer different aspects of virulence. In addition, genetic factors in the host may play a key role in innate non-immune resistance to infection with *M. tuberculosis*. The existence of this genetically-related resistance is suggested by the differing degrees of susceptibility to tuberculosis in different populations (Raviglione and O’Brien 2005). It is generally believed that virulence of this pathogen in a population decreases over time.

When a new pathogen is introduced into a susceptible population, morbidity and mortality rates can be predicted using epidemic wave models (Dutt 2006). The general shape of this wave consists of sharply ascending limb to a peak, then a gradually descending limb. While for many
infectious diseases this wave can be plotted in terms of weeks or months, for TB this wave is plotted over decades and centuries and runs its course over approximately 300 years (Dutt 2006). The tubercle bacilli can remain viable in human tissue for many years, and when disease is produced, the course is generally chronic and protracted. Any resistance in an individual gained from initial infection is not absolute and the duration uncertain, although infection does not necessarily entail progressive disease (Dubos and Dubos 1952). Production of disease can happen decades after initial infection, which exacerbates the long term epidemiological trend. Thus, the infection becomes endemic when a large proportion of the population is infected, even though clinical presentation of the disease may be absent.

Current epidemiological trends of tuberculosis

Table 1.1 Tuberculosis data** in regions designated by the World Health Organization (2004)

<table>
<thead>
<tr>
<th>WHO Region</th>
<th>Incidence</th>
<th>Prevalence</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Rate*</td>
<td>Number</td>
</tr>
<tr>
<td>Western Pacific</td>
<td>1,925,332</td>
<td>111</td>
<td>3,764,564</td>
</tr>
<tr>
<td>Southeast Asia</td>
<td>2,967,328</td>
<td>182</td>
<td>496,479</td>
</tr>
<tr>
<td>Europe</td>
<td>444,777</td>
<td>50</td>
<td>575,448</td>
</tr>
<tr>
<td>East Mediterranean</td>
<td>644,531</td>
<td>122</td>
<td>1,090,434</td>
</tr>
<tr>
<td>Americas</td>
<td>363,246</td>
<td>41</td>
<td>466,232</td>
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<tr>
<td>Africa</td>
<td>25,272,988</td>
<td>356</td>
<td>3,740,695</td>
</tr>
<tr>
<td>TOTAL</td>
<td>8,918,203</td>
<td>140</td>
<td>14,602,353</td>
</tr>
</tbody>
</table>

*Rate represents the number of cases per 100,000 in the given population  
**Cases include those infected with HIV

Global tuberculosis control is facing major challenges today (Lienhardt and Rustomjee 2006; Raviglione and Uplekar 2006). Currently, tuberculosis control demands a comprehensive
and sustained response that complements measures to address the social and environmental factors that increase the risk of developing the disease (Raviglione and Uplekar 2006).

Treatment of tuberculosis involves multi-drug chemotherapy with the dual goals of preventing acquired drug resistance and increasing efficacy. Rifampin and isoniazid are the primary pharmaceuticals used to treat TB.

Table 1.2 Tuberculosis data** for Oceania by country and conventional cultural region (World Health Organization 2004)

<table>
<thead>
<tr>
<th>Country</th>
<th>Incidence</th>
<th>Prevalence</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Rate*</td>
<td>Number</td>
</tr>
<tr>
<td>American Samoa</td>
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<td>28</td>
<td>31</td>
</tr>
<tr>
<td>Cook Islands</td>
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<td>28</td>
<td>9</td>
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<td>French Polynesia</td>
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<td>28</td>
<td>142</td>
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<tr>
<td>New Zealand</td>
<td>424</td>
<td>11</td>
<td>432</td>
</tr>
<tr>
<td>Western Samoa</td>
<td>52</td>
<td>28</td>
<td>78</td>
</tr>
<tr>
<td>Tonga</td>
<td>29</td>
<td>28</td>
<td>43</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>600</strong></td>
<td><strong>25</strong></td>
<td><strong>735</strong></td>
</tr>
<tr>
<td>Guam</td>
<td>98</td>
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<tr>
<td>Kiribati</td>
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</tr>
<tr>
<td>Marshall Islands</td>
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<tr>
<td>Federated States of Micronesia</td>
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<td>56</td>
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<td>Nauru</td>
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<td>28</td>
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<tr>
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<tr>
<td>Vanuatu</td>
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<tr>
<td><strong>TOTAL</strong></td>
<td><strong>13,988</strong></td>
<td><strong>102</strong></td>
<td><strong>26,558</strong></td>
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**TOTAL FOR OCEANIA** | **15,144** | **54** | **28,021** | **80** | **2,656** | **7** |

*Rate represents the number of cases per 100,000 in the given population

**Cases include those infected with HIV

usually with an introductory combination of streptomycin and/or ethambutol. The course of treatment is generally six to nine months for non-drug resistant TB (Chan and Iseman)
2002) and is considered curable (Raviglione and O'Brien 2005). Inability to complete the course of therapy exacerbates the development of drug-resistant tuberculosis strains, as well as jeopardizes the health of infected patients.

Figure 1.2 Conventional regions of the Pacific

It should be noted that in addition to chemotherapy regimens, there is a limited-use vaccine available. Bacillus Calmette Guérin (BCG) vaccines utilize an attenuated strain originally derived from bovine tuberculosis and was first administered to humans in 1921. It causes a mild self-limited infection that induces partial resistance. This resistance only lasts for several years, at most, and immunity is never absolute. Consequently, there are many physicians who feel that the degree of immunity afforded by vaccination in the case of tuberculosis is far too low to be of practical significance (Dubos and Dubos 1952). Many BCG vaccines are available worldwide; although all are derived from the original strain, the vaccines vary in efficacy. In fact, estimates of efficacy from randomized, placebo-controlled trials have ranged from 80% to nil (Raviglione and O'Brien 2005). The higher rates of efficacy are usually correlated with the protection of infants and young children from relatively serious forms of
tuberculosis, such as tuberculous meningitis and miliary tuberculosis. When BCG is administered, the local tissue response begins two to three weeks after vaccination, with scar formation and healing within three months. Side effects are most commonly ulceration at the vaccination site and regional lymphadenitis, and these occur in 1 to 10% of vaccinated persons. Some vaccine strains have caused osteomyelitis in one case per million doses administered. Infection and death associated with the administration of BCG have occurred in one to ten cases per ten million doses administered, although this problem is restricted almost exclusively to persons with impaired immunity, such as HIV infection. BCG vaccine is recommended for routine use at birth in countries with high tuberculosis prevalence. However, the vaccine has never been recommended for general use in the United States because of the low risk of transmission of tuberculosis in the United States and the unreliable protection afforded by BCG (Raviglione and O’Brien 2005).

To address these issues of non-adherence to drug regimens and achieve higher detection and treatment success rates, the World Health Organization (WHO) implemented the directly observed therapy short course (DOTS) program worldwide in the early 1990s. This program utilizes a third party, usually a nurse or surrogate, to deliver and observe patients taking all doses of their drugs. These DOTS regimens are intermittent instead of daily and continue over six months. This strategy has been shown to be highly effective in promoting successful treatment of tuberculosis as consistently high treatment success rates are reported (Chan and Iseman 2002; Dye et al 2007). The 2005 targets for case detection and treatment success entail 70% case detection and an 85% cure rate of these new cases. These targets were apparently reached in 26 countries, including some identified as high burden countries. However, national TB control programs overall failed to meet these goals (Dye et al. 2007; Lienhardt and Rustomjee 2006).
is noted that implementation of this program and evaluation of its success is problematic in regions whose populations are co-infected with HIV, have drug resistant strains of TB, and have weak health infrastructure (Dye et al. 2007).

The WHO (2006) issued its Global TB Control Report for data gathered in 1990 and 2004. Among the statistics in this report, there is information on global TB burden in terms of incidence (the number of new cases reported each year), prevalence (the total number of cases in a given population at a specific time), and mortality. In 2004, over 8.9 million new cases of tuberculosis were reported worldwide, with prevalence at over 14.6 million. Overall mortality rates in 2004 were recorded at almost 1.7 million. These numbers are based on detected cases that include co-infection with HIV. For the Western Pacific Region, which includes Oceania and East Asia, approximately 1.9 million new cases were reported in 2004 with a prevalence of approximately 3.8 million. Over 300,000 people died of tuberculosis, including those co-infected with HIV, in this region. In 2004 in the areas that comprise Oceania, 15,144 new cases of TB were reported with prevalence of 28,021 and 2,656 deaths. In comparison with the data from 1990, when DOTS was first implemented, all rates of incidence, prevalence, and mortality have decreased in Oceania with the exception of New Zealand and Papua New Guinea.

Factors influencing susceptibility

Diet is a significant factor in determining tuberculosis susceptibility. Adequate nutrition is extremely important in developing and maintaining a resilient immune system. Poor nutrition can influence the incidence, severity, duration, and outcome of tuberculosis (Roberts and Buikstra 2003). In addition to diet, population density and living conditions are relevant to the occurrence of tuberculosis. Due to the mode of transmission (aerosolized droplets) there is a distinct correlation between population density and tuberculosis frequency.
Finally, migration and travel are also noted as important for the transmission potential of tuberculosis. Infective risks for both traveler and visited population exist. This plays an important role in contemporary populations given the relative frequency and ease of travel. This may also have played an important role for prehistoric groups, such as Polynesians and their ancestors, who due to the geographic and geologic characteristics of the Pacific, were significantly engaged in maritime migration and travel. Roberts and Buikstra (2003) note that the highest rates of tuberculosis are reported immediately after arrival for modern immigrants and this can be attributable to stress and compromised immunity, as well as malnutrition. It could be speculated that similar stressors and effects may have affected migrating Polynesians as well, especially considering the often lengthy sea voyages.
CHAPTER 2
Introduction of Disease

In an attempt to refute the presupposition that tuberculosis was introduced into Polynesia by European explorers, a discussion of the phenomenon of disease introduction in general can provide a comparative template. European exploration of the New World changed the course of history, not only for the explorers and their countries of origin, but especially for the indigenous populations that already existed in these “newly discovered” lands. Historical accounts of European contact in places such as the Americas, as well as certain regions of Oceania, are heavily burdened with accounts of dramatic decline in native populations. These population declines are inextricably tied to the infectious diseases introduced by the European colonizers. These phenomena produce demographic patterns that can be compared in an effort to assess, when possible, the roles of specific diseases, such as tuberculosis. In addition, tuberculosis has been shown to have existed in regions of the Americas before Europeans arrived. Therefore, an overview of the American experience can serve as a basis for comparing the patterns found in Polynesia and provide further support for the presence of tuberculosis before European contact.

The Americas

The phenomenon of disease introduction to “virgin soil” populations is an important topic in the discussion of indigenous populations of the New World, i.e. the Americas. European contact through explorers such as Columbus, Cortés, and Pizarro has changed the demographic and cultural countenance of these populations dramatically. The incredible decline in population size of various aboriginal North and Central American groups, as well as the Aztecs, Maya, and Inca is to a great extent attributed to infectious diseases introduced by Europeans. With the early explorers came the highly contagious diseases of European cities, such as smallpox, measles, typhus, and scarlet fever, to which these native populations had never been exposed. These
diseases are believed to have caused “virgin soil epidemics,” defined as those in which populations at risk have had no previous contact with the disease that affects them and are therefore immunologically defenseless (Thornton 1987). These are especially devastating since nearly all of the afflicted population is infected simultaneously. While historians agree that Old World diseases caused an incredible population crash, there continues to be debate as to the magnitude, rate, and timing of this hemispheric depopulation (Roberts 1989).

It has been suggested that native American populations were relatively disease-free due to the “cold screen” provided by the cold arctic climates that initial human migrants endured. Not only was the cold inhospitable to numerous infectious pathogens, it also ensured that only the fittest humans survived to eventually spread through the rest of the Americas. However, it is also noted that aboriginal populations that did not move through this “germ filter” were also free of European diseases and suffered greatly after contact with Europeans, i.e. the aboriginal populations of Australia. Therefore, perhaps it was the lack of domesticated animals in the Western Hemisphere that was the basis for the comparative lack of disease (Thornton 1987). Without domesticated animals, zoonotic potential is reduced, as are animal vectors and/or reservoirs for disease.

It is important to note that the Americas were not completely disease-free before European contact. Thornton (1987) mentions that bacillary and amoebic dysentery, viral influenza and pneumonia, viral fevers, American leishmaniasis, trypanosomiasis, endoparasites such as roundworm, and salmonella were all present before 1492. However, it is widely agreed that the diseases which caused the most deaths in epidemic proportions, such as smallpox, cholera, measles, diphtheria, typhoid fever, and the plague, were definitely brought from Europe and Africa. Thornton (1987) notes:
Europeans brought smallpox, measles, the bubonic plague, cholera, typhoid, pleurisy, scarlet fever, diphtheria, mumps, whooping cough, colds, the venereal diseases gonorrhea and chancroid, pneumonia and some unusual influenza and respiratory diseases, quite probably typhus and venereal syphilis, and only remotely possibly, tuberculosis. (Thornton 1987:44)

In addition, these diseases did not strike singularly or only once; instead there were often repeated outbreaks of a disease. Smallpox, for example, can re-infect a population after a considerable time lapse which produces a new generation of susceptible hosts. Also, epidemics of one disease often coincided and/or made possible infections and outbreaks of different diseases concurrently.

In North America alone, it is estimated that as many as ninety three serious epidemics and pandemics of Old World diseases occurred from the early sixteenth century to the beginning of the twentieth century (Thornton 1987). The first recorded epidemic of smallpox, the first known Old World disease in the Americas, occurred in Hispaniola in 1507. However, the first recorded epidemic in North America took place from 1520-24 (Thornton 1987). Continual colonization by Europeans and continual interaction with these colonists exacerbated aboriginal exposure to Old World pathogens and numerous smallpox epidemics ensued over the next three hundred years (Thornton 1987). Documented reports on these epidemics describe drastic population reductions with estimates of up to 90% being wiped out. The Atlantic coast populations suffered greatly due to the higher concentration of Europeans. However populations in the West and Southwest also suffered epidemics of smallpox, cholera, and bubonic plague throughout the sixteenth century delivered by Spanish contacts from Central America. Numerous smallpox epidemics are continually recorded throughout the seventeenth and eighteenth centuries among North American natives, usually accompanied by dire death
tolls. In addition to smallpox, several epidemics of measles, influenza, typhoid, typhus, diphtheria, and scarlet fever are noted during the eighteenth century.

It is difficult to know in absolute numbers the effect that introduced infectious diseases had on native North American populations. It is even more difficult to ascertain the relative impact on population size of these epidemics, for this would entail knowing population sizes at the time of European contact. Unfortunately, sources for this information are scarce and/or unreliable, and always debatable. Thornton (1987) describes ethnohistorical sources that include early explorer and colonist accounts, as well as missionary records, and these are of questionable accuracy. Archaeological data can provide information from excavation sites and settlement patterns, but these are subject to interpretation as well as sampling and dating error. Physical anthropological data based on skeletal remains can provide demographic information, but this is also subject to issues of sampling and provenience. Ecological evidence can be inferred from “carrying capacity” approaches, although this is also fraught with complications. Estimates of North American native populations are usually derived from extrapolations of a combination of these data sources, as well as back-projecting based on depopulation ratios and epidemiological information, such as mortality rates.

Thornton (1987) reviews the population estimates using these methods and data, and they are quite varied. This is due, in part, to the time frame addressed by the scholars making these estimates. For example, Mooney (1928, as cited by Thornton 1987) suggests a total population 1.153 million for aboriginal North America; however this number is for the periods of extensive European contact. These periods do not account for the effects of infectious disease epidemics prior to ca. 1680. Initial European contact
resulted in important population losses as well, and this is not addressed. Alternatively, Roberts (1989) notes historian Henry F. Dobyns' assertion that North America had a much larger native population of 18 million that was virtually wiped out by disease after the Spanish landed in 1492. By 1900 the population had dropped to 500,000 or less. Part of the problem in interpreting what actually occurred is that, in some places, Old World diseases preceded actual contact with explorers by decades or longer, as pathogens were carried inland along existing trade routes. This suggests that some populations had already been decimated by the time the Europeans made their first estimates (Roberts 1989). The debate around population size at the time of European contact is important in the larger endeavor to understand the real demographic and cultural impact of introduced disease on these populations.

Reff (1991) also subscribes to the idea of “virgin soil epidemics” in his review of disease and depopulation in northwestern New Spain, now the Mexican states of Baja California, Sinaloa, Durango, Chihuahua, Sonora, as well as regions of southern Arizona, New México, and Texas. According to Reff, these native populations lacked the genetic traits that promote resistance to diseases such as smallpox, malaria, and influenza. Reff also cites the added effects of starvation, secondary infection, and “debilitating emotional states” (1991:2) that contributed to overall population decline. The geographic area of Reff's study differs from studies of North American aboriginal decline because there is abundant and early documentary evidence for population sizes. The region of his study, often termed the “Greater Southwest,” presents population densities based on sedentary agriculture that are more similar to other areas of the New World than central México and
Peru, perhaps serving as a better model for regions in the Americas for which there is little documentary data.

The cultures and societies in the Greater Southwest region at the time of European contact were well developed. Reff (1991) concludes that there was an abundance of permanent towns and villages. Agricultural techniques used ranged from floodwater farming to canal irrigation, which were successful enough in exceeding subsistence requirements that craft production and trade could be supported. Hierarchical social structures that were used in productive and organizational endeavors were also in place. There is abundant evidence to support that native populations in this region had sophisticated religious systems as well.

In terms of a chronology of epidemics in this region, Reff (1991) mentions the first smallpox epidemic that occurred between 1518 and 1525. This event is also referred to as pandemic, as native populations throughout the Caribbean, Puerto Rico, Cuba, Central America, South America, and arguably in North America were affected. It is this sweep of smallpox that preceded Cortés into the Aztec capital of Tenochtitlan, decimated its inhabitants including its ruler Cuitláhuac, and allowed Cortés to capture the city (Reff 1991; Cook 1998). In this window, the areas of the Yucatán peninsula, Guatemala, and Panama also experienced epidemic proportions of this disease. Interestingly, many of the societies in the Greater Southwest region were organized into independent political entities. While these people were engaged in local and long-distance exchange at the time of European contact, there is little or no evidence that this commerce involved regular exchanges with populations in the Valley of México or other areas of
Mesoamerica. Therefore, the native populations of this area apparently escaped this first recorded pandemic of smallpox (Reff 1991).

However, in 1530 another Spanish expedition brought a number of infectious diseases, such as malaria, dysentery, and typhoid. Reff (1991) reports that there was a tropical storm that preceded this epidemic. This storm, in addition to the heavy summer rains typical of this region, would have provided an excellent environment for the anopheline mosquito that acts as the malaria vector. In addition, dysentery and typhoid were common among European armies during this time. Those who contract typhoid can retain the Shigella bacteria for years, continually disseminating it in their feces. The case is similar for those who acquire amoebic dysentery and survive; they can harbor the disease for months and sometimes years. An outbreak of malaria, combined with dysentery and typhoid, would explain the high case frequency and mortality rates that were reported in the areas of Nayarit and Sinaloa at this time.

The first New World measles epidemic also occurred in 1530 and lasted for approximately four years. Again, this disease raged for several years in southern México before spreading northward. Measles is a highly contagious viral disease that is communicable through direct contact as well as aerosol transmission. Reff also notes that measles is both highly contagious and lethal among virgin soil populations with mortality rates as high as 50%. This mortality rate is exacerbated by concurrent infections of dysentery and/or typhoid.

In 1545 another epidemic hit the Greater Southwest region, the symptoms of which suggest typhus. Typhus is a group of related diseases caused by bacteria of different species of Rickettsia and are transmitted to humans by various arthropod
vectors, such as lice and fleas. The toll of this epidemic may be as high as five sixths of the native population of the New Spain region (Reff 1991).

By 1550 Reff (1991) claims that the aboriginal population of central México had been reduced by at least half. Those who survived presumably acquired active immunity to many of the aforementioned diseases. For a period of approximately thirty years (1548-76), there is a general epidemiological calm for the region. During this time, Spanish silver mining activity along the eastern slopes of the Sierras led to the development of extensive transportation networks which linked central México with the Greater Southwest region. It is believed that these networks brought disease agents that had become endemic in the south to the unexposed populations in the north. In 1576 this became apparent in an epidemic of what is believed to be a concert of typhus, typhoid, and dysentery. Jesuit missionary accounts report that the two thirds of the native population died in this epidemic.

Reff (1991) enumerates a number of succeeding epidemics well into the late seventeenth century. The same diseases occur and recur, exacting significant death tolls with each appearance. Based on estimates of population sizes in 1500 compared with 1764 in seventeen different regions of the Greater Southwest, the loss of life due to introduced disease is approximated at 954,000 people, which is roughly 89% of the estimated initial population. In central México, the estimate is more dramatic since this area had much higher population densities and interconnected trade networks. According to Reff (1991), the population of this region declined from 25 million to less than one million over the same time span. This is an almost 99% reduction in population. It is important to note that these population declines are not due solely to deaths caused
directly by disease. Epidemics of introduced disease also lowered birth rates and increased infant and child mortality rates.

Another region of the Americas affected by the 1518 pandemic of smallpox was the Andean empire of the Inca. Notably, this disease took the life of then Incan ruler Huayna Capac, as well as his infant son that he had named his heir. This is an example of the deterioration of cultural systems and structures that is noted as one of the effects of epidemic disease, as well as a factor that allowed the Spanish to conquer with greater ease. It is suggested that this disease also traveled along pre-Columbian exchange networks that linked Mesoamerica with Andean societies (Cook 1998). The Andean region also experienced the measles epidemic of 1532. Cook (1998) also mentions that the number of deaths caused by these epidemics is exacerbated by the lack of social support for the ill, sudden and swift migrations leaving areas nearly abandoned, disruption of kinship structures due to death and migration, and starvation due to the lack of those able to procure and produce food.

Hawai‘i

European contact had equally devastating effects on the native populations of Hawai‘i. Bushnell (1993) proposes two hypotheses, neither of which he finds provable, to explain Hawaiian susceptibility to introduced infectious disease. The first of these is that a significant portion of the native population may have been inbred to the point that they were genetically predisposed to experience great suffering from contagious diseases introduced by Europeans. This hypothesis is derived from the assumption that initial settlers of the Hawaiian islands were a relatively small group, making the entire population descendants from a very small gene pool. Bushnell also suggests that genetic drift may have contributed to a genetic deficiency that causes
its inheritors to produce insufficient amounts of human lymphocyte antigens (HLA) which are part of the human immune response system.

The second of these hypotheses is that before 1778, Hawaiians were not acquainted with the major infectious diseases that were common in other areas of the world. Like the authors that discuss native populations of the Americas, Bushnell does not suggest that Hawaiians were completely disease-free. The biogeographic patterns of macroscopic animal and plant diversity in the Pacific show that diversity declines significantly between the regions of Near Oceania (from Papua New Guinea east to the edge of the Solomon Islands) and Remote Oceania, which includes the region of Polynesia. Kirch (2000) notes that microorganism diversity follows these same patterns, suggesting that the islands within Remote Oceania harbored fewer species of pathogenic organisms. He attests to this through his own field experience, as well as the numerous diseases, including malaria, dysenteries, and parasites that afflict populations in the Near Oceania region (Kirch 2000). However, any germs that coexisted with early Hawaiians would have become adapted to their hosts, making infection without disease more common (Bushnell 1993). In support of this hypothesis, one of the six factors that must exist for an epidemic to arise is that there must be a relatively high proportion of susceptible individuals in the exposed population (Bushnell 1993).

Despite the reasoning, it is well documented that Cook’s first voyage to the Hawaiian Islands in 1778 had traumatic health consequences for the native population. One of the best-documented repercussions of this anchorage is the venereal diseases of gonorrhea and syphilis that struck with acute virulence. Bushnell (1993:145) notes the written thoughts of Captain Clerke upon the second visit to the Hawaiian archipelago in 1779:

Here are many of these good Folks both Men and Women about the Ship miserably afflicted with the Venereal disease, which they accuse us of introducing among them during our last visit, they say it does not go away, that they have no Antidote for it, but
that they grow worse and worse, explaining the different symptoms in the progress of the disorder till it totally destroys them....It is certainly a most unfortunate and ever to be lamented incident...”

Interestingly, Bushnell (1993) notes that the inhabitants of Tonga and Tahiti were also introduced to these same diseases as a result of contact with Cook’s crew. However, Bushnell believes that the effect of syphilis in these islands may have been mitigated by endemic exposure to a related spirochete organism that causes yaws. Additional introduced diseases to Hawai‘i include dysentery, meningitis, measles, whooping cough, smallpox, typhoid, and influenza (Stannard 1990; Bushnell 1993). Recorded epidemics include typhoid in 1804, measles, whooping cough, influenza, and dysentery from 1848-49, and smallpox in 1853 (Stannard 1990).

Bushnell (1993) also asserts that tuberculosis was brought to the Hawaiian Islands via the several ships and crew members who came through. Captain Charles Clerke and Surgeon William Anderson, both members of Cook’s crew, were reportedly in advanced stages of tuberculosis when they landed the second time in 1779. A surgeon by the name of Archibald Menzies, part of Captain George Vancouver’s expedition in 1792, describes a Native Hawaiian:

...lying on a litter in a double canoe. A chair was lowered down for him in which he came into the ship, and appeared very weak and emaciated from a pulmonary complaint that now produced hectic symptoms, for which I gave him some medicines, accompanied with some general directions how to manage his complaint.

Again in 1793, a member of Vancouver’s crew describes a Hawaiian woman as “Only a few years ago...she was one of the handsomest little girls on the island, she was now indeed wonderfully altered, she was in appearance far gone in consumption...” (Bushnell 1993:278)

The population decline brought by these diseases is difficult to assess. Again, it is dependent on unreliable documented accounts of explorers, missionaries, and various projection methods. Like the situation in the Americas, contact with explorers and missionaries that may
have taken head counts often occurred decades after initial European arrival. The interim, then, becomes a matter of great dispute (Kirch 2000).

The effect of diseases brought through European encounters also varies based on the contexts within which they occur. The size, distribution, and character of populations, as well as settlement patterns, social organization, and levels of subsistence all help shape the outcome of European contact. For example, Samoans did not suffer as severely as Hawaiians, and this is attributed to the different patterns of land seizure by the colonizing Europeans (Jones 2003). Similarly, the Navajo fared better than the neighboring Hopi because their pastoral lifestyle was more easily adapted to the challenges posed by European settlers (Jones 2003).

While there are competing opinions for most islands of Polynesia as to actual population size and subsequent decline, no area is more zealously debated than the islands of Hawai‘i. Population size on the eve of European contact has been estimated between 110,000-150,000 (Dye 1994) to 800,000-1 million (Stannard 1989). Stannard’s vastly larger estimate is based on a number of factors, including accounts of early visitors and residents, archaeological evidence that speaks to “carrying capacity,” revisions in population statistics for natives of the Americas that favor larger numbers than previously accepted, analogous situations derived from population data in other regions of the world, and finally the rapid population declines of aboriginal peoples due to European colonization throughout the world. In the most recent publication by Kirch and Rallu (2007), the matter is still unresolved for Hawai‘i. Whatever the numbers may be, by 1885 the Hawaiian population had been reduced to 40,000 (Jones 2003).

It remains undisputed, however, that diseases introduced by Europeans had a devastating effect on the population, both demographically and socially. Even with the low-end estimate of Dye (1994), this represents a decrease of approximately 63%, which is still significant. In
addition, like many aboriginal American populations, the Marquesan people on the island of Nukuhiva experience a population decline from 17,700 to 5,000 (by 72%) in the century following initial European contact (Bushnell 1993).

A number of peripheral effects contribute to the overall devastation caused by introduced diseases. For example, the breakdown of traditional cultural taboos and protocols that maintained sanitation and hygiene may have contributed to the increased spread of infectious pathogens. Also, with disease and death, the family system and structures deteriorated, which may have impacted care-giving practices and child care. Venereal diseases affect fertility and child mortality so that the birth rate continually decreased. In fact, Bushnell (1993) notes that after European contact, the birth rate in Hawai‘i never again exceeded the mortality rate. Stannard (1990) also contends that infertility from disease, stress, and malnutrition may have been the primary cause of the catastrophic population decline in both native Hawaiian and American populations. This conclusion is based on continual population decline in Hawai‘i in years where major epidemics were absent. Similar to the situation in the Americas, malnutrition and/or starvation would have been intensified by significantly fewer adult producers, both in agriculture, fishing, and hunting, as well as preparation. Malnutrition can also have adverse effects on female fertility (Stannard 1990). Psychological distress, although difficult to assess or quantify, should also be mentioned as contributing to the infectious process. Current medical opinion notes that stress is an important factor in a weakened immune response (Padgett and Glaser 2003). If this is true today, it is quite plausible that facing cultural demise would produce an analogous form of stress and have similar detrimental effects on immune response.

Tuberculosis during European contact
In attempting to assess available data to evaluate disease introduction into a population, the epidemiology of modern infectious diseases are often used as an analogue for past events. Because the native populations of the Americas and the native populations of Polynesia present similar (although not identical) circumstances in the context of European contact, i.e. “virgin” populations, an assessment of tuberculosis as a significant disease during the period of European contact in North America can serve as a proxy evaluation for a similar event in Polynesia.

It should be noted that this approach presents at least two hurdles that make it problematic. Part of this approach entails assuming that descriptions of modern disease parallel its past manifestations. Ramenofsky (1987) accurately notes that using modern disease as an analogue for past diseases contradicts certain evolutionary principles. Microbes such as bacteria evolve throughout time, sometimes extremely rapidly. Therefore, it becomes problematic to assume that current descriptions and epidemiology of a disease, such as tuberculosis, are entirely applicable to manifestations of this species over three hundred years ago. Secondly, the written information available that includes descriptions of disease around the time of European contact in Polynesia is a questionable source of accurate information. Medical knowledge was not well developed during this time which renders descriptions and identification of disease from these sources somewhat convoluted (Ramenofsky 1987).

With this in consideration, generally host-parasite interactions can become very complex in the infectious disease process. A number of evolutionary pathways can develop between the host and parasite, such as mutual extinction from overly successful colonization of the host, or parasite extinction from effective host immune response. Of particular interest to this discussion, Ramenofsky (1987) notes that another possibility is that parasite and host co-evolve to form a kind of commensal relationship. This can then lead to endemicity of the disease in which the
host population indefinitely maintains a parasitic species. Due to repeated exposure there is a population level of immunity that is maintained. Individuals may experience an infectious episode, but these are isolated events and the population at large is not generally at risk. It should be noted that tuberculosis may need higher population densities to evolve and become epidemic, but the pathogen can persist and be maintained endemically in small isolated populations (Black 1975).

With this taken into account, consider the situation of tuberculosis in the Americas before European contact. Evidence is accumulating that strongly suggests, if not definitively concludes in some regions, that this pathogen was present during this time period (Roberts and Buikstra 2003; Konomi et al. 2002; Rothschild 1996; Clark et al. 1987). This evidence contradicts common notions that the large settled populations that tuberculosis requires did not exist in pre-contact America. However, there were major population centers in North America during late prehistory, and it is in these locations that evidence of ancient tuberculosis is found (Roberts and Buikstra 2003). Roberts and Buikstra (2003) find clusters of archaeological sites, a total of thirty-nine, where at least one probable case of pre-contact tuberculosis is found focus in the mid-continent and the southwestern United States. In the southwest, the earliest examples of tuberculous lesions coincide with the formation of large pueblos and permanent agricultural settlements. Clusters also exist in Perú, Chile, and Colombia. These authors suggest that American tuberculosis may have developed in South America with camelids such as llamas as the reservoir, and subsequently spread to North America by 1000 AD.

Accounts and analyses of diseases that caused severe epidemics among aboriginal American populations, both in the north and the south, are unable to exclusively identify tuberculosis in any specific epidemic. This may be due in part to the pathogenesis and
epidemiological patterns of this disease. This may also be due to the increasing likelihood that tuberculosis was already present in many regions of the Americas, and that consequent outbreaks of tuberculosis were due to co-infection with introduced diseases and possibly stress-related factors. Given its general course of infection and disease development, it is plausible that the multiple effects of European contact allowed a pathogen that was once in epidemiological “check” to reach new epidemic proportions. Descriptions of tuberculosis following European contact in Polynesia reveal interesting similarities. In Hawai‘i there are descriptions of cases that have been identified as tuberculosis that occur after European contact (Bushnell 1993). However, there is a paucity of firm data that discusses actual case load during the time period during and directly following Captain Cook’s landing. This task is further complicated by the concurrent devastation caused by other diseases, such as measles, syphilis, and gonorrhea.

In summary, the Americas and Polynesia both experienced devastation caused by introduced diseases. In addition, reports of tuberculosis post-European contact in both of these regions occur in the midst of the epidemiological havoc caused by other diseases. As the existence of tuberculosis in the Americas has been generally accepted, the similarity between its effects in the Americas and in Polynesia support the hypothesis that tuberculosis was also present in Polynesia before European contact.

The problematic assumptions of “virgin soil”

In the discussion of introduced diseases to unexposed populations, the commonly accepted idea of “virgin soil” populations is not without critique. Jones (2003) notes that claims of “no immunity” in aboriginal populations conflates and convolutes multiple distinct factors, such as genetics, antibody production, as well as compromised immune systems due to psychological, social, and nutritional factors. Jones finds that the actual contribution of genetic
or developmental factors is unknowable. Rather, the fates of these populations in regard to epidemics of Old World diseases are contingent on their physical, economic, social, and political environments. Jones goes on to claim that the epidemics among native populations in the Americas were caused by “the same forces of poverty, social stress, and environmental vulnerability that cause epidemics in all other times and places” (2003:705). The social chaos that ensued with European contact includes famine, overcrowding, warfare and stress that probably left these American natives extremely vulnerable to disease.

While theories of genetics and natural selection have a logical appeal in the explanation of the severe population decline due to new diseases, there is argument that the immune systems of these “virgin soil” populations were equally operational to their European counterparts. Jones (2003) cites a study by geneticist James Neel among the Yanomami of Venezuela and Brazil in which he found no evidence for innate susceptibility to measles. High rates of infection could be attributed to co-infection with other parasites typical of the region. In the case of TB there is evidence for specific genes that affect the behavior of immune system components which may influence an individual’s resistance to initial infection and progression of the disease. These genetic differences may provide mechanisms to describe perceived differences in susceptibility to tuberculosis among different populations.

In the case of native American populations, the evidence for diseases that existed before European contact such as tuberculosis, pneumonia, possibly herpes and chicken pox, calls into question the notion of vulnerability due to the lack of acquired immunities (Jones 2003). In addition, truly protective immunity only applies to a small number of viral infections. Some viruses, such as influenza, can infect the same individual repeatedly and most bacterial diseases produce little or no protective immunity (Jones 2003).
Furthermore, Jones (2003) reviews the ideological implications of "virgin soil" epidemic explanations. Importantly, the inevitability of decimation due to lack of immunity asserts that the outcome was unavoidable. The inherent inferiority of the victims relieves Europeans, and their descendants, of responsibility for the mortality that occurred. In summary, the issue of depopulation is a complex question that is often simplified by calling on immunological determinism. It is important to remember that there are a number of factors associated with disease epidemics that cannot be disregarded in the discussion of European-introduced diseases to the aboriginal populations of the Americas and of Polynesia.
CHAPTER 3
The physical anthropology of tuberculosis

The discussion of tuberculosis in pre-European contact Polynesia is centered on the data collected from the analyses of skeletal samples. The differential diagnoses of tuberculosis in bones dated before 1778 provides the most significant indication that tuberculosis was present and that this possibility calls for further investigation.

Osteological consequences of tuberculosis

It is recognized that tuberculosis infection can leave osteological markers (Zink et al. 2005). Because the bacilli disseminate through the blood, they can establish themselves in any bone of the body. In fact, tuberculosis has been reported in most bones of the body (Watts and Lifeso 1996), although the incidence of skeletal involvement in tuberculosis cases before the advent of antibiotics is approximately 3%-7% (Aufderheide and Rodriguez-Martín 1998; Ortner 2003). However they tend to gravitate toward red bone marrow, most likely due to the high oxygen tension of these areas (Roberts and Buikstra 2003). Because ribs and vertebrae contain a lot of this kind of tissue, these are usually the most commonly affected sites. Once established, the bacilli lead to the formation of tubercles which are distinct from surrounding tissue. In the caseous necrotic center of these tubercles are multi-nucleated cells surrounded by lymphocytes. Encapsulation of these caseous foci can lead to the replacement of the tubercle with scar tissue, calcification, and/or ossification. Generally, infection from mycobacteria of the *M. tuberculosis* complex in archaeological skeletal remains is associated with the presence of lytic lesions of the ribs (although bone proliferation can be observed on the internal aspects of ribs) and thoracolumbar vertebrae, which is often described as Pott’s disease and/or kyphosis. Tuberculous skeletal lesions have been described as “concave, smooth-walled reaction areas that are oval, circular, or coalesced, ranging from 5 to 32 mm diameter in vertebral and long bone articular
areas, but less than 3 mm in rib and nonarticular surfaces” (Aufderheide and Rodriguez-Martín 1998:134). Tuberculosis of the ribs generally involves four to eight adjacent ribs, and rib lesions are more common in the left hemithorax (Aufderheide and Rodriguez-Martín 1998).

In the spine, which is usually noted as the most commonly affected area of skeletal tuberculosis, paradiscal lesions can cause erosion of the cartilage endplate. This can result in the narrowing of the disc space and damage to adjacent vertebrae, and eventually leads to kyphosis, or the collapse of these vertebral bodies. Spinal tuberculosis usually involves up to four vertebrae, bone regeneration is rare, and extravertebral abscesses are common (Aufderheide and Rodriguez-Martín 1998; Ortner 2003).

Additionally, osteomyelitic lesions from tuberculosis can also be found in the ilia, ischia, femora (including the greater trochanter), and tibiae as well as wrist and ankle joints. Presentation in the hip, knee, and other joints is typically classified as tuberculous arthritis, which is caused by the spread of TB through the blood affecting the synovium and causing erosive deforming arthritis (Pietrusewsky et al. 1991; Trembly 1997; Roberts and Buikstra 2003; Leonard, Jr. and Blumberg 2006). The second most frequent (20%) skeletal lesions due to tuberculosis occur in the hip. Destruction of the femoral head, acetabulum, cavitating lesions and large triangular foci with a spongiosa sequestrum in the center are also characteristic. The round ligament is destroyed by the infection and the hip becomes dislocated. Pelvic infection can reveal destruction of the sacroiliac joint with reactive osteosclerosis. Healing with bony fusion can lead to asymmetrical pelvic deformity. Sacroiliac involvement is reported to occur in up to 2% of skeletal tuberculosis cases (Aufderheide and Rodriguez-Martín 1998; Ortner 2003).

The type of bone tissue associated with lesions is also indicative of the possible chronic nature of infection: woven bone tissue indicates lesions active at the time of death, whereas
sclerotic tissue indicates a "healed" disease process (Roberts and Buikstra 2003). It is important to remember, however, that archaeological remains only present the possibility of a differential diagnosis of skeletal tuberculosis due to the absence of supplementary evidence, i.e. bodily fluids such as sputum, presentation of symptoms, etc.¹ There are numerous pathological conditions considered in the differential diagnoses of spinal changes associated with tuberculosis. These include osteomyelitis, actinomycosis, brucellosis, fungal infection, osteoporosis, septic arthritis, and histoplasmosis (Roberts and Buikstra 2003; Buckley 2000; Tayles and Buckley 2004).

Case studies

Evidence of tuberculosis in bone tissue has been established through case studies on both ancient and modern skeletons. Roberts and Buikstra (2003) review numerous reports, both published and unpublished, on the extent of tuberculosis based on archaeological remains from Northern Europe, the Mediterranean, Asia, North America, Central America, South America, and to a lesser extent, the Pacific Island groups of Papua New Guinea, the Solomon Islands, and Tonga. Each of these regions presents data that reflect the presence of tuberculosis determined through osteological indicators from as early as the Neolithic in Poland and Spain up to the 13th century A.D., with the exception of the Pacific Islands (possibly excluding islands of Hawai‘i).

¹ The development of molecular techniques to test for mycobacterial DNA in bone tissue provides a method that allows for specific diagnoses, the details of which are discussed in Chapter 5.
Of particular consequence for this paper is any skeletal evidence for the presence of tuberculosis in Polynesia before European contact. Unfortunately, there is a noticeable lack of data within these parameters. Buckley (2000) published findings from two burial mounds in Tonga considered to be pre-European contact based on the form and size of the burial mounds. While the analyses reveal both proliferative and lytic lesions of the bronchial aspect of the ribs in two adults consistent with pulmonary TB, the author maintains that these lesions do not necessarily indicate the presence of tuberculosis in this sample due to the difficulty in making such a diagnosis based on skeletal data.

Pietrusewsky (1976) also notes two individuals from the Marquesas that exhibit similar vertebral fusion indicative of spinal tuberculosis dated approximately A.D. 1110 to A.D. 1635. These individuals are part of a larger sample of forty-two burials, considered to be well provenienced, excavated from the Hane dune site (MUH-1) on the island of Ua Huka, part of the Marquesas Islands.
The strongest evidence for tuberculosis in Polynesia before European contact is presented by Pietrusewsky et al. (1991) in skeletons from the Honokahua burial site on the island of Maui, Hawai‘i. None of the affected burials are associated with historic artifacts, and in situ charcoal and volcanic glass dating suggest use of this area from A.D. 610 to A.D. 1800. As the time of European contact in Hawai‘i is considered the year 1778 with the arrival of Captain Cook, these burials likely represent a pre-European population. Four individuals exhibit lytic rib lesions which, due to their similarity in appearance, are likely caused by the same agent. According to the authors, the rib lesions appear to fit the pattern of tuberculosis infection. In addition, infectious lesions in the thoracic and lumbar vertebrae, ilia, ischia, femora, tibiae, and greater trochanter in other individuals also suggest tuberculous infection. A total of 16 individuals exhibit possible osteological evidence of pre-contact tuberculosis in this skeletal series from Maui.

The scarcity of substantial data on pre-European tuberculosis in the Pacific can be attributed to a number of limiting factors. Human remains from archaeological sites are often fragmentary and poorly preserved (Roberts and Buikstra 2003). The high levels of degradation and ability to show only a limited range of changes render osseous tissue problematic in diagnosing individuals from the past (Houghton 1996). Burial practices, e.g. cremation, and the local environments can also affect what remains can be excavated and studied. Even if there are suitable osteological samples, the disease load in a skeletal sample does not necessarily represent the health burden of an entire population. Because tuberculosis more commonly affects soft tissue, it may not cause bone changes before a person dies, or the immune system may have been strong enough to resist skeletal/extrapulmonary infection. In Hawai‘i and New Zealand repatriation concerns also limit the availability of skeletal material that can be evaluated.
CHAPTER 4

Archaeology and demographics of pre-European Polynesia

In assessing the possibility that tuberculosis may have existed in the Pacific prior to European introduction, it is necessary to consider supporting factors such as population size, density, and settlement patterns as these are important factors in the epidemiology of tuberculosis. These factors can further scaffold the claim that tuberculosis may have existed in these populations. In order to pursue this task, paleodemographic studies and archaeological data are key sources of information needed to reconstruct the aforementioned features of these past populations. If instances of substantial population density and settlement patterns coincide geographically with findings of skeletal remains that present lesions consistent with tuberculosis, the likelihood of tuberculosis increases.

Pacific paleodemography has been traditionally based on documentary accounts of Western voyagers and missionary census records (Kirch 2007). Archaeological evidence has recently emerged as a second avenue of exploration as an alternative and/or additional perspective in addressing the question of population size and density in various Pacific islands on the eve of European contact. Direct archaeological evidence provides a proxy measure of past populations that can be applied through the categories of osteological demography, settlement demography, dating curves, and productivity/carrying capacity models (Kirch and Rallu 2007). A combination of these applications in pre-European Polynesian populations can offer a more highly resolved representation of the demographic circumstances that may have supported the presence of pathogens such as M. tuberculosis.

Analyses of Polynesian sites

44
Demographic analyses are directed by the locations of osteological findings that suggest the presence of pre-European tuberculosis. For the Marquesas Rallu (2007) uses retrodiction analysis based on an 1887 census to estimate a contact population of 45,000-63,000. Conte and Maric (2007) present a case study of Hokatu Valley on the island of Ua Huka (the Hane dune site is located on this same island, see Pietrusewsky 1976). The authors focus on data presented by surface remains, i.e. length of the sleeping area of thirty-four house sites, as well as ethnographic accounts of Marquesan houses and sleeping habits. The result is a population estimate for this valley of 315 to 473 (high) and 153 to 170 (low).

Buckley (2000) briefly considers the pathogenesis of tuberculosis as a possible cause of some of the resorptive lesions on the ribs from adult samples excavated in Tongatapu, Tonga. Tongatapu is the largest island of the Tongan archipelago and was the political center of the Tongan chiefdom by 950 AD. Burley (2007) notes that population densities in Tonga were most likely the highest in western and central Tongatapu. This is supported by carrying capacity estimates based on traditional sedentary agricultural practices centered on mixed-crop dryland farming, animal husbandry, and highly productive marine resources. These factors yield a maximum population density estimate of between 124 and 221 individuals/km². Burley (2007) extrapolates a pre-Contact population of 18,195 for Tongatapu and 34,057 for the entire archipelago.

Archaeological work in Hawai‘i has revealed important demographic characteristics of these Polynesian populations before 1778. The islands of Hawai‘i supported large human populations in complex societies that were based on intensive agriculture. The factors of climate and soil fertility combined to constrain large dryland agricultural systems and the societies they supported to well-defined portions of the younger islands within the Hawaiian archipelago, while
societies on the older islands were based on irrigated wetland agriculture (Vitousek et al. 2004). Societies supported by intensified agricultural systems most likely present the demographic conditions sufficient for the spread of infectious disease, such as tuberculosis, and Hawai‘i was home to the largest single population of Polynesians (Kirch 2007). Kirch (2007) mentions the use of radiocarbon dates by Dye and Komori as a proxy measure of population that results in a logistic growth curve and an actual pre-Contact population estimate of 141,787 to 192,606, although the methodology is not without critique. Aside from actual numbers, Kirch (2007) asserts that by AD 1600 humans occupied virtually all of the lowland zones throughout the Hawaiian archipelago. He also notes that there was a period of rapid exponential population growth between AD 1100 and 1500. Interestingly, the overall trend in Hawaiian population growth demonstrates a kind of plateau between AD 1500 and 1778, which is attributed to both a leveling off and achievement of stability or perhaps regional reductions in population size. It is expected that during this time period, cultural controls on fertility and mortality would have been most influential, assuming a density-dependent situation (Kirch 2007).

In terms of specific islands of the archipelago, it is the pre-European Maui burial site of Honokahua in western Maui that provides strong osteological evidence for tuberculosis (Pietrusewsky et al. 1991). Meanwhile, the permanent settlements along the southern flank of Haleakala Volcano on Maui suggest a maximum population density of 43-57 persons/km² achieved by A.D. 1700-1800 (Kirch et al. 2004). This settlement is identified as a marginal landscape for Polynesian agricultural practices based on its boundaries of aridity and soil nutrient depletion. Tuljapurkar et al. (2007) note that movement of populations into marginal areas is understood to be a reflection of substantial population increase and Malthusian limits in more fertile areas. Such a sizeable population, which permanently exploited Kahikinui’s resources
despite its marginality for agriculture, testifies to the demographic pressures building within Hawaiian sociopolitical systems in the years immediately before European contact (Kirch et al. 2004).

In addition to Maui, demographic estimates have been made for regions on the island of O‘ahu and the island of Hawai‘i at the time of European contact. Cordy (2007) looks at the districts of Wai‘anae and ‘Ewa on the island of O‘ahu using permanent house sites as a measure of household in conjunction with actual census counts. Contact population estimates range from 4606-6108 people in the Wai‘anae district to 9510 people in the ‘Ewa district. In addition, Cordy (2007) looks at contact population estimates for the Hāmākua district on the island of Hawai‘i and suggests 10,508 people over an area roughly 740 square miles. These data suggest that certain populations in Hawai‘i may have had sufficient numbers and/or densities to support the presence of the *M. tuberculosis* pathogen.

It is surprising to note that the population patterns of Hawai‘i as they relate to tuberculosis are not considered in greater detail in the published literature. The accepted model of tuberculosis epidemiology stems from its rise in Medieval Europe. When people began living in larger communities and population densities increased, TB may have become epidemic. Higher population densities affected how people lived, social class disparities, sanitation, ventilation, piped water, etc. Overcrowding in urban centers also increased the transmission of tuberculosis via droplet infection (Roberts and Buikstra 2003).

Typically, small hunter-gatherer groups do not fit this model and are therefore not likely prospects in finding ancient tuberculosis. These types of societies do not keep domesticated animals (cited as a potential source of TB infection from zoonotic *M. bovis*), and are continuously shifting residence. However, Roberts and Buikstra (2003) note that in modern
foraging populations, such as the !Kung San, tuberculosis has been a significant and common cause of death. In archeological skeletal series, an analysis of a hunter-gatherer population from Kentucky dated to 3000-2000 B.C. indicates that rib lesions associated with lung infection were present in 21.3% of the sample (Roberts and Buikstra 2003). Skeletal evidence from past hunter-gatherer populations is scarce, but it is important to remember that absence of evidence is not evidence of absence. These data suggest that these types of societies, even though they do not conform to the typical epidemiological population patterns of TB, may still be able to sustain this pathogen's existence (see Black 1975).

**Origins of TB in the Americas**

Evidence for the existence of *M. tuberculosis* is present in Europe and western Asia no later than 2000 BC (Ortner 2003; Zink at al. 2004; Zink et al. 2005). At the same time, tubercular-like infections have been found in skeletons and soft tissue that predate European contact in the Americas (Konomi et al. 2002; Buikstra and Cook 1981). This would suggest, perhaps definitively, that tuberculosis did, in fact, exist in the Americas before European contact.

The next inquiry that develops must examine how the same genus of bacillus was present on two or more continents simultaneously. This becomes a bit more difficult to account for. One hypothesis supported by Buikstra and Cook (1981) is that tubercle-causing Mycobacteria independently and autochthonously evolved in North America during the approximate dates of 1400-1540 AD (Ramenofsky 1987). Because tuberculosis is considered density-dependent and Mississippian populations were large and centralized (Ramenofsky 1987), this is plausible. This is further supported by the presence of resorptive spinal lesions from this time period and the absence of these from earlier time periods where settlement patterns were different and
populations were smaller. The difficulty with this possibility arises from the apparent application of the concept of convergence to explain the presence of the same genus/species of pathogen. Convergence is a morphological response to selection by phylogenetically unrelated organisms (Ramenofsky 1987). Therefore, to suggest that the same genus of bacteria causes tuberculosis in each different region contradicts this definition, unless a nonhuman reservoir for Mycobacteria can be found in the Americas. Ramenofsky (1987) notes that domestic cattle may have been the reservoir for tuberculosis in Europe (perhaps an ancestor of *M. bovis*). Buikstra (1981) suggests that bison could have potentially acted in this capacity for American populations.

Interestingly, Rothschild et al. (2001) report the presence of the *M. tuberculosis* complex in a fossilized bison (*Bison cf. antiquus*) metacarpal found in Wyoming dating to approximately 17,000 years BP. This sample displayed lesions indicative of granulomatous infection, as did samples from bighorn sheep and extinct musk ox from the same site. DNA sequencing of the bison sample provides a definitive diagnosis of the *M. tuberculosis* complex.

A second hypothesis that accounts for the presence of tuberculosis in multiple regions entails diffusion from the Old World, i.e. Europe and Asia. This suggests that tubercle-causing Mycobacteria were part of the disease load of the original Asian migrants to the Americas. Because tuberculosis is a long-lived and frequently chronic infection, this is plausible. In addition, this pathogen can be maintained in relatively small and isolated populations (Black 1975; Ramenofsky 1987). However, this hypothesis requires a disease continuity over 10,000 years to when these original migrations took place. Existing evidence for tuberculosis in the Old World dates back only as far as 2000 BC, so this hypothesis must be accepted with some reservation.
Given this information, it seems possible that the ancient bovids that migrated to the Americas across the Bering Strait brought, carried, and acted as the reservoirs for the Mycobacteria that eventually became part of the *M. tuberculosis* complex (Rothschild et al. 2001). With bovid reservoirs on both continents, the hypothesis suggested Buikstra and Cook (1981) that convergent evolution of the tuberculosis pathogen seems increasingly plausible. It is likely, then, that the *M. tuberculosis* complex was already present in North America when the first human settlers arrived (Rothschild et al. 2001).
CHAPTER 5
Molecular biology and tuberculosis

Despite the uncertainty in defining the pre-historic/historic nature of the burials at Honokahua, Trembly (1997) and Roberts and Buikstra (2003) firmly suggest that tuberculosis was present in pre-European contact Hawai‘i. While these claims are based on strong skeletal evidence (see Pietrusewsky et al. 1991), this evidence can at best attest to the strong likelihood of the presence of this disease. In order to narrow the margin of uncertainty as to the presence of \textit{M. tuberculosis} in pre-contact skeletal samples, the field of molecular genetics offers strong support.

**Tuberculosis genome**

In 1998, Cole and colleagues published the complete genome sequence of \textit{Mycobacterium tuberculosis}. These researchers used the H37Rv strain of \textit{M. tuberculosis}, first isolated in 1905, which has extensive application in biomedical research because it has retained full virulence in animal models of tuberculosis, it is susceptible to drugs, and it is amenable to genetic manipulation. Like most prokaryotes, the DNA of \textit{M. tuberculosis} is structured as a single circular chromosome. The entire sequence of this bacterium’s genome consists of 4,411,529 base pairs. This is the largest bacterial genome sequence currently available, second only to that of \textit{Escherichia coli}. In addition, this genome has a guanine-cytosine content of 65.6%. This relatively and unusually high percentage of G-C pairs is significant because these base pairs are able to form three hydrogen bonds with each other instead of two, as is the case with the other complementary pair of adenine and thymine. The three hydrogen bonds increase the strength of the double helix because these are more difficult to hydrolyze, or degrade, and this contributes greatly to the DNA’s temporal stability.
The decoding of this bacterium's genome has revealed interesting and important genetic components related to how this organism provokes disease in its host. Two genes encoding hemoglobin-like proteins, which may protect against oxidative stress or be involved in oxygen capture, were found. This may allow the bacillus to adapt its metabolism to environmental change. This is significant because the bacillus not only has to compete with the lung for oxygen but must also adapt to the low-oxygen and/or anaerobic environment at the core of a proliferating granuloma. The presence of storage proteins in the bacillus, such as these hemoglobin-like oxygen captors, points to its ability to stockpile essential growth factors, thus allowing it to persist in the nutrient-limited environment of the granuloma. In addition, *M. tuberculosis* is naturally resistant to many antibiotics, making treatment difficult. Cole and colleagues (1998) believe that this resistance is due primarily to the highly hydrophobic cell envelope, which acts as a barrier to permeability. However, many potential resistance determinants are also encoded in the genome. These include hydrolytic or drug-modifying enzymes and many potential drug efflux systems.

These researchers also find that the cell envelope of *M. tuberculosis* contains an additional layer beyond the peptidoglycan that is exceptionally rich in unusual lipids, glycolipids, and polysaccharides. Cell wall components such as mycolic acids, mycocerosic acid, phenolthiocerol, lipoarabinomannan and arabinogalactan may contribute to this mycobacteria's longevity, play a role in triggering inflammatory response in the host, and act in pathogenesis. Little is known about the mechanisms involved once the bacillus is within the macrophage. The extent, nature, and source of virulence factors produced by the bacillus and their contribution to disease are also somewhat enigmatic.
It is thought that the progenitor of the *M. tuberculosis* complex, comprising *M. tuberculosis, M. bovis, M. canetti, M. africanum,* and *M. microti,* arose from a soil bacterium and that the human bacillus may have been derived from the bovine form following the domestication of cattle (Cole et al. 1998). This complex lacks overall genetic diversity and nucleotide changes are very rare. This may prove to be important in terms of immunity and the development of a vaccine, as most of the proteins will be identical in all strains and antigenic drift will be restricted. From this, it is concluded that the genome of *M. tuberculosis* is either unusually inert or that the organism is relatively young in evolutionary terms.

Some factors that may contribute to virulence are becoming apparent. Before the completion of the genome sequence, only three virulence factors had been described. These include catalase-peroxidase, which protects against reactive oxygen species produced by the phagocyte in an effort to eradicate the pathogen. There is also a macrophage-colonizing factor, as well as a sigma factor gene, mutations in which can lead to attenuation. In addition to these single-gene virulence factors, the mycobacterial cell wall is also important in pathology but the complex nature of its biosynthesis makes it difficult to identify key genes whose inactivation would lead to attenuation. From the genome sequence, a number of secreted proteins have been identified that could act as virulence factors. These include a series of phospholipases C, lipases, and esterases, which might attack cellular or vacuolar membranes.

**Polymerase chain reaction**

The use of molecular genetic techniques, such as polymerase chain-reaction (PCR) amplification, is a burgeoning field in laboratory and retrospective disease diagnosis, confirmation of diagnosis based on skeletal changes, as well as phylogenetic studies of microorganisms themselves. Since its development in the 1980s, PCR has provided a relatively
quick and inexpensive method to replicate a given nucleotide sequence within any strand of DNA that contains it (Mullis and Faloona 1987). This technique utilizes the enzyme DNA polymerase, which is a naturally-occurring enzyme required and used in DNA replication. Normally, the DNA double helix is “unzipped” and the polymerase synthesizes a new strand of nucleic acid along the original strand, which now acts as a template. The polymerase achieves accuracy through the principle of complementary base pairs within DNA. This particular enzyme also requires a primer in order to carry out the synthesis, which in PCR is an oligonucleotide designed by the experimenter, although normally in a cell it is a small segment of RNA (Mullis and Faloona 1987).

The DNA is heated to 94-98°C for a specified time interval, usually less than one minute. This denatures the DNA double helix by disrupting the hydrogen bonding between complementary base pairs. Then the reaction mixture is cooled to 50-65°C for less than one minute. This allows the oligonucleotide primer to anneal, or hybridize, to the now single-stranded DNA in the location specified by the sequence of the primer. It is during this phase that the polymerase carries out synthesis of a new DNA segment. This thermal cycle is repeated multiple times thus producing an adequate amount of a single DNA segment which can then be optimized with gel electrophoresis and other techniques. Due to the thermal cycling used in PCR technique, it is usually the polymerase from a thermophilic bacterium (Thermus aquaticus) called “Taq” that is used (Mullis and Faloona 1987).

The diagnosis of tuberculosis is often dependent on laboratory culture and/or tissue samples, in conjunction with clinical presentation. Due to the slow growth of this organism, this method is not ideal in terms of expedient diagnoses. Many researchers suggest the use of PCR to test for the detection of mycobacterial sequences in clinical specimens (Lucchini and Altwegg
1994). Although the cell wall composition of *M. tuberculosis* makes lysis, and hence access to the DNA, difficult, Lucchini and Altwegg (1994) propose alternative methods for isolating the DNA which are successful in their trials. The use of PCR in this way results in a sensitivity of 92%.

**PCR and ancient DNA**

While PCR and other techniques can be utilized to diagnose clinical specimens, these techniques can also be employed in significant research on the ancient DNA (aDNA) of *M. tuberculosis*. In addition to phylogenetics and population genetics, the DNA of animal and plant remains can reveal clues as to social and agricultural practices. Coprolites (ancient feces) can give information about health status and diet. Nuclear DNA from human beings can reveal their sex, which may not be able to be determined from skeletal morphology. Analysis of mitochondrial DNA can provide information about kinship relatedness and the data to test hypotheses on population migrations in prehistory (Donoghue et al. 2004). However there are numerous challenges and complications unique to working with ancient DNA. Within living cells, the integrity of DNA molecules is continually maintained by enzymatic repair processes. After the death of an organism, cellular compartments that normally sequester catabolic enzymes break down. As a consequence, the DNA is rapidly degraded. In addition, normal decomposition involving bacteria, fungi, and insects continue to degrade the DNA. Under certain circumstances, such as rapid desiccation, low temperatures or high salt concentrations, the DNA can become adsorbed to a mineral matrix and/or nucleases (enzymes that break down nucleic acids) can themselves be destroyed or inactivated before all nucleic acids are reduced to mononucleotides (Hofreiter et al. 2001; Pääbo et al. 2004). In these cases the DNA may escape enzymatic and microbial degradation. On such occasions, slower but still persistent chemical
processes start affecting the DNA. Many of these processes are similar or identical to those that affect the DNA in the living cell, such as depurination and deamination. However, once the organism has died, these processes are no longer counterbalanced by cellular repair processes. The damage accumulates progressively until the DNA loses its integrity and decomposes. This results in the irreversible loss of nucleotide sequence information. It is now possible with PCR to salvage the information from the rare samples in which the disintegration of DNA is not yet complete. Yet even with ideal preservation conditions, the upper limit for PCR studies on ancient DNA is approximately 100,000 (Donoghue et al. 2004) to one million years (Hofreiter et al. 2001; Pääbo et al. 2004). It should be noted that in the context of Polynesia, the upper limit of human-related aDNA presence is 3000 years, so this does not become an issue.

In the case of ancient DNA, available fragments are generally between 100 and 500 base pairs in length. The reduction in size is due to both to the processes mentioned above as well as the nonenzymatic hydrolytic cleavage of phosphodiester bonds in the phosphate-sugar backbone that produce nicks in the strand. The glycosidic bonds between nitrogenous bases and the sugar backbone are also subject to hydrolytic cleavage that results in abasic sites. These sites are gaps in the chain that resemble missing teeth. The length of the DNA sequences that can be amplified by PCR is limited also by lesions induced by free radicals such as peroxide radicals, hydrogen peroxide, and hydroxy radicals. These lesions present blocks to the elongation of DNA strands by the Taq polymerase. Oxidative attack occurs commonly at the double bonds of both pyrimidines and purines, leading to ring fragmentation. In addition, the chemical bonds of the deoxyribose residues are susceptible to oxidation resulting in fragmentation of the sugar ring (Pääbo et al. 2004).
In addition to fragmentation and DNA modifications that impede the action of DNA polymerases, there are other types of damage are common in ancient DNA. Some of these are problematic because although they allow the amplification of the template molecules, they cause incorrect bases to be incorporated during the PCR. The most common form of such modification is the loss of amino groups from the bases adenine, cytosine, and 5-methyl-cytosine. The resulting bases become hypoxanthine, uracil, and thymine respectively. These deamination products are of particular importance for the amplification of ancient DNA since they cause incorrect bases (A instead of G, and C instead of T) to be inserted when new DNA strands are synthesized by a DNA polymerase (Pääbo et al. 2004).

Further complications in the study of ancient DNA involve the little DNA that survives in ancient tissues compared with contemporary DNA that is pervasive in both the external and internal laboratory environment. Therefore, stringent precautions need to be taken to avoid the presence of extraneous DNA in the PCR. The greatest difficulties are encountered in the study of human remains. Human DNA is particularly prevalent in the environment of laboratories and museums, and cannot easily be distinguished from the DNA endogenous to ancient human remains. Therefore, contamination of ancient specimens with contemporary human DNA is a significant issue (Hofreiter et al. 2001; Donoghue et al. 2004; Pääbo et al. 2004). Ancient animals pose less of a problem as the sequences are more distinct from humans. The sequences retrieved from ancient animals are often testimony to their own authenticity if they are distinct from, yet related to, extant species in the same taxonomic group (Hofreiter et al. 2001). Also, multi-copy DNA sequences, like those from organelles such as mitochondria and plant chloroplasts, are more likely to survive in ancient specimens, most likely due to their overall greater frequency per cell. Therefore, most ancient DNA sequences that have been retrieved are
from mitochondria in mammals and from chloroplasts in plants. This creates a near complete reliance on mitochondrial DNA sequences for phylogenetic studies of extinct animals. This is not problematic for species not very closely related to each other because after sufficient time between speciation events, all parts of the genome are expected to show the same phylogeny. However, when closely related species or population genetic questions are studied, it is important to remember that the mitochondrial DNA represents a single genetic locus that may or may not reflect the overall history of the genome (Hofreiter et al. 2004).

**Tuberculosis aDNA**

Mycobacteria, including the tuberculosis complex, are the ideal microorganisms for studying the aDNA of pathogens (Donoghue et al. 2004). Lesions left in the bone often contain residual microbial DNA, which due to the high levels of guanine and cytosine is more stable and can survive longer than other nucleic acids, due in part to their ability to form three hydrogen bonds instead of two. In addition, the lipid-rich cell walls of mycobacteria protect the DNA from enzymes and other processes that may act on the host post-mortem. Therefore, mycobacterial DNA can be detected even when genomic and mtDNA of the host is severely degraded. This is particularly significant for the possibility of recovering pathogen aDNA from skeletal samples in the Pacific that present severe degradation, as is often the case.

*Mycobacterium tuberculosis* aDNA (MTB aDNA) has been extracted and identified from a number of ancient non-European samples, including Egyptian and South American mummies (Konomi et al. 2002; Zink et al. 2004). DNA of tuberculosis was recovered and identified from tissue samples of South American mummies dating from A.D. 140 to 1220 (Konomi et al. 2002). Zink et al. (2004) describe ancient tuberculosis research conducted on a number of Egyptian mummies using bone tissue. In order to identify the skeletal remains that might contain MTB
aDNA samples, various morphological characteristics are sought. These include pleural adhesions to the chest wall, and severe spondylitis with intraosseous lytic destruction and kyphotic collapse of vertebral bodies in the lumbar and thoracic vertebral column. MTB DNA amplification and spoligotyping techniques are employed and provide the ability to characterize a small series in these Egyptian cases.

But skeletal evidence of pathological morphology is no longer a necessary condition for extracting MTB aDNA (Baron et al. 1996; Zink et al. 2005). Work by Baron and colleagues (1996) indicates that MTB aDNA can be extracted from unremarkable bone tissue as well. In this study, bone tissue that did not show any tuberculosis lesions was assessed for the aDNA of *M. tuberculosis*. Because the pathogen travels through the blood, DNA should be retrievable from any bone tissue. According to these researchers, individuals who exhibit tuberculosis lesions in certain areas should have tuberculosis DNA in unremarkable bone tissue as well. Using PCR amplification techniques, the authors detect *M. tuberculosis* complex-specific DNA in bone tissue without lesions. The detection of tuberculosis-specific DNA from bone tissue without lesions is significant because this could potentially increase the sampling potential for ancient tuberculosis exponentially, as well as provide a means to test bone tissue that does not display pathogenic morphology. In addition, the ability to find MTB aDNA in unremarkable bone tissue could shed greater light on the true disease load of a sampled population.

In addition to the possibility of identifying tuberculosis genetically (and allowing for more certainty in its diagnosis), molecular genetic studies of ancient tuberculosis also provide important genomic information about this pathogen. The sequencing of the tuberculosis genome may reveal deletions that are associated with virulence, which may have important implications on host-pathogen relationships. Genetic “fingerprinting” of different strains using a particular
DNA polymorphism has been instrumental in describing particular strains and the various epidemics they cause (Van Soolingen et al. 1991).
CHAPTER 6

Discussion of the evidence

The areas of physical anthropology, archaeology, and paleodemography highlight multiple aspects that support the presence of tuberculosis in Polynesia before European contact. This section attempts to articulate these holistically and mention additional contingencies and possibilities that should be considered.

Demography

Population size, density, and settlement patterns in Hawai‘i on the eve of European contact are somewhat disputed. Stannard (1989) contends that the population of the Hawaiian islands on the eve of contact could easily have been as high as 800,000 to 1,000,000 people. However, more conservative estimates support a contact-era population of between 110,000 and 250,000 (Kirch 1990; Dye 1994). Kirch (1990) notes that although the average population density may have been approximately 120 persons/km², local population densities could be significantly higher, e.g. 250 persons/km² in Halawa Valley on the island of Moloka‘i. These population estimates rival those of intensive irrigation regimes of the Asian tropics. Intensified production systems such as irrigation, dryland field agriculture, and aquaculture, supported general and local population concentrations significantly greater than that of societies such as the !Kung San, whose population density is estimated at 6 persons/km² (Minnis 1985). If small hunter-gatherer groups, such as the !Kung San, are capable of sustaining \textit{M. tuberculosis}, it is conceivable that the higher population densities of Hawaiian groups centered on intensive agriculture may have been able to maintain the presence of \textit{M. tuberculosis} as well.

Skeletal lesions
It is particularly interesting that tuberculous changes seen in the skeleton are usually chronic healed lesions which may indicate slow development of the disease and/or a strong immune response that is able to resist acute infection and death. Roberts and Buikstra (2003) note that a population with no previous exposure to infection will succumb very quickly to death. A pathogenic organism like *M. tuberculosis* can cause an epidemic when introduced into a population in which only a small portion is immunologically protected. The TB epidemic pattern indicates that at its peak, morbidity is still greater than mortality. This allows for the population to gradually acquire adequate immune response (Dutt 2006). After generations of exposure the immune response strengthens, allows survivability and, consequently, visible changes in the bones of an individual. Based on this information, it would seem that the lesions present in the skeletal sample from the burials at Honokahua indicate a long-term presence of tuberculosis. Pathogenesis of skeletal TB is related to reactivation or spread from adjacent paravertebral lymph nodes, which are usually infected after primary infection of the lungs has already taken place (Raviglione and O’Brien 2005). If tuberculosis had been a recent introduction at the time of death for these 16 individuals (see Pietrusewsky et al. 1991), it is most probable that no skeletal changes would be apparent as they would have died before these could be sustained.

Another consideration is the latency of TB infection. Pietrusewsky and Douglas (1994) note that there is an approximate 40-year gap between actual European contact in 1778 and cultural changes reflected in burial practices. This results in some ambiguity when dating remains from these temporal parameters. The findings at the Honokahua burial site on Maui (Pietrusewsky et al. 1991) date to this time period, so although the burial context indicates pre-European, it is not necessarily so. This ambiguity obscures the legitimate possibility that the
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61
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lesions associated with TB found in this sample may, in fact, be caused by a tuberculosis pathogen introduced before contact with Europeans in 1778.

It has been noted that an individual may not present symptoms while infected with *M. tuberculosis* for decades. While it is possible that these remains reflect post-contact bone lesions, it is equally possible that these lesions are manifestations of a chronic and/or latent development of tuberculosis infection that was present before contact. It is important to remember that the skeletal evidence does not necessarily reflect disease events that occur at or near the time of death. In the case of tuberculosis, bony lesions may have been formed years before death. Proliferative lesions on the pleural side of the ribs suggest acute infection, while sclerotic fused collapsed vertebrae are usually indicators of chronic infection. This suggests a considerably longer timeline from infection to osteological presentation. If this is the case, this pushes back the time of infection even further to a point that is conceivably before 1778, even among the sample from Honokahua.

**Diet of Polynesians**

It is also possible that immune responses among these Polynesians were adequate to resist acute infection causing a more chronic appearance of the disease. Diet is considered an important factor in the strength of immune response and the ability of an individual to resist infection. The diet of Polynesians is noted to be highly adequate in nutrition (Houghton 1996). Miller (1974) reviews in detail the principal foods of Hawaiians based on explorer accounts, writings of old Hawaiians, recollections of modern Hawaiians, and other ethnographic observations. Miller then assesses the nutritive value of these foods based on their ability to provide calories, protein, minerals, and vitamins. It is concluded that although the diet of ancient Hawaiians may have been lacking in great variety, it was of sufficiently high nutritive value to
promote and maintain good health. Because nutrition or lack thereof is an important factor in an individual’s ability to contain an tuberculosis infection (Dubos and Dubos 1952; Bushnell 1993; Roberts and Buikstra 2003), it is possible the overall high level of health and nutrition of most Hawaiians prevented tuberculosis from reaching epidemic levels in a pre-contact setting.

**Summary**

Tuberculosis is often grouped with a number of other diseases, such as influenza, syphilis, and cholera, as being responsible for the dramatic and rapid population decline of Native Hawaiians after European contact. It is then usually concluded that this must indicate high virulence of the tuberculosis pathogen, which in turn indicates no prior exposure of this population to the disease. It is thus assumed that tuberculosis must have been a European introduction. However, there are documented accounts of explorers who name venereal disease as a “ravaging epidemic decimating the native population” (Stannard 1989). There is also mention of “probably the worst epidemic ever recorded in Hawai‘i- the catastrophic ma‘i ‘oku‘u or “squatting sickness- variously thought to have been cholera, typhoid fever, or bubonic plague” (Stannard 1989:55). The diseases that had dramatic effects on the population and could be identified do not include tuberculosis. Tuberculosis is implicated in the decimation of the native population based on circumstantial evidence. Stannard (1989) notes that Cook’s ships brought “tuberculosis, an influenza virus or some other [my emphasis] deadly upper respiratory infection…” (70). In addition, Cook left England at a time when tuberculosis morbidity was peaking, and so it is assumed that the epidemic is carried over to the islands he visited, including Hawai‘i.

This is not to say that Cook and his sailors did not bring TB with them, nor is this meant to say that TB was not an important disease in the depopulation of Hawai‘i and other Pacific
Islands. The point is that tuberculosis could have already been present. Increased prevalence of TB after European contact could be due to: 1) compromised immune function due to the exposure to other truly new virulent diseases, such as influenza, cholera, etc. 2) prolonged contact with highly infectious cases, i.e. Cook's sailors (prolonged contact is necessary to acquire infection, see Roberts and Buikstra 2003), 3) the introduction of a different, perhaps more virulent strain of TB, 4) disruption of nutritional patterns and collapse of social structures, and/or 5) physiological, cultural, and psychological stressors associated with colonization which could have weakened immune systems.

**Alternative origins for TB in Polynesia**

Trembly (1997) suggests the possibility that TB may have been introduced to Hawai‘i by shipwrecked Spaniards before Cook. It is plausible that two of Saavedra’s ships may have wrecked in Hawai‘i in 1528. However, there is no official report made in Spanish history of this event. In addition, there is no trace of Spanish influence in religious customs, art, or language, nor have there been any Spanish artifacts found in Hawai‘i. This postulated introduction of TB is therefore possible, but it is with little empirical support.

In the exploration of possible pre-European Polynesian contacts, the issue of the sweet potato (*Ipomoea batatas*) must be addressed and considered. It has been well-established that the sweet potato was first domesticated in the Americas, most probably in Central or northern South America, i.e. northern Peru (Ballard 2005; Green 2005). European explorer accounts attest to the well-established presence of this crop upon their arrival, most notably in the Society Islands, Mangaia (Cook Islands), Rapa Nui, and Hawai‘i. In addition, archaeological evidence of the sweet potato in Hawai‘i dates to 1358-1626 AD and in New Zealand to 1150-1250 AD,
and charred sweet potato has been positively identified in Mangaia and dated to approximately 1000 AD (Ballard 2005; Green 2005).

With these significant indicators, the question then becomes how did the sweet potato get to Polynesia? Ballard (2005) notes that there is a limited scope of natural dispersal for this plant, especially over the distances between Polynesian islands and the Peruvian coast. Green (2005) concurs that human agency is the only credible method for this to have taken place. A "tripartite hypothesis" has been proposed to explain the patterns of plant variation found in the Pacific (Yen 1974 as cited in Ballard 2005). This hypothesis proposes a prehistoric movement from Peru to central Polynesia, movement from the Caribbean to Southeast Asia in the 15th and 16th centuries, and from México to the Philippines during the 16th century. Importantly, these proposed movements create hubs of secondary distribution, the Marquesas being at the core of the Polynesian hub. Diffusion from here would have been to Hawai‘i, New Zealand, and Rapa Nui.

In spite of Heyerdahl’s Kon Tiki expedition in 1947, there is general consensus that it was Polynesian rather than American voyagers who are responsible for the initial sweet potato transfer. Green (2005) suggests a Polynesian voyage launched from the area between Mangareva and Rapa Nui. He also cites the Hokule‘a, the Polynesian experimental sailing canoe, as undertaking the voyage from Mangareva to Rapa Nui which supports this claim. According to Green, this journey should take about seventeen to twenty days, and conjectures that from there to the South American coast would take no more than another month. It is proposed that since the Mangareva-Temoe-Pitcairn interaction sphere was settled by 900 AD, it would be feasible to have accomplished a voyage to South America in the following few centuries. Once this first trip was complete, it is very likely that it would have been repeated.
Although the evidence is slight in this regard, Green (2005) notes that this possibility should not be disregarded.

In addition to the logistic possibilities, Green (2005) reviews extensive linguistic evidence that points to sweet potato introduction via Polynesians. The Peruvian and Ecuadorian word for sweet potato, “kumar,” is the very likely root of the proto-word “kuumala” that numerous Polynesian cultures use for this crop. For example, the initial Marquesan form would have been “kuumara,” “umara” in Tahiti, and “kuuara” in Mangaia.

In summary, Green (2005) proposes an initial introduction of sweet potato to the Cook Islands around 1000-1100 AD, and from there diffused to New Zealand by 1150-1250 AD, Hawai‘i by 1100-1200 AD, and to Rapa Nui by 1300 AD. It is most likely Polynesian voyagers that sailed to the coast of South America, most likely Peru and/or Ecuador. It is also likely that repeated voyages were made.

Although the migration of the sweet potato is not directly analogous to the movement of a pathogen like *M. tuberculosis*, the evidence discussed above presents a strong case for contact between Polynesians and various South American populations before European contact in either region. The presence of tuberculosis in Peru is established through PCR analysis of tissue samples from twelve mummies in the American Museum of Natural History collection in New York. The mummies were excavated in the Andes Mountain region of South America, and radiocarbon dating estimates that the mummies date from A.D. 140 to 1200. DNA extracted from these tissues reveal the *M. tuberculosis* complex (Konomi et al. 2002). Therefore, it is important to consider that tuberculosis may have been introduced to Polynesia before European contact via this link.
Conclusion

The global significance of infectious diseases, such as tuberculosis, calls for a multidisciplinary approach to understanding the complex interaction between sociocultural, biological, and ecological factors in the etiology and prevalence of these diseases. Medical anthropology applied through bioarchaeology allows for a multi-faceted approach to health and disease questions in the archaeological record. By using these techniques and methods to reconstruct cultural behavior and past disease events, the efficacy of these behaviors, as well as the antiquity of these pathogens, can be assessed vis-à-vis the evidence available.

A review of the physical anthropological data and archaeological data present factors that indicate the possibility that *M. tuberculosis* was present in Polynesia, i.e. Hawai‘i, before European contact. The strongest osteological evidence comes from a prehistoric burial site on Maui. While the prehistoric classification of this site can be debated, it is still significant that there is osseous presentation of TB. These skeletal markers point toward the possible chronic nature and/or decreased virulence of the disease in this skeletal series. If this is the case, this would indicate long-term presence; otherwise the immune response would be insufficient to prevent acute infection and fairly rapid death (which would prevent development of bony changes).

In addition to skeletal data, there is supporting archaeological data concerning the population size and density of some areas, such as Hawai‘i, which contribute to the likelihood of the presence of pathogens like *M. tuberculosis*. The accounts of early explorers neither support nor refute the presence of TB before their arrival. Due to the pathogenesis of tuberculosis— the need for prolonged exposure, susceptibility due to stress, malnutrition, and compromised immune function— it is possible that explorers either introduced a more virulent strain of TB or
created conditions that allowed an already existing pathogen to have greater health
consequences.

In order to narrow the margin of uncertainty in regard to the presence of tuberculosis
before European contact, molecular genetic tests should be carried out on available osseous
samples. Ironically, it is in Hawai‘i that the strongest possibility of tuberculosis is presented, and
it is also one of the few places in the Pacific where studies of Native Hawaiian skeletal series are
no longer possible at present. However, skeletal samples from other areas of the Pacific are still
available for analysis. Samples from French Polynesia, Tonga, and the Marquesas may provide
important molecular data which could be extrapolated, hypothetically, to Hawaiian populations.
In addition, if the biomolecular presence of TB can be established in pre-European populations
in the central and western regions of the Pacific, it would be possible to make inferences based
on peopling events and migrations as to the likelihood of its presence in Polynesia.

In the paleopathological study of tuberculosis in the Pacific, it is important to suspend the
assumptions often made and review the available evidence, considering the possibility that
tuberculosis could have been present before European contact. The lack of definitive proof or a
conclusive answer justifies further inquiry into this question. Future research must involve
techniques from the field of molecular genetics, which offers a strong tool in providing more
conclusive evidence. In addition, the testing of unremarkable osseous samples, i.e. those that do
not display characteristic lesions, from Pacific sites for MTB aDNA should be expanded in order
to ascertain a more accurate description of the true disease load represented in a sample. The
presence of tuberculosis in pre-contact Polynesian skeletal series, if established, could have
significant implications for understanding population movements, social structure and
environment, and population interaction, as well as the co-evolution of the *M. tuberculosis*
pathogen with human hosts in the Polynesian region. In addition, amplification of any mycobacterial aDNA found in Pacific samples may reveal phylogenetic relationships within the *M. tuberculosis* complex that may have further implications on ideas and models of peopling events. This information can be further extrapolated to aid in the understanding of current tuberculosis epidemiology.
References


