Population Decline Induced by Gonorrhea and Tuberculosis Transmission: Micronesia during the Japanese Occupation, 1919 – 1945

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A pressing global health concern in the 21st century is the risk of rapid and widespread disease transmission and its negative demographic consequences. For example, the global HIV/AIDS pandemic has caused significant demographic change. In Botswana the HIV/AIDS adult prevalence rate has exceeded 35% and has contributed to a substantial decrease in life expectancy, dropping from 65 years in 1985-1990 (before the epidemic) to 40 years in 2000-2005 (UNAIDS/WHO 2004). Indeed, most discussion about introduced disease, disease transmission, and depopulation centers on mortality; however, certain diseases such as gonorrhea can greatly influence population growth by means of fertility decline.

The interface between disease transmission and demography is of central importance today; however, understanding the interrelationships among these factors is frequently difficult due to confounding factors. Thus there are many benefits of studying historical epidemiology and demography, particularly in island populations. First, populations were usually isolated and had existed for a long period of time without foreign influence. Once foreigners arrived during colonial periods, changes in the health and demography of populations were often drastic and clearly related to introduced disease. Another benefit is the opportunity to observe the full magnitude of an epidemic and measure the demographic consequences. Disease can play a large role in demography, but it is often hard to tell *how much* of a role. Today, infections are frequently treated, and we rarely see uninhibited epidemics without interventions.

An episode of severe depopulation in Micronesia during the Japanese occupation (1919 – 1945) provides a unique opportunity to study the links between disease transmission and population growth in a simplified setting. At this time the Yapese population was suffering from a severe gonorrhea epidemic, and nearly half of all deaths were due to tuberculosis. The objective of this paper is to quantify the individual and combined impact of gonorrhea and tuberculosis on population growth on the island of Yap in Micronesia during the Japanese colonial period. We document a very unique demographic phenomenon—depopulation at an

annual rate of -2.0%, build a unified simulation model of reproduction that incorporates disease-induced infertility and mortality, predict the role of gonorrhea and tuberculosis in reducing population growth, and identify risks of depopulation from introduced disease for more complex and contemporary settings.

Micronesia – A brief history

Archeological evidence suggests that initial human settlement in the Micronesian islands occurred around 1500 BC (Kirch 2002). The island populations were almost completely isolated until the first western contact in 1521. Contact became more regular with the arrival of missionaries in the 1800's. The Spanish were the first to colonize the islands; they arrived in Micronesia in 1886 and controlled the islands until Germany took over in 1899. Japan, as part of their "southward advance" and major colonial endeavors during WWI, arrived in 1914 and quickly built a self-sufficient Japanese colony in Micronesia (Hezel 1983; Hezel 1995; Yanaihara 1940). Officially, the League of Nations mandated the islands to Japan in 1919, but Japan essentially controlled the Micronesian islands from 1914 to 1945 (the end of WWII).

During the Japanese occupation, Micronesia was referred to as the South Sea Islands. The islands were divided into six districts: Saipan, Palau, Yap, Truk, Ponape, and Jaluit. See Figure 1 for a map from 1935 of Japanese-occupied Micronesia (i.e. the South Sea Islands). Today, Micronesia is an umbrella term for the Federated States of Micronesia (FSM), the Republic of the Marshall Islands, the Republic of Palau, and the Commonwealth of the Northern Marianas.

- Insert Figure 1 about here – (**Note, we could switch this map with a current, clearer map showing where Micronesia is.)

The native population in Micronesia grew at an annual rate of 0.27% during the Japanese occupation, staying nearly steady at 50,000 inhabitants. Nonetheless, an individual look at the six districts reveals heterogeneity: Three districts grew slightly (Saipan, Palau and Ponape), two districts were relatively stable (Truk and Jaluit), and Yap's population dropped drastically at a rate of -2.0% per year (see Figure 2). Before the Japanese occupation, nearly all native populations in the area experienced a decline. During the Japanese occupation, all of the populations except for Yap had recovered or were in the process of recovering. Indeed, Yap's population was still declining because it had the *lowest* crude birth rate and the *highest* crude death rate among the six districts (South Seas Government 1920 - 1937).

- Insert Figure 2 about here -

The causes of depopulation on Yap during the Japanese occupation are well known: high death rates due to tuberculosis and amebic dysentery especially in infants, and low birth rates caused by venereal disease, especially gonorrhea (Gorenflo and Levin 1991; Lessa and Myers 1962; Peattie 1988; Pirie 1972). But many unanswered questions remain, including: how much did tuberculosis mortality affect population decline; how much did gonorrhea-induced infertility affect population decline; which disease was a more significant factor in depopulation; and what was the combined demographic affect from both diseases? These questions are answered in three sections. The first uses a Monte-Carlo simulation model of reproduction to quantify the impact of gonorrhea-induced infertility on the net reproductive rate (NRR). In the second section, a multi-decrement life table is built along with an associated single-decrement life table in order to predict how long someone in Yap would have lived in the absence of tuberculosis, as well as how much tuberculosis contributed to the reduction in life expectancy. In the third section, the newly predicted mortality schedule, with tuberculosis deaths discounted, is added to the initial simulation model. The results of this model allow the impacts of gonorrhea and tuberculosis on population growth to be compared separately and in combination. In answering these questions, we also consider contextual affects, i.e. how and why Yap was different than the other districts, and the role of labor migration, enforced upon Micronesians by the Japanese, in disease transmission.

Data

Data for this paper are from 17 years of annual reports (1920 – 1937), submitted to the League of Nations from the South Seas Government. Besides a large amount of other data, the Annual Reports include demographic data and mortality and morbidity data by age. Most of the data are stratified into the six districts of Micronesia at the time.

Included in the League of Nations report is an in-depth study of native health on the main island of Yap, conducted from 1929 to 1931 by Dr. Fujii, the director of the Yap hospital. This study provides extensive and reliable age and sex-specific data on tuberculosis and gonorrhea prevalence in Yap. Out of the total population of 3,996, the health of 3,787 people was examined between November 1929 and February 1930. Dr. Fujii's medical examination showed that 15.4% of the entire population suffered from tuberculosis (of any form). 1,091 people were determined to be in good health, and 2,696 were determined to be in bad health.

Dr. Fujii's special survey in Yap identified a much higher prevalence of tuberculosis than previous reports. In 1930, 80 natives on the island of Yap (2% of the population) died from tuberculosis, down from 83 the previous year. The majority of deaths (20) were aged 50-59, and those aged 60-69 experienced the next highest number of deaths (14) (see Figure 3, sex-specific data were not reported). Only two (2) deaths from tuberculosis were reported for children aged 0-9. Overall, tuberculosis was responsible for 47.9% of all deaths in 1930 (South Seas Government 1920 - 1937).

- Insert Figure 3 about here-

After the tuberculosis investigation, Dr. Fujii was able to ascertain the causes of the high death rate in Yap. He then conducted an investigation into the low birth rate. From April 1930 to July 1931, Dr. Fujii and his team carefully examined 2,354 people (1,252 males and 1,102 females) between the ages of 8 and 60 of the 3,884 inhabitants on Yap for gonorrhea. He found, contrary to previously reported data that indicated lower prevalence, that 33.3% of the Yap population was suffering from gonorrhea (312 or 24.9% of males and 472 or 42.8% of females). Young women had the highest gonorrhea prevalence, reaching 63% of women aged 16-20 and 51% of women aged 20-25 (see Figure 4) (South Seas Government 1920 - 1937). The prevalence of gonorrhea in men peaked later than women, in the age-group 31-35 years (39%).

- Insert Figure 4 about here -

Existing quantitative data show the magnitude of the epidemics, but lacking are data on sexual behavior, social norms, labor migration, and other social processes that potentially contributed to enhanced disease spread. Therefore, the objective of Cassels' 2004 qualitative fieldwork was to gather first hand information about sexual behavior, disease, and labor migration in Yap and Palau (the location of the phosphate mines) during the Japanese occupation and fill in gaps that the quantitative data have left open. The fieldwork was conducted in Yap and Palau from February 26th to March 16th, 2004. 15 interviews were conducted; 11 in Yap and 4 in Palau. The age of the respondents ranged from 68 to 85. Most respondents were male (12), but three females contributed priceless perspective.

Methods

A Monte Carlo simulation model, based on a model used by Barrett (1971) that creates and tracks the reproductive histories of a cohort of women, is used to answer our question regarding the role of disease in population growth. The model includes mortality rates, thus the output of the model is the net reproductive rate (NRR)—or the number of female offspring a woman can expect to have in her lifetime. Unlike the total fertility rate (TFR), the NRR incorporates both age-specific fertility and mortality rates. A NRR greater than one implies a growing population, while an NRR less than one means the population is shrinking. Gonorrhea-induced sterility is added to the model to calculate the affect of infertility on population growth. The model aims to characterize fertility patterns in Yap, Micronesia during the Japanese occupation in order to understand why population growth was negative.

Secondly, multi-decrement and associated single-decrement life table models were built to predict the mortality schedule discounting all TB deaths. Lastly, when this mortality schedule is added to the simulation model, it predicts how much TB mortality affected population growth. Detailed descriptions of the models are found in their respective sections of the paper.

Simulation models are very useful tools. Unlike deterministic models, stochastic simulation models allow complex *individual* dynamics and behavior to be modeled in a transparent way. Additionally, since one can not run experiments when studying historical data, simulation models can be used to recreate historical situations and allow for assumptions to be examined one at a time and in combination. These models can be used to "experiment" with different sets of assumptions about migration, to see how sensitive certain parameters are. Lastly, simulation models can be quite flexible: modifications can be introduced without affecting the global structure of the system (Wachter, Hammel, and Laslett 1978).

Gonorrhea, infertility, and population growth

The first objective of this paper is to measure how much fertility, and ultimately population growth, decreased from gonorrhea-induced sterility. However, delays in childbearing and lengthy birth spacing were commonly practiced in Yap at this time (Cassels' fieldwork) and are known to significantly influence fertility rates (Bongaarts and Potter 1983; Davis and Blake 1956). The model therefore incorporates additional changes of fertility from these proximate determinants to see what was ultimately responsible for negative population growth.

Past models

A large set of theoretical and empirical papers has shown the links between gonorrhea and infertility (Arya, Taber, and Nsanze 1980; Brunham, Garnett, Swinton, and Anderson 1992;

Garnett and Anderson 1993; Garnett, Swinton, Brunham, and Anderson 1992; Hethcote and Yorke 1984; Kramer and Reynolds 1981; Larsen 1996; Swinton, Garnett, Brunham, and Anderson 1992). Using sets of ordinary differential equations, past work has predicted a somewhat linear relationship between growth rates and gonorrhea prevalence (Brunham, Garnett, Swinton, and Anderson 1992; Swinton, Garnett, Brunham, and Anderson 1992). A growth rate of -2.0%—as seen in Yap—should correspond with a gonorrhea prevalence rate near 55% in women according to these studies. They predict that a 20% prevalence of gonorrhea could lower net population growth rates by 50% or more. (In Yap during the Japanese occupation, female gonorrhea prevalence was closer to 43%). However, another study using a microsimulation model concludes that sterility is not likely to cause a very large decrease in population growth rates (Zaba and Campbell 1994). Using the same gonorrhea prevalence as the earlier papers, they find a 20% drop in growth rates from gonorrhea-induced sterility, about a third found in the Swinton *et al.* paper.

In these models, a set of ordinary differential equations describes how individuals move between states of susceptibility and infection. These models do not incorporate age specific fertility, mortality, or other fertility-reducing determinants, which is an unrealistic simplifying assumption (Zaba and Campbell 1994). A simulation model, on the other hand, can incorporate many small details while remaining fairly simple and straightforward (Hammel, McDaniel, and Wachter 1979; Wachter 1987). This type of model also allows multiple parameters to be varied individually and in concert with others while the impact on fertility is measured. In this model, the key parameters are delays in childbearing, birth spacing, and ultimately sterility from gonorrhea.

Additionally, studies looking at the impact of disease induced sterility on fertility have been inconclusive because of numerous and confounding fertility inhibiting factors (Zaba and Campbell 1994). The present paper characterizes fertility on Yap, accounting for the aforementioned proximate determinants of fertility and gonorrhea-induced sterility. A simulation model of the whole reproductive process allows the impact on fertility from each variable to be measured individually and in combination with others. Another unique contribution of this paper is that the historical data from Yap provide a glimpse into uninhibited gonorrhea transmission and infection since no barrier-method contraception or treatment was available at the time (Cassels' fieldwork). Therefore, the results from the model represent a true estimate of the unrestrained impact of gonorrhea infection on population growth.

Monte Carlo simulation model

The Monte Carlo simulation model of reproduction and gonorrhea infection used in this paper incorporates many types of data. Data such as the risk of gonorrhea infection by age and mortality rates come from historical reports (South Seas Government 1920 - 1937), while other data have been estimated from the results of recent qualitative work in Yap (Cassels' fieldwork). These point estimates include the length of post-partum insusceptibility and the age of first birth (i.e. birth spacing and delays of childbearing). Lastly, biological parameters for the model come from existing literature on the reproductive process, such as age-specific fecundity (Barrett 1971; Trussell and Olsen 1983), naturally-occurring sterility (Menken, Trussell, and Larsen 1986), and the probability of various pregnancy outcomes (Trussell and Olsen 1983). For a list of model parameters, see Table 1.

- Insert Table 1 about here –

- Insert Figure 5 about here –

Figure 5 shows a schematic outline of the Monte Carlo simulation model of reproduction and gonorrhea infection. The salient features of the model are outlined below:

(i) A woman ages through the model in one-month intervals. Each month, she is subject to a numbered set of risks, and the outcomes affect the course of her reproductive path. For example, the question is asked: Does she conceive? If the answer is yes, then the question is asked: Does the conception end in fetal death, stillbirth, or a live birth? The length of time until the women is once again susceptible to conception depends on the answer to the previous question.

(ii) From the beginning of the simulation (birth) to the end of the reproductive span (age 40), each woman is subject to age-specific mortality. These data are taken directly from historical data from Yap in 1930; see the Yap life table (Table 3). Later, to ascertain the impact of tuberculosis on population growth, the mortality schedule is exchanged for one that excludes all deaths from TB.

(iii) Fecundability (p), or the probability that a woman will conceive in any month, starts at 0.2, which translates to an average waiting time of $(1-p)/p$ or 4 months. Each woman's fecundability declines linearly after age 30 to zero at age 50. In subsequent model runs, fecundability starts at 0.1.

(iv) Sterility due solely to aging is modeled after results cited in Menken *et al*. (1986). The natural risk of being unable to bear a child rises from 5.7% for women aged 20-24, to 9.3% for women aged 25-29, 15.5% for the 30-34 age group, 29.6% for 35-39 and 63.6% for women aged 40-44.

(v) Possible pregnancy outcomes in the model are live birth, stillbirth, and fetal death. The probabilities do not vary by individual, but are age-dependent. The probability of a fetal death is 0.24 + 0.005(age – 30), and is 0.03 + 0.001(age – 30) for a stillbirth (Trussell and Olsen 1983).

(vi) Fetal deaths are distributed exponentially from month zero to eight, with an average of four months. Live births and stillbirths are associated with a nine month pregnancy.

(vii) The length of post-partum insusceptibility is two months for fetal death or a stillbirth. However, the length of post-partum insusceptibility after a live birth varies in the model anywhere from zero to 24 months. Post-partum insusceptibility is a key parameter in the model.

(viii) Age at first birth is another significant parameter in the model. Often, simulation models of reproduction use age at marriage to signify when an individual becomes at risk of conception. In Yap, however, women would delay childbearing until their mid-twenties regardless of age at marriage. In the model, age at first birth varies from constant at age 15, to a gradual risk (40% of women aged 15-20, 80% of women aged 20-25, and 100% of women 25 and older), to constant at age 25.

(ix) Gonorrhea-induced sterility is the last parameter introduced to the model so that the drop in fertility solely from gonorrhea can be evaluated. Age-specific risk of gonorrhea is shown in Figure 4. The risk of sterility resulting from gonorrhea is 0.12 times the age-specific probability of contracting gonorrhea. 0.12 equals the probability that a gonorrheal infection leads to pelvic inflammatory disease (PID) multiplied by the probability that PID leads to infertility (0.2 * 0.6 = 0.12) (Anderson 1994; Swinton, Garnett, Brunham, and Anderson 1992).

(x) All women begin the model at birth and are observed at age 40. If at any point prior to age 40 the individual dies or becomes sterile, she drops out of the simulation and her characteristics are recorded. Each simulation consists of a cohort of 1,000 women. Note that a woman may be at risk of gonorrhea before she is at risk of conception in the model. Qualitative research in Yap has shown that women often engaged in pre-marital and extra-marital sex, but were greatly encouraged to delay childbearing. Thus, this model

assumes that young women practiced natural contraception or aborted unwanted children in order to delay childbearing until the expected age.

Results

The first run is the baseline model (see Table 2, model 1). This run is meant to provide a glimpse of uninhibited fertility: no contraception, no delay in childbearing, and no period of post-partum insusceptibility. As in all the models, each woman is subject to age-specific mortality as experienced on Yap in 1930. Plus, each model assumes the same probabilities of natural sterility and birth outcomes (live birth, stillbirth, and fetal death) for all women. In the baseline model, fecundability is 0.2, all individuals begin reproducing at age 15 and stop at age 40, and there is no period of insusceptibility after a live birth. The results of later runs can be compared to the baseline in order to evaluate parameter sensitivity.

- Insert Table 2 about here –

In the baseline model, as with every subsequent model, gonorrhea-induced sterility is introduced. In the baseline model, births decreased from 4,504 to 4,024. Translating the number of births to the NRR (NRR = (# births/1000)*0.4886), gonorrhea-induced sterility reduced the NRR from 2.20 to 1.97, a 10.7% reduction.

Models 2 through 6 represent the sensitivity analysis. In models 2 and 3, the length of post-partum insusceptibility (birth spacing) is varied. Consistent with well known results (Bongaarts and Potter 1983; Trussell 1986) birth spacing can have a profound effect on fertility and population growth. Comparing model 2 with the baseline shows that 12 months of post-partum insusceptibility decreased the NRR by 28%. However, if the individual waits 24 months until participating again in coitus the NRR drops by 42%. Results from Cassels' qualitative work suggests that traditional expectations kept couples from sexual contact from the third month of pregnancy until about two years after the child was born. Numerous respondents mentioned that the benchmark for allowing sexual intercourse was when the new child was able to jump over a log or a small ditch.

Models 4 and 5 show how sensitive fertility rates are to delays in childbearing. In model 4, the age of first birth is no longer constant at age 15; rather, women begin risk of conception at age 25. According to qualitative work, women were expected to wait until their mid-twenties to bear their first child. When risk of first birth is delayed ten years, the NRR (including gonorrhea) drops 58% from 1.97 to 0.83. Most likely, some women did not wait until age 25 to give birth;

therefore, model 5 assumes a gradual entry into the risk of childbearing: 40% begin at age 15, 80% by age 20, and everyone after age 25. The NRR, accounting for gonorrhea, drops to 1.40, or a 29% drop from the baseline model.

Additionally, contraception or reduction in coital frequency reduces the birth rate, but the relative reduction in the birth rate is less than the efficacy of contraception. For example, say that 100% of women practiced the withdrawal method, and it was 50% effective. Then the monthly probability of conception would be (0.2)*(1 - 0.50) = 0.1. Reducing the monthly probability of conception from 0.2 to 0.1, while keeping the rest of the parameters constant, resulted in a 22% drop in the NRR.

During the Japanese occupation of Micronesia before WWII, the growth rate in Yap was negative—implying an NRR less than one. In fact, annual population growth during the Japanese occupation averaged -2.0%, which loosely translates to an NRR near 0.57.[1] However, in 1930 the Yap population dropped from 6,486 to 6,410, which is a -1.17% change (NRR near 0.72). Thus models 7 through 9 are attempts to realistically vary the parameters in order to characterize the reproductive situation in Yap, with an NRR close to 0.72.

Model 9 is the best characterization of the expected fertility schedule on Yap. In this model, the NRR with gonorrhea-induced sterility is 0.73. This indicates depopulation at a rate extraordinarily close to the actual change in 1930. Model 9 represents a 63% drop in NRR from the baseline, and gonorrhea-induced sterility lowered the NRR by 13%.

Depending on the combination of parameters used, the percent drop in NRR from gonorrhea-induced sterility varied from 7% in model 7 to 17% in model 8. Although gonorrhea-induced sterility was not the largest determinant of fertility, without it the NRR would have been much closer to one. The model does not predict substantial negative population growth as seen on Yap without including gonorrhea.

Discussion

Some of the variation of NRR reduction from gonorrhea-induced sterility is the result of model dependency. In these models, women are not engaging in coitus from the third month of pregnancy until the end of the post-partum insusceptibility period. Therefore, the percent sterile from gonorrhea among those alive at menopause is lowest for the models with a long period of insusceptibility, and highest in the models with no post-partum insusceptibility (models 1, 4, 5, 6). NRR reductions due to gonorrhea are also generally the lowest for the models with a long

[1] r = ln(NRR)/T, where T is the mean length of a generation, or very similar to the average of the mean age at childbearing in a stable population. In the present model, the mean age at childbearing is 27.7.

period of insusceptibility after a live birth because exposure to gonorrhea is also lowered. However, this does not always hold true since some of the variation is simply due to model stochasticity. The average drop in the NRR is 12%, which is close to the drop in NRR from gonorrheal sterility in model 9. This result falls in line with previous demographic work which showed that sterility does not affect fertility rates as much as other determinants. However, the importance of gonorrhea in depopulation in Yap can not be understated; without gonorrhea-induced sterility in combination with the other determinants, population growth would have been stable or increasing in Japanese-occupied Yap.

Which determinants were the most significant in influencing population growth? In model 3, 24 months of post-partum insusceptibility accounted for a 42% drop in the NRR. If women delayed childbearing until age 25, this parameter would be more influential, accounting for a 56% drop in the NRR. However, the more realistic assumption of delays in childbearing—that the waiting time was distributed from age 15 to 25, as in model 5—accounted for a 29% reduction in the NRR. Gonorrhea-induced sterility accounted for a 12% reduction in the NRR, which is not as large as expected, but still significant. Finally, a drop in the probability of conception from 0.2 to 0.1 resulted in a 22% drop in the NRR. Therefore, birth spacing was the most important, followed by delays in childbearing, conception probabilities, and gonorrhea-induced sterility in explaining why Yap was experiencing such extreme depopulation. All four factors, when combined, were sufficiently influential to reverse the sign of population growth from positive to negative.

These results are consistent with past work in showing that birth spacing and delays in first birth can have significant impacts on fertility, partly because of the large variability inherent in the parameters. In certain situations, gonorrhea-induced sterility can also have significant impacts on population growth. However, when other determinants of fertility are accounted for, the impact of gonorrhea-induced sterility is lessened. This paper shows that gonorrheal sterility at the levels seen on Yap can decrease the NRR by 12%; Swinton *et al*. (1992) suggested a much larger effect: that 20% gonorrhea prevalence could decrease population growth rates by 50%. This disparity is due to the fact that Swinton *et al*. did not control for other causes of sterility, and did not include age-variation in fertility and mortality levels or other fertility determinants. Ignoring these other fertility-limiting behaviors results in an over-estimate of the impact of sterility on population growth (Zaba and Campbell 1994). The results of this paper are more similar to, but still smaller than Zaba and Campbell's (1994) results, which found a 20% drop in fertility rates with 20% gonorrhea prevalence. This disparity is due to different model specifications: Zaba and Campbell do not include naturally occurring sterility in their model. In

fact they specify that "if the primary sterility levels in [their] simulated population were due in part to factors other than STD's, then the effect of removing disease-induced sterility would be even lower." These specifications increase the effect of gonorrhea-induced sterility in their model compared with the specifications of the present model.

There are a few reasons why fertility in Yap was the lowest among the six districts, contributing to continued depopulation in Yap while the other districts were stable or growing: Yapese customs encouraging delays in childbearing and birth spacing, which kept fertility rates low, and the gonorrhea epidemic. But why was gonorrhea so prevalent? Although at this time many Yapese men were forced to work at phosphate mines in Palau, their circular migration did not significantly impact gonorrhea prevalence at home (Cassels 2005). Labor migration in Micronesia during the Japanese occupation was most likely conducive to gonorrhea transmission at the mines; a large number of men lived in close quarters at the mine and shared female sexual partners. However, migration did not contribute to a great increase in gonorrhea prevalence rates in Yap because of the combination of short infectious periods and high transmission probabilities. With time and without treatment, prevalence rates quickly increased and the effect from introduced infections from migrants was overshadowed by the epidemic.

A better explanation for the extraordinary gonorrhea prevalence rates has to do with social change during the Japanese occupation. Many interview respondents recalled that at this time, alcohol became available and the Yapese began to mix more freely with others throughout the island. Beforehand, the Yapese tended to stay close to home and only interacted with their local villagers. Many respondents mentioned that people moved around the island much more frequently during the Japanese occupation. Additionally, they engaged in much more extra- and pre-marital sex, which was likely associated with the newfound availability of alcohol. Both of these trends likely boosted gonorrhea prevalence on the island.

Tuberculosis, mortality, and population growth

Tuberculosis—an infectious disease that has plagued humans since antiquity and is still responsible for nearly 2 million deaths a year—was widespread in Micronesia during the Japanese occupation. In Yap, a district in Japanese-occupied Micronesia, one out of every two deaths in 1929 was attributed to a tuberculosis infection. At this time, Yap was experiencing severe depopulation while population growth in other districts in Micronesia was either close to zero or increasing. Although no similar in-depth study of tuberculosis took place in the other districts in Micronesia, it seems safe to assume that TB prevalence was higher in Yap than the other districts since 1) TB accounted for half of all deaths in Yap and 2) Yap had the highest

crude death rate among all districts. Since tuberculosis was a significant cause of death on Yap, it must have played an important role in depopulation. In this analysis of tuberculosis on Yap, we measure how much tuberculosis decreased life expectancy, and consider why tuberculosis was prevalent.

Multiple-decrement and associated single-decrement life tables

The goal of the following counterfactual "thought experiment" is to see how long someone in Yap during the Japanese occupation would have lived in the absence of tuberculosis deaths. The answer will allow us to predict how much TB contributed to the overall population decline in Yap. To answer the former question, we build a simple period life table for the Yap population in 1930. Second, we construct a multiple-decrement life table by cause of death (TB and other) to see the age-specific probabilities that a person will die from TB. Third, we construct an associated single-decrement life table to predict life expectancy if death from TB were eliminated and compare it to the initial life table (Keyfitz 1977; Preston, Heuveline, and Guillot 2001).

- Insert Table 3 about here –

Given historical data from Yap including total number of deaths and age-specific population estimates, a life table can be constructed. See Table 3 for the estimated life table for Yap in 1930. Life expectancy increases from 28 years at age 0 (e_0) to almost 35 years at age 5 (e_5), meaning that the expected age of death for a newborn is 28 years but the expected age of death for a 5 year old (given that she survived to age 5) is 40. The initial increase of life expectancy with age is due to high infant mortality. However, mortality at all ages stays quite high. By age 25, 50% of the initial population has died (l_{25}), and the probability of dying between ages 25 and 30 is 8% ($_5q_{25}$). For comparison, the same probability of dying between ages 25 and 30 in the U.S. in 2002 is less than 0.5%, life expectancy at birth is 77.3 years for the entire population, and 50% of the initial population does not die until after age 80. In 1930 U.S., estimated life expectancy at birth was 59.7 (Arias 2004).

Next, cause-specific deaths are used to produce a multiple-decrement life table. In this formulation, we assume that causes of death are exhaustive and mutually exclusive, meaning that all deaths are attributed to a single cause and each cause of death is exclusive from the others, i.e.:

$$nDx = nDx^A + nDx^B + nDx^C + \ldots,$$

where nDx is the number of deaths from age x to x+n, and nDx^A is the number of deaths due to cause A from age x to x+n. Now, we assume that the population at risk (nKx) is the same for all causes, so

$$nMx = (nDx^A + nDx^B + nDx^C + \ldots)/nKx,$$

thus

$$nMx = nMx^A + nMx^B + nMx^C + \ldots$$

- Insert Table 4 about here –

Table 4 shows the multiple decrement life table for Yap in 1930, where nqx is the probability of dying between ages x to x+n from any cause, and nq^ix is the probability of dying from x to x+n from cause i, in this case tuberculosis.

It is important to note that the formulas for nqx and nq^ix are very similar; the denominators are identical. The difference is that nq^ix has the cause-i specific death rate (nm^ix) in the numerator. This clarifies the dependent competing risks in multiple decrement life tables. Given a certain death rate from tuberculosis in the age interval 40 – 44, for example, the proportion of 40 years olds who die from tuberculosis between ages 40 – 45 will be lower given higher death rates from all other causes. When the death rate from causes other than tuberculosis is higher, more potential victims of tuberculosis will die from other causes (Preston, Heuveline, and Guillot 2001).

If the probability of dying from one cause declines, the probability of dying from some other cause(s) must increase. This does not mean that, for example, when the death rate from causes other than TB declines that the death rate from TB must rise. People must eventually die, but they do not have to die in a certain age-interval. Rather, the number of person-years of exposure to risk of tuberculosis deaths will increase when other causes decline, so the number of tuberculosis deaths will increase (or vice-a-versa).

A child on the island of Yap during the Japanese occupation in 1930 had no risk of dying from tuberculosis before age 5. Then, the probability of dying from TB between age 5 to 10 is 0.028 ($_5q^i_5$). The probability of dying from TB within a five year interval more or less steadily increases, reaching 0.431 from 75 to 80 years.

The conditional probability of eventually dying from TB (for survivors at age x) ranges from 0.39 at age 0 to 0.61 at age 70 ($\pi^i_x = l^i_x/l_x$); the average is a little higher than 0.51. However, TB deaths accounted for 48% of deaths in 1930. This disparity comes from the fact that many deaths from causes other than TB occurred at early ages. Therefore, the cumulative probability of

eventually dying from TB for survivors at age x is higher than 48% at almost all ages above 5 years. For example, 58% of people who survive to age 40 will die from TB in this model life table.

Now that we have the probability of dying from TB—the multiple decrement life table—we must assume that risk of death from tuberculosis and risk of death from all other causes are independent in order to calculate life expectancy without TB deaths, or the associated single decrement life table. This will estimate the probability of dying from all other diseases given the absence of TB. The associated single decrement probability of dying between x and x+n from cause i is:

$$*nq^{i}x = 1 - (lx+n/lx)*\exp(nD^{i}x/nDx)$$

Since risk of death from tuberculosis is in fact not independent from risk of death from other causes, assuming independence will over-estimate the impact of removing TB deaths on life expectancy. In actuality, decreasing the probability of death from TB to zero will increase the probability of death from other causes.

- Insert Table 5 about here –

The most useful results come from comparing the associated single decrement life table (Table 5) with the all-causes life table (Table 3). Note that Table 5 shows the associated single decrement life table for all causes of death *other than* tuberculosis. This answers the question: what would the mortality schedule look like without deaths from TB? The probabilities of surviving to age x with and without TB are fairly similar until around age 20, and then they start to diverge. The probability of surviving to age 20 for all causes combined is 0.58 (lx), however the probability of surviving to age 20 in the absence of TB is 0.64 ($*l^{-i}x$). In the absence of TB, more than 40% of the population would still be alive at age 50, compared to only 23% with TB deaths included. Not surprisingly, life expectancy between the models with and without TB deaths varies drastically. Life expectancy at birth excluding TB deaths is almost 38 years, compared to 28 with TB deaths, a 10 year difference. Due to high childhood mortality from causes other than TB and no TB deaths before age 5, life expectancy at age 5 is longer than life expectancy at birth. Additionally since no children die of TB before age 5, the difference in life expectancy between the two models reaches its peak at age 5. A child aged 5 is expected to live an additional 49 years in the model excluding TB deaths, as opposed to only 35 more years in the all-causes of death model. This analysis suggests that in the absence of TB, life expectancy would have been 14 years longer.

Discussion

Why was Yap's mortality rate so much higher that the others? Tuberculosis was the driving factor behind the high mortality rates, and in this case, labor migration most likely played a large role in tuberculosis transmission, as well as certain Yapese customs that were conducive to disease transmission.

Past literature has shown an association between TB, socioeconomic status, and household overcrowding (Antunes and Waldman 2001; Murphy, Singer, Anderson, and Kirschner 2002), immigration (Antunes and Waldman 2001), family size, malnutrition, poor access to health care, and poverty (Elender, Bentham, and Langford 1998). Poverty is often associated with malnutrition, overcrowding, poor quality homes, and physical stress, all of which are significant risk factors for the disease. Poor nutrition and lack of protein in the diet are known to be associated with reduced immune function (Elender, Bentham, and Langford 1998). Many of these factors were commonly found on Yap during the Japanese occupation.

Two unique features of traditional Yapese lifestyle were most likely affiliated with increased risk of tuberculosis. They included 1) the tradition of men's clubhouses (*faluw*), and 2) housing style. *Faluw*, or men's clubhouses, are traditional houses on Yap where the village men would gather every day and night. Before modern schooling the *faluw* acted as a schoolhouse; village men would teach the younger boys how to fish, fix nets, and build boats in these clubhouses. The clubhouse was also a place of socialization. In fact, men would spend almost all of their time in the *faluw*: they would go home to eat and possibly help out with household chores during the day but then return to sleep in the *faluw*. Each village would have at least one *faluw*, and on average there would be around 45 men per *faluw*; the size of the membership and the number of clubs in a village depended on the village population.

Faluw's were built on high platforms along the shore with beams always facing east to west. The walls were made of bamboo and the roof made from palm fronds; the windows were also fitted with palm fronds; therefore, the clubhouses were fully enclosed. The fact that the men would sleep in close, enclosed quarters every night suggests that the tradition of the *faluw* was a factor in tuberculosis transmission.

The traditional style of houses—both the *faluw* and family homes—on Yap could have exacerbated tuberculosis transmission as well. According to Japanese reports, native housing on Yap was the worst of all districts. Yapese houses had few doors, poor ventilation, and little light, and the Yapese would sleep on the bare ground. After heavy rains the palm-frond roofs and coconut mats would stay damp for a long time.

In contrast with the minimal impact of labor migration on gonorrhea prevalence at home, labor migration most likely played a much larger role in tuberculosis transmission, due in part to different disease etiology and transmission dynamics. Due to the living and working conditions at the mines, many of the circular migrants most likely returned home infected with TB. Because of the long latent period, these men could become sick and infectious after returning home and transmit the disease easily to others, in part because of the local *faluw* and housing conditions.

Labor migration under colonial regimes has been associated with tuberculosis transmission via poor living and working conditions, inadequate diets, and circular migration. Native labor conditions under colonial regimes were mostly crowded, unsanitary, and conducive to the spread of disease (DeLancey 1978; Dumett 1993; MacPherson and Gushulak 2001; Manderson 1996; Packard 1989; Patterson and Hartwig 1978). Often laborers caught infectious diseases at their place of work and then brought their newly acquired afflictions back home with them (Patterson and Hartwig 1978). In some cases laborers were repatriated because they were too sick to work, but in other cases the laborers were sent home after their work contract expired, only to become sick years later, which was often the case with tuberculosis (Dumett 1993; Packard 1989).

In Micronesia during the Japanese occupation, migrant laborers would work for an average of six months and then return home as a new batch of workers replaced them. All of the migrant laborers lived in single-sex barracks according to their island of origin; about 50 men would stay in one barrack. Migrant laborers worked long hours, six days a week. According to one interview respondent who used to work at the mines, the labor was very hard; the men had to shovel the phosphate by hand from the ground into the train cars, and the phosphate was very dry and dusty, like sand. Poor diets, crowded living, and daily phosphate dust inhalation increase both the exposure to infection and susceptibility to TB progression due to a compromised immune system.

Clearly, the TB epidemic altered Yap's population drastically. The next section will show results of the net reproduction rate (NRR) model using age-specific mortality rates from the associated single-sex life table excluding TB deaths. This will enable the comparison between the impacts of TB mortality on population growth with gonorrhea impacts on infertility and population growth to see which effect was larger, as well as the combined effects of TB and gonorrhea on population size.

Gonorrhea + tuberculosis: relative and combined affects on population growth

This last section will show 1) which disease had a greater impact on population growth and 2) the combined demographic impact of tuberculosis and gonorrhea. The model is identical to the original simulation model (model 9, Table 3), but mortality rates are varied in order to estimate population growth without deaths from TB. The monthly probability of conception is 0.1; the length of insusceptibility after a live birth is set at 24 months; and women delay risk of childbearing according to the schedule presented earlier. The model is run four times to estimate the NRR in four different scenarios: 1) without TB or gonorrhea, 2) without TB and with gonorrhea, 3) with TB and without gonorrhea, and 4) with both TB and gonorrhea. Table 3, column 'nqx' shows the mortality schedule used when tuberculosis deaths are included, and the column labeled *nq-ix in Table 5 is the mortality schedule used when tuberculosis deaths are excluded.

- Insert Table 6 about here-

Table 6 shows the NRR from four scenarios. The first scenario predicts the NRR without any deaths from TB and no sterility from gonorrhea. The model estimates the NRR to be 1.06, which translates loosely to a population growth rate equal to 0.20%, assuming the mean age of childbearing to be 27.7 years. Thus without TB or gonorrhea, the Yap population would have been growing during the Japanese occupation.

Scenario 2 predicts the NRR excluding deaths from TB but including gonorrhea-induced sterility. This scenario predicts the impact of gonorrhea-induced sterility on population growth, given no deaths at all from TB. The NRR dropped from 1.06 to 0.96, or by 9.5%. Essentially, this is the same result as before: the role of gonorrhea in population decline, but under a different mortality schedule. We will compare this result with scenario 3, which measures the role of TB in population growth.

Scenario 3 is the reverse of scenario 2: We exclude gonorrhea-induced sterility but include deaths from TB. Note that this model is the same as in Table 3, model 9 without gonorrhea-induced sterility. The NRR is 0.84, which is a 21% drop from scenario 1 (1.06). Therefore, this scenario predicts that deaths from TB lowered the rate of population growth about twice as much as did sterility from gonorrhea.

Lastly, scenario 4 includes both gonorrhea and tuberculosis. Again, this is the same as model 9 in Table 3, including gonorrhea-induced sterility. This scenario shows the combined impact of TB and gonorrhea on population growth. The NRR in scenario 4 is 0.73, which

translates to a population growth rate of -1.14%. TB and gonorrhea together were potentially responsible for a 31% drop in the NRR (1.06 down to 0.73).

Mortality was very high in Yap during the Japanese occupation. Without any mortality at all, the NRR would have been 1.78 without gonorrhea-induced sterility, and 1.52 with gonorrhea-induced sterility (data not shown). Therefore, mortality alone decreased the NRR substantially. Mortality solely due to TB still decreased the NRR by 21%, and was a greater factor in depopulation on Yap during the Japanese occupation than was gonorrhea, which decreased the NRR by 9 to 13% (depending on the other model assumptions).

Conclusion

The results of the gonorrhea analysis revealed that gonorrhea-induced infertility accounted for a 12% reduction in the net reproduction rate (NRR). Without gonorrhea infections, population growth would have been much closer to zero. Analogous to classic demographic work, birth spacing and delays in childbearing were the most important determinants of fertility levels and thus population growth. The model did not predict significant depopulation without including risk of gonorrhea-induced sterility. When risk of infertility from gonorrhea infections was added to the model, the NRR dropped significantly, implying negative population growth.

Gonorrhea was rampant in Yap during the Japanese occupation—more than 40% of females aged 8 to 60 were suffering from gonorrhea in 1930. However, tuberculosis accounted for almost half of all the deaths in the same year. In 1930, a newborn had only a 33% chance of surviving to age 40. According to the TB analysis, if all deaths from TB were eliminated, a newborn would have had nearly a 50% chance of surviving to age 40. Overall, the results suggested that tuberculosis accounted for 14 years of life expectancy lost in Yap during the Japanese occupation.

Population growth is generally more sensitive to changes in fertility than mortality (Coale 1974; Demeny 1986; Lutz 1994). In the case of Yap during the Japanese occupation, gonorrhea-induced sterility affected women during their childbearing years, whereas middle-aged to elderly men and women were dying from tuberculosis. The fact that tubercular mortality affected many people after their childbearing years (40 and above) while gonorrhea affected many women before age 40 might enhance gonorrhea's impact on population growth. However, tubercular mortality was extremely high in Yap during the Japanese occupation, and was a more important driver of negative population growth than gonorrhea-induced infertility.

[Lessons for today??]

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Figure 1: Maps of the South Sea Islands and principle islands under the Japanese occupation.

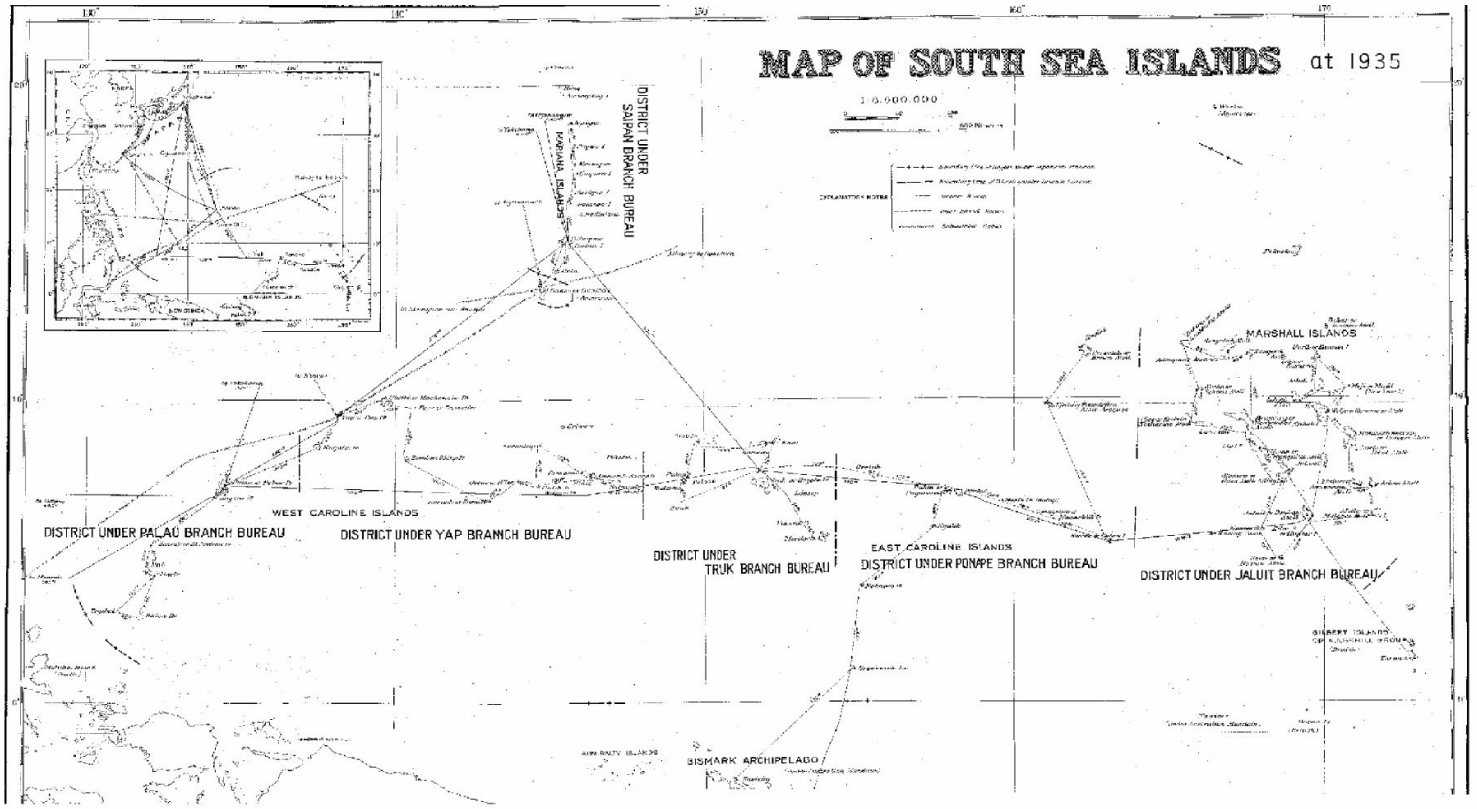
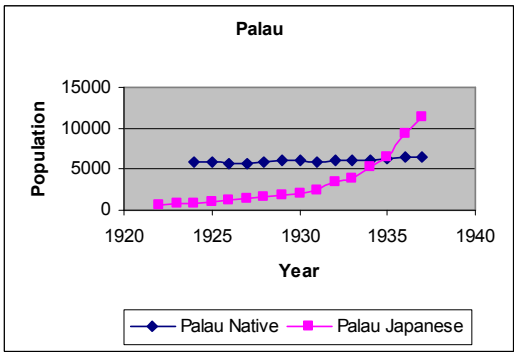
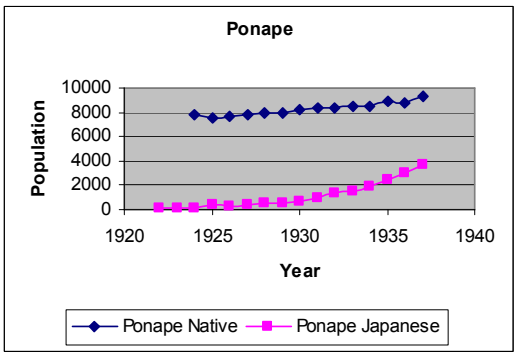
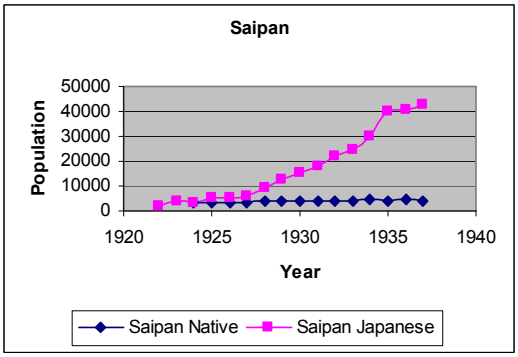
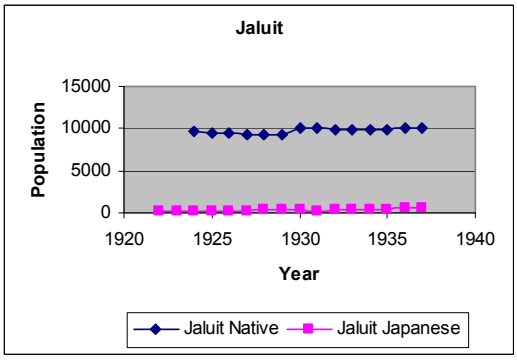


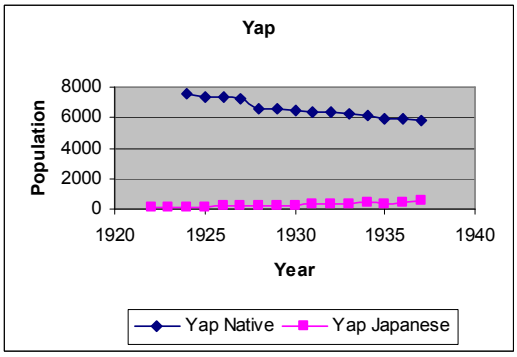
Figure 2: Population trends for Japanese and Native Micronesians, by district.











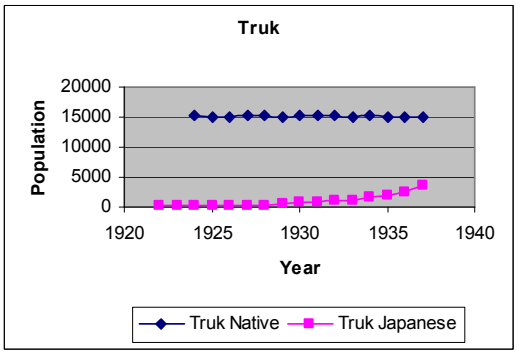
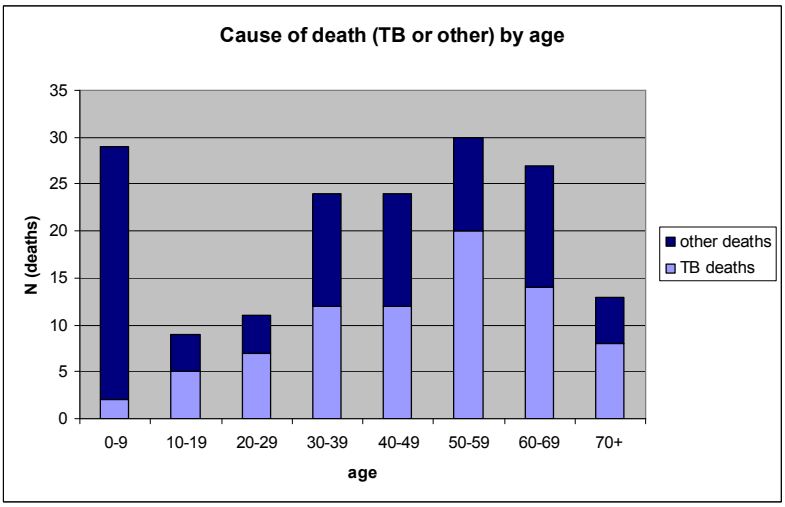
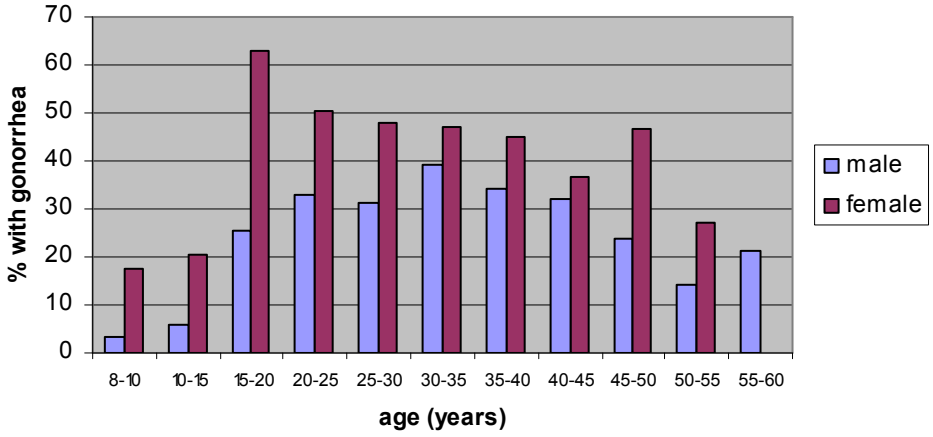


Figure 3: Deaths due to tuberculosis of the lungs and other causes on the main island of Yap in 1930, by age



Source: (South Seas Government 1920-1937)

Figure 4: Gonorrhea prevalence in the native population, by age, on the main island of Yap in 1930



Source: (South Seas Government 1920-1937)

Figure 5: Flow chart of simulation model

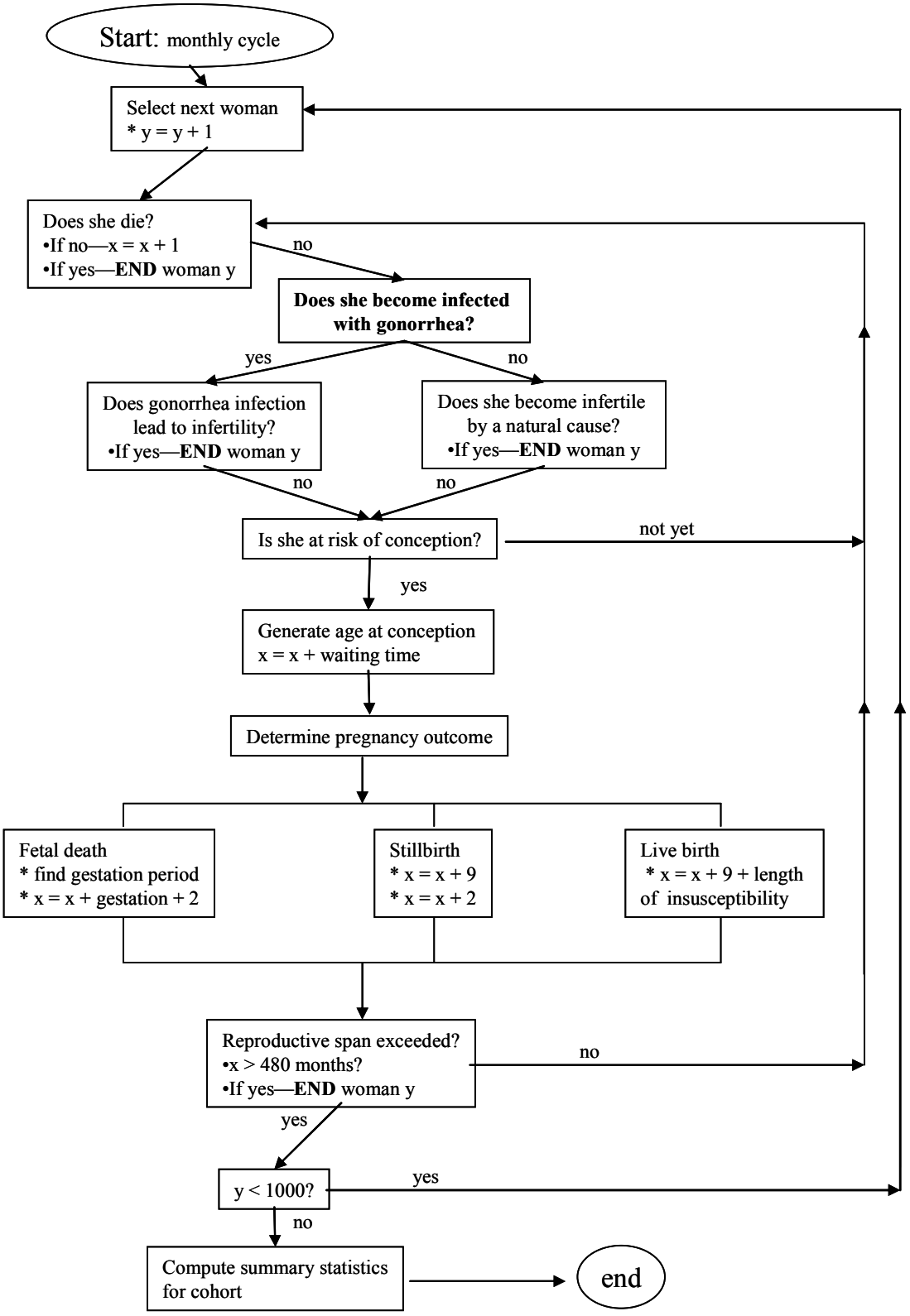


Table 1: Parameters used in simulation model

Monthly parameters	
Monthly probability of conception	p
Probability of fetal death	$0.24 + 0.005*(\text{age} - 30)$
Probability of stillbirth	$0.03 + 0.001*(\text{age} - 30)$
Length of infertility after fetal death or stillbirth	2 months
Probability of live birth	1 - P(fetal death) - P(stillbirth)
Length of post-partum insusceptibility	s_2
Age of menopause	480 months
Age-specific parameters	
Cumulative risk of first birth	a_x
Probability of gonorrhea infection	${}_nm_x$
Probability of infertility	$0.12*{}_nm_x$
Mortality	${}_nq_x$

Table 2: Simulated results from a model of reproduction

Input assumptions	*model 1*	*model 2*	*model 3*	*model 4*	*model 5*	*model 6*	*model 7*	*model 8*	*model 9*
Probability of conception (monthly)**	0.2	0.2	0.2	0.2	0.2	0.1	0.2	0.2	0.1
Insusceptibility after live birth (months)	0	12	24	0	0	0	12	24	24
Age of first birth	15	15	15	25	gradual	15	gradual	gradual	gradual
Implications									
# births/1000 women	4,504	3,134	2,614	1,950	3,301	3,660	2,404	1,997	1,720
with gonorrhea									
# births/1000 women	4,024	2,888	2,334	1,694	2,856	3,147	2,245	1,658	1,494
# women infertile/1000 women	98	75	59	103	108	97	95	84	97
# deaths/1000 women	587	596	585	554	605	597	569	611	590
% sterile among those alive at menopause	23.73	18.56	14.22	23.09	27.34	24.07	22.04	21.59	23.66
NRR	2.20	1.53	1.28	0.95	1.61	1.79	1.17	0.98	0.84
NRR with gonorrhea-induced sterility	1.97	1.41	1.14	0.83	1.40	1.54	1.10	0.81	0.73
NRR % drop from baseline (model 1)	0.00	28.23	42.00	57.90	29.03	21.79	44.21	58.80	62.87
NRR % fall due to gonorrhea	10.66	7.85	10.71	13.13	13.48	14.02	6.61	16.98	13.14

** Geometrically distributed delay

Table 3: Life table (all causes) for Yap population, 1930

Age x	${}_nK_x$	${}_nD_x$	${}_nM_x$	${}_na_x$	${}_nq_x$	l_x	${}_nd_x$	${}_nL_x$	T_x	e_x
0	72	14	0.194	0.130	0.166	1.000	0.166	0.855	27.883	27.883
1	232	11	0.047	1.700	0.171	0.834	0.143	3.007	27.027	32.419
5	345	4	0.012	2.500	0.056	0.691	0.039	3.358	24.021	34.756
10	432	5	0.012	2.500	0.056	0.652	0.037	3.169	20.662	31.682
15	322	4	0.012	2.500	0.060	0.616	0.037	2.985	17.493	28.421
20	249	7	0.028	2.500	0.131	0.578	0.076	2.702	14.508	25.082
25	241	4	0.017	2.500	0.080	0.502	0.040	2.412	11.806	23.496
30	384	8	0.021	2.500	0.099	0.462	0.046	2.198	9.394	20.315
35	353	16	0.045	2.500	0.204	0.417	0.085	1.871	7.196	17.273
40	374	14	0.037	2.500	0.171	0.332	0.057	1.517	5.325	16.048
45	253	10	0.039	2.500	0.180	0.275	0.049	1.252	3.808	13.847
50	239	14	0.059	2.500	0.255	0.226	0.058	0.984	2.557	11.332
55	170	16	0.094	2.500	0.382	0.168	0.064	0.680	1.572	9.358
60	167	14	0.084	2.500	0.347	0.104	0.036	0.430	0.893	8.590
65	92	13	0.142	2.500	0.523	0.068	0.035	0.251	0.463	6.821
70	65	5	0.077	2.500	0.322	0.032	0.010	0.136	0.212	6.554
75	20	6	0.303	2.500	0.863	0.022	0.019	0.062	0.076	3.478
80	9	2	0.216	2.500	1.000	0.003	0.003	0.014	0.014	4.628

Table 4: Multiple decrement life table
Cause i = death from tuberculosis of the lungs

Age x	${}_nD_x$	${}_nD^i_x$	l_x	${}_nq_x$	${}_nq^i_x$	${}_nd^i_x$	l^i_x	π^i_x
0	14	0	1.000	0.166	0.000	0.000	0.393	0.393
1	11	0	0.834	0.171	0.000	0.000	0.393	0.471
5	4	2	0.691	0.056	0.028	0.019	0.393	0.568
10	5	2	0.652	0.056	0.022	0.015	0.373	0.573
15	4	3	0.616	0.060	0.045	0.028	0.359	0.583
20	7	3	0.578	0.131	0.056	0.033	0.331	0.572
25	4	4	0.502	0.080	0.080	0.040	0.298	0.594
30	8	6	0.462	0.099	0.074	0.034	0.258	0.559
35	16	6	0.417	0.204	0.076	0.032	0.224	0.538
40	14	6	0.332	0.171	0.073	0.024	0.192	0.579
45	10	6	0.275	0.180	0.108	0.030	0.168	0.610
50	14	11	0.226	0.255	0.201	0.045	0.138	0.612
55	16	9	0.168	0.382	0.215	0.036	0.093	0.553
60	14	8	0.104	0.347	0.198	0.021	0.057	0.547
65	13	6	0.068	0.523	0.241	0.016	0.036	0.535
70	5	5	0.032	0.322	0.322	0.010	0.020	0.614
75	6	3	0.022	0.863	0.431	0.009	0.009	0.431
80	2	0	0.003	1.000	0.000	0.000	0.000	0.000

Table 5: Associated single decrement life table for causes of death other than tuberculosis (-i)

Age x	${}_nD^{-i}_x/{}_nD_x$	${}^*{}_nq^{-i}_x$	${}^*l^{-i}_x$	${}_na_x$	${}^*{}_nd^{-i}_x$	${}_nL_x$	T_x	${}^*e^{-i}_x$
0	1.000	0.166	1.000	0.13	0.166	0.855	37.976	37.976
1	1.000	0.171	0.834	1.70	0.143	3.007	37.121	44.526
5	0.500	0.029	0.691	2.50	0.020	3.406	34.114	49.361
10	0.600	0.034	0.671	2.50	0.023	3.300	30.708	45.739
15	0.250	0.015	0.648	2.50	0.010	3.217	27.408	42.267
20	0.571	0.077	0.638	2.50	0.049	3.069	24.191	37.890
25	0.000	0.000	0.589	2.50	0.000	2.946	21.122	35.855
30	0.250	0.026	0.589	2.50	0.015	2.908	18.177	30.855
35	0.625	0.133	0.574	2.50	0.076	2.680	15.269	26.604
40	0.571	0.102	0.498	2.50	0.051	2.363	12.590	25.287
45	0.400	0.076	0.447	2.50	0.034	2.151	10.227	22.869
50	0.214	0.061	0.413	2.50	0.025	2.002	8.076	19.548
55	0.438	0.190	0.388	2.50	0.074	1.755	6.074	15.659
60	0.429	0.167	0.314	2.50	0.052	1.441	4.318	13.739
65	0.538	0.329	0.262	2.50	0.086	1.094	2.878	10.987
70	0.000	0.000	0.176	2.50	0.000	0.879	1.783	10.140
75	0.500	0.630	0.176	2.50	0.111	0.602	0.904	5.140
80	1.000	1.000	0.065	2.50	0.065	0.301	0.301	4.628

Table 6: NRR from 4 scenarios of TB mortality and gonorrhea-induced infertility: Simulated results are from the reproduction model presented in Chapter 4

		gonorrhea	
		no	yes
tuberculosis	no	Scenario 1: 1.06	Scenario 2: 0.96
	yes	Scenario 3: 0.84	Scenario 4: 0.73