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Population decline induced by gonorrhoea and tuberculosis transmission: Micronesia during the Japanese occupation, 1919–45

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Abstract

The islands of Yap in Micronesia survived a period of severe depopulation during the Japanese occupation from 1919 to 1945. Using data from historical documents, supplemented by ethnographic evidence, we calibrate a simulation model that accounts for this phenomenon. Our model tracks the reproduction histories of a synthetic cohort of women in Yap, including effects of infertility due to gonorrhoea as well as tuberculosis mortality, and predicts the net reproduction rate (NRR). In this particular case and throughout history, human migrations and associated social and cultural interactions have frequently been accompanied by dramatic changes in patterns of disease transmission and substantial demographic consequences. Despite the broad emphasis on mortality as a measure of demographic consequences in the historical and contemporary literature, there are important instances where life expectancy at birth, fertility rates, and total population size are important demographic consequences. We find that gonorrhoea may have significantly contributed to depopulation during the Japanese occupation of Micronesia, due to repeated infections and high risk of sterility. Results of our model suggest that gonorrhoea alone could have reduced the net reproduction rate by 82%, whereas deaths from tuberculosis may have contributed to a 17% decline.

Keywords

Simulation model; Historical demography; Yap; Health policy; Labour migration

Introduction

Human migrations and the social and cultural interactions associated with them have frequently been accompanied by dramatic changes in patterns of disease transmission and substantial demographic consequences (Cliff et al. 2000; Crosby 2003; Hirsch 1883; Wilson 1991). Classic examples are the epidemic of smallpox introduced into Mexico by Spanish conquistadors in 1520, decimating the indigenous Aztec population (Crosby 2003); the first introduction of measles into Fiji in 1875 that led to the death of 25–30% of the islands' population within a two-month period (Cliff and Haggett 1985); and the cholera epidemic of

1902–1904 in the Philippine Islands that left 200,000 people dead, following a loss of 800,000 Filipino lives—out of a population just under eight million—in the Philippine-American War (Smallman-Raynor and Cliff 1998). These examples are illustrative of a broad emphasis on mortality as a measure of demographic consequences in the historical and contemporary literature on epidemics. There are, however, important instances where life expectancy at birth, fertility rates, and total population size are important demographic consequences of epidemics. A contemporary instance of this phenomenon is the HIV/AIDS pandemic in sub-Saharan Africa. For example, the adult prevalence rate in Botswana has exceeded 35% and contributed to a decrease in life expectancy at birth from 65 years in 1985–1990 (before the epidemic) to 40 years in 2000–2005 (UNAIDS/WHO 2004).

Underlying the coarse descriptive statistics cited above are complex social and political factors that regulate exposure of susceptible individuals to infectious persons and progression to the ultimate demographic consequences. Identifying the detailed processes that lead to changes in life expectancy, net reproduction rate, and total population size is often difficult because of heterogeneity in exposure patterns of large populations and a multiplicity of confounding factors that can mask important details. Characterizing the complex behavioural and disease transmission patterns accompanying migration-induced mixing of populations is easier for islands where geographical closure is obtained (Cliff et al. 2000).

In this context, we investigate the processes that underlie the depopulation (at an annual rate of—2.0%) of the islands of Yap in Micronesia during the Japanese occupation (1919–1945). At this time the Yapese population was suffering from a severe gonorrhoea 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 gonorrhoea and tuberculosis on population growth on the islands of Yap during the Japanese colonial period. We utilize a diversity of historical documents, supplemented by ethnographic evidence, to provide a rationale for a simulation model that represents reproduction and gonorrhoea-induced infertility together with tuberculosis mortality. The potential for high impact from gonorrhoea is a consequence of the unavailability of both barrier-method contraception and antibiotic treatment during this historical period. We also characterize the complex social and economic forces, linked to inter-Micronesian island circular migration, that promoted the transmission of tuberculosis in Yap. Finally, we conclude with a discussion of the implications of our analysis for monitoring and surveillance of emerging infectious diseases in contemporary settings.

Micronesia: a brief history

Archaeological 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 1800s. 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. Shortly thereafter (1900–1911), the population of Yap experienced major outbreaks of influenza, leprosy, and typhoid (Hunt et al. 1954). Japan, as part of their ‘southward advance’ and major colonial endeavours during WWI, arrived in 1914 and quickly built a self-sufficient Japanese colony in Micronesia (Hezel 1983, 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. 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.

The native population in Micronesia grew at an annual rate of 0.27% during the Japanese occupation, thereby remaining steady at approximately 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 Fig. 1). 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. It had the lowest crude birth rate and the highest crude death rate among the six districts (South Seas Government 1920–1937; Taeuber and Han 1950).

In past anthropological and demographic research, hypotheses were put forth regarding depopulation on Yap. After the American occupation in 1945, Harvard University sent an expedition team as part of the Coordinated Investigation of Micronesian Anthropology (CIDA), and carried out a census of the island of Yap from 1947 to 1948 (Hunt et al. 1954). After considering many hypotheses for Yapese depopulation including disease mortality, male absenteeism, malnutrition, intestinal parasites, abortions, and cultural patterns of sexual behaviour that led to low birth rates, they concluded that the main causes were sexually transmitted infections (STI), coital taboos and abortion (Hunt et al. 1954). Another paper was published in the mid-1970s that used census data from the CIDA study as well as anthropometric data from the mid-1960s (Underwood 1973). This research clearly rejected the hypothesis that abortion was a main driver of depopulation and concluded that STIs, particularly gonorrhoea, were more important. These early studies taken together with more recent evidence suggest that the primary causes of depopulation on Yap during the Japanese occupation were high death rates due to tuberculosis and amoebic dysentery especially in infants, and low birth rates caused by STI, especially gonorrhoea (Gorenflo and Levin 1991; Lessa and Myers 1962; Peattie 1988; Pirie 1972; Taeuber and Han 1950; Hunt et al. 1954; Underwood 1973).

Beyond this coarse-level associational literature, there are many unanswered questions regarding the magnitude of the individual and combined effects of gonorrhoea and tuberculosis. In particular, we do not know how much tuberculosis mortality by itself or gonorrhoea-induced infertility alone affected population decline. Quantitative estimates of the demographic effect of both diseases together are also unavailable. We provide answers to these questions in two steps. First 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. The second step uses a stochastic microsimulation model of reproduction to quantify the impact of gonorrhoea-induced infertility on the net reproductive rate (NRR) with the original or the newly predicted mortality schedule—with tuberculosis deaths discounted—included. The results of this model allow the impacts of gonorrhoea and tuberculosis on population growth to be compared separately and in combination. In answering these questions, we also consider contextual effects, i.e. how and why Yap was different from the other districts, and the role of labour migration—enforced upon Micronesians by the Japanese—in disease transmission.

Previous literature

A large set of theoretical and empirical papers has shown the links between gonorrhoea and infertility (Arya et al. 1980; Brunham et al. 1992; Garnett and Anderson 1993; Garnett et al. 1992; Hethcote and Yorke 1984; Kramer and Reynolds 1981; Larsen 1996; Swinton et al. 1992). Using sets of ordinary differential equations, past work has predicted a somewhat linear relationship between growth rates and gonorrhoea prevalence (Brunham et al. 1992; Swinton et al. 1992). A growth rate of -2.0% —as seen in Yap—should correspond to a

gonorrhoea prevalence rate near 55% in women according to these studies. They predict that a 20% prevalence of gonorrhoea could lower net population growth rates by 50% or more. (In Yap during the Japanese occupation, female gonorrhoea prevalence was closer to 43%). However, another study using a micro-simulation model concludes that sterility is not likely to cause a very large decrease in population growth rates (Zaba and Campbell 1994). Using the same gonorrhoea prevalence as the earlier papers, they find a 20% drop in growth rates from gonorrhoea-induced sterility, about one-third of that found in the Swinton et al. paper.

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 other proximate determinants of fertility and gonorrhoea-induced sterility such as birth spacing. A simulation model of the whole reproductive process allows the impact on fertility from each factor to be measured individually and in combination with others. An important feature of our analysis is that the historical data from Yap provide a view of uninhibited gonorrhoea transmission and infection since no barrier-method contraception or treatment for the disease was available at the time (SC interviews). Therefore, the results from the model represent an estimate of the unconstrained impact of gonorrhoea infection on population growth.

Data

Data for this paper derive from 17 years of annual reports (1920–1937), submitted by the Japanese government to the League of Nations, as part of their mandate for governing the South Sea Islands. The Annual Reports include demographic data, and notably, age-specific mortality and morbidity data. Most of the data are stratified by the six districts of Micronesia at the time.

Included in the League of Nations reports 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 data on tuberculosis and gonorrhoea prevalence in Yap. Out of the total population of 3,996 on the main island, the health of 3,787 people was examined between November 1929 and February 1930. The medical examinations showed that 15.4% of the entire population suffered from tuberculosis of any form. Dr. Fujii's 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 Fig. 2, 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).

Following the tuberculosis survey, Dr. Fuji and his team investigated the basis of the low birth rate on Yap. From April 1930 to July 1931, they examined 2,354 people (1,252 males and 1,102 females) between the ages of 8 and 60 of the 3,884 total inhabitants on Yap for gonorrhoea. The survey found that 33.3% of the Yap population was suffering from gonorrhoea (312 or 24.9% of males and 472 or 42.8% of females). Young women had the highest gonorrhoea prevalence, reaching 63% of women aged 16–20 and 51% of women aged 20–25 (see Fig. 3). Details on survey methodology were not reported, and some have challenged the validity of the results (Taeuber and Han 1950). However, other evidence suggests that they may be valid. Yap had an extraordinarily low birth rate. Although practically all women were married by age 21, 35% of married women had never conceived, and the number of conceptions per woman who had conceived was only 2.5 (Taeuber and Han 1950). These figures, consistent with evidence from previous census data and age

distributions, could be due to an extraordinarily high prevalence of gonorrhoea. Additionally, the League of Nations report details the ‘particulars of persons suffering from venereal diseases’, ranging from acute and chronic urethritis, vaginitis, cervicitis and endometritis, suggesting that a clinical examination with a speculum and microscope was used to make the diagnosis (South Seas Government 1920–1937; Hunt et al. 1954).

Although the official records provided quantitative information about the prevalence of gonorrhoea and tuberculosis on Yap, there are no survey data on sexual behaviour, social norms, labour migration, and other social processes that potentially contributed to enhanced disease spread or low birth rates. Key input on these topics and context to frame the results of the model were obtained from past ethnographic evidence (Hunt et al. 1954; Underwood 1973) as well as face-to-face interviews by one of us (SC) with residents in Yap and Palau, who lived in Micronesia during the Japanese occupation and were born no later than 1936. Eleven interviews were conducted on Yap and four on Palau. There were twelve male and three female interviewees. Their ages ranged from 68 to 85. The interviews were conducted from 26 February to 16 March 2004.

Methods

Standard life table analyses and microsimulation models of reproduction are used to assess the impact of gonorrhoea-induced infertility and tuberculosis mortality on population growth.

Life tables

Multi-decrement and associated single-decrement life table models were utilized to predict the mortality schedule, discounting all tuberculosis deaths. When the mortality schedule is added to the simulation model, then prediction of the impact of TB mortality on population growth is feasible.

We have two primary objectives: (1) to ascertain how long someone in Yap during the Japanese occupation would have lived in the absence of tuberculosis deaths; and (2) to estimate how much TB contributed to population decline in Yap. To answer the former question, we first build a simple period life table for the Yap population in 1930. Second, we constructed a multiple-decrement life table by cause of death (TB and other) to obtain the age-specific probabilities that a person will die from TB. Third, we constructed an associated single-decrement life table to predict life expectancy under the assumption that death from TB is eliminated. We compare the prediction to the initial life table (Keyfitz 1977; Preston et al. 2001).

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.:

$${}_nD_x = {}_nD_x^A + {}_nD_x^B + {}_nD_x^C + \dots,$$

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

$${}_nM_x = ({}_nD_x^A + {}_nD_x^B + {}_nD_x^C + \dots) / {}_nK_x,$$

satisfies

$${}_nM_x = {}_nM_x^A + {}_nM_x^B + {}_nM_x^C + \dots$$

Next we 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 facilitates estimation of 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:

$${}_nq_x^i = 1 - \left(\frac{l_{x+n}}{l_x} \right)^{{}_nD_x^i / {}_nD_x}$$

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

Microsimulation model

We developed a stochastic microsimulation model to generate and track the reproductive histories of a synthetic cohort of women. A cohort of 1,000 women age through the stochastic microsimulation model in one month intervals, from birth to the end of their reproductive life span at 40 years. Each month every woman is subject to a specified set of risks; namely, mortality, natural sterility, conception and childbirth, gonorrhoea, and gonorrhoea-induced infertility among women who have already contracted the disease. For each of these conditions, a random number is drawn from the uniform distribution on the unit interval and compared to the probability of the condition/event occurring. If the random number is less than the probability, we stipulate that the event occurs (e.g. conception). The status of each woman is updated monthly, and, as detailed below, the outcomes affect the course of her reproductive path. The model applies period mortality and gonorrhoea prevalence rates to the cohort-based model, thus assuming that women experience these risks as they age through the life course. Therefore the results of the model suggest how population growth would be affected if the 1930 period rates persisted at a stable rate. The principal indicator is the net reproductive rate (NRR). The NRR is a useful summary measure since it can be interpreted as the average number of daughters born to a woman subject to age-specific maternity and mortality rates throughout her lifetime. Thus if the NRR is greater than one, each generation will be larger than the previous one, implying population growth.

The model is parameterized primarily from data from the League of Nations reports, including mortality data, risk of gonorrhoea infection, and information on birth spacing, length of postpartum insusceptibility, and age at first birth. Other biological parameters are from the general literature. Table 1 presents the parameters included in the model.

In the model, women are susceptible to conception sometime between 11 and 18 years of age. Once susceptible, the probability that a woman will conceive in any month is 0.2, which

translates to an average waiting time of $(1 - 0.2)/0.2$ or 4 months (Barrett 1971). Throughout the course of the model, women are also at risk of naturally-occurring sterility (Menken et al. 1986). Possible pregnancy outcomes in the model are live birth, stillbirth, and fetal death. The probabilities do not vary by individual, but are age-dependent. Fetal deaths occur after three months gestation. Live births and stillbirths are associated with a nine month pregnancy. The length of postpartum insusceptibility is two months after fetal death or a stillbirth and nine months after a live birth.

Gonorrhoea-induced sterility is the key parameter in the model. Age-specific gonorrhoea prevalence is shown in Fig. 3. To predict risk of acquiring a gonorrhoeal infection, we assume that a typical untreated infection lasts six months (Hook and Handsfield 2008). Thus we arrive at estimates of monthly gonorrhoea incidence, or the age-specific monthly risk of acquiring gonorrhoea, by dividing the age-specific prevalence by six months. The risk of sterility resulting from gonorrhoea is assumed to be cumulative, at 6% risk per infection, which equals the probability that a gonorrhoeal infection leads to pelvic inflammatory disease (PID) multiplied by the probability that PID leads to infertility ($0.1 * 0.6 = 0.06$) (Swinton et al. 1992; Anderson 1994; Hook and Handsfield 2008). Given n infections, the probability of infertility equals:

$$1 - (1 - 0.06)^n$$

For example, if not already sterile, a woman has a 27% chance of becoming sterile after her fifth infection. Note that a woman may be at risk of gonorrhoea before she is at risk of conception in the model. Women in Yap often engaged in pre-marital and extramarital sexual intercourse, including non-penetrative sex or 'outercourse' (Hunt et al. 1954), but were greatly encouraged to delay childbearing (South Seas Government 1920–1937; Taeuber and Han 1950; Hunt et al. 1954; Underwood 1973) (SC interviews). Thus, this model assumes that young women practised natural contraception or aborted unwanted children in order to delay childbearing until the expected age.

Each simulation consists of a cohort of 1,000 women, and each model scenario was run 100 times. The mean NRR and 95% bounds of the runs are reported. A secondary outcome is percentage of childless women by age. Model outcomes are compared to reports of percentage childless, average number of children per woman who ever conceived, the mortality schedule, and reported gonorrhoea prevalence to ensure model validity.

Results

Tuberculosis mortality

Table 2 contains an 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 was less than 0.5%. Life expectancy at birth was 77.3 years for the entire population, and 50% of the initial population did not die until after age 80. In 1930 U.S., estimated life expectancy at birth was 59.7 (Arias 2004).

Table 3 shows the multiple decrement life table for Yap in 1930, where ${}_nq_x$ is the probability of dying between ages x to $x + n$ from any cause, and ${}_nq_x^i$ 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 ${}_nq_x$ and ${}_nq_x^i$ are very similar; the denominators are identical. The difference is that ${}_nq_x^i$ has the cause ' i ' specific death rate (${}_nm_x^i$) in the numerator. This specifies the dependent competing risks in multiple decrement life tables. Given a certain death rate from tuberculosis in the age interval 40–45, for example, the proportion of 40 year olds who die from tuberculosis between ages 40 and 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 et al. 2001).

A child on the island of Yap during the Japanese occupation in 1930 had no risk of dying from tuberculosis before age 5. The probability of dying from TB between age 5 to 10 is 0.028 (${}_5q_5^i$). 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 ($\pi_x^i = l_x^i / l_x$); 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.

The most important results derive from a comparison of the associated single decrement life table (Table 4) with the all-causes life table (Table 2). Note that Table 4 shows the associated single decrement life table for all causes of death other than tuberculosis. 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 (l_x), however the probability of surviving to age 20 in the absence of TB is 0.64 (${}_x l_x^i$). 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 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.

Gonorrhoea and infertility

The second step of our analysis was to run the stochastic microsimulation model with the reported mortality schedule and risk of gonorrhoea. On average, 68% of women were dead by the age of 40 in the model, which is very similar to the predicted life table presented in Table 2 ($1 - l_{40} = 0.67$). At age 40, on average 31% of women were childless (95% bounds: 0.23, 0.40) (see Fig. 3). Again, these results coincide closely to the League of Nations reports that 35% of all married women had never conceived, and 28% of women aged 36 and up had never conceived (Taeuber and Han 1950; South Seas Government 1920–1937). Among women who had ever conceived, the average number of children in the model was 2.67 (95% bounds: 2.37, 2.98). According to the League of Nations, the average number of conceptions among women who had ever conceived was 2.5 (South Seas Government 1920–

1937; Taeuber and Han 1950). Of the total number of conceptions in the model, an average of 1% were stillbirths and 17% were miscarriages or fetal deaths.

Among women who survived to age 40, the average number of gonorrhoea infections was slightly less than one infection per year from age 10 to 40. This roughly coincides with an average gonorrhoea prevalence of 42% and six months duration per infection. The average NRR in the model was 0.58 (95% bounds: 0.46, 0.69). 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 our model falls within the bounds of actual population growth in Micronesia during this time (Fig. 4).

Gonorrhoea + tuberculosis: relative and combined effects on population growth

We specify a model that is identical to the original simulation model, but mortality rates are varied in order to estimate population growth without deaths from TB. The model is run four times to estimate the NRR in four different scenarios: (1) no gonorrhoea or TB infection, (2) no TB infection but gonorrhoea at reported levels, (3) no gonorrhoea infection but TB at reported levels, and (4) gonorrhoea and TB infection at reported levels (model presented above). Monthly risk of mortality is derived from Table 3 column ${}_nq_x$ and Table 4 column ${}_nq_x^{*-i}$ for the scenarios with and without tuberculosis, respectively.

Figure 5 shows the distribution of NRR from four scenarios. The first scenario predicts the NRR without any deaths from TB and no sterility from gonorrhoea. The model estimates the NRR to be 3.44 (95% bounds: 3.22, 3.66), which translates loosely to a population growth rate equal 4.5% , assuming the mean age of childbearing to be 27.7 years. Although this is not a realistic scenario, we can nonetheless conclude that the growth rate on native Yapese would have been positive without tuberculosis or gonorrhoea infection.

The second scenario predicts the NRR excluding gonorrhoea-induced sterility but including deaths from TB. The mean NRR from 100 runs is 2.84 (95% bounds: 2.65, 3.07), which equates to a 17% drop from scenario 1. In other words, the model predicts that tuberculosis mortality alone could have been responsible for a 17% drop in the net reproduction rate. Nonetheless, the NRR is still above one; therefore tuberculosis alone was most likely not responsible for depopulation.

Once we include gonorrhoea-induced sterility the NRR drops below one, signifying depopulation. The third scenario includes gonorrhoea but excludes TB mortality. The mean NRR dropped from 3.44 to 0.62 (95% bounds: 0.46, 0.78), or by 82%. Lastly, scenario 4 includes both gonorrhoea and tuberculosis, and was presented above. This scenario shows the combined impact of TB and gonorrhoea on population growth. The mean NRR in scenario 4 is 0.58 (95% bounds: 0.46, 0.69), which translates to a population growth rate of -2.0% . TB and gonorrhoea together were potentially responsible for an 83% drop in the NRR. Tuberculosis accounted for a 6% decrease in the NRR in the presence of gonorrhoea (scenario 3 vs. 4) compared to a 17% drop without gonorrhoea (scenario 1 vs. 2), implying that the impact of TB is moderated in the presence of gonorrhoea. However, the 95% bounds significantly overlap between scenarios 3 and 4. Therefore we conclude that TB did not significantly affect population growth in the presence of gonorrhoea. Overall, the impact of gonorrhoea overwhelms the effect of TB on reducing population growth.

¹ $r = \ln(\text{NRR})/T$, 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.

Discussion

The importance of gonorrhoea in depopulation in Yap cannot be understated; without gonorrhoea-induced sterility in combination with the other determinants, the rate of population growth most likely would have been positive in Japanese-occupied Yap. Our findings fall in line with past demographic research that suggests that prolonged exposure to sexually transmitted infections is a common cause of infertility (Rutstein and Shah 2004). Our results are consistent with past work suggesting that 20% gonorrhoea prevalence could decrease population growth rates by 50% (Swinton et al. 1992). While assessing the impact of gonorrhoea-induced infertility is a key objective, fertility rates are also influenced by delays in childbearing and lengthy birth spacing (Bongaarts and Potter 1983; Davis and Blake 1956), both of which were commonly practised in Yap at the time of the Japanese occupation (Hunt et al. 1954; Underwood 1973) (SC interviews).

In terms of relative impact, we observe that 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, gonorrhoea-induced sterility affected women during their childbearing years, whereas middle-aged to elderly men and women were dying from tuberculosis. The modest impact of tuberculosis on population growth can be seen from the fact that in 1930, a newborn had a 33% chance of surviving to age 40. We find that if all deaths from tuberculosis had been eliminated, a newborn still would have had only a 50% chance of surviving to age 40.

Model limitations

Our model simply compares NRR with and without gonorrhoea or tuberculosis infection, with all else being equal. We did not consider how other behaviours or risks might change in the absence of gonorrhoea or tuberculosis. Additionally, we do not model the behaviours that lead to gonorrhoea and tuberculosis infection, but rather the consequences of those behaviours. In this manner our model does not attempt to explore the underlying determinants of infection that lead to decreased population growth.

We only had data on gonorrhoea and TB prevalence at one point in time. The outcome of our model must be considered in the context of how population growth might have been affected if rates of infection (a) were as high as reported, and (b) did not change over time. For the sake of simplicity, we only considered women. The lack of male infertility from gonorrhoea most likely led to an underestimate of the predicted role of gonorrhoea infection in depopulation. Lastly, since we did not have sex-specific death rates from TB, we assumed that the mortality schedule for women was equal to the total mortality schedule. If, for instance, men suffered disproportionately from tuberculosis, our model may overestimate the role of TB in depopulation in Yap.

Social and environmental context in Yap: conducive to infectious disease

The extraordinary gonorrhoea prevalence rates during the Japanese occupation were driven by substantial social change. Many of the respondents of the qualitative interviews indicated that alcohol became available, and the Yapese began to mix more freely with others throughout the island. Before the Japanese occupation, Yapese tended to stay close to home and only interacted with their local villagers. There was substantially more extra- and premarital sexual activity, linked to the new and widespread availability of alcohol. Indeed the Japanese investigation into the low birth rates mentioned that 'sexual intercourse is excessively indulged in for sheer pleasure' (p. 174, South Seas Government 1920–1937). Ethnographic work also suggested that Yapese engaged in 'surreptitious extramarital sexual affairs', although there were many cultural restrictions placed on married couples

identifying periods of abstinence, such as during menstruation, pregnancy, or, for example, when a man is fishing or constructing a canoe (Hunt et al. 1954). Lastly, gonorrhoea is quite contagious, and without antibiotic treatment, durations of infectiousness were longer.

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, one out of every two deaths in 1929 was attributed to a tuberculosis infection. A key issue before us is to explain why tuberculosis rates on Yap were so extraordinarily high. Past literature has shown an association between TB, socio-economic status, and household overcrowding (Antunes and Waldman 2001; Murphy et al. 2002), immigration (Antunes and Waldman 2001; Acevedo-Garcia 2001), family size, malnutrition, poor access to health care, and poverty (Elender et al. 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 et al. 1998). Many of these factors were commonly found on Yap during the Japanese occupation. Note that these factors may have led to high rates of TB, but we find that TB was not the main driver of depopulation. Thus these factors should not be interpreted as key drivers of depopulation as well.

Two unique features of traditional Yapese lifestyle might possibly have facilitated transmission of tuberculosis. They included (1) the tradition of men's clubhouses (*faluw*), and (2) housing style, particularly when construction of both kinds of structure was of poor quality. *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 (SC interviews).

However, the critical feature for tuberculosis transmission would have been the poor quality and traditional style of the *faluw* and family homes, as documented in Japanese reports. 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.

Lastly, labour migration may have played a role in tuberculosis transmission as well. Labour migration under colonial regimes has been associated with tuberculosis transmission via poor living and working conditions, inadequate diets, and circular migration. Native labour living conditions under colonial regimes were mostly crowded, unsanitary, and conducive to the spread of disease (DeLancey 1978; Dumett 1993; Manderson 1996; Packard 1989; MacPherson and Gushulak 2001; Patterson and Hartwig 1978). Often labourers 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 labourers were repatriated because they were too sick to work, but in other cases the labourers 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 labourers would work for an average of six months at the Japanese-run phosphate mines in Palau, and then return home as a new

batch of workers replaced them. All of the migrant labourers lived in single-sex barracks according to their island of origin; about 50 men would stay in one barrack. Migrant labourers worked long hours, six days a week. According to one interview respondent who used to work at the mines, the labour 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. We thus argue that labour migration may have led to high rates of TB, but not to depopulation since TB was not a main driver of depopulation.

Conclusion

Our model allowed us to examine how certain hypothetical scenarios could have resulted in alternative demographic consequences in this historical example. Our findings concur with many of the previous studies that gonorrhoea was a major driver in the depopulation of Yap during the Japanese occupation. Our work added to the literature by deriving quantitative estimates of the individual and combined impact of gonorrhoea and tuberculosis.

The modelling strategy, linked to ethnographic appraisal, we have employed could serve as a prototype for developing health impact assessments of contemporary economic development projects. In this regard, the health impact assessments for such major projects as the Chad-Cameroon petroleum development and pipeline project (Uttinger et al. 2005), the Nam Theun 2 hydroelectric project in Laos (Nam Theun Power Company 2005) and the Sakhalin Island natural gas project (<http://www.Sakhalin.Environment.Ru/en/2006>) could all have benefited from a more comprehensive assessment of alternative scenarios about the downstream health impacts of major population relocations and ecosystem transformations. For industrial projects, the International Finance Corporation (IFC) guidelines (International Finance Corporation, & World Bank Group 2009) on health impact assessments provide a framework within which this kind of modelling can be included. In addition, this kind of analysis would add greater specificity to the WHO guidelines (Health impact assessment 2006).

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Fig. 1.
Population trends for Japanese and native Micronesians, by district

Fig. 2.
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)

Fig. 3.
Gonorrhoea prevalence in the native population on the main island of Yap in 1930, by age and sex. Source: (South Seas Government 1920–1937)

Fig. 4. Simulated proportion of childless women by age: simulation begins at month 0 (birth) and ends at month 480 (40 years). Data are from 100 runs

Fig. 5. Simulated net reproduction rate under four scenarios: (1) no gonorrhoea or TB infection, (2) no TB infection but gonorrhoea at reported levels, 3) no gonorrhoea infection but TB at reported levels, and (4) gonorrhoea and TB infection at reported levels

Table 1

Parameters, point estimates, and sources used in the microsimulation model

Parameter	Value	Source
Birth outcomes		
Monthly probability of conception	0.2	(Barrett 1971; Menken et al. 1986; Trussell and Olsen 1983)
Probability of fetal death	$0.24 \cdot 0.0005^{*}(\text{age in months} - 360)$	(Trussell and Olsen 1983)
Probability of stillbirth	$0.03 \cdot 0.0001^{*}(\text{age in months} - 360)$	(Trussell and Olsen 1983)
Probability of live birth	$1 - P(\text{fetal death}) - P(\text{stillbirth})$	(Trussell and Olsen 1983)
Length of postpartum insusceptibility	9 months	(South Seas Government 1920–1937; Hunt et al. 1954)(Cassels' interviews 2004)
Gonorrhoea disease dynamics		
Monthly probability of acquiring gonorrhoea infection	Derived from Fig. 3	(South Seas Government 1920–1937)
Probability of infertility	$1 - (1 - 0.06)^n$, where n = number of gonorrhoea infections	(Anderson 1994; Swinton et al. 1992)
Population dynamics		
Age at risk of first birth	Uniformly distributed from 11 to 18 years	(South Seas Government 1920–1937; Underwood 1973)(Cassels' interviews 2004)
Age at menopause	40 years	(Barrett 1971; Menken et al. 1986)
Monthly mortality	Derived from Fig. 2	(South Seas Government 1920–1937)
Natural sterility	5.7% for women age 20–24; 9.3% age 25–29; 15.5% age 30–34; 29.6% age 35–39; and 63.6% age 40–44	(Menken et al. 1986)

Table 2

Life table (all causes) for the population of the main island of Yap, 1930

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

Table 3

Multiple decrement life table for the main island of Yap, 1930: Cause i = death from tuberculosis of the lungs

Age x	nD_x	nD_x^i	l_x	nq_x	nq_x^i	$n d_x^i$	l_x^i	π_x^i
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 4

Associated single decrement life table for causes of death other than tuberculosis (−i), main island of Yap, 1930

Age x	D_x^{-i} / nD_x	${}^*q_x^{-i}$	${}^*l_x^{-i}$	$n a_x$	${}^*d_x^{-i} / n$	$n l_x$	T_x	${}^*e_x^{-i}$
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