INTRODUCTION

It is postulated that humans have been subject to infectious disease pressure throughout evolutionary history, it is not clear to what extent, if any, communicable disease (transmitted via direct contact with an infected individual or indirectly through a vector) was important to shaping mortality prior to the rise of agriculture and urban centers about 10,000 years ago. Many anthropologists believe that communicable diseases did not represent an important source of pre-agricultural mortality. The most compelling “evidence” for lack of communicable diseases pre-agriculture is that population sizes would have been too small to maintain communicable diseases without burning out.

The majority of existing tuberculosis (TB) models are deterministic, they define states for individuals within a population—essentially assigning individuals to subpopulation groups based on characteristics such as stage of disease—and specific rules by which individuals move among states through a series of differential equations. The risk of transmission per contact is given an average value across the population. Using a model like this one, TB cannot be maintained in a population of 10 persons, indeed, it is able to take hold at all.

However, we know that humans behave in complex variations and patterns. Contact is not equally distributed across a population. To create a more realistic and agent-based model, which average contact rates, the results of my preliminary model clearly demonstrate that a large population size is needed to maintain communicable disease when the population exhibits structured mixing. For the initial parameters used, and a stationary population, TB can remain endemic for 500 years.

CURRENT METHODS

I have used the statistical software R to program a stochastic social network model, the nodes in which represent individuals defined by a set of attributes. Presence and strength of a tie between any two individuals is based upon the intersection of the attributes of that pair of individuals: stronger ties indicate greater contact and risk of an airborne infectious exposure. This differs from previous models because I allow for a highly structured population by allowing contact among individuals to vary, rather than assuming equal contact across levels of the population. Over time, individuals are born, age, experience a natural mortality rate, and can transition between disease states. Initially, I maintain a stationary population of 10 people, new individuals are introduced into the population with every death and are assigned to a household based on the number of households having at least two members. Between discordant pairs (i.e., one susceptible and one infectious person) there exists some probability of disease transmission that is correlated with the usual CSI experienced by that pair.

CONTACT STRENGTH INDEX (CSI)

The contact strength index function (CSI) is a numerical score between 0 and 1 that captures relative contact among individuals, which can be predicted based on their respective age, sex, and household and village statuses. Within a small population, all individuals are tied to one another, but the strength of each tie depends on attributes of the individuals involved. For each specific pair-type, there is a beta distribution (Figure 1) of actual contact levels, from which a number is estimated for each specific pair of people at each time step. Within a particular village, the means of the beta distributions (α) are scaled relative to the degree of contact between a mother and newborn (Table 1).

The probability density function of the beta distribution is

\[
\Gamma (x; \alpha, \beta) = \frac{\Gamma (\alpha + \beta)}{\Gamma (\alpha) \Gamma (\beta)} x^{\alpha-1} (1-x)^{\beta-1}
\]

where \( \Gamma \) is the gamma function. If \( x \) is a positive integer, \( \Gamma (n) = (n-1)! \)

The cumulative distribution function is

\[
F(x; \alpha, \beta) = \frac{B(\alpha, \beta)}{B(\alpha, \beta) + I(\epsilon) \alpha, \beta}
\]

Where \( I(\epsilon) \) is the regularized incomplete beta function.

The mean of the beta function is \( \alpha/\alpha + \beta \)

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Ongoing Questions

What is the relationship between disease (DM) and non-disease mortality (NDM) in pre-agricultural settings?

- Modern human mortality hazard curves are similar across populations (Figure 2), and include DM+NDM. To create life tables for anthropological populations, Weiss (2) relies on a number of pre-agricultural settings, is DMDNM correct?

- If these rates (2) are applied in pre-agricultural settings, is DMDNM correct?

- Is the contact structure reasonable, and how can it be enhanced?

- Does the beta matrix (Table 1) capture important aspects of within-village contact?

- Should between-village contact be a multiple of within-village contact, as done by McGrath (3)?

- I am including group membership as an additional attribute.

- The simplest of all transmission models is (4), but TB mortality to them, implying that the “natural” Weiss rates do not include DM. Is this correct?

- How was the effect of TB on the population be characterized from those of the population on TB?

- Create an explicitly ecological model, with birth and death rates fluctuate in response to the population size’s proximity to a given environmental carrying capacity.

- Explore both the threshold population sizes needed to maintain TB, and the levels of disease-mortality above which a population cannot effectively recover.

- Should TB directly affect birth rates? How?

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Literature Cited


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Figure 1. Beta probability density function (1).

Figure 2. Age-specific hazard of mortality for four modern populations (4).

Table 1. Matrix of relative means of CSI beta distributions.