Malaria Mortality Favors Patriliny

James Holland Jones *
Department of Anthropology
Stanford University

Emilio Zagheni
Max Planck Institute
for Demographic Research

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Abstract

In this study we analyze how demographic forces might constrain social organization by examining the impact of infectious disease mortality on human stocks. Infectious diseases have been a major source of mortality and an important selective force throughout human history. We test the hypothesis that high mortality in the characteristic age-sex pattern of countries where malaria is endemic favors patrilineal social organization. We use demographic microsimulation (the SOCSIM platform) calibrated with a variety of data sources including the INDEPTH African model. Preliminary results show that, as a result of higher mortality for males in pre-reproductive age, the average relatedness through patrilineal kin is higher than through matrilineal kin, thus favoring social organization based on patrilineal descent.

*Correspondence Address: Department of Anthropology, 450 Serra Mall, Building 50, Stanford, CA 94305-2034; phone: 650-723-4824, fax: 650-725-0605; jhj1@stanford.edu

EXTENDED ABSTRACT

1 Introduction

Using data from the Standard Cross-Cultural Sample (SCCS), we see that patrilineal kinship is considerably higher than expected in Sub-Saharan Africa. Sub-Saharan Africa is also subject to very high levels of malaria endemicity.

Demography clearly plays a central role in social structure since it is the stocks and flows of people that are the raw material of social structure. In a foundational analysis, Hammel (1976) re-considered the classic arguments of alliance theory cross-cousin marriage preference (e.g., Levi-Strauss 1949) which maintains that the predominance of MBD cross-cousin marriage arises because of its effect of reticulating diverging kindreds, permitting the formation of larger kin-based alliances. Using a simple logical model, Hammel showed that a bias for MBD marriages can arise simply from age consistent preferences of men for wives younger than themselves. When such marriage age preferences are in place, on average, the only cousin who will be consistently younger than ego is his MBD. Hammel applied demographic microsimulation to confirm this prediction.

Wachter et al. (1978) showed that demography greatly constrained the possibility of stem families in early modern Europe, providing evidence that the nuclear family was the predominant form of social organization at this critical phase of European history.

Ellison (1994) developed a model based on branching-process theory (Galton and Watson 1874) that predicted members of patrilines to be generally more related to each other than members of matrilines. This pattern results from the depth of the respective lineages. Because men's reproductive success (RP) is generally more variable than women's, the branching process model predicts greater rates of lineage extinction in patrilines (Ellison 1994). When more individual lineages go extinct, the remaining individuals are necessarily more closely related. Ellison (1994: 160) argues that "to the extent that male RS variance exceeds female variance, coalitions formed on the basis of patrilineal descent will, on average, be much larger than those formed on the basis of matrilineal descent, or any other single rule of tracing descent, at a given degree of relatedness." If power follows from the numerical strength of coalitions, then patrilineal descent provides a powerful mechanism for mobilizing kin for a variety of ends (e.g., political, economic, defensive).

We can apply similar reasoning to that of Ellison (1994) in thinking about the kinship consequences of different forms of mortality. In particular, consider a source of mortality that shows consistent patterns in age and sex-specific patterns. A source of mortality that systematically affects pre-reproductive males, for example, will reduce the size of any given male cohort. As long as the population does not decline, the following generation will be more closely related on average through their patriline than their matriline simply because fewer men sire the replacement (of larger) cohort. For a fixed cohort, a smaller number of sires means that the cohort will be on average more closely related than the same size cohort with a larger number of sires. Indeed, this is just a specific mechanism by which high variance in male RS arises. Male variance is large because all the males who die in childhood have zero reproductive success.

A number of sources of mortality show pronounced biases in their age-sex distribution. In this paper, we test the hypothesis that the pattern of mortality characteristic of malaria endemic

areas favors patrilineal social organization. To test this hypothesis, we employ demographic microsimulation. Following Ellison (1994), we expect patriliny to be favored when patrilineal kin are more closely related on average than matrilineal kin or, equivalently, when the number of patrilineal kin (e.g., cousins, uncles, aunts) is greater than the number of matrilineal kin. Do malarial mortality patterns yield distributions of kin biased toward patrilines? Are there interactions with sex-specific fertility patterns and malaria mortality that make patriliny more likely?

It is fairly obvious that male-biased differential mortality during childhood would favor polygynous marriage systems where cultural norms permit them. What is perhaps less obvious is the fact that this pattern of mortality increases the relative power of patrilines.

2 Materials and Methods

2.1 Model Life Tables

We use the INDEPTH model lifetables (MLTs) (INDEPTH Network 2002; Clark et al. 2010) to generate malaria-dominant mortality schedules. INDEPTH model lifetables are component mortality models, similar in concept to the well know Coale-Demeny MLTs (Coale et al. 1983), but constructed using a much more transparent and intuitive methodology. INDEPTH MLTs are constructed by first assembling a collection of high-quality empirical mortality schedules from the developing world (primarily Sub-Saharan Africa). Mortality components are estimated using multivariate statistical techniques. In particular, empirical estimates of ${}_nq_x$ are first logit-transformed and then an $n \times n$ correlation matrix of $\text{logit}({}_nq_x)$ values is constructed for the n empirical lifetables. Principal components (PCs) are extracted from this correlation matrix and specific mortality clusters are determined.

To then construct the actual MLTs, the empirical $\text{logit}({}_nq_x)$ values are regressed on the 18×15 matrix of PCs $\mathbf{C}$. To add an intercept to the regression model, the matrix $\mathbf{C}$ was appended with a trailing column of ones, yielding a new matrix $\mathbf{C}'$. Let the vector of coefficients from this regression be $\mathbf{a}$. This is a column vector of 16 elements representing the regression coefficients on the 15 mortality components (i.e., the PCs) plus a constant. This constant sets the level of mortality, while the coefficients on the components determine shape of the schedule. Let $\mathbf{m}$ be the logits of the ${}_nq_x$ schedule for the desired model lifetable. We generate these $\mathbf{m}$, and consequently the desired MLT, through a simple matrix multiplication,

$$\mathbf{C}'\mathbf{a} = \mathbf{m}. \tag{1}$$

Fixing the first 15 components of $\mathbf{a}$ will generate a family of mortality schedules for which the level can be changed by altering the constant (i.e., the 16th element of $\mathbf{a}$). Different families of MLTs can be generated by using different PCs, $\mathbf{C}$, themselves arising from reducing different clusters of empirical life tables.

Clark and co-workers (INDEPTH Network 2002; Clark et al. 2010) identified seven mortality clusters in the empirical INDEPTH life tables. One of these clusters includes primarily mortality schedules from West Africa. They show a relative excess of infant and early child mortality, a feature indicative of malaria mortality (Coale et al. 1983; INDEPTH Network 2002). Indeed, the populations that comprise this cluster are largely from hyper-endemic malaria areas.

The published INDEPTH MLTs are only available for fixed values of e_0 (INDEPTH Network 2002). However, it is relatively straightforward to generate model life tables with arbitrary e_0 from the published PCs and coefficients from equation 1. Using the matrix of the first 15 principal components of the empirical life tables included in the INDEPTH collection, we could vary the constant and then pick the value that yields a MLT with the desired e_0 by means of numerical optimization.

2.2 Fertility

We picked two fertility schedules for comparison. The first comes from a Gambian population in the 1990s (Ratcliffe et al. 2000). This was a very fecund population with strikingly different patterns of male and female fertility. Total fertility rates of men and women were 12 and 6.8 respectively. Men achieved such high fertility through serial and polygynous marriages and the extension of reproduction well beyond the age of last reproduction for women. Age-specific fertility rates (ASFRs) were taken from figure 2 of Ratcliffe et al. (2000) using DataThief software. We smoothed the resulting ASFR curve using smoothing splines using a smoothing parameter of 0.4.

The second fertility schedule comes from the demographic reconstructions of the forest-period for Aché hunter-gatherers of eastern Paraguay (Hill and Hurtado 1996). The Aché are also very fecund, but are characterized by a largely monogamous marriage system. Data on Aché ASFRs were taken from the table in Hill and Hurtado (1996). We applied smoothing splines to the Aché ASFR data as well both to smooth the slightly jagged male schedule (due to small sample sizes for older men) and to make the results maximally comparable. Again, the smoothing parameter was set to 0.4.

Rapidly growing populations will have more kin of all kinds than slowly growing or stationary populations. To control for this potential confound, we standardized all the fertility schedules so that they yielded stationary populations when combined with the specific mortality schedule of the simulated population. The net maternity ratio, R_0, is the expected number of newborn offspring that a woman in a particular population can expect to have. It is also the ratio of population size from one generation to the next. R_0 is the sum of the age-specific net maternity schedule:

$$R_0 = \int_{\alpha}^{\beta} l(x)m(x)dx, \tag{2}$$

where α is age at first reproduction, β is age at last reproduction, $l(x)$ is the probability that a woman survives to exact age x, and $m(x)$ is the ASFR at age x.

We can standardize fertility for a fixed $l(x)$ schedule by simply dividing by R_0: $m'(x) = m(x)/R_0$. Like the construction of MLTs, this preserves the shape of the fertility schedule while changing the total level of fertility. By doing this, we control for the potentially confounding effect of differences in net fertility on the number of kin under different mortality and fertility regimes.

2.3 Demographic Microsimulation

We simulate populations with different mortality/fertility combinations using the demographic microsimulation software SOCSIM (Wachter et al. 1978; Wachter 1987; Wachter et al. 1997). Our simulations are individual-based, using an event-competition approach described, for example, by Gillespie (1977). For each individual in the simulation, there are a number of events that are possible, given the individual's age, sex, and marital status. The waiting time to each event for which the individual is at risk follows a piecewise exponential distribution and is generated randomly from the input demographic rates. A given individual's next event is the one with the shortest waiting time. Because marriage is a two-sided process, marriage formation is slightly more complicated. The population simulated by SOCSIM is 'closed': all partners must be drawn from within the existing population. To deal with this, SOCSIM employs a two-stage process to pair eligible women and men from within the simulated population. When the next scheduled event for an individual is 'marriage', then the person is placed in a pool of eligible members to form a union. If a member of the opposite sex with appropriate demographic characteristics is available in the pool, then the two individuals are paired. Otherwise the person remains in the available pool until an appropriate mate is found. Like all other events, marriages are based on a random process with probabilities dependent on demographic characteristics of the potential spouses.

The output from a SOCSIM simulation run are two large tables consisting of all the individuals and marriages, respectively, from the simulated population. The information output in the individual table is (1) the unique person identifier ("person id") (2) sex, (3) group (for populations with specific group structures, such as ethnicities), (4) the next scheduled event in the simulation (or death if the individual is already dead), (5) birth date; (6) mother's person id, (7) father's person id, (8) eldest sibling via mother's person id, (9) eldest sibling via father's person id, (10) last-born child's person id, (11) marriage identifier ("marr id") of last union, (12) marital status, (13) death date, (14) fertility multiplier (to model heterogeneity), or 0, if male. For items (6)-(9) the value is 0 if the person was in the initial seed cohort. From the person id's and marr id's it is a straightforward to calculate the relationships of all members of the population except those of of the initial seed cohort.

3 Preliminary Results

We ran several different simulations with SOCSIM, comparing combinations of fertility rates from the Gambia and the Aché, and mortality patterns from INDEPTH model lifetables. Table 3 shows the average number of paternal and maternal cousins for different combinations of fertility and mortality profiles, in presence and absence of polygamy. Table 3 shows that there is a potentially important confounder in our analysis, polygyny, which is associated with patriliny. With a smaller breeding sex ratio (i.e., smaller number of fathers relative to mothers) in the P1 generation, members of the F1 will be more related, on average, than in a population with an even breeding sex ratio. When we condition the marriage system to be monogamous, we observe that the average relative abundance of cousins on the paternal side vs maternal side is higher when the pattern of mortality is typical of regions where malaria is endemic.

Simulation work presents a number of challenges for statistical inference. In particular,

	Ache fertility profile			
	Malarial mortality		Non-malarial mortality	
	Paternal cousins	Maternal cousins	Paternal cousins	Maternal cousins
Monogamy	15.1	11.12	6.78	5.9
	(0.54)	(0.38)	(0.29)	(0.25)
Polygamy	6.22	2.2	2.52	0.86
	(0.31)	(0.13)	(0.15)	(0.08)

Table 1: Average number of paternal and maternal cousins for different combinations of fertility and mortality profiles, in presence and absence of polygamy. The numbers between parentheses are the standard errors for the means, based on 2000 observations.

hypothesis tests are problematic because standard errors can be made arbitrarily small by simply increasing the number of simulations (this observation lies at the heart of Bayesian critiques of frequentist hypothesis testing (e.g., Berger and Delampady 1987)). To compare the different outputs, we fix the number of observations to 2000. These 2000 observations are obtained by sampling 50 individuals out of each of the 40 simulation runs with different random seeds and counting their kin along patrilines and matrilines. By fixing the number of observations for all comparisons, we are then able to present mean numbers of kin with internally meaningful 95% confidence intervals. It is important to note that such confidence intervals will be different for different number of simulation replicates.

A second important confounder is related to population growth. Different combinations of fertility and mortality rates are associated to different population growth rates. The faster the population grows, the larger the average number of members of the same kin. We control for that by adjusting the level of the fertility schedules upward or downward in order to make the population stationary. Figure 2 shows a comparison of matrilineal vs. patrilineal kin using the INDEPTH malaria mortality schedule, a stationary fertility schedule, and a monogamous marriage system . The relative abundance of patrilineal kin is generally higher than the one of matrilineal kin. These preliminary results support our hypothesis. To the extent that the size of coalitions is representative of power, then the pattern of mortality characteristic of regions where malaria is endemic favors patrilineal social organizations.

Acknowledgments

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Figures

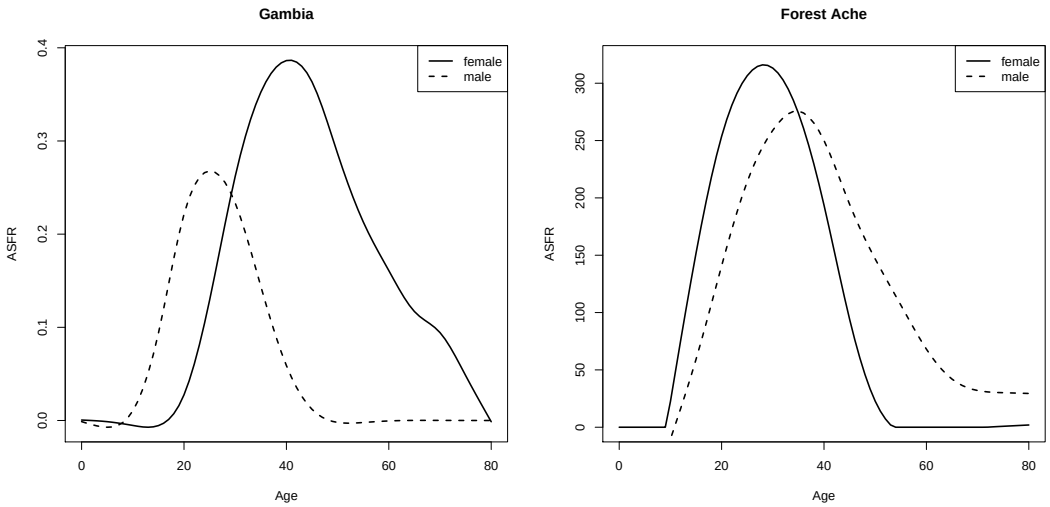

Figure 1: The smoothed sex-specific ASFRs for the Gambia and Aché.

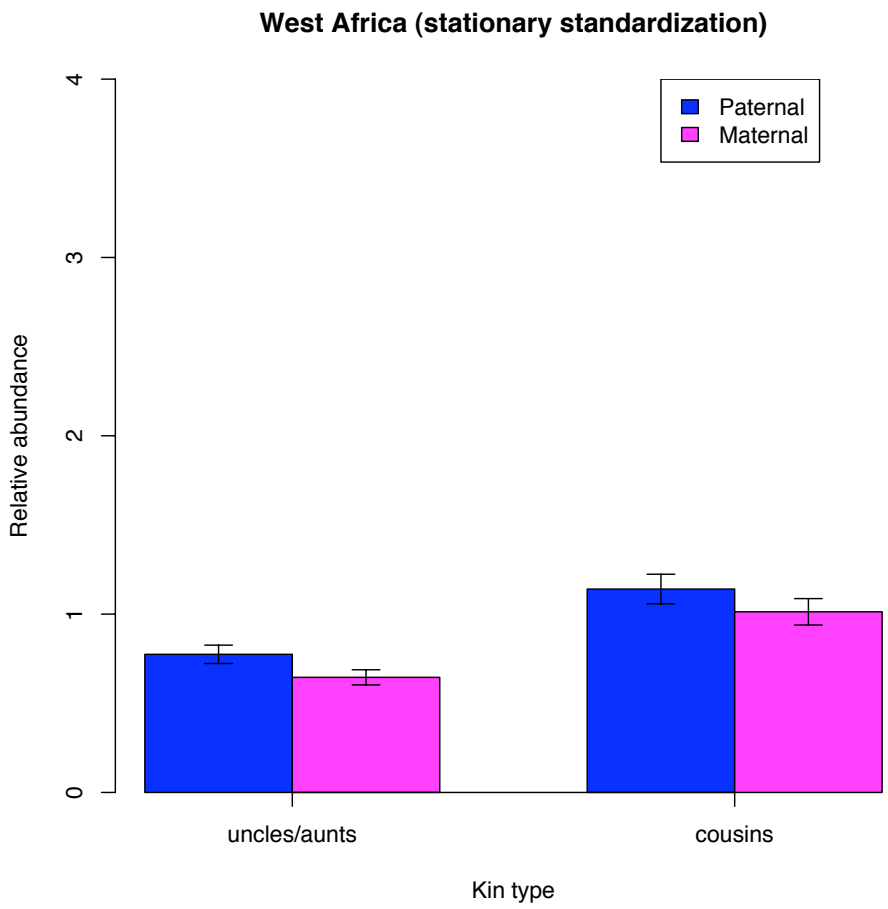

Figure 2: Comparison of matrilineal vs. patrilineal kin using the INDEPTH malaria mortality schedule, a stationary fertility schedule, and a monogamous marriage system.