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Pathogen Intensity Cross-Culturally

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1. INTRODUCTION

Serious pathogens represent a special category of environmental uncertainty, and one likely to have more far-reaching effects than climatic or other physical unpredictabilities with which humans must cope (e.g. Low 1988a, 1989a). Above and beyond the obvious difficulties serious pathogens represent, they have been implicated in sexual selection (Hamilton and Zuk 1982, Read 1987, 1988, Read and Harvey 1989, Low 1988a, 1990) both through intersexual competition and female choice (Fisher 1958, Williams 1966), as well as in the evolution of sexual reproduction itself (Hamilton 1980, Hamilton et al. 1981, Seger and Hamilton 1987). Pathogen stress is a possible example of Zahavi's (1975) handicap principle: that sexual selection may force males to pay obvious, discernable costs (e.g., costly advertisements), simply to prove that they can pay. As Pomiankowski (1987, 1988) has shown, when additive genetic variance in viability is maintained over several generations, the male handicapping trait is favored. Thus (Pomiankowski 1988), preference is more likely to evolve through the handicap principle for traits that are reliable indicators of heritable viability and do not excessively penalize the males that bear them.

In human societies, there is a positive non-linear relationship between the intensity of pathogen stress and the intensity of polygyny (Low 1988a, 1990). These patterns do not rely on the heritability of resistance to pathogens, which is difficult to test (Hamilton 1980, Mosseau and Voge 1981, Pomiankowski 1989, Read and Harvey 1989), though the sample here will be restricted to those pathogens meeting Hamilton and Zuk's criteria, for consistency. Polygyny, being profitable for those men who can manage it, (e.g., see Betzig 1986, Low 1988b) occurs in all sorts of ecological types, including relatively pathogen-free areas, but monogamy and mild polygyny are absent from areas of high pathogen stress.

These results are not simply due to co-variance of pathogens and polygyny geographically (Low 1990). This relationship appears to derive from the fact that as pathogen stress increases, fewer men are healthy, appropriate providers (a form of the polygyny threshold hypothesis; Verner and Willson 1966, Orians 1969). Further, as pathogen stress increases, the capture of women, and the choice of non-sororal (rather than the more common sororal) polygyny, become more common; both of these increase the genetic variability of offspring (Low 1990). In fact, 52% of the cross-cultural variation in female capture is explained, using logistic regression (Finney 1971), by pathogen stress (Low 1990).

Culturally augmented ornaments show no pattern with pathogen stress. Societal formalizations or customs may give less accurate information about worth as a mate than direct evidence of health from appearance and behavior, and even physical size (e.g., dimorphism) may reveal only that a potential mate was healthy during childhood and adolescence. Further, humans accumulate, make heritable, and use resources in connection with both mating and parental effort to a degree unknown in other species (e.g., Flinn and Low 1986), and these resources use may have significant value independent of health (Low 1989b).

Major pathogens may well have been an important selective force during human evolutionary history, shifting the polygyny threshold. New work could profitably focus on within-society patterns of pathogen stress and reproductive success of individuals, and on associated problems of ornamentation in sexual selection (Kurland personal communication).

Here, the sample and the descriptions are offered, to help facilitate further work.

2. Sample

The sample comprised the 186 societies of the Standard Cross-Cultural Sample (Murdock and White 1969), stratified for geographic region and language group, and for which ethnographies are available by qualified ethnographers resident with the society for a substantial period. This is the sample typically used by cross-cultural investigators, to represent the breadth of cultural diversity known, and to minimize potentially confounding (e.g. geographically localized) effects (see Naroll 1961, Thornhill 1990, and Low 1990 on Galton's problem).

3. Pathogens

The pathogens chosen for analysis were leishmanias (Leishmania tropica, L. braziliensis, L. donovani); trypanosomes (Trypanosoma gambiense, T. rhodesiense); malaria (Plasmodium vivax, P. ovale, P. maliariae, P. falciparum); shistosomes (Schistosoma japonicum, S. mansoni, S. haematobium); the filariae (Wucheria bancrofti, Brugia malayi); spirochetes (Borrelia duttoni, B. recurrentis, Treponema), and leprosy (Mycobacterium leprae). These pathogens meet the criteria of an acute, possibly fatal initial stage of infection, and long-term chronic debilitation or recurrence of acute episodes; further, good worldwide geographic records exist for them. All have sufficiently short generation times relative to humans to produce the requisite unpredictability (cf. Low 1988a). Their characteristics (Beaver et al. 1984, Markel and Voge 1981, Faust and Russell 1964, Craig and Faust 1970) are as follows:

Leishmanias (Leishmania tropica, L. braziliensis, L. donovani).

> L. tropica produces cutaneous leishmaniasis, or Oriental Sore. Infestation involves blood cells and lymph nodes. Necrosis and ulceration at cutaneous sites are common, as are secondary bacterial infections, with fever, chills, and other usual symptoms of infection. Sores may be single or multiple, "moist" or "dry." L. braziliensis also produces lesions with hyperplasia and intense dermal inflammation, with edema. In some areas, secondary lesions become very common, with edema and extensive erosion of both hard and soft tissues. L.

donovani produces visceral leishmaniasis (kala-azar), with malaise, headache and fever, bleeding of mucous membranes, dysentery or diarrhea, and marked enlargement of the spleen and liver. Death may occur in the acute stages in the leishmanias, or as a result of complications during the chronic stages.

Trypanosomes (Trypanosoma gambiense, T. rhodesiense, T. cruzi).

T. gambiense produces an initial almost symptomless period, an acute febrile period, a chronic stage, and a sleeping sickness stage with central nervous system involvement. Lymph nodes are invaded, with an accompanying attack of fever lasting about a week. Later quiescent periods alternate with febrile periods, typically accompanied by dyspnea, cardiac pain, disturbed vision, anemia, and loss of strength. With invasion of the nervous system, the sleeping-sickness phase begins, with severe headache, mental dullness, apathy, muscle spasms, trembling, and sometimes mania, melancholia, and delusions. Death may result from complications like malaria, schistosomiasis, amoebic or bacillary dysentery, bronchopneumonia, meningitis, or by simple heart failure. T. rhodesiense has similar symptoms, but the symptoms develop more rapidly, and fatal termination is often a matter of months rather than years. T. cruzi, which produces Chagas' Disease, has acute (more prevalent in children, but also found in adults) and chronic phases, and similar symptoms.

Malaria (Plasmodium vivax, P. ovale, P. maliariae, P. falciparum).

The symptoms of various forms of malaria are among the best-known of the pathogens considered here: definite paroxysms of chills, fever and sweating, with different rhythms for the different Plasmodium species. Relapses are typical; death is not uncommon in untreated cases of P. falciparum malaria. Congenital malaria is rare in endemic areas, and is more common in non-immune infected mothers. There is debate whether transmission is through the placenta, or at birth itself.

Schistosomes (Schistosoma japonicum, S. mansoni, S. haematobium).

The three species of schistosomes have almost identical pathogenesis; the intestinal tract and the liver are most affected, and the degree of symptoms depends on the worm burden and host reaction. During the incubation period, symptoms comprise dermatitis associated with the cercaria penetrating the skin, infiltrative involvement of the lungs with hemorrhage, acute hepatitis, hyperemia in the wall of the small intestine, trauma with hemorrhage in the intestine as the eggs escape into the intestinal canal, and eosinophilia. The cellular immune response of the host continues for years, although it may become less evident during the chronic stage; the functions of the small intestine and the liver are impaired as pseudotubercule form around eggs. Other manifestations include dysentery, hepatic fibrosis, splenomegaly, appendicitis, intestinal obstruction, vascular occlusion, myelitis, and cerebral syndrome.

Filariae (Wucheria bancrofti, Brugia malayi).

The filarial infections may have an incubation period of over a year. In non-endemic areas, there is an acute inflammatory reaction, with reddened, swollen lymph areas, malaise, fever and pain; these are exacerbated by exertion. Attacks tend to be recurrent. In endemic areas, the initial stages may be asymptomatic. The chronic stage follows, accompanied by swelling and fibrosis, as the worms die and are absorbed or become calcified. Severe elephantiasis occurs in a relatively small percentage of individuals in endemic regions.

Spirochetes (Borrelia duttoni, B. recurrentis, Treponema).

These tick- and louse-borne pathogens produce relapsing fever, characterized by episodes of fever lasting from a few days to two weeks, separated by afebrile periods of 2-4 days. The louse-borne variety is the more severe, with a mortality rate of over 50% in untreated individuals.

Leprosy (Mycobacterium leprae).

Leprosy occurs principally in two forms, a more malignant and progressive lepromatous type, and a relatively benign and stable tuberculoid type, but there is transition between the two types. The incubation period can last for years, with skin lesions and neurological disturbances, including anaesthesia and painful neuralgia. Systemic manifestations include lymphadenopathy, anemia, and lepra fever (an acute febrile episode of several weeks' duration, with evanescent skin lesions). Spontaneous remissions can occur in the tuberculoid type.

The lepromatous type, if untreated, shows a progressive course, and death results from direct damage of leprosy or from complications of tuberculosis, intercurrent disease, etc.

Each of these pathogens represents a risk of serious debilitation, even death. For each pathogen, a three-level code was constructed: 1=absent or not recorded; 2=present, no indication of severity; 3=present and serious, widespread, or endemic. Societies were located by latitude and longitude, and data for coding were taken from Beaver et al. (1984), Markel and Voge (1981), Faust and Russell (1964), Craig and Faust (1970) and maps of the American Geographical Society of New York (1950-1955), constructed from WHO data. When data existed from more than a single time period, data from the period closest to that of the ethnography were used, although previous tests established that there is no relationship between date of ethnography and either pathogen stress or degree of polygyny (Low 1988a). Naive coders worked from these sources, and intercoder agreement in codes was over 95%.

In previous work on pathogen stress, sexual selection, and marriage systems (Low 1988a, 1990), total pathogen stress was important, and there was no a priori reason to predict a pattern between any of the proposed dependent variables and any particular pathogen. Thus, in that work, the sum of the scores for all pathogens was used as a measure of total pathogen stress for each society (Low 1988a). This variable ranged from a score of 7 (none of the pathogens recorded for the area) to a score of 21 (all seven pathogens endemic and serious); it was further reduced to a three-level ordinal code (Low 1988a). STDS64.DAT and STDS64.COD contain the original codes.

1253. LEISHMANIAS

N	Code	Meaning
117	1	= Absent or not recorded
35	2	= Present, no indication of severity
4	3	= Present and serious, widespread, or endemic

1254. TRYPANOSOMES

N	Code	Meaning
134	1	= Absent or not recorded
38	2	= Present, no indication of severity
14	3	= Present and serious, widespread, or endemic

1255. MALARIA

N	Code	Meaning
55	1	= Absent or not recorded
20	2	= Present, no indication of severity 111 3 = Present and serious, widespread, or endemic

1256. SCHISTOSOMES

N	Code	Meaning
129	1	= Absent or not recorded
16	2	= Present, no indication of severity
41	3	= Present and serious, widespread, or endemic

1257. FILARIAE

N	Code	Meaning
89	1	= Absent or not recorded
1	2	= Present, no indication of severity
96	3	= Present and serious, widespread, or endemic

1258. SPIROCHETES

N	Code	Meaning
76	1	= Absent or not recorded
45	2	= Present, no indication of severity
65	3	= Present and serious, widespread, or endemic

1259. LEPROSY

N	Code	Meaning
70	1	= Absent or not recorded

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 72    2 = Present, no indication of severity
 44    3 = Present and serious, widespread, or endemic

1260.  TOTAL PATHOGEN STRESS

 N    Code      Meaning

 30    7 = Sum of variables 1253-1259 is 7
  9    8 = Sum of variables 1253-1259 is 8
  9    9 = Sum of variables 1253-1259 is 9
  4   10 = Sum of variables 1253-1259 is 10
 20   11 = Sum of variables 1253-1259 is 11
 20   12 = Sum of variables 1253-1259 is 12
 13   13 = Sum of variables 1253-1259 is 13
 20   14 = Sum of variables 1253-1259 is 14
 16   15 = Sum of variables 1253-1259 is 15
 16   16 = Sum of variables 1253-1259 is 16
  9   17 = Sum of variables 1253-1259 is 17
  9   18 = Sum of variables 1253-1259 is 18
  6   19 = Sum of variables 1253-1259 is 19
  3   20 = Sum of variables 1253-1259 is 20
  2   21 = Sum of variables 1253-1259 is 21
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