PROCEEDINGS OF THE ROYAL SOCIETY B

Proc. R. Soc. B (2008) **275**, 2587–2594
doi:10.1098/rspb.2008.0688
Published online 29 July 2008

Assortative sociality, limited dispersal, infectious disease and the genesis of the global pattern of religion diversity

Corey L. Fincher* **and Randy Thornhill**

Department of Biology, University of New Mexico, Albuquerque, NM 87131, USA

Why are religions far more numerous in the tropics compared with the temperate areas? We propose, as an answer, that more religions have emerged and are maintained in the tropics because, through localized coevolutionary races with hosts, infectious diseases select for three anticontagion behaviours: in-group assortative sociality; out-group avoidance; and limited dispersal. These behaviours, in turn, create intergroup boundaries that effectively fractionate, isolate and diversify an original culture leading to the genesis of two or more groups from one. Religion is one aspect of a group's culture that undergoes this process. If this argument is correct then, across the globe, religion diversity should correlate positively with infectious disease diversity, reflecting an evolutionary history of antagonistic coevolution between parasites and hosts and subsequent religion genesis. We present evidence that supports this model: for a global sample of traditional societies, societal range size is reduced in areas with more pathogens compared with areas with few pathogens, and in contemporary countries religion diversity is positively related to two measures of parasite stress.

Keywords: cross-cultural psychology; ethnocentrism; limited dispersal; religion; social behaviour; xenophobia

1. INTRODUCTION

Why does the country Cote d'Ivoire have 76 religions while Norway has 13, and why does Brazil have 159 religions while Canada has 15 even though in both comparisons the countries are similar in size? One potential answer, that we pursue here, is simply that the genesis of religions has occurred at different rates across the world. We suggest that the genesis of religions has varied spatially across the globe because religion manifests from evolved behavioural strategies for the avoidance and management of infectious disease. And, importantly, infectious disease stress varies across the globe.

(a) *Model: the parasite-driven wedge*

The process we propose works in a human group to generate discontinuity and hence religion diversity as follows.

(*a*) Initially, the group has a geographical distribution and a uniform cultural repertoire and distribution of immunity.

(*b*) Over time, parasite–host antagonistic coevolutionary races (Red Queen races; Van Valen 1973; Ridley 1993; Ewald 1994; Graham *et al.* 2005) become spatially distinct and localized across the range of the culture. This spatial variation is the result of localized emergence of new parasites and the evolution of locally adaptive immunity. The new parasites may be new species or novel varieties of already present species.

* Author for correspondence (fincher@unm.edu).

Electronic supplementary material is available at http://dx.doi.org/10.1098/rspb.2008.0688 or via http://journals.royalsociety.org.

An important condition of this model is that parasite–host coevolutionary races are spatially variable, generating spatial variance in the immunobiology of human host populations and groups. The distribution of human parasites and human responses to them actually do vary spatially. This variation is seen on regional and latitudinal scales as well as more fine-grained scales such as nearby villages (see review in Fincher & Thornhill 2008).

(*c*) At this point, there is spatial variation across the culture's range in people's ability to meet the immune challenge of infected individuals. Thus, antipathogen behaviours are selected and manifest as the avoidance of humans who are infected or potentially infected with dangerous parasites. These behaviours are limited dispersal reducing the interaction with out-group members and assortative sociality favouring local conspecifics (and hence immunologically locally adapted individuals).

(i) *Limited dispersal*

Limited dispersal refers to behaviours that reduce movements away from a central location (also called 'philopatry'). In areas of high pathogen stress compared with those of low pathogen stress, limited dispersal will be favoured by selection owing to the correspondent increase in association with the immunologically similar individuals and decreased contact with more distant, and differently parasitized, other individuals (Freeland 1979). Freeland (1976), Møller *et al.* (1993) and Loehle (1995) all discuss how limited dispersal may reduce exposure to a diversity of types of infectious diseases, and also argue for the importance of territoriality and

Received 20 May 2008
Accepted 9 July 2008

restricted home ranges, forms of limited dispersal, as means for reducing contact with dissimilar conspecifics that may carry novel diseases. Fincher *et al.* (2008) provided evidence of a positive association between country-wide measures of pathogen prevalence and the cultural value unidimension of collectivism–individualism, arguing that collectivism is fundamentally antipathogen psychology (i.e. the empirical finding is high collectivism, high parasite prevalence; low collectivism (=high individualism), low parasite prevalence). In addition, collectivism is associated with higher ethnocentrism (i.e. significant attraction to and support of in-group members) and xenophobia (i.e. avoidance of and dislike towards out-group members) than individualism (Fincher *et al.* 2008). Alesina & Giuliano (2007) demonstrate across countries that individuals who are more family-oriented (and demonstrably more collectivist) disperse for shorter distances and are, hence, more philopatric. These studies are consistent with our notion that infectious diseases contribute to human dispersal behaviour—where infectious disease is more stressful, individuals disperse over shorter distances than where infectious disease is less stressful.

(ii) *Assortative sociality*

Assortative sociality refers to the alliance with similar individuals including for mating or other social contact (e.g. religious service, reciprocity, cooperative hunting, cooperative breeding, etc.). It operationalizes as *contact bias* (or *selective contact*) referring to behaviours that promote contact with particular individuals and not others. Assortative sociality is similar to Wilson & Dugatkin's (1997) 'assortative interactions'. There is ample evidence that the psychology of xenophobia and ethnocentrism—two forms of assortative sociality—is importantly related to avoidance and management of infectious disease (Faulkner *et al.* 2004; Navarrete & Fessler 2006; Navarrete *et al.* 2007; Park *et al.* 2007; Schaller & Duncan 2007; Fincher *et al.* 2008; Schaller & Murray 2008). Other forms of assortative sociality with respect to disease avoidance include assortative mating or other assortative alliances based on the presence of similar normative behaviour (norms), religious convictions, adornment, values, dialects and language use, or major histocompatibility complex genes (MHC; see Lewis (1998) on MHC assortative interactions). We propose the following: the interactions with out-group or in-group members will vary along a continuum. At one end of the continuum would be an individual who interacts with only genetically or immunologically similar individuals (i.e. in the extreme this would be complete avoidance of unrelated individuals) and at the other end would be an individual who interacts only with genetically or immunologically dissimilar individuals (i.e. in the extreme this individual would interact only with strangers). An individual is predicted to fall along this continuum based on the local levels of infectious disease stress, such that under high levels of disease stress the optimal strategy is contacting relatively immunologically similar individuals while an individual under low levels of infectious disease stress can benefit from contact with dissimilar individuals. A more fundamental way to envision this is to consider the optimal level of contact between ego and the genetic relatedness of individuals contacted over a lifetime. The average coefficient of relatedness of ego's interactions over the lifetime should be positively related to local levels of infectious disease stress.

It is not necessary for our model that individuals have actual knowledge of the infective status of unknown or out-group individuals. Rather what is important is that limited dispersal and assortative sociality are the solutions for contagion avoidance, which have been favoured by selection.

(*d*) As individuals adaptively contact local others preferentially (contact bias in altruism and mating), cultural divergence ensues. This assortative sociality towards local or in-group members and limited dispersal can—via the erection of an intergroup boundary—reduce or eliminate the flow of values and ideas. The reduced flow of ideas and values will promote cultural isolation generating religion diversity. This provides a context for cultural divergence-absent geographical physical barriers.

(*e*) The localized host–parasite races will endure if the parasites do not go extinct. Such races are potentially strong evolutionary mechanisms that can increase or maintain the divergence across the original range of the host culture. In addition, this localized antagonistic coevolution will drive divergence among the parasites themselves, further generating novel contagions that the hosts experience.

(*f*) The higher the parasite richness of a host population the more opportunity for spatial variation in parasite–host coevolutionary races. The frequency, duration, and intensity of (*b*)–(*e*) above will covary positively with the infectious disease diversity. That is, disease richness will positively yield more localized disease problems across geographical ranges of human societies, increased parasite-driven isolation by adaptive local assortative sociality and reduced dispersal and increased intergroup divergence.

Our model does not rely on a dismissal of cultural divergence through vicariance processes. For example, Wilson (2007) suggested that, inevitably, religions will diversify because, as a collection of survival strategies, religions will become adapted to local environmental conditions. This model of religion (or cultural) diversification is different from ours. Such a model makes no predictions about the development of xenophobic and ethnocentric psychology and related limited dispersal and assortative sociality that are paramount in ours.

As modelled by others, assortative interactions are linked in important ways to the genesis and maintenance of languages (Nettle 1999*a*), ethnic markers (McElreath *et al.* 2003) and species (Hochberg *et al.* 2003). We have argued, and supported with data, that these assortative interactions are driven in their intensity by the prevalence of infectious diseases and are fundamental to the genesis and maintenance of language richness patterns in humans across the globe (Fincher & Thornhill 2008). We propose that the worldwide pattern of religion diversity will be tied to infectious disease severity similarly. Hence, we predict that religion diversity is the highest where infectious disease severity is also the highest and the lowest where disease severity is also the lowest. Relatedly, Nettle *et al.* (2007) found that language and religion diversity positively covary across the globe.

Our approach builds on the prior theory that religions (or languages (Nettle & Dunbar 1997; Nettle 1999*b*; Fitch 2004) and other ethnic markers; McElreath *et al.* 2003) are markers of commitment to and membership of a single group of likewise individuals (Irons 2001; Wilson 2002; Sosis 2003; Sosis & Alcorta 2003; Wilson 2005). This theory has focused on these markers serving as honest signals of group membership and commitment, because learning a religion or language or an ethnic code of conduct and incorporating it fully into an individual's life requires major effort, time and experience, making unlikely credible faking by someone who did not also have an ontogeny in the same region or with the same people. This honest marking of individuals is important for the avoidance of cheaters (those who would take advantage of group benefits without paying for them in currency of altruism). We are attempting to expand this line of reasoning to include a context that can explain important variation in the empirical patterns of religion diversity: honest marking of group membership by religious values in the context of spatially variable coevolutionary host–parasite races. We are not attempting to explain whether religious experience is an adaptation itself or a by-product of adaptations for purposes other than religion. That is a separate research issue that has been illuminated by Richerson & Boyd (1998), Kirkpatrick (1999), Boyer (2001), Irons (2001), Atran (2002), Wilson (2002), Alcorta & Sosis (2005) and others.

In this article, we show that societal range size for traditional human societies is negatively related to pathogen stress and that the contemporary global pattern of religion diversity correlates with infectious disease stress in ways that support our model of parasite-driven diversification described above.

2. MATERIAL AND METHODS

(a) *Range size: traditional societies*

According to our model, the area of land that individuals use should negatively correlate with pathogen stress, reflecting limited dispersal in the face of high contagion risk associated with out-group contact. In order to test this proposition, we examined the correlations between Binford's (2001) measure of societal range size, reflecting the aggregate use of space by individuals within a society, for his sample of 339 traditional societies, and an estimate of pathogen stress for each of these societies.

We estimated pathogen stress by first establishing the linear equation that best predicted pathogen prevalence as measured by Low (1994) for the 186 societies in the standard cross-cultural sample (SCCS, Murdock & White 1969). We examined absolute latitude, mean annual temperature and mean yearly rainfall as predictor variables because they were also provided by Binford (2001) for each of the 339 societies in his sample. The data for the SCCS were collected from the *World Cultures* journal website (http://eclectic.ss.uci.edu/~drwhite/worldcul/world.htm). The best single predictor of pathogen prevalence was absolute latitude (adjusted $r^2=0.51$; $F_{1,184}=191.35$; $p<0.0001$). Both temperature and rainfall significantly predicted pathogen prevalence but with lower coefficients and F statistics (annual temperature: adjusted $r^2=0.42$, $F_{1,178}=131.50$, $p<0.0001$; annual rainfall: adjusted $r^2=0.04$; $F_{1,184}=9.13$; $p=0.003$); therefore, we generated a linear equation to predict pathogen prevalence based on absolute latitude: pathogen prevalence $=16.097-0.152$ (absolute latitude). Then, we used this equation to estimate pathogen prevalence for each of the 339 societies in Binford (2001). See appendix 1 in the electronic supplementary material for these pathogen-stress estimates.

Societies with more individuals might also have larger home ranges, thus we examined the potentially confounding effect of population size (ln transformed to reduce skewness and kurtosis) provided by Binford (2001). Also, societal range size might be positively related to reliance on hunting of terrestrial animals for subsistence; thus, we examined the potentially confounding effect of the proportion of subsistence derived from hunting of terrestrial animals as provided by Binford (2001). These values were normally distributed and not transformed for analysis. Additionally, we considered the patterns of group mobility with respect to estimated pathogen prevalence because high levels of infection and associated lethargy and incapacitation may reduce mobility generating a negative relationship between societal range size and pathogen prevalence irrespective of psychological design to minimize dispersal (see Waguespack 2002). We did this by analysing the average distance moved annually and the ln-transformed average annual number of moves (1 was added to the number of moves prior to transformation to eliminate negative values). Lastly, there was an analytical concern that societies near each other would be similar due to spatial autocorrelation. Thus, we used an ANOVA with state/country for each society as provided in Binford (2001) as the independent variable predicting societal range size to examine this influence. All values analysed for each society are contained in appendix 1 in the electronic supplementary material.

(b) *Religion diversity*

We were interested in whether there was a positive correlation between religion diversity and infectious disease stress throughout the world. To this end, we correlated the number of religions per contemporary country/governmentally autonomous territory (=religion richness) with pathogen stress measured in two complementary ways (see below). Appendix 2 in the electronic supplementary material contains religion richness, both pathogen-stress measures, world area and longitudinal band designations (see below) for each country/territory.

(i) *Religion richness*

We used tallies of the total number of religions per country/territory ($n=219$ countries or territories), which include the number of major religions and ethnoreligions, from Barrett *et al.*'s (2001) *World Christian Encyclopedia*. Barrett *et al.* (2001) is a highly regarded source among religious scholars (Grim & Finke 2006). Religion is defined by Barrett *et al.* (2001) as '…a grouping of persons with beliefs about God or gods, and defined by its adherents' loyalty to it, by their acceptance of it as unique and superior to all other religions, and by its relative autonomy'. The average religion richness per country was 30.8 ± 69.4 (mean$\pm$s.d.) and ranged from 3 to 643. The number of religions was ln transformed prior to analysis to reduce skewness and kurtosis.

(ii) *Pathogen richness*

We acquired human pathogen richness scores (number of all infectious diseases listed) for all contemporary countries/territories worldwide listed in the Global Infectious Disease

and Epidemiology Network (GIDEON; www.gideononline.com) April–August 2007. GIDEON is a continually updated database available to the medical community and researchers. GIDEON has been used lately to explore the ecological correlates (Guernier *et al.* 2004) and globalization of human diseases (Smith *et al.* 2007). The average parasite richness score per country/territory was 200.1±15.2 (mean±s.d.), $n=229$, range=178–248.

(iii) *Pathogen prevalence*

We also compiled a pathogen prevalence value for a subset of infectious diseases for each country using data extracted from GIDEON. We used a set of parasites similar to those used in prior research (e.g. Low 1990; Gangestad *et al.* 2006), using the same seven classes of diseases but expanded to include all entries in GIDEON for each class (a total of 22 parasites). We classified the country-wide disease level of seven groups of parasites: leishmanias; trypanosomes; malaria; schistosomes; the filariae; spirochetes; and leprosy. We used GIDEON's three-point scale of parasite prevalence (3=endemic, 2=sporadic and 1=not endemic) based on distribution maps provided in GIDEON. Leprosy was handled differently because GIDEON does not map the precise distribution. Rather the infection rates are presented. Thus, we coded infection rates *per capita* of 0–0.01/100 000 as 1, 0.01–1/100 000 as 2 and >1/100 000 as 3. Trypanosomiasis–African was given in GIDEON in a similar way. We used coded infection rate 'not endemic' as 1, >0–0.25/100 000 as 2 and >0.25/100 000 as 3. The values for the different parasites were summed and provided our index (mean=31.5±6.8, $n=225$, range=23–48). These data were collected from April–August 2007. The correlation between disease richness and pathogen prevalence was $r=0.80$ ($p<0.0001$, $n=224$).

(iv) *Confounding variables*

We included in our analysis potentially confounding variables. There has been very little research on the development of religion diversity, and, therefore, there are no established paradigms for explaining religion diversity. However, given the general importance of latitude to the study of parasites and diversity (Guernier *et al.* 2004; Hillebrand 2004), we considered absolute latitude measured at the midpoint of each country as a control variable. A larger population (irrespective of country land area) may have more religions than a smaller population; therefore, we included the effect of human population size. In addition, some countries may have more religions within their borders simply because they are larger countries; therefore, we included a country's land area. Some political systems restrict the ability to form religions and worship freely. Hence, we included Vanhanen's (2003) index of democratization. Higher values indicate greater democracy while lower values indicate greater autocracy. Values range from 0 (e.g. Myanmar) to 44.2 (=Denmark).

The secularization hypothesis for explaining religiosity (reviewed in McCleary & Barro 2006*a*,*b*) argues that a religion's vitality (commitment to and participation in by adherents) decreases under increasing economic development, because individuals have less 'need' for religion and its benefits when economic resources are favourable. An extension of this hypothesis to explain religion diversity in an area would be that, under conditions of increasing economic development, religion diversity should decrease due to fewer adherents while reduced economic conditions would increase religion diversity. If correct, then we would expect to find a negative relationship between economic development and religion diversity. We used gross domestic product (GDP) *per capita* in terms of purchasing power parity (GDP *per capita*) and Gini coefficients to explore this. Gini is a measure of wealth inequity within a country where larger values represent greater inequity. Thus, we would expect a negative relationship between religion richness and GDP *per capita* and a positive relationship between religion richness and Gini.

Data for these potential confounders except for Vanhanen's (2003) Index of Democratization came from the *World factbook, 2007* (www.cia.gov). GDP *per capita*, population size and land area were ln transformed to reduce skewness and kurtosis prior to analysis.

(v) *Other analyses*

It is possible that the distribution of religions across the world is due to different patterns of human settlement on the continents (e.g. more recent colonization; Nettle 1999*c*) or different patterns of conquest (McNeill 1980; Diamond 1998). We attempted to account for this effect using two methods. First, we explored the predicted positive correlation between parasite richness and prevalence and the number of religions in each of six world culture areas. Murdock (1949) divided the world's societies into six world culture areas based on a shared historical and geographical range. Murdock's division of the world reduces interdependence of cultures between regions. We divided the countries of the world into Murdock's six world regions (North America, South America, West Eurasia, East Eurasia, Africa and Insular Pacific) and examined the correlation between parasite richness and prevalence and religion richness in each region. Second, to further account for different histories of conquest and colonization, we compared the pattern of the correlations between parasite richness and prevalence and religion richness within three quasi-independent longitudinal geographical bands similar to Collard & Foley's (2002) analysis of the latitudinal distribution of cultures. The bands were American, Europe–Africa and Asia–Australia.

3. RESULTS

(a) *Range size: traditional societies*

Societal range size was negatively correlated with pathogen prevalence ($r=-0.48$, $p<0.0001$, $n=339$). After partialling the effect of population size or the proportion of subsistence from hunting, the correlation between range size and pathogen prevalence remained negative and significant (partialling population size: $r=-0.42$; partialling proportion hunting: $r=-0.38$; $p<0.0001$, $n=339$ for both correlations). Removing the effects of population size and the proportion of subsistence from hunting simultaneously, the correlation between pathogen prevalence and home range size was negative and significant ($r=-0.27$, $p<0.0001$, $n=339$). Thus, there is a robust reduction in range size in areas with greater pathogen prevalence.

In general, a society's range size was predicted by the state/country that a society resided in ($F_{66,272}=7.75$, $p<0.0001$). Thus, to reduce the effect of spatial autocorrelation, we computed the average value of home range size, pathogen prevalence, population size and the proportion of subsistence from hunting for each state/country and conducted correlations with these composite values. At the state level, the correlation

Table 1. Zero-order correlations between religion richness, disease richness, pathogen prevalence, and control variables for the countries of the world.

	correlation with the natural log of religion richness		
	r	*p*	*n*
disease richness	0.75	<0.0001	214
pathogen prevalence	0.62	<0.0001	211
absolute latitude	−0.37	<0.0001	218
population size (ln)	0.59	<0.0001	214
land area (ln)	0.58	<0.0001	214
democracy index	−0.14	0.08	167
GDP *per capita* (ln)	−0.33	<0.0001	213
Gini	0.38	<0.0001	122

between home range size and pathogen prevalence was −0.60 ($p<0.0001$, $n=67$). After partialling the effects of population size and proportion of subsistence from hunting, the correlation between home range size and pathogen prevalence at the state level was −0.43 ($p=0.0004$, $n=67$). Considered separately, partialling the effect of proportion of subsistence from hunting and the effect of population size, the correlation between home range size and pathogen prevalence was −0.51 ($p<0.0001$; $n=67$) and −0.52 ($p<0.0001$; $n=67$), respectively.

Considering mobility patterns, the number of moves (ln) annually was positively correlated ($r=0.12$, $p=0.03$) while the distance moved annually was negatively correlated ($r=-0.21$, $p<0.0001$) with pathogen prevalence ($n=339$ for both). Moreover, partialling the effect of distance moved increases the correlation between the number of moves and pathogen prevalence to 0.45 ($p<0.0001$), while partialling the effect of number of moves increases the correlation between pathogen prevalence and distance moved to −0.47 ($p<0.0001$). Thus, people in traditional societies move more often but over shorter distances in high pathogen prevalence areas. Conversely, they move less often but over longer distances in areas with fewer pathogens.

(b) *Religion diversity*

The zero-order correlations between religion richness and the explanatory or control variables are presented in table 1. Religion richness was positively related to disease richness ($r=0.75$, $p<0.0001$, $n=214$; figure 1) and pathogen prevalence ($r=0.62$, $p<0.0001$, $n=211$). Religion richness was also significantly related to population size ($r=0.59$), GDP *per capita* ($r=-0.33$), Gini ($r=0.38$), absolute latitude ($r=-0.37$) and land area ($r=0.58$). Democracy ($r=-0.14$, $p=0.08$) was close enough to conventional statistical significance for retention in subsequent analyses.

We conducted first-order partial correlations between religion richness and disease richness or pathogen prevalence while controlling each of the confounding variables (table 2). The correlation between religion richness and disease richness was robust to the effects of the confounding variables (*r* values ranged from 0.59 to 0.73; table 2); as well, the correlation between religion richness and pathogen prevalence was robust to the confounding variables (*r* values ranged from 0.45 to 0.58; table 2). Only absolute latitude was significantly related to religion richness while partialling the effects of disease richness ($r=-0.27$, $p<0.0001$, $n=214$). Thus, we regressed religion richness on disease richness and absolute latitude. This multiple regression was significant (adjusted $R^2=0.59$, $F_{2,211}=156.72$, $p<0.0001$) with both disease richness and absolute latitude contributing distinct effects (standardized coefficients: disease richness = 0.71; absolute latitude = −0.19, both *p* values <0.0001). Population size, land area and democracy were significantly related to religion richness when partialling the effects of pathogen prevalence (respective *r* and *p* values: $r=0.37$, $p<0.0001$; $r=0.34$, $p<0.0001$; $r=0.21$, $p=0.006$). Thus, we regressed religion richness on pathogen prevalence, population size, land area and democracy. This multiple regression was significant (adjusted $R^2=0.44$, $F_{4,162}=33.49$, $p<0.0001$). Only pathogen prevalence and population size contributed significant distinct effects to the regression (standardized coefficients: pathogen prevalence = 0.50, $p<0.0001$; population size = 0.24, $p=0.006$; land area = 0.14, $p=0.11$; democracy = 0.12, $p=0.08$).

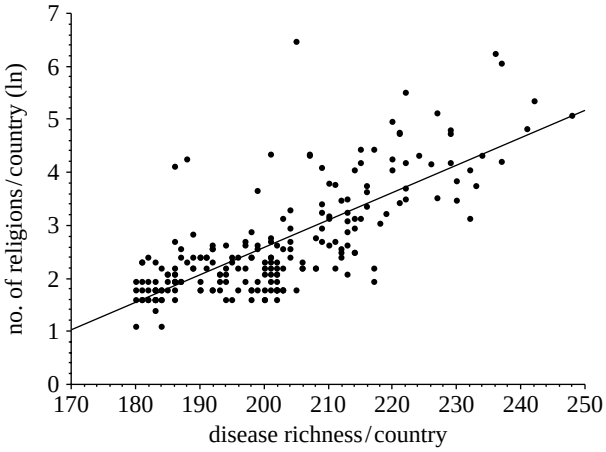

Figure 1. The correlation between disease richness and religion richness ($r=0.75$, $p<0.0001$, $n=214$). The line is the regression line.

Religion richness was positively related to disease richness (and significantly so) in all six world regions (*r* values range from 0.26 to 0.91; table 3) and all three longitudinal bands (American: $r=0.93$, $n=56$; Asia–Australia: $r=0.65$, $n=45$; Europe–Africa: $r=0.78$, $n=113$; all *p* values <0.0001). Religion richness was positively related to pathogen prevalence in all six world regions (*r* values range from 0.15 to 0.79; table 3); however, the correlation in West Eurasia was not significant. A sign test on the direction (+ or −) of each correlation shows that this relationship is not spurious ($0.5^6=0.016$). The correlation between religion richness and pathogen prevalence was significantly positive in all three bands (American: $r=0.78$, $n=54$; Asia–Australia: $r=0.63$, $n=45$; Europe–Africa: $r=0.70$, $n=112$; all *p* values <0.0001).

4. DISCUSSION

(a) *Range size: traditional societies*

The main finding of our analysis of range size was that range sizes for traditional societies are smaller in areas of the world where pathogen stress was estimated to be more

Table 2. First-order partial correlations between religion richness (ln) and disease richness or pathogen prevalence while partialling the effects of potentially confounding variables. (All p values are <0.0001; sample sizes are in parentheses following the correlation coefficients.)

	correlation between disease richness and religion richness	correlation between pathogen prevalence and religion richness
variable partialled		
absolute latitude	0.73 (214)	0.54 (211)
population size (ln)	0.59 (210)	0.47 (207)
land area (ln)	0.60 (210)	0.46 (207)
democracy	0.72 (167)	0.58 (167)
GDP *per capita* (ln)	0.72 (209)	0.56 (206)
Gini	0.68 (121)	0.45 (121)

Table 3. Zero-order correlations between religion richness and disease richness or pathogen prevalence split by the six world regions.

	disease richness			pathogen prevalence		
world region	r	p	n	r	p	n
North America	0.90	<0.0001	30	0.45	0.01	29
South America	0.91	<0.0001	18	0.79	<0.0001	18
Africa	0.79	<0.0001	56	0.62	<0.0001	55
West Eurasia	0.26	0.04	61	0.15	0.25	61
East Eurasia	0.71	<0.0001	24	0.70	<0.0001	24
Insular Pacific	0.75	<0.0001	25	0.72	<0.0001	24

intense. And, this relationship is not confounded by population size or the proportion of subsistence from hunting. This finding is consistent with the notion that societal range size (or species' range size; Rapoport's rule; Stevens 1989) is generally reduced in the tropics. We suggest that this general pattern results from the evolved response of high levels of limited dispersal in pathogen-rich areas owing to strong selection against out-group contact (see also Fincher & Thornhill 2008).

According to the data, individuals in societies in areas with high pathogen prevalence move often but over shorter distances. We suggest that this is an aspect of evolved antipathogen behaviour. Individuals within these societies may strategically move often within a restricted territory to optimally distance themselves from parasites, such as helminths, that persist in the soil (McNeill 1980; Loehle 1995). The findings herein certainly negate the notion that limited range size is owing to incapacitation and lack of movement.

(b) *Religion diversity*

As predicted, we found that religion diversity is the highest where disease diversity is also the highest and the lowest where disease diversity is also the lowest. This pattern is robust to the effects of other significant correlates of religion diversity. This finding supports our hypothesis that religions emerge from intergroup cultural boundaries that form in response to the spatial variation of infectious disease stress and associated assortative sociality and limited dispersal.

To our knowledge, previous evolutionary models do not offer an explanation for why religion diversity varies spatially across the globe. If, according to these models, religions fundamentally are for avoidance of cheaters and for group coordination (e.g. Richerson & Boyd 1998; Irons 2001; Wilson 2002; Sosis 2003) and/or arise from incidental effects of other psychological design elements (e.g. Kirkpatrick 1999; Boyer 2001; Atran 2002; Dawkins 2006), they must explain why anti-cheating psychology and the need for group coordination lead to greater genesis of religions in the tropics compared with temperate regions or why incidental effects generate different religion diversity in the tropics versus temperate regions. The previous models do not seem to do this. We suggest that the importance of group coordination is actually the manifestation of the importance of in-group assortative sociality and limited dispersal for infectious disease avoidance and management. And the intensity of this selective regime in the tropics outpaces that found in the temperate areas owing to the greater threat of infectious disease in tropical areas.

Our analyses indicate that infectious disease appears to be a more important driver of religion diversity than economic conditions. Furthermore, country-level differences in democratization did not have a major effect on the overall pattern that we present. Likewise, the effects of different histories of colonization and conquest did not confound our general patterns of finding more religions where there are also more infectious diseases.

In our analysis, disease richness is more strongly correlated with religion richness than is pathogen prevalence. This, we argue, is because disease richness better represents the history of antagonistic coevolutionary arms races across the world than pathogen prevalence. Each infectious disease, irrespective of its prevalence, has the potential to generate localized and spatially variable coevolutionary races and thereby render out-group contact very costly and in-group favouritism much more beneficial.

Given that one probable benefit of outbreeding is the diversification of offspring in order to cope with parasite threats (Tooby 1982; Trivers 1985; Hamilton *et al.* 1990; Ridley 1993), it appears counter-intuitive to suggest that

individuals living in conditions of high parasite stress should avoid distant others. However, as argued in Fincher & Thornhill (2008), because Red Queen host–parasite races build complex locally adaptive host immune adaptations, including coadapted gene complexes that work well only if not disrupted by distant outbreeding, both coadapted gene complexes and local adaptation render some degree of inbreeding adaptive (Shields 1982; Kokko & Ots 2006). Thus, although close inbreeding is maladaptive under high parasite stress levels, distant outbreeding is too. The optimal balance between inbreeding and outbreeding depends on the local levels of infectious disease stress.

It may also be that assortative interactions and limited dispersal increase inclusive fitness because they promote the acquisition and maintenance of an individual host's mutualistic and commensalistic microbial community (Dethlefsen *et al*. 2007). Humans begin acquiring their microbial community at birth, but the development and maintenance of this community occurs over the lifetime. Benefits provided by mutualistic and commensalistic symbionts can include provision of metabolic by-products that can be used as fuels (e.g. butyrate) or acting as a defence system through competition with pathogenic symbionts preventing colonization and infection by pathogens (reviewed in Dethlefsen *et al*. 2007). Owing to the localized coevolutionary races between hosts and parasites and because in the tropics these races occur in smaller areas within isolated populations, it is probable that humans living in low latitude areas will experience greater specificity and local adaptation on the part of their mutualist and commensalist symbionts. Interacting widely with out-group members has the potential to disrupt these communities as well as leading to the acquisition of dangerous pathogens. We consider that both parasitic and mutualistic/commensalistic interactions may be driving the assortative sociality and limited dispersal that operate in the genesis of religions.

In sum, religion diversity appears to be tied importantly to infectious disease stress across the globe, and the global pattern is consistent with our model of religion genesis. Our analysis suggests that the nature of religion needs to be reconsidered. Although religion apparently is for establishing a social marker of group alliance and allegiance, at the most fundamental level, it may be for the avoidance and management of infectious disease.

We thank Nils Chr. Stenseth for encouraging comments, two anonymous referees for their thoughtful and useful criticism and Devaraj Aran, Keith Davis and Phuong-Dung Le for their assistance with data processing. We also thank the University of New Mexico's Biology Department and Biology Graduate Student Association for funding to support the publication of this manuscript. The use of data from public and published sources is exempt from Human Subjects Review.

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