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A parasite-driven wedge: infectious diseases may explain language and other biodiversity

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Parasite–host coevolutionary races are spatially variable across species' or human cultural ranges. Assortative sociality, biased toward local conspecifics, and limited dispersal (philopatry) in humans and other organisms can be adaptive through reduced contact with dangerous contagions harbored by distant/non-local conspecifics. These factors can generate cultural or population divergence. Thus, parasites are like a wedge driving groups apart through their effective creation of anticontagion behaviors. If this proposition is correct, then biological diversity should positively correlate with parasite diversity. Here we show that the worldwide distribution of indigenous human language diversity, a form of biodiversity, is strongly, positively related to human parasite diversity indicative of a legacy of parasite-mediated diversification. The significant pattern remains when potential confounds are removed. The pattern too is seen in each of the six world regions and is not confounded by regional differences in their history of colonization and conquest. We hypothesize that variation in limited dispersal and assortative sociality with conspecifics in response to the worldwide spatial variation in pathogen diversity provides a fundamental mechanism of population divergence explaining many important aspects of the geographic patterns of biodiversity. This hypothesis has broad implications for a diversity of research topics including language diversity, cultural evolution, speciation, phylogeny and biogeography.

There is little doubt that biological diversity declines with increasing latitude (Hillebrand 2004). Numerous hypotheses have been proposed to explain this pattern with little consensus on causal mechanisms (Gaston 2000, Schemske 2002, Willig et al. 2003, Mittelbach et al. 2007). Because the pattern is so widespread amongst taxa, many scholars appeal to a single causal mechanism (MacArthur and Connell 1966) while others like Gaston (2000) argue there is no a priori reason to suppose that a single process must explain this general pattern for all taxa. However, at the most general level, a single causal process can explain the distribution of biological diversity on earth: differential reproductive success. Such differential reproductive success can arise through disparate evolutionary processes like selection or drift and at varying levels of biological organization (i.e. individuals, populations, species, clades, cultures). A challenge is to describe the context(s) in which differential reproductive success is realized and how this generates or maintains biological diversity differently across the world.

We hypothesize that an important context is the variable magnitude of infectious diseases that organisms have faced and continue to face throughout the world. Such diseases have been and are ubiquitous environmental hazards as demonstrated by numerous behavioral adaptations to avoid and manage infection (Freeland 1976, Hart 1990, Møller et al. 1993, Ewald 1994, Loehle 1995, Schaller and Duncan 2007). We propose that the adaptive behavioral avoidance and management of infectious disease can reproductively isolate populations and culturally isolate human groups (e.g. by language) providing the necessary conditions for the genesis of species as well as languages.

Model

We propose the process works as follows in a population or cultural group to generate discontinuity and hence diversity:

1) Initially, the population or culture has a geographical distribution and a uniform distribution of immunity with normal phenotypic variation.

2) Then, parasite–host antagonistic coevolutionary races (Red Queen races; Van Valen 1973, Ridley 1993) become spatially distinct and localized across the range of the population or 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 species that are new to a locale or novel varieties of already present species.

An important condition of this model is that parasite-host coevolutionary races are spatially variable generating spatial variance in the immunobiology of a host population and/or cultural group. Because human language diversity is the basis of our empirical analysis we focus our discussion on humans and their parasites. The distribution of human parasites and human responses to them are known to vary

spatially worldwide. Examples include Guernier et al.'s (2004) finding that human-disease species richness increases with decreasing latitude; Low's (1990) finding that pathogen severity levels experienced by traditional societies varied across 186 societies distributed throughout the world (Law 1990 and Cashdan 2001 showed this measure of pathogen severity was related negatively to latitude); leishmaniasis resistance at the genetic level is different in adjacent villages in Sudan (Miller et al. 2007); genetically based malaria resistance differs among sympatric African ethnicities (Modiano et al. 1996); among Amazonian Indian tribes, parasites and immunological defenses vary among spatially separated groups (Eveland et al. 1971, Black 1975); and, genetic diversity at the HLA complex (human leukocyte antigen; also known as the major histocompatibility complex or MHC) is positively correlated with local pathogen richness (Prugnolle et al. 2005). Additionally, spice use in food recipes is positively related to mean annual temperature within a country, assuming higher parasite activity in warmer environments (Sherman and Billing 1999).

3) At this point, there is spatial variation across the population or culture in ability to meet the immune challenge of an infected conspecific. Consequently, anti-pathogen behaviors are favored by natural selection and manifest as the avoidance of conspecifics that are infected or potentially infected with dangerous parasites by assortative sociality favoring local conspecifics (and hence immunologically locally-adapted individuals) and, we propose, also by limited dispersal. Assortative sociality refers to the alliance with similar others including for mating or other social contact (e.g. grooming, nepotism, reciprocity, cooperative hunting, cooperative breeding). Assortative sociality may operationalize as contact bias (or selective contact) referring to behaviors that promote contact with particular individuals and not others. It is similar to what Wilson and Dugatkin (1997) refer to as 'assortative interactions'. Examples of the kinds of behaviors we consider assortative sociality with respect to disease avoidance include in-group mating or other in-group social alliances. In humans, these include behaviors of contact bias based on the presence of similar normative behavior (norms), styles of adornment, values, religious convictions, dialects and language use, or MHC genes (see Lewis 1998 on MHC assortative interactions). Limited dispersal refers to behaviors that reduce movements away from a central location. In areas of high pathogen severity compared to areas of low pathogen severity, limited dispersal/philopatry will be favored by selection because of the correspondent increase in association with immunologically close individuals and decreased contact with more distant, and differently parasitized, conspecifics (Freeland 1979). Belliure et al. (2000) also recognized the importance of limited dispersal for generating population divergence. They provide evidence that, in British birds, species characterized by limited dispersal also had higher numbers of associated subspecies indicative of relatively greater population diversification.

In many ways limited dispersal is assortative sociality. However, selective local contact with conspecifics is promoted by psychological adaptations for associating with these individuals and not others. This, at a minimum, requires the ability to recognize and evaluate local social markers. Limited dispersal on the other hand requires a different set of adaptations that function specifically to restrict movement. Thus we consider limited dispersal separate from assortative sociality. Freeland (1976), Møller et al. (1993) and Loehle (1995) all discussed how limited dispersal may reduce exposure to a diversity of infectious diseases, and also argued for the importance of territoriality and restricted home ranges – forms of limited dispersal – as means for reducing contact with dissimilar conspecifics that may carry novel diseases unlike those carried by local conspecifics. Møller et al. (2004) showed that birds with more extensive dispersal distances had more responsive immune systems suggesting that species with longer characteristic dispersal pay a higher cost of maintaining their immune systems. Freeland (1979) provided evidence consistent with the thesis that reduced dispersal is a response to local parasite levels. In a study of rain forest-dwelling versus savannah-dwelling primates, he showed that savannah baboons, with characteristically higher rates of intergroup movement of individuals, shared protozoan faunas while rain forest primates, with lower rates of intergoup movements of individuals, had more unique protozoan faunas. Freeland argued that because of increased parasite severity for rain forest primates, out-group contact was too costly to promote more frequent exchange of individuals. Meanwhile because of the lower parasite severity for the savannah primates, greater interchange of individuals and associated out-group contact had low costs.

It is not important for our model that individuals have actual knowledge of the infective status of unknown or outgroup individuals. Rather what is important is that assortative sociality and limited dispersal/philopatry are the solutions for contagion avoidance that have been favored by selection.

4) As individuals adaptively contact local conspecifics preferentially (contact bias in altruism, associative behavior, mating) population or culture divergence ensues. This assortative sociality toward local or in-group conspecifics and limited dispersal can reduce or eliminate gene or cultural information flow. Reduced gene flow will promote gene pool isolation which may generate species diversity while the reduced flow of ideas and values will promote, in the case of humans, culture isolation generating cultural diversity including language diversity and diversity in other cultural features. Thus, the parasite-driven wedge is formed by the effective creation of anticontagion behaviors that behaviorally isolate populations or cultures. The ubiquity of parasites for all living organisms suggests this is a common driver of speciation. Furthermore, this provides a context for population and cultural divergence absent geographic barriers and thus forms a basis for widespread parapatric divergence.

5) Even after divergence, the localized host–parasite races can endure. Such races are potentially strong evolutionary mechanisms that can increase or maintain the divergence across the original range of the host population or culture. Too, this localized antagonistic coevolution will drive population divergence among the parasites themselves further generating novel infectious parasites that the hosts experience.

6) The higher the parasite richness of a host species or cultural group the more opportunity for spatial variation in parasite–host coevolutionary races. The frequency, duration, and intensity of 2)–5) above will covary positively with the infectious disease diversity. That is, relatively high disease richness yields more localized disease problems across geographic ranges and hence increased parasite-driven intergroup divergence by adaptive local assortative sociality and limited dispersal/philopatry.

According to our model, biological diversity should be highest where parasite diversity is highest marking a legacy of parasite-host antagonistic coevolutionary races and providing a context for the maintenance of isolated populations/cultures: locally high parasite richness maintains the high cost of out-group contact through dispersal or other behavioral means.

Language

We focus our model on its ability to explain the distribution of one form of biological diversity: human language diversity. Although we confine our analysis to a single species, this gives us an advantage in exploring group diversification within a restricted phenotypic landscape. Nevertheless, humans are found worldwide in various ecologies providing a backdrop for exploring, in a general way, the ecology of diversification. And, much more is known about human parasites than those of other species.

Language diversity has received much interest from researchers employing evolutionary theory (Mace and Pagel 1995, Nettle 1999a, Harmon 2002, Sutherland 2003, Maffi 2005). Mace and Pagel (1995) demonstrated a latitudinal gradient in the density of indigenous languages on the North American continent: higher density of languages at low than high latitudes. Sutherland (2003) showed that language diversity is related negatively to latitude worldwide. Nettle (1999a) provided a mechanistic argument for the distribution of languages across the globe arguing that the primary environmental correlate of language diversity is ecological risk (climatic factors) which he measured using mean growing season. He suggested that areas with long growing seasons provide conditions that allow for self-reliance on local resources within a particular ethnic group. In contrast, people in areas with short growing seasons must participate in contact and trade with other ethnic groups to buffer the effects of ecological risk. Under these conditions, many languages emerge and persist in areas with long growing seasons while few languages emerge from areas with short growing seasons and associated ecological insecurity because individuals must be able to speak with many different people across an expansive area. Therefore, in Nettle's view, high ecological risk is a homogenizing mechanism leading to low language diversity. However, Sutherland (2003) reported that mean growing season was not an important predictor of language diversity throughout the world.

In this article, we provide evidence supportive of our hypothesis that a significant mechanism underlying the latitudinal diversity gradient is the adaptive avoidance and management of pathogens through assortative sociality and limited dispersal (i.e. the parasite-driven wedge).

Material and methods

We examined the correlation between contemporary measures of human parasitic disease richness and human language richness across most countries of the world (the global analysis). We then examined the same correlation in each of six world regions and three longitudinal geographical bands.

We acquired, during August 2007, parasite richness scores for all contemporary countries (and a few territories) worldwide listed in the regularly updated database, Global infectious disease and epidemiology network (GIDEON; <www.gideononline.com>). Our tally includes all infectious diseases listed for a country. 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 201 ± 15 (mean $\pm$ SD; $n = 229$).

We acquired the language richness values for each country and territory from Ethnologue (Gordon 2005), a resource commonly used in the study of human language diversity (Harmon 1996, Nettle 1999a, Sutherland 2003). We confined our analyses to the number of living indigenous languages per country/territory reported in Ethnologue (38 ± 94, mean $\pm$ SD; $n = 225$) because this is the category of languages that arise within a region and hence the type to which our model applies. Because the language richness scores were highly skewed we use ln-transformed values for analysis.

In our analysis we examined the influence of potentially confounding factors. Given that latitude is negatively related to language richness (Sutherland 2003) and the general importance of latitude to the study of biodiversity (Hillebrand 2004), we included absolute latitude measured at the midpoint of each country as a variable. Because country-level wealth may affect language diversity within a country (Nettle 2000), we entered gross domestic product/capita in terms of purchasing power parity (GDP/capita) as a potential confounder. A larger population (irrespective of country land area) may have more languages than a smaller population; therefore, we include the effect of human population size. Lastly, some countries may have more languages within their borders simply because they are larger countries; therefore, we include a country's land area. Data for these potential confounders came from the World factbook 2007 (<www.cia.gov>). GDP/capita, population size and land area were all ln transformed to reduce skewness and kurtosis prior to analysis.

It is possible that the distribution of languages across the world is due to different patterns of human settlement on the continents (e.g. more recent colonization; Nettle 1999b), or different patterns of conquest (Diamond 1998). We accounted for this effect using three methods. First, we explored the predicted positive correlation between parasite richness and language richness in each of six world culture areas. Second, we considered the correlation between parasite richness and language richness using the means for both variables from the six world regions. Third, we compared the pattern of the correlation among three longitudinal geographical bands. Murdock (1949) divided the world's societies into six world culture areas based on a shared historical and geographic range. Murdock's division

of the world reduces interdependence of cultures between regions; analyzing culture areas separately reduces the effects of spatial autocorrelation between countries and may resolve Galton's problem as it is called in anthropology. 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. Primarily, the countries were easy to separate into these world regions; however, the division between West Eurasia and East Eurasia was less distinct. Russia, Kazakhstan, Tajikistan, Kyrgystan, Pakistan, and India were placed in the East Eurasia category; Uzbekistan, Afghanistan, Turkmenistan and Iran were placed in the West Eurasia category. Similar to Collard and Foley's (2002) analysis of the latitudinal distribution of cultures, we analyzed the relationship between disease richness and language richness across three longitudinal geographical bands that have been subjected to different histories of conquest and colonization (Diamond 1998, Collard and Foley 2002). The American band incorporates those countries and territories from 180°W to 30°W; the Europe-Africa band incorporates the countries and territories from 30°W to 60°E including Iran and Iceland; the Asia-Australia band incorporates the countries and territories from 60°E to 180°W including Kazakhstan, Russia, Turkmenistan and Uzbekistan. The bands provide three quasi-independent latitudinal gradients.

One analytical concern is that because any two adjacent countries share a border, a measured effect may arise because of their nearness in space. According to the model we present above, we expect such spatial autocorrelation to the extent that adjacent nations share disease profiles. This sharing of infectious disease burdens should be reflected in each nation's language richness profiles. Removing this effect might remove the effect we are predicting and testing. The most suitable analysis then to test our model as it applies to language diversity involves using separate analyses in world cultural regions and longitudinal bands.

Results

In the global analysis, human parasite richness was positively correlated to living indigenous language richness ($r = 0.78$, $p < 0.0001$, $n = 221$; Fig. 1). The zero-order correlations between language richness, disease richness and the control variables are presented in Table 1. All of the control variables were correlated significantly with language richness – absolute latitude (–0.21), GDP/capita (–0.27), population size (0.69), land area (0.70) – thus each was considered in partial correlations between disease richness and language richness.

The correlation between disease richness and language richness was 0.77 when partialling absolute latitude ($n = 217$), 0.75 when partialling GDP/capita ($n = 215$), 0.54 when partialling population size ($n = 217$), and 0.54 when partialling land area ($n = 217$). All significance levels were $p < 0.001$. Population size and land area were significantly correlated with language richness when partialling disease richness ($n = 217$; population size: $r = 0.21$, $p = 0.001$; land area: $r = 0.26$, $p < 0.001$). Therefore, language richness, disease richness, population size, and land area were all considered in a subsequent partial correlation (absolute latitude and GDP/capita were not considered in further analyses because they did not correlate significantly with language richness when partialling disease richness). In this partial correlation ($n = 217$), population size was no longer a significant correlate with language richness ($r = 0.05$, ns), but land area ($r = 0.16$, $p = 0.016$) and disease richness ($r = 0.51$, $p < 0.001$) were significant correlates of language richness. We then regressed language richness on both disease richness and land area. This regression was significant (adjusted $R^2 = 0.63$, $F_{2,214} = 185.60$, $p < 0.0001$) with both disease richness and land area contributing distinct effects (standardized coefficients: disease richness = 0.60, land area = 0.25, both $p < 0.0001$).

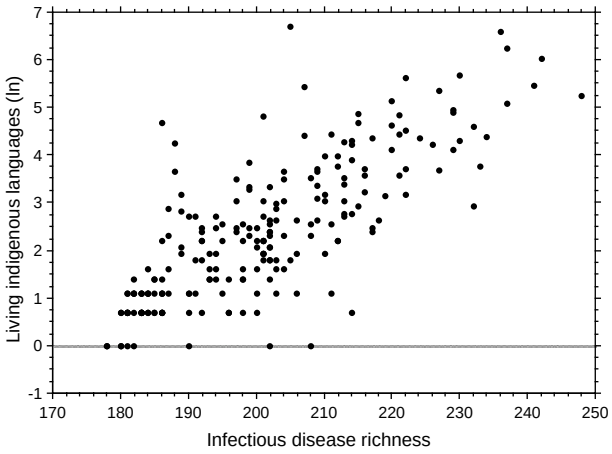

Figure 1. The correlation between human parasite richness and the natural log of indigenous living languages per country found across the world ($r = 0.78$, $p < 0.0001$, $n = 221$).

The correlations between language richness and disease richness for the six world regions were all positive and significant (r ranged from 0.58 to 0.92; all $p \leq 0.002$; Table 2). The correlation between language and disease richness using the means from the world regions was 0.84 ($p < 0.03$, $n = 6$). And, language richness was positively correlated to disease richness in all three longitudinal bands (American: $r = 0.92$, $p < 0.0001$, $n = 60$; Europe–Africa: $r = 0.81$, $p < 0.0001$, $n = 115$; Asia-Australia: $r = 0.61$, $p < 0.0001$, $n = 46$).

We conducted partial correlations of language richness, disease richness, and land area in all six world regions and three bands. In all six regions or three bands, disease richness and language richness were positively correlated when partialling the effect of land area (for the six world

Table 1. Zero-order correlations between living indigenous language richness, disease richness and control variables for the countries of the world.

	Correlation with living indigenous language richness (ln)		
	r	p	n
Disease richness	0.78	<0.0001	221
Absolute latitude	−0.21	0.0014	220
GDP/capita (ln)	−0.27	<0.0001	218
Population size (ln)	0.69	<0.0001	220
Land area (ln)	0.70	<0.0001	220

Table 2. Zero-order correlations between living indigenous language richness and disease richness split by the six world regions.

World region	Correlation between living indigenous language richness (ln) and disease richness		
	r	p	n
North America	0.92	<0.0001	30
South America	0.92	<0.0001	19
Africa	0.83	<0.0001	56
West Eurasia	0.58	<0.0001	63
East Eurasia	0.59	0.0021	24
Insular Pacific	0.75	<0.0001	29

regions, r ranged from 0.29 to 0.82; for the three bands, r ranged from 0.30 to 0.76; Table 3). The correlation was not significant in the world region East Eurasia. However, the significance probability would support a directional test ($p = 0.043$, one-tailed) which our hypothesis predicts.

Discussion

According to our model, localized parasite–host coevolutionary races select for limited dispersal (philopatry), in-group favoritism in social interactions because of immunological similarity, and out-group avoidance because of infectious disease risks. These circumstances together provide an engine of speciation and of ethnogenesis in humans. The finding that human language richness across countries positively correlates with parasite richness provides evidence consistent with our model. Furthermore, our model accounts for many aspects of the latitudinal biodiversity gradient: as infectious disease levels increase so too does biodiversity; as infectious disease levels diminish so too does biodiversity.

Other researchers have found positive correlations between parasite diversity and clade diversity that support our model, but they attributed the correlation to different causes. Krasnov et al. (2004) found that small mammal species richness was correlated positively with the species richness of their fleas. In their words (Krasnov et al. 2004, p. 1861), "This positive correlation suggests that diversification of parasites is a response to diversification of hosts".

Table 3. First-order partial correlations between language richness and disease richness when partialling the effect of land area in the six world regions and three longitudinal bands.

World region	Correlation between living indigenous language richness (ln) and disease richness partialling the effect of land area (ln)		
	r	p	n
North America	0.82	<0.001	28
South America	0.82	<0.001	19
Africa	0.66	<0.001	54
West Eurasia	0.29	0.021	63
East Eurasia	0.36	0.085	24
Insular Pacific	0.37	0.046	29
Longitudinal band			
American	0.76	<0.001	57
Europe–Africa	0.66	<0.001	114
Asia–Australia	0.30	0.045	46

Nunn et al. (2004) used similar reasoning to explain their finding that the more speciose a primate clade the more speciose the clade's parasites. These findings and ideas, while generally supportive of our model, do present a logical challenge because we could interpret our findings similarly. However, we reiterate the alternative framework that we propose: the presence of a parasite generates selection for mechanisms to prefer in-group, immunologically similar conspecifics and avoid dissimilar ones (i.e. mechanisms that facilitate assortative sociality and limited dispersal). While this discriminative social engagement generates population divergence among hosts, it also generates population divergence among parasites. Simultaneously, parasites and hosts are involved in antagonistic, coevolutionary races that can lead to further divergence within host and parasite populations. Short of clade extinction this positive feedback is unending and has presumably been so since the first infective parasite attacked a host.

Insomuch that ethnic diversity is similar to language diversity, Cashdan (2001) provided findings supportive of our hypothesis. Using a different measure of infectious disease burden than we do that taps the relative severity of a select set of pathogens (based on Low 1990) for each society, Cashdan showed that for 186 traditional human societies (the standard cross-cultural sample) ethnic diversity was related positively to pathogen stress. Explaining this finding, Cashdan (2001) suggested pathogen severity was a limiting factor in the successful dispersal of humans to different areas of the world. She provided examples (mostly from McNeill 1990) arguing that variable levels of pathogen immunity among conquerors and the conquered, in some cases, facilitated invasion of new territory; whereas, in other cases differences in immunity staved off invasion by conquerors. Cashdan did not suggest as we do that spatial differences in pathogen severity provided the necessary conditions for populations or cultures to diverge.

Collard and Foley (2002) along with Cashdan (2001) showed that human culture diversity declines with increasing latitude. This along with the already mentioned negative correlation between latitude and human parasite richness (Guernier et al. 2004) and pathogen stress (Cashdan 2001) is also generally supportive of our hypothesis.

Similar to Nettle's argument for the role of ecological risk for the distribution of languages, Cashdan (2001) and Collard and Foley (2002) argued for the role of ecological uncertainty in the distribution of human groups. Our hypothesis agrees with the basic thesis that there will be greater intergroup contact (via more dispersal and reduced out-group avoidance) in areas with high ecological risk (high latitudes). We suggest, however, that local levels of pathogen severity determine to a large extent whether and what forms of intergroup interactions are adaptive and will take place. For example, we predict that human groups in areas of high pathogen severity will be characterized by very little exchange of goods, norms and other cultural items. Moreover, widespread networks of exchange (trade, culture) may only persist in areas of low parasite severity. We expect widespread social networks in high pathogen severity areas to be relatively uncommon and impermanent.

A common argument for the current distribution of human cultures and their languages is that the pattern is due to differences in patterns of colonization and conquest and the diseases carried along (McNeill 1976, Diamond 1998, Nettle 1999a, Cashdan 2001). The common positive correlation between language richness and disease richness we found in the six world regions and three longitudinal bands strongly speaks against this as the primary factor for language distribution on a worldwide scale.

We expect there are more languages and more species in the tropics because parasites are better able to thrive in tropical environments and thus parasite severity is greatest in the tropics (on the topic of greater tropical parasite severity see Møller 1998, Cashdan 2001, Guernier et al. 2004, Wolfe et al. 2007). We consider our proposed model of parasite-mediated diversification an important link between previous work showing that warmer temperatures, greater precipitation or amount of tropical forestation were positively related to species richness (Reed and Fleagle 1995, Allen et al. 2002, Hawkins et al. 2003).

Additionally, while our analysis is limited to human cultures the same mechanism, we propose, can lead to the genesis and maintenance of cultures in other animals. For example, the development of song dialects in birds involves assortative mating within spatially distinct dialects. Apparently, immunological barriers may generate the adaptive value of within-group (-dialect) preference (MacDougall-Shackleton et al. 2002).

Our model and supportive evidence provide significant context for three other models of divergence in the literature: Nettle's (1999a,1999c) social-marker model of language divergence, McElreath et al.'s (2003) model describing the evolution of human ethnic markers, and Hochberg et al.'s (2003) model of socially-mediated speciation.

Nettle (1999a, 1999c) argued that languages serve as markers (rather targets) for individuals to direct reciprocal exchange (Nettle and Dunbar 1997). (Similarly, Fitch (2004) suggested language dialects serve as markers of kinship.) Nettle (1999a) recognized the apparent inability to explain language diversity based solely on a geographic isolation model and suggested that through the action of the social selection of language (i.e."[the language] learner does not just pick up all the language activity going on around him or her, but instead homes in specifically on that of a target group"; Nettle 1999a, p. 29), languages can emerge and remain distinct without geographic barriers. We suggest that while this may be correct, language as a social marker is for the direction of assortative in-group sociality as pathogen avoidance, not merely reciprocal exchange (and not merely for kin recognition as in Fitch's hypothesis).

McElreath et al. (2003) argued that, in humans, normative social behaviors are markers that create cultural boundaries through in-group coordination and cooperation; they argued this relationship between marker and normative behavior provides reliable in-group identification and thereby gives a resolution to the freerider/cheater problem inherent in any social system that involves altruism. We suggest social norms, as markers, function more importantly in the context of antipathogen behavior (Schaller and Duncan 2007). McElreath et al.'s model does not offer an explanation for the worldwide distribution of cultural diversity as we do. Furthermore, we predict that the spatial variation in pathogen severity will predict the distribution of adherence to social norms (conformity to traditional values) with greater adherence in areas of high pathogen severity and lower adherence in areas of low pathogen severity.

Hochberg et al.'s (2003) social-marker-based model showed that prezygotic reproductive isolation can emerge without geographic barriers through the action of directing altruism to similarly marked individuals. Recent research by Blais et al. (2007) is supportive of Hochberg et al. (2003) and our suggested compendium of our model with that of Hochberg et al. (2003). Blais et al. (2007) showed that sympatric speciation of a pair of African cichlid fish species was related to assortative mating based on adaptive divergence in MHC genes. These MHC genes are purportedly important social markers and function in parasite defense.

While we consider Hochberg et al.'s socially-mediated speciation model to be a strong starting point, it doesn't explain why the rates of speciation have differed worldwide. Furthermore, it gives no context for the presence of relevant markers in the first place; their existence is assumed. Missing from their model is incorporation of the worldwide spatial variation of parasites and the resultant variation in assortative sociality between conspecifics.

Moreover, we argue that markers per se are not always needed to generate reproductive isolation. Required are organismal features that facilitate selective contact with conspecifics. For example, behavioral syndromes that reduce disease infection by limited dispersal (Freeland 1976, Møller et al. 1993, Loehle 1995), and hence lead to population divergence, can be favored by selection without marker formation.

All three models (Nettle, McElreath et al., Hochberg et al.) reveal the ability of assortative sociality to generate divergence without geographic isolation. We suggest that varying infectious disease levels have been the selective context that differs across the world and hence have largely led to the geographic patterns of biological diversity. While not denying the validity of allopatric divergence for speciation and ethnogenesis (i.e. divergence resulting from geographic barriers), we argue that the dominant mode of speciation and population/culture divergence in high parasitic stress regions (low latitudes) has been parasite-driven parapatric speciation/ethnogenesis (on parapatric speciation see review by Endler 1977). If so, hybrids between closely related species will be inferior specifically because of their lowered fitness in the context of parasite resistance, not in other fitness components that diverge in allopatry across barriers. Allopatric speciation should generate hybrid disadvantage across all fitness components with equal likelihood.

Parasites play an important role in the distribution of species through their effects on range expansion by host species. For example, parasites are known to facilitate range expansion by serving as biological weapons that invading species or groups carry along, effectively eliminating competition by residents (Freeland 1983, Diamond 1998). Hosts have also been able to successfully invade new territories by leaving their parasites behind (Wolfe 2002, Mitchell and Power 2003, Torchin et al. 2003). Prior

research has also implicated parasites as important moderators of species coexistence and community composition (Freeland 1983, Pagel et al. 1991). While certainly important for understanding the distribution of species, parasites in these roles, in contrast to their role in our model, do not provide the types of general mechanisms that explain the latitudinal diversity gradient.

Parasites, in general, are also featured in other models of speciation that might explain the geographic distribution of species. One popular theme is that parasites are strong selective agents on hosts (similar to predators as selective agents on prey) and hence may be important in generating the evolution of diversity (Haldane 1949, Price et al. 1986). This form of divergence is important but our model casts parasites in a more encompassing role as depicted in the model's sequential events (Introduction). Our model, too, is different than the theory of pest pressure (Gillett 1962, Janzen 1970, Connell 1971, Gilbert 2005). This theory is based on the idea that high density-dependent mortality due to parasites at low latitudes creates a condition whereby no species can be exceptionally common. Thus, high levels of tropical diversity arise and persist because many more species can coexist in low latitude areas than in temperate areas. The theory of pest pressure might be supported by the data we present: more parasites, more diversity. However, research has shown that similar levels of density-dependent mortality are experienced in temperate areas (Hille Ris Lambers et al. 2002) reducing the likelihood that density-dependent mortality due to pathogens is a general explanation for the latitudinal biodiversity gradient.

Future research can best distinguish between our model and the theory of pest pressure by examining the history of effective natural selection by searching for adaptations predicted uniquely by our parasite-driven wedge model. This characterization of functional design is the fundamental path for identifying historically relevant selection pressures (Williams 1966, Thornhill 1990). Our parasite-driven wedge model predicts that the psychology of xenophobia (or its opposite, xenophilia) and of philopatry will differ in accord with parasite intensity levels. The theory of pest pressure makes no such predictions. For example, our model predicts that individuals will exhibit greater xenophobia (facilitating avoidance of out-group contact) in areas with high levels of pathogen severity. Xenophobia that functions for avoidance of hybrid matings should exist independent of pathogen intensity levels if allopatric speciation/divergence with subsequent range overlap and hybrid disadvantage in contexts other than inferior immunity is involved. Evidence supporting this prediction that xenophobia in humans is functional as antipathogen psychology and that it is strongly linked to local levels of infectious disease can be found in Faulkner et al. (2004), Navarrete and Fessler (2006), Navarrete et al. (2007), Park et al. (2007), Schaller and Duncan (2007), Fincher et al. (2008) and Schaller and Murray (in press). Similar evidence for xenophobia is predicted by our model to be found among closely related non-human species and especially in low latitude/high parasite stress areas.

It may appear counterintuitive to anchor our model on the proposition that parasites lead individuals to avoid distant conspecifics. Indeed, one hypothesized benefit of sexual reproduction and outbreeding is the diversification of a brood in order to combat parasite threats (Tooby 1982, Trivers 1985, Hamilton et al. 1990, Ridley 1993). Shields' (1982) review and recent modeling by Kokko and Ots (2006), however, indicate that inbreeding may be adaptive under a range of circumstances that give rise to outbreeding depression. We suggest that the costs and benefits of inbreeding and outbreeding will vary spatially primarily in accordance with parasite severity. In areas of high parasite severity, inbreeding is costly because it reduces variation in the molecular milieu from which an individual can mount an immune response (Penn and Potts 1999) but beneficial through the maintenance of locally adaptive immune responses as well as the avoidance of infectious diseases that are not already harbored by the local group. Thus, although close inbreeding is maladaptive under high parasite severity levels, distant outbreeding is too (Wegner et al. 2003). This is 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 and Ots 2006).

The recognition of spatial variation of parasite richness and the resultant contingent expression of antipathogen mechanisms (assortative sociality and limited dispersal) across the globe may have broad implications for patterns of cultural evolution and biogeography. We highlight a few cases here.

The genetic distance between nearby populations should be greater in areas of high parasite severity. This, we pose, results from the history of strong selection against out-group contact in these areas. In low parasite areas, out-group mating will have been less costly and may manifest as genetically more similar adjacent populations. This prediction about genetic distance is supported by a recent study of the genetic divergence of populations in a study of 62 vertebrate taxa by Martin and McKay (2004) that shows that the genetic divergence is negatively related to latitude (and hence parasite problems). Also, these patterns across taxa are consistent with a negative relationship between dispersal and latitude. Similarly, spatial differences in parasite prevalence might produce patterning in hybrids and hybrid zones: because hybrids provide avenues for unfamiliar parasites to travel between host populations we expect hybrids among species to be more common in low parasite severity regions, and because parasites increase the cost of hybridization the extent of hybrid zones are predicted to vary along parasitic gradients with wider zones found in low parasite severity areas and narrow zones found in high parasite severity areas.

A current debate centers on whether the transmission of culture (including language) proceeds primarily through vertical transmission (parent to offspring) or horizontal transmission (society to society), and whether a bifurcating phylogeny and associated analytical techniques is the best method for studying human cultural evolution (Borgerhoff Mulder et al. 2006, Collard et al. 2006). Vertical transmission results in branching cultural phylogenies (strictly bifurcating) while horizontal transmission results in blending cultural phylogenies (reticulate). The spatial variation of parasite intensities may help resolve this debate. Cultures

that emerge from high parasite severity regions are expected to produce strictly bifurcating phylogenies representing significant in-group assortative sociality. While cultures that come from low parasite severity regions may be best represented by reticulate, blending phylogenies resulting from lower levels of assortative sociality and increased exchange of ideas and values with out-groups. We add that phylogenies for other species may also contain histories of blending and branching in accord with variation in parasite intensities. We predict that the phylogenies of species that emerge at high latitudes (low parasite severity) will appear more reticulate than phylogenies of species that emerge at low latitudes (high parasite severity) which will appear more bifurcate. The spatial variation in parasite intensities may have significant implications for the phylogenetic analysis of both cultures and species.

Because of the selective advantage of limited dispersal under severe parasite levels, we suggest that the general finding that species' geographic ranges are smaller in the tropics than in temperate areas (Rapoport's rule; Stevens 1989) derives from the greater pathogen severity found at low latitudes. Thus, dispersal variation in response to local pathogen severity may provide the context for Rapoport's rule.

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